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Review of contaminants of potential human health concern in wastewater and stormwater

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ABBREVIATIONS AND ACRONYMS

ADI	Acceptable daily intake
AMR	Antimicrobial resistance
APEOs	Alkylphenol ethoxylates
APs	Alkylphenols
ARfD	Acute reference dose
ATSDR	Agency for Toxic Substances and Disease Registry
BDE	Brominated diphenyl ethers
BFRs	Brominated flame retardants
BMDL	Benchmark dose (lower confidence limit)
BPA	Bisphenol A
CEPA	California Environmental Protection Agency
CSO	Combined sewer overflows
DDT	Dichlorodiphenyltrichloroethane
DVFA	Danish Veterinary and Food Administration
DWSNZ	Drinking-water Standards for New Zealand
ECHA	European Chemicals Agency
EDCs	Endocrine-disrupting compounds
EFSA	European Food Safety Authority
EU	European Union
FAO	Food and Agriculture Organization of the United Nations
FST	Faecal source tracking
GC	Gene copies
HAV	Hepatitis A virus
HBCD	Hexabromocyclododecane
HUS	Haemolytic uraemic syndrome
IARC	International Agency for Research on Cancer
IPCS	International Programme on Chemical Safety
JECFA	Joint FAO/WHO Expert Committee on Food Additives
JMPR	Joint FAO/WHO Meeting on Pesticide Residues
LOD	Limit of detection

LOQ	Limit of quantification
MAV	Maximum allowable value
MCL	Maximum contaminant limit
MfE	New Zealand Ministry for the Environment
MoH	New Zealand Ministry of Health
MPN	Most probable number
NMPs	Nanoplastics and microplastics
NOAEL	No observable adverse effects limits
NP	Nonylphenol
NPEOs	Nonylphenol ethoxylates
OCs	Organochlorine pesticides
OECD	Organisation for Economic Co-operation and Development
OP	Octylphenol
OPEOs	Octylphenol ethoxylates
PAEs	Phthalic acid esters
PAHs	Polycyclic aromatic hydrocarbons
PBBs	Polybrominated biphenyls
PBDEs	Polybrominated diphenyl ethers
PCBs	Polychlorinated biphenyls
PCDDs	Polychlorinated dibenzo-p-dioxins
PCDFs	Polychlorinated dibenzofurans
PEs	Phthalate esters
PFAAs	Perfluoroalkyl acids
PFAS	Per- and polyfluoroalkyl substances
PFCAs	Perfluorocarboxylic acids
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonate
PFSAs	Perfluorosulfonic acids
PFU	Plaque-forming units
POP	Persistent organic pollutant
ppb	Parts per billion
PPCPs	Pharmaceuticals and personal care products
ppm	Parts per million
PTWI	Provisional tolerable weekly intake

REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
RfD	Reference dose
SCF	Scientific Committee on Food (European Union)
STEC	Shiga toxin-producing *E. coli*
TBBPA	Tetrabromobisphenol A
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TDI	Tolerable daily intake
TEF	Toxic equivalency factor
TEQ	Toxic equivalent
TWI	Tolerable weekly intake
US EPA	United States Environmental Protection Agency
UWRRC	Urban Water Resources Research Council
WHO	World Health Organization
WWTP	Wastewater treatment plant

EXECUTIVE SUMMARY

Municipal wastewater and urban stormwater contain a myriad of contaminants, both microbiological and chemical, many of which are known or suspected to pose a risk to environmental and/or human health. This report has been prepared in response to a request from Te Whatu Ora / Health New Zealand to provide an overview of those contaminants in municipal wastewater and urban stormwater that have the potential to pose a risk to human health, based on international grey and published scientific literature.

Untreated or partially-treated wastewater may be discharged to the environment through overflows, spills or illegal activity, and stormwater is often deliberately discharged untreated. Further, different wastewater and stormwater treatment processes vary significantly in their ability to remove specific contaminants. Thus, this report focuses on untreated wastewater and stormwater to provide a baseline understanding of what contaminants may be present in these matrices in the first instance and that may constitute a hazard, including in the event of direct discharge or inadequate treatment.

The inclusion of contaminants in this report was guided by priority contaminant lists prepared by the United States Environmental Protection Agency (US EPA) and European Commission, and several reviews that have been recently prepared for New Zealand Regional Councils (Tremblay and Northcott 2015; Stewart et al. 2016, 2017; Gadd 2019; Stewart and Tremblay 2020). Contaminants were assessed on two criteria: that they were known to be present in untreated municipal wastewater and/or stormwater, and that they were known (or highly likely) to be a human health hazard. Contaminants that occur in municipal wastewater but are predominantly associated with industrial effluents (ie, 'trade wastes') are reviewed separately by Eaton (2022). For the purposes of this report, the contaminants have been grouped into nine broad classes: microbial pathogens, heavy metals, per- and polyfluoroalkyl substances (PFAS), polycyclic aromatic hydrocarbons (PAHs), pesticides, pharmaceuticals and personal care products (PPCPs), endocrine-disrupting compounds (EDCs), brominated flame retardants (BFRs), and microplastics. For each contaminant, an overview of the adverse human health effects associated with exposure is provided with a general focus on oral exposure, although route and magnitude of exposure are not specifically considered in this report. Where information was readily accessible, health-based guideline values for exposure (eg tolerable or acceptable daily intakes) from national or international regulatory or advisory organisations have been included.

Many of the contaminants discussed in the report have been detected in both wastewater and stormwater, due to a combination of their diverse industrial, civil and consumer applications, the presence of combined wastewater and stormwater networks in some regions, and/or the cross-contamination of separated networks through cross-connections, damaged infrastructure, inflow and/or infiltration. However, the specific contaminant profiles of both wastewater and stormwater are highly variable, being influenced by characteristics of the catchment (eg, land use, population density, population behaviour and consumption patterns), characteristics of the network (eg combined vs separated, network integrity, industrial contributions) and climatic conditions (rainfall frequency and intensity, season). For some contaminants such as heavy metals, their presence in wastewater and/or stormwater has been well-documented over several decades and is supported by a large body of literature; however, for emerging contaminants such as pharmaceuticals or microplastics, their presence is not well characterised. For many contaminants, their continuous emission to wastewater in particular, which in turn is continuously discharged to the environment, may provide the opportunity for chronic exposure to contaminants, even if they are not inherently environmentally persistent (ie, they may exhibit a 'pseudo-persistence').

Similarly, the human health effects of exposure to some contaminants, including microbial pathogens and heavy metals, are well characterised. However, for the large number of emerging contaminants the potential impacts on human health are unclear, especially with regards to the effects of chronic exposure to low or trace-level concentrations, and little to no consolidated data on health-based exposure guidelines for these contaminants was identified. Further, significant knowledge gaps remain concerning the potential for additive or synergistic effects that may result from exposure to multiple contaminants, such as the mixtures that occur in both wastewater and stormwater.

The report has also considered Te Ao Māori perspectives on contaminants in wastewater and stormwater, but should only be considered the briefest of introductions and is not a substitute for engaging directly with Māori communities. Traditionally, Māori waste management practices were highly prescriptive and underpinned by key cultural principles including tapu, noa, mana and mauri, and the discharge of wastes to water considered highly offensive. Contemporary wastewater and stormwater matrices and management systems present multiple challenges for Māori, including the inability to exercise tikanga regarding waste separation and management, and the unknown risks presented by contemporary contaminants; compliance with regulatory standards is insufficient to ensure cultural safety. The contamination or loss of mahinga kai or wai māori resulting from the discharge of wastewater and/or stormwater may result not only in impacts on physical health, but also on cultural, social and spiritual wellbeing.

As this report constitutes a high-level overview based on international literature, no assertion is made that all of the contaminants discussed in this report will be present in untreated wastewater and/or stormwater in New Zealand, and/or would persist in treated wastewater. Similarly, no assertion is made that this review covers of all potential contaminants of potential health concern; the sheer diversity of possible contaminants in these matrices means that omissions are inevitable. Further work will be required to assess the presence of priority contaminants in municipal wastewater and stormwater in the New Zealand context, review treatment efficacy for New Zealand-relevant technologies and contaminants, undertake specific risk characterisation and assessment, and to consider what regulation, if any, exists and/or may be appropriate for managing human health risks associated with these contaminants.

1. INTRODUCTION

1.1 BACKGROUND

Wastewater and stormwater may contain a myriad of contaminants, both microbiological and chemical, that may present a risk to human health, especially where they are discharged to receiving environments without adequate treatment (whether unintentionally through spills, overflows or treatment failure, or through unconsented or illegal activity). The composition of both wastewater and stormwater streams may vary significantly depending on the nature of the catchment, including population size, density and behaviour; land use (eg urban, rural); design and integrity of wastewater and stormwater networks (eg combined or separated networks, cross connections, leaks); climatic conditions; and contributions from industrial and trade wastes (Beca et al. 2020).

Wastewater and stormwater are key sources of contaminants to aquatic and terrestrial environments (Makepeace et al. 1995; Gasperi et al. 2012; Zgheib et al. 2012; Loos et al. 2013; Luo et al. 2014; Margot et al. 2015; Masoner et al. 2019). New Zealand is currently undergoing a period of regulatory change with regards to 'Three Waters,' encompassing the drinking-water, wastewater and stormwater sectors. Among other objectives, these reforms are intended to improve the performance of wastewater and stormwater systems to better protect environmental and public health (DIA 2021). New Zealand utilises an 'effects-based approach' to setting discharge standards that considers both the nature of the treatment plant and the receiving environment (Beca et al. 2020); however, the recent Three Waters Review identified that the sector is currently challenged by the lack of a clear national framework for regulating and reporting on the performance of these sectors[1]. A comprehensive understanding of the hazards that may be associated with wastewater and/or stormwater discharge is therefore necessary to help ensure that any interventions, including policy or regulation, are appropriately designed and underpinned by a strong evidence base, in order to achieve desired environmental and public health outcomes.

[1] https://www.dia.govt.nz/diawebsite.nsf/Files/Three-waters-documents/$file/Cab-Paper-November-2018.pdf.

1.2 WASTEWATER AND STORMWATER

1.2.1 Municipal wastewater

Humans create large volumes of wastewater during their daily lives, for example, through flushing toilets, showering, preparing food, washing dishes and doing laundry (Figure 1). This domestic wastewater, also known as sewage, is more than 95% water, and includes organic matter including human waste (ie, urine and faeces), food scraps, fats and oils, detergents, and dusts and debris such as sand, grit and plastics (Stewart et al. 2017; Beca et al. 2020). For residential and commercial properties that are connected to a reticulated wastewater system, wastewater is conveyed to the wastewater treatment plant (WWTP) where the entire waste stream is treated, and subsequently discharged to the environment. Wastewater treatment will remove some, but not all, of the various contaminants present in the influent wastewater; removal efficiency will vary significantly depending on the specific contaminant and the treatment process(es) employed (Beca et al. 2020).

Many of the contaminants present in municipal wastewater have the potential to cause adverse effects on receiving environments and human health, however few are routinely monitored (Loos et al. 2013; Luo et al. 2014; Margot et al. 2015; Stewart et al. 2017). Typical contaminants present in municipal wastewater can include microorganisms (bacteria, viruses, protozoa, helminths), biodegradable organic materials, nutrients (nitrogen, phosphorus), heavy metals (arsenic, cadmium, chromium, copper, lead, mercury, nickel, zinc), detergents and surfactants (alkylphenols), plasticisers (bisphenol A (BPA), phthalates) pharmaceuticals and personal care products, and various other legacy and emerging organic contaminants (hydrocarbons, chlorobenzenes, pesticides, dioxins, polychlorinated biphenyls (PCBs), flame retardants, solvents, microplastics) (Zgheib et al. 2010; Loos et al. 2013; Luo et al. 2014; Margot et al. 2015; Stewart et al. 2017).

Industrial effluents may also be discharged to municipal wastewater networks (see Section 1.3). For the purposes of this report, efforts have been made to focus on municipal wastewaters without significant industrial contribution; however, it is not always clear in the literature whether a network being studied receives trade or industrial inputs. Contaminants associated with industrial effluents have been reviewed separately by Eaton (2022).

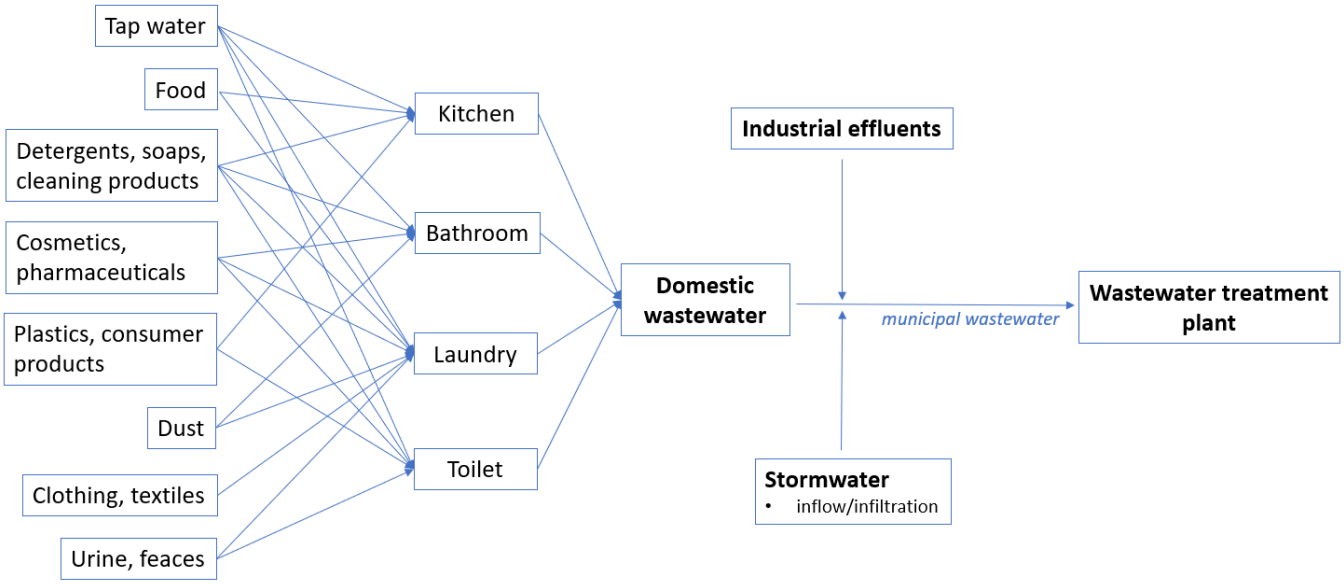

Figure 1: Sources of pollutants to domestic and municipal wastewater systems. Adapted from Moriyama et al. (1989).

1.2.2 Stormwater

Stormwater runoff is generated by rainfall (and in some areas, snowmelt) events where water is unable to soak into the ground, especially in urban areas where much of the land area is covered by buildings and impervious surfaces (roofs, roads, carparks, footpaths etc). During periods of heavy rainfall, excess water may also run off from lawns, parks and green spaces (UWRRC 2014; Müller et al. 2020). Stormwater carries with it an array of contaminants that have built up on these various surfaces, including litter, sediments, hydrocarbon fuels and oils, domestic and wild animal faeces, pesticides, and air- and dust-borne contaminants. Stormwater may also contain materials spilt or dumped into drains, or compounds leached from the aforementioned hard surfaces (Stewart et al. 2017; Müller et al. 2020).

In urban areas, stormwater is commonly collected in a series of gutters and drains, then discharged untreated to nearby waterways, although some newer networks may contain soak pits or constructed wetlands (Figure 2) (UWRRC 2014; Masoner et al. 2019). In rural areas, stormwaters may be collected in grassy swales, or wash into waterways directly. Although stormwater is typically an episodic discharge associated with rainfall, some stormwater systems experience a dry-weather ‘base flow,’ associated with the infiltration of

groundwaters and/or run-off derived from ‘non-storm sources’ such as the irrigation of lawns and gardens, vehicle washing, and other power-washing flows (UWRRC 2014; Müller et al. 2020).

Urban stormwater is well known to transport large quantities of contaminants, and is a major contributor of pollution to aquatic receiving environments (Pitt et al. 1995; Barbosa et al. 2012; Zgheib et al. 2012; Masoner et al. 2019; Müller et al. 2020). The international literature on stormwater demonstrates substantial differences in contaminant profiles between studies, reflecting variation in factors that affect the build-up of contaminants on surfaces (eg land use, traffic intensity, antecedence dry period, atmospheric contribution, adjacent soil types) (Ingvertsen et al. 2011; Müller et al. 2020), and study design (eg sampling directly from stormwater infrastructure or from receiving environments themselves, the contaminants screened for). Further, because of the episodic nature of stormwater flows, contaminant profiles differ during the course of a storm event, with contaminant loading typically being highest during the early phase of the event as accumulated contaminants are washed from surfaces, known as the ‘first-flush’ phenomena (Barbosa et al. 2019).

Stormwater may contain an array of ‘conventional’ and emerging contaminants, including heavy metals, pathogens, pesticides, PAHs, PCBs, phenols and cresols, phthalates, organotins, dioxins, per- and polyfluoroalkyl substances (PFAS) and volatile organic compounds (Makepeace et al. 1995; Eriksson et al. 2007; Zgheib et al. 2010; Masoner et al. 2019; Müller et al. 2020). The US National Stormwater Quality Database[2] contains data from more than 9,000 urban runoff events across different urban land uses, and describes more than 100 different contaminants. Whilst heavy metals and pathogens are thought to be the main drivers of human health risk associated with exposure to stormwater (Chong et al. 2013; Ma et al. 2016; Ahmed et al. 2019), the potential implications of exposure to other contaminants in stormwater remain to be fully determined (Masoner et al. 2019).

As urban stormwater may also be contaminated by municipal wastewater as described below (Section 1.2.3), or by runoff from industrial sites failing to implement best practice containment, contaminants associated with these waste streams may also be present in stormwaters (Müller et al. 2020).

[2] https://bmpdatabase.org/national-stormwater-quality-database

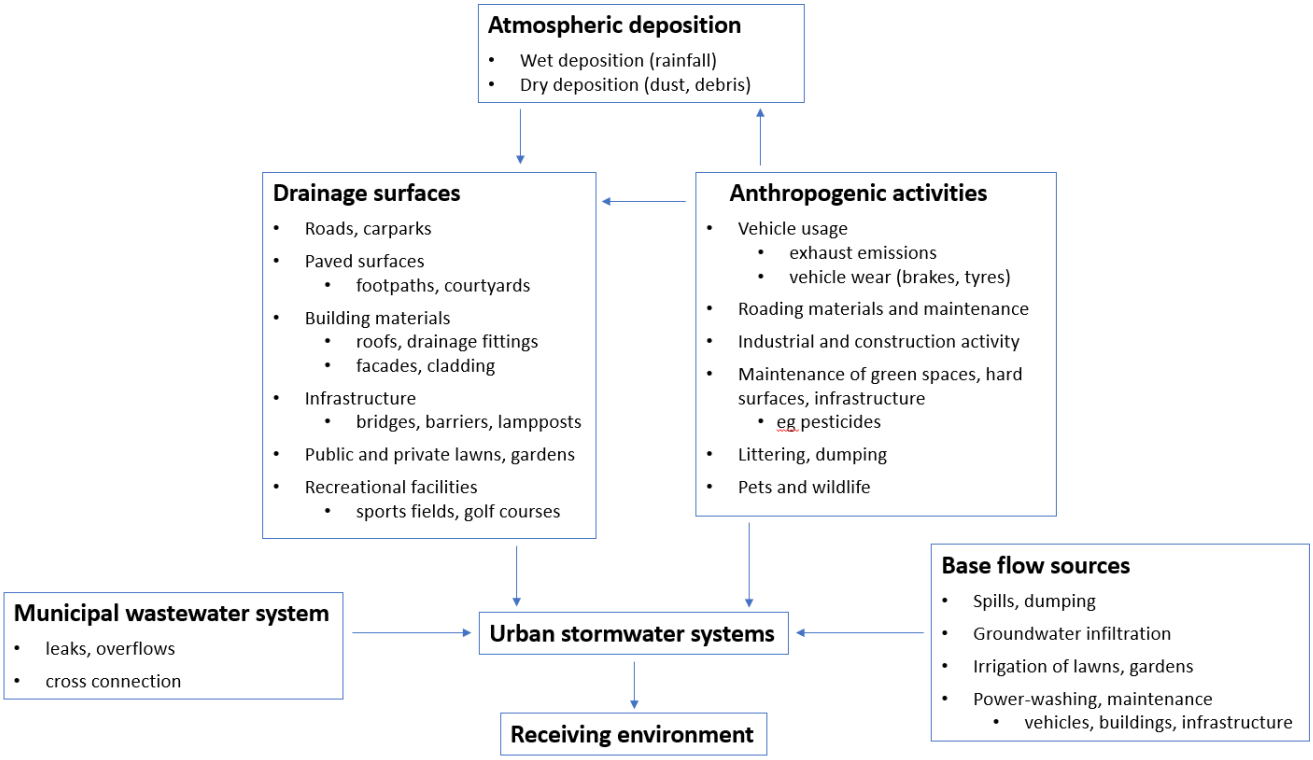

Figure 2: Sources of pollutants to urban stormwater systems, with transportation pathways indicated by arrows. Adapted from Müller et al. (2020).

1.2.3 Interaction between wastewater and stormwater networks

In most developed cities, reticulated wastewater and stormwater systems comprise separate networks. However, in precincts with older infrastructure (including some New Zealand networks dating to early 20th century)[3] these systems may be combined, collecting both wastewater and stormwater and conveying the combined stream to the WWTP. These systems are prone to 'combined sewer overflows (CSOs),' whereby increased stormwater flow during periods of rain overwhelms the capacity of the network, and the combined wastewater-stormwater stream is discharged untreated to the environment at designated overflow points (Beca et al. 2020).

Even where networks are separated, stormwater systems are frequently contaminated by sewage through accidental or illegal cross connections, damaged infrastructure, or blocked and overflowing sewers. Although this is a well-recognised problem in older cities with aging

[3] https://www.environmentguide.org.nz/activities/stormwater-and-wastewater/managing-stormwater-and-wastewater/. Accessed 28 February 2022.

infrastructure (eg, Paris, Gasperi et al. 2012; Zgheib et al. 2012), it is also widely reported in newer cities including Brisbane (Sidhu et al. 2012; Chong et al. 2013), Sydney (Sidhu et al. 2013) and Milwaukee (Sauer et al. 2011), as well as in rural towns (Stea et al. 2015).

Conversely, stormwaters may cause issues for wastewater networks through inflow and infiltration. Inflow describes stormwater entering the wastewater system through cross connections or flooded manholes or gully traps, while infiltration describes groundwaters seeping into the wastewater network through damaged infrastructure (eg cracked pipes or pipe joins) (UWRRC 2014; Beca et al. 2020). When these additional inputs overwhelm the capacity of the wastewater network, wastewater overflows to the stormwater network and/or directly to receiving waters through engineered overflow points, resulting in the discharge of diluted but untreated wastewater to the environment, known as a 'wet weather overflow' (Water New Zealand 2021). Stormwater inflow and infiltration can also cause issues at the WWTP by increasing the volume of effluent requiring treatment and reducing overall treatment efficacy (Beca et al. 2020).

1.2.4 Overview of contaminants in municipal wastewater and stormwater

An overview of different classes of contaminants that are commonly identified in municipal wastewater and/or stormwater is presented in Table 1. Specific examples of the occurrence of contaminants in each matrix, and of the potential health effects associated with those contaminants, are described in the following chapters. It is important to note that contaminants may be present in wastewater and/or stormwater in their dissolved, colloidal or particle-associated forms (Eriksson et al. 2007), which will influence both their fate through these networks (and hence potential human exposure), and their bioavailability (and hence toxicity); however, this has not been specifically considered in this report.

Table 1: Overview of contaminant classes present in municipal wastewater and/or stormwater that are of potential concern to human health, with common examples.

Contaminant class		Examples	Municipal wastewater	Stormwater
Microbial pathogens		*Campylobacter* spp. *Salmonella spp.* Norovirus Adenovirus *Cryptosporidium* spp. *Giardia* spp.	✓✓	✓✓
Heavy metals		Arsenic Cadmium Chromium Mercury Lead Zinc	✓✓	✓✓
Perfluoroalkyl substances		PFOA PFAS	✓✓	✓✓
Pesticides	Organochlorine pesticides	DDT Aldrin Chlordane	✓	✓✓
	Organophosphate pesticides	Chlorpyrifos Malathion		
	Carbamate pesticides	Aldicarb Carbofuran		
	Pyrethroids	Permethrin Cyfluthrin		
	Triazines	Atrazine		
	Phenoxy alkonates	2,4-D Mecoprop		
Polycyclic aromatic hydrocarbons		Naphthalene Fluorene Pyrene Benzo(a)pyrene	✓✓	✓✓
Endocrine-disrupting compounds	Plasticisers	BPA Phthalates	✓✓	✓✓
	Corrosion inhibitors	Benzotriazole		
	Surfactants	Nonylphenol		
	Dioxins and dioxin-like PCBs	TCDD PCB-138		
Brominated flame retardants	Polybrominated diphenyl ethers	BDE-47 BDE-99 BDE-153 BDE-209	✓✓	✓
	Polybrominated biphenyls	BB209		
	Hexabromocyclododecanes	HBCD		
	Tetrabromobisphenol A	TBBPA		

Table 1 continued.

Contaminant class		Examples	Municipal wastewater	Stormwater
Pharmaceuticals and personal care products	Antibiotics	Trimethoprim Sulfamethoxazole Ciprofloxacin	✓✓	✓
	Analgesics and anti-inflammatories	Acetaminophen Naproxen Diclofenac Ibuprofen		
	Steroids and hormones	17β-estradiol		
	Other prescription medications	Clofibric acid Atenolol Carbamazepine Gemfibrozil		
	UV filters	Benzophenone Camphor		
	Parabens and other preservatives	Methyl paraben 2-phenoxyethanol		
	Fragrances	Galaxolide Tonalide		
	Disinfectants	Triclosan Triclocarban		
Microplastics			✓✓	✓✓

✓✓ - Are consistently detected and/or at high concentrations.

✓ - Are detected less frequently and/or at low-level/trace concentrations.

1.3 TRADE WASTES

During the normal course of business, commercial and industrial premises generate a variety of liquid waste streams. These wastes may be treated on site before being discharged to receiving waters, or discharged to the municipal wastewater network (with or without pre-treatment); wastes that are discharged to the municipal wastewater network are referred to as trade wastes[4]. Trade wastes include wastes from restaurants, service stations, meat works, dairy factories, tanneries, metal fabrication and finishing, chemical manufacturing, fertiliser production, mills, and hospital wastes. They can also include cooling or condensing waters, stormwaters that cannot be reasonably separated, and very

[4] Internationally, the term industrial effluents is commonly used to describe all commercial and industrial liquid wastes. This term may also be used in New Zealand, although 'trade waste' is generally used to describe wastes that are specifically discharged to the municipal network.

occasionally, domestic wastes (Beca et al. 2020). The composition of trade wastes can be markedly different depending on the industry of origin. Many of the pollutants that have been identified in various industrial effluents and trade wastes may pose a risk to human health (Eaton 2022).

The discharge of trade wastes to municipal wastewater networks means that the contaminants from those waste streams may be present in municipal wastewater. For example, approximately 25% of the wastewater received at the Morrinsville WWTP originates from industrial sources (Beca et al. 2020). However, because the composition of trade wastes can be so diverse, and the relative contribution of trade wastes to municipal wastewater is highly variable between catchments,[5] contaminants that may be present in trade wastes and of potential concern to human health have been reviewed separately by Eaton (2022).

1.4 APPROACH AND SCOPE

This report represents the first stage of an analysis of the potential human health risks associated with municipal wastewater and urban stormwater, and how those risks might be better managed in New Zealand, especially considering opportunities presented by the current sector reform. For this report, a broad, high-level approach has been taken in reviewing international scientific publications and grey literature to identify those contaminants that may be of concern for human health. Key aspects of the review include:

- A review of contaminants of concern to human health that may be present in untreated municipal wastewater.
- A review of the contaminants of concern to human health that may be present in untreated urban stormwater.
- A broad overview of the human health risks associated with exposure to the contaminants identified above, including information on health-based exposure guidelines or standards, where these have been determined and are readily available.

It was initially intended that information on health-based discharge limits for municipal wastewater and/or stormwater from key international jurisdictions (eg, Australia, US, EU, Canada) would also be included. However, the various regulatory frameworks of different jurisdictions were diverse and complex, there was little consolidated information available, and the derivation of such limits (ie, whether they were health-based or ecosystem-based)

[5] https://www.waternz.org.nz/WWTPInventory Accessed 22 March 2022.

was seldom obvious. Further, relatively few emerging contaminants are regulated. It was therefore not possible to compile meaningful information on discharge limits within the time and resource constraints of this report.

The report focuses on contaminants in untreated ('raw') municipal wastewater and stormwater streams; the potential health risks associated with sludges, biosolids or onsite waste management systems (eg, septic tanks) are beyond the scope of this review. The inclusion of contaminants in this report was guided by priority contaminant lists prepared by the United States Environmental Protection Agency (US EPA) and European Union (EU) (Appendix A), New Zealand Biomonitoring Surveys (Mannetje et al. 2018), and several reports that have been recently prepared for Regional Councils (eg, Tremblay and Northcott 2015; Stewart et al. 2016, 2017; Gadd 2019; Stewart and Tremblay 2020). Contaminants were briefly assessed against two criteria: whether the contaminant is known to be present in municipal wastewater and/or stormwater, and whether the contaminant likely constitutes a human health hazard. The potential for a contaminant to cause adverse health effects in humans was based on conclusions reached by the World Health Organisation (WHO), International Programme on Chemical Safety (IPCS), the Joint FAO/WHO Expert Committee on Food Additives (JECFA), the European Food Safety Authority (EFSA), the US EPA and the Agency for Toxic Substances and Disease Registry (ATSDR), when such assessments were available. However, for some contaminants, there was little to no information available or the purported health effects remain contentious. Contaminants that are known to present a risk to ecological health but not human health have not been considered here[6].

While the route of exposure to contaminants has not been specifically considered here, the potential risks associated with occupational exposure to wastewater and/or stormwater are the responsibility of WorkSafe and therefore outside the scope of this review. The general population is likely to be exposed to wastewater and/or stormwater only once these have been discharged to the environment (intentionally or otherwise); thus, information provided on health effects or exposure limits has focused on oral ingestion as the most likely means of exposure. For example, drinking-water sources may become contaminated, contaminated recreational waters may be accidentally ingested, or foods (shellfish, fish, aquatic plants) may be harvested from contaminated receiving environments. However, it should be noted that inhalation and/or dermal exposures are also possible, for example by inhalation of aerosols produced by rapids or surf at contaminated receiving environments, or swimming in contaminated waterways, respectively.

[6] Contaminants presenting a risk to ecological health are the responsibility of the Ministry for the Environment.

Because of the potential for significant overlap or interaction between municipal wastewater and stormwater streams (eg, through inflow or infiltration of stormwater into wastewater networks, overflow of wastewater into stormwater networks, or the possible use of combined networks), the two matrices have not been presented separately in this report. Instead, contaminants have been grouped into nine classes and presented as separate sections: microbial pathogens, heavy metals, per and polyfluoroalkyl substances (PFAS), pesticides, PAHs, pharmaceuticals and personal care products (PPCPs), endocrine-disrupting compounds (EDCs), brominate flame retardants (BFRs), and microplastics. Within each section, data is provided as to the occurrence of these contaminants in both wastewater and stormwater, and an overview of the health risks associated with the contaminant(s) follows. In addition, consideration has been given to Te Ao Māori perspectives regarding contaminants in wastewater and stormwater. This section has been positioned at the front of the report in order to highlight the importance of considering mātauranga Māori (Māori knowledge) alongside the scientific literature, so that the reader may bear both perspectives in mind whilst reading the report.

This report is not intended as a complete review of all potential contaminants in wastewater and/or stormwater, nor is it intended as a human health risk assessment. The focus on untreated wastewater and stormwater allows for understanding of the hazards that may exist in the event of accidental or illegal discharge, treatment failure or overflow. Further work will be required to assess the presence of specific contaminants in New Zealand wastewaters and stormwaters, their removal by treatment processes commonly used in New Zealand WWTPs, and to consider the route(s) and magnitude of exposure in further detail, in order to better characterise the potential risks to public health.

2. TE AO MĀORI PERSPECTIVES AND TIKANGA FOR CONTAMINANTS IN WASTEWATER AND STORMWATER

In considering the various contaminants present in wastewater and stormwater that may present a risk to human health, it is important to consider not only the knowledge that the scientific literature provides, but also the mātauranga (knowledge), whakaaro (thoughts, perspectives) and tikanga (correct procedure, custom, principle) of mana whenua and Te Ao Māori. The following section should be considered only the briefest of introductions and is not a substitute for direct engagement with Māori communities.

2.1 TIKANGA MĀORI

To understand Māori perspectives on contaminants in wastewater and stormwater, it is important to understand the profound connection between Māori and their environment, as well as key cultural principles such as tikanga, tapu, noa, mauri and mana – themselves linked to a suite of other cultural values, mātauranga and intergenerational life experiences that together inform philosophical and spiritual frameworks within Te Ao Māori. As Afoa and Brockbank (2019) note, many of these concepts are best understood in their own language and culture, as reinterpretation or translation across cultural boundaries can result in the loss of context and meaning. Further, the views and perspectives of iwi, hapū and whānau are not homogeneous, but are seen through the lens of generations past, present and future, of personal and shared experiences, and traditions, resources and priorities of the time, and will therefore differ based on their own unique experiences and environment (Ataria et al. 2016). Nonetheless, some common values and perspectives regarding the management of wastewaters and stormwaters are evident. This section will attempt only to highlight key themes as they have been identified from the literature. The reader is referred to Pauling and Ataia (2010), Ataria et al. (2016), Durie et al. (2017) and Afoa and Brockbank (2019), and most importantly, to engage directly with Māori stakeholders, for more in-depth discussion of the relationship between Māori and wai, of tikanga Māori, and the concerns that Māori have regarding wastewaters and stormwaters.

Tikanga has been defined in a number of ways: for example, as the “values, standards, principles or norms to which the Māori community generally subscribe for the determination

of appropriate conduct" (Durie 1996), and "the right Māori ways" (Metge 1995). Underpinned by the notion of what is right and moral, tikanga is the system of core values and the application and practice of those values,[7] that guide Māori in ways of being and enable them to live efficiently and safe from harm (Georgia Bell (Ngāti Maniapoto, Ngāti Pū, me Ngāi Te Rangi), ESR, personal communication). Tikanga is pragmatic, open-minded and flexible, allowing adaptation to the needs of a particular time or situation.[8]

Tapu and noa are fundamental traditional constructs in Māori philosophy and spirituality (Ataria et al. 2016). They are complementary opposites, which together constitute a whole. There are many meanings and conditions associated with tapu, which is often translated to mean sacred, prohibited, restricted or forbidden. All things (ie, objects, people, places) possess tapu, although tapu is not equal in all things. Tapu can describe an intrinsic quality that requires a permanent exercise of tikanga, or it can also have temporal or fluxing dimensions that change with time, situation or protocol (Ataria et al. 2016).[9] Noa is the counter to tapu, describing a state that is deemed safe or ordinary, free from the danger or restriction that is associated with tapu (Ataria et al 2016). Tapu and noa are in essence social codes focused on avoiding risk[10] and ensuring the safety of people and the environment (Feltham 2021).

For Māori, wai (water) is taonga (a treasure) and an essential ingredient of life, both physically and spiritually. It is a living entity, being both the source of life for all things, and a gift from the Atua (gods) to sustain that life. As such, all waters have their own mauri (essence or life force), mana (authority, status, spiritual power) and hau (vitality), and tāngata whenua have a responsibility to protect them (Pauling and Ataria 2010; Durie et al. 2017). Mauri is inherently linked to other physical and metaphysical characteristics including tapu, noa and wairua (spirit or soul). Wai is an integral part of Māori wellbeing and identity, being able to sustain life as drinking water, support healthy mahinga kai (traditional foods and the associated habitats and resources), increase or reduce tapu, and providing rongoā (traditional medicine) and wai tapu (sacred waters) for physical and spiritual cleansing and healing (Durie et al. 2017; Afoa and Brockbank 2019). The whakapapa (genealogical)

[7] Gallagher (undated). https://nzetc.victoria.ac.nz/tm/scholarly/tei-Bid001Kahu-t1-g1-t1.html Accessed 31 August 2022.
[8] Gallagher (undated). https://nzetc.victoria.ac.nz/tm/scholarly/tei-Bid001Kahu-t1-g1-t1.html Accessed 31 August 2022.
[9] For example, certain places (eg urupa, burial grounds) or people (rangatira or tohunga) are always tapu, while rahui is a temporary restriction on an area or resource (eg for conservation of a depleted resource, or when death has polluted the land or water through tapu) and is lifted following an agreed period of time. A person may be considered tapu when they are ill, as is a woman when she is menstruating.
[10] Gallagher (undated). https://nzetc.victoria.ac.nz/tm/scholarly/tei-Bid001Kahu-t1-g1-t1.html Accessed 31 August 2022.

connection between Māori and the environment is captured in the following whakataukī (proverb)[11]

Ko au te awa, ko te awa ko au

(I am the river, the river is me); the welfare of each is inextricably linked (Ataria et al. 2016).

2.2 TIKANGA AND CONCERNS REGARDING WASTEWATER AND STORMWATER

From the principles of tapu and noa, Māori established highly prescriptive traditions and practices regarding the management of all types of waste, from human waste to food waste and other material wastes (eg kaimoana shells, wood shavings from carvings), each of which was handled separately and in accordance with specific protocols (Pauling and Ataria 2010). Tikanga Māori did not permit the discharge of waste of any kind to water; the contamination of water with waste was not just hē (wrong), but a spiritual offence that could result in serious misfortune to offenders and their hapū (Durie et al. 2017). Human waste in particular was considered tapu and harmful, and meticulous separation between the human food chain and human wastes is a consistent theme of both traditional and contemporary Māori society (Pauling and Ataria 2010). This separation of a known risk to people's health (waste) from a primary route of exposure (food and water) demonstrates the pragmatic nature of tikanga Māori and tapu, being grounded in keeping people safe (Georgia Bell, personal communication).

Water may become hāparu (contaminated, impure, unsanitary) when its natural flow is disturbed or waters with different mauri are mixed by unnatural means (Durie et al. 2017). The mauri of water used to carry wastes is considered to have been degraded or even destroyed, and thus Māori consider the discharge of sewage, wastewater and stormwater directly to waterways also degrades the mauri of these receiving waters and is highly offensive (Pauling and Ataria 2010; Durie et al. 2017; Afoa and Brockbank 2019). This is especially true where receiving waters may be used for mahinga kai or are considered wāhi tapu (sacred place). This includes treated effluents, and even where these comply with regulatory water quality standards, the discharge of wastewater to water is not acceptable from a cultural perspective (Durie et al. 2017). This is not strictly a question of compliance with modern health standards but is a consequence of the extension of the tapu associated with human waste, in that water bodies receiving wastewater also become tapu. For example, Feltham (2021) discusses how it may be possible to remove some of the

[11] Many such whakataukī exist; this is but one example.

restrictions associated with tapu, without overcoming the intrinsic state of tapu; biowastes may be treated to a point where they are 'safe' from a scientific perspective but the intrinsic tapu of human waste persists. Waters receiving wastewaters therefore may not be recognised as safe in the traditional sense as wai māori, making them inappropriate for use as drinking-water, in food preparation, for bathing or other daily use (Afoa and Brockbank 2019). Further, mahinga kai from such waters cannot be harvested or eaten (Pauling and Ataria 2010; Afoa and Brockbank 2019). The concerns that Māori have regarding the discharge of wastewater and stormwater therefore extend beyond the potential impacts of contaminants on ecosystem and human health; the loss of access to wai māori and mahinga kai impacts on the ability of iwi/hapū/whānau to obtain physical sustenance from these resources or to engage in traditional cultural and spiritual practices, and detracts from core Māori values including manaakitanga (to care and provide for manuhiri/guests) and kaitiakitanga (guardianship, stewardship). Indeed, numerous claims to the Waitangi Tribunal highlight these concerns, and the associated impacts on physical, spiritual, cultural and social wellbeing (Pauling and Ataria 2010; Drurie et al. 2017; Afoa and Brockbank 2019).

The mauri of water can only be restored by Papatūānuku (Mother Earth). By infiltrating and interacting with the whenua (land), wai goes through a process of transformation from tapu to noa, returning it to a state that is safe for contact and consumption (Pauling and Ataria 2010; Afoa and Brockbank 2019). This process is the basis for Māori expressing a general preference for wastewaters and stormwaters to be treated using land-based methods such as wetlands – allowing for both the removal of contaminants and the transformation from tapu to noa, before being discharged to waterways (Pauling and Ataria 2010; Afoa and Brockbank 2019).

In contemporary society, reticulated wastewater and stormwater systems present challenges for Māori, both in terms of scale and the inability to exercise traditional controls of tapu and noa (Ataria et al. 2016). Unlike traditional waste streams, there is seldom a clear knowledge of the various constituents of contemporary waste streams, and it is not possible to maintain waste separation. Municipal wastewaters, stormwater, and trade wastes – themselves presenting a complex mixture of different types of waste[12] – combine in a way that make it impossible to achieve proper separation and disposal (Ataria et al. 2016). Further, although traditional kōrero does not tend to focus on specific contaminants (eg pathogens, metals etc) within waste streams, the changing nature of these wastes – in particular the proliferation of

[12] For example, in addition to urine, faeces, food and commercial wastes, municipal wastewater contains menstrual waste and often hospital and mortuary wastes, which present significant cultural challenges for hapū in being assured that treatment processes can adequately facilitate transition from tapu to noa (Ataria et al. 2016).

chemical contaminants – presents further challenges for tikanga Māori. For example, the following passage from a Ngā Kaihautū Tikanga Taiao (Māori Advisory Committee to the Board of the Environmental Protection Authority) report discusses the issue of tapu and noa in relation to persistent chemicals (in Ataria et al. 2016):

> *"Many Māori consider that within the realms of Papatūānuku and Ranginui there exist a range of established processes and relationships that continuously cycle chemicals through the spiritual states of tapu (restricted state) and noa (relaxed or normalised state). In a scientific context these processes could be termed bio- and physico-chemical transformation which acts to breakdown and modify chemical compounds to basic building blocks for other uses or re-partitioning back into the environment. Compounds that have been synthesised with properties that convey resistance to these natural processes are often met with opposition – particularly if their intended use involves direct deployment into the environment or at some point during the life cycle of these products environmental exposure occurs."*

Restrictions of tapu may be placed on situations, material, places or people not only where potential risks are known, but also where potential risks are unknown (Georgia Bell, personal communication; Ataria et al. 2016). As many of the contaminants in contemporary wastewater, stormwater and trade waste streams are emerging contaminants, there is no existing tikanga regarding them, and there remain many uncertainties regarding their environmental fate and potential impacts on human or environmental health. The unknown nature of the risks to people or the environment that may be associated with these recalcitrant and/or emerging contaminants means Māori may consider that these waste streams remain tapu, even when many of the known risks have been mitigated, and new tikanga may be required for this contemporary setting and its associated challenges (Georgia Bell, personal communication).

3. PATHOGENIC MICROORGANISMS

A pathogen is a microorganism (eg, bacterium, virus, protozoa, fungus or helminth) that causes disease. To become infected by a pathogen, a person must be exposed to some minimum number of pathogenic organisms, known as an infectious dose (UWRRC 2014). This number can vary significantly for different pathogens – viruses and protozoa generally have very low infectious doses, often estimated to be between 1 and 100 organisms (Fong and Lipp 2005; Yezli and Otter 2011), while bacterial pathogens have higher infectious doses, ranging from 10 to >1,000,000 (Schroeder and Wuertz 2003). Individuals may also differ significantly in their susceptibility to infection, so that the same dose may cause infection in some people but not others (Teunis et al. 2020). Routes of transmission also vary between different pathogens: for example, infection by some pathogens requires direct contact with infected blood or body fluids, while other pathogens are transmitted via inhalation of infected droplets or aerosols, or ingestion of contaminated food or water[13]. Not all individuals who become infected will become ill; the probability and severity of illness also depends on the specific pathogen, the strain of pathogen, the dose ingested, and the overall health of the individual (Teunis et al. 2020). The risk of severe illness is greatest for people with lower immunity, including children, the elderly, pregnant women, or people who are immunocompromised or have underlying health conditions (Ministry for the Environment and Ministry of Health, 2003). At-risk individuals may also develop illness following infection by environmental or commensal microorganisms that do not typically cause issues in healthy individuals; these microorganisms are known as opportunistic pathogens (Martinez 2014).

Despite large advances in water and wastewater treatment in recent decades, waterborne disease still poses a major risk to public health globally (Toze 1997; WHO 2022b). Many of the key causes of waterborne illness are associated with human or animal faeces. These ‘enteric pathogens’ reproduce in the gastrointestinal tract, where they cause illness (typically self-limiting gastroenteritis) either by damaging cellular structures or by producing cytotoxins (Petri Jr et al. 2008). They are shed in high numbers in the stool of both individuals with acute illness and those with asymptomatic infection, and transmission subsequently occurs through the faecal-oral route, including the ingestion of contaminated food or water (Toze 1997; Fong and Lipp 2005). There is evidence to suggest that the human health risks associated with faecal contamination differ depending on the source of contamination (ie, human, cattle, wildfowl etc), due to differences in the presence, strain and concentration of

[13] https://www.cdc.gov/csels/dsepd/ss1978/lesson1/section10.html Accessed 3 May 2022

specific pathogens that may be present in each host group (Soller et al. 2010, 2014). Human faecal material is considered to present the greatest risk to public health, since any pathogens present are assumed to be capable of infecting humans, particularly viruses, which are both highly infectious and host-specific (Soller et al. 2010, 2015). Conversely, only some animal pathogens are capable of causing infection and illness in humans. In addition, some microorganisms that are commensal inhabitants of animal hosts may be pathogenic in humans. Animal-associated microorganisms that cause infection in humans are referred to as zoonotic (Schroeder and Wuertz 2003).

Some environmental microorganisms may also be pathogenic in humans. These organisms naturally inhabit soils, surface waters, foods, and vegetation but can cause infection and illness when humans come into contact with them (eg *Vibrio* spp., *Legionella* spp.) (Cangelosi et al. 2004). Some of these organisms are primary pathogens and cause disease in normal healthy hosts, while others are opportunistic and cause disease only in individuals with predisposing conditions such as reduced immunity or open wounds (Cangelosi et al. 2004; Martinez et al. 2014; WHO 2022b).

Unlike chemical contaminants, pathogens are living organisms that may persist, die off or grow, depending on environmental conditions (UWRRC 2014). Environmental pathogens are able to survive and thrive to a greater extent than strict human or zoonotic pathogens (Cangelosi et al. 2004).

3.1 PATHOGENS IN WASTEWATER

A wide variety of waterborne, foodborne, bloodborne, respiratory and sexually-transmitted diseases may be shed in urine, faeces, vomit, sputum and other bodily wastes, and may therefore be detected in sewage (Gerardi and Zimmerman 2005; Sinclair et al. 2008; Chahal et al. 2016; Garcia-Aljaro et al. 2018; Table 2). As pathogens are only shed by infected individuals, the pathogens present in municipal wastewater will reflect the diseases circulating within the community (Gerardi and Zimmerman 2005). Most of the microbial pathogens present in wastewater are of enteric origin, with human faeces being the primary source (Toze 1997; Chahal et al. 2016). Pathogens may also enter the wastewater network in contaminated water associated with household uses including bathing and laundry (Gerardi and Zimmerman 2005). Trade waste from animal processing facilities (eg meatworks) or veterinary clinics can also be a source of zoonotic pathogens, as can soils or

animal wastes entering the network through stormwater inflow/infiltration (Chahal et al. 2016; Garcia-Aljaro et al. 2018).

Of the range of bacterial pathogens present in wastewater, microorganisms commonly recognised as 'frank' primary pathogens, with known transmission via contaminated water include *Campylobacter* spp., *Salmonella* spp., *Shigella* spp, *Vibrio parahaemolyticus* and *Yersinia* spp. (Chahal et al. 2016). Other bacteria are also transmitted via the faecal-oral route but have very high infectious doses (eg *Vibrio cholera* at 10^6 organisms; Schroeder and Wurtz 2003), or have not yet been conclusively demonstrated to show waterborne transmission (eg, *Helicobacter pylori*; WHO 2022b), and may present a lesser risk to public health through exposure to wastewater. A number of opportunistic bacterial pathogens may also be present in wastewater, causing wound infections (eg *Pseudomonas aeruginosa, Vibrio vulnificus*) or respiratory infections following inhalation (eg *Legionella pneumophila*[14], *Mycobacterium* spp.[15]).

Viruses also show considerable diversity in wastewaters, and concentrations up to 10^{10} viral particles per litre are reported (Table 2; Corpuz et al. 2020). The major viral pathogens in wastewater are enteric viruses, including hepatitis A virus, norovirus, rotavirus, adenovirus 40/41, astrovirus, and various enteroviruses (coxsackieviruses, poliovirus and echovirus) (Chahal et al. 2016). Other enteric viruses such as polyomavirus are widely detected but commonly associated with asymptomatic infection, while bloodborne viruses such as HIV and hepatitis B virus may be detected in wastewater but are not transmitted through contaminated water (Gerardi and Zimmerman 2005). Respiratory viruses are also commonly detected, however their viability and/or the potential for their transmission from wastewater remains uncertain (eg SARS-CoV-2; Giacobbo et al. 2021).

Protozoan parasites *Cryptosporidium* and *Giardia* are commonly detected in sewage, although their abundance may be highly seasonal; in New Zealand, higher cryptosporidiosis case notifications are reported in spring and associated with lambing and calving (ESR 2021). Other protozoan pathogens including *Cyclospora*, *Entamoeaba hystolitica* and *Isopora helii* are also detected in sewage (Chahal et al. 2016).

Internationally, helminths are known health hazard associated with sewage and wastewater, however these are not considered to be of concern in New Zealand (NZWERF 2002).

[14] https://www.who.int/news-room/fact-sheets/detail/legionellosis. Accessed 3 May 2022
[15] https://bmcpublichealth.biomedcentral.com/track/pdf/10.1186/s12889-022-12527-z.pdf Accessed 2 May 2022

Table 2: **Occurrence and indicative maximum concentrations of human pathogens that are commonly detected in municipal sewage, and the primary illness(s) (excluding sequelae) they cause.**

Pathogen	Disease	Maximum concentration in sewage (100/ml)
Viruses		
Adenoviruses	Respiratory illness, gastroenteritis	10^{10} GC
*Human adenovirus 40/41	Gastroenteritis	10^{9} GC; 10^{3} PFU
Astroviruses	Gastroenteritis	10^{7} GC; 10^{4} PFU
*Enteroviruses	Range of illnesses, respiratory and gastrointestinal illness	10^{6} GC
Poliovirus	Poliomyelitis	nr
Coxsackievirus	Respiratory illness, meningitis, fever	nr
Echovirus	Meningitis, encephalitis, fever	nr
*Hepatitis A virus	Hepatitis	10^{9} GC
Hepatitis E virus	Hepatitis	10^{7} GC
HIV	Acquired immunodeficiency syndrome (AIDS)	nr
Influenza virus	Influenza	nr
*Noroviruses	Gastroenteritis	10^{9} GC
Norovirus GI		10^{9} GC
Norovirus GII		10^{9} GC
Reoviruses	Respiratory illness, gastroenteritis	10^{4} GC
*Rotaviruses	Gastroenteritis	10^{8} GC
Sapovirus	Gastroenteritis	10^{5} GC
Protozoa		
Balantidium coli	Balantidiasis	nr
**Cryptosporidium* spp.	Cryptosporidiosis	10^{4} oocysts
C. hominis		nr
C. parvum		nr
Cyclospora cayetanesi	Persistent diarrhoea	nr
Entamoeba histolytica	Amoebiasis, acute dysentery	100
**Giardia intestinalis*	Giardiasis	10^{6} cysts
Toxoplasma gondii	Toxoplasmosis	nr
Bacteria		
Bacillus anthracis	Anthrax	nr
**Campylobacter* spp.	Campylobacteriosis, gastroenteritis	10^{6} MPN
C. jejuni		nr
C. coli		nr
Clostridium perfringens	Gastroenteritis, tissue necrosis	nr
*Enteropathogenic *E. coli (eg E. coli* O157*)*	Gastroenteritis	10^{2} MPN
Francisella tularensis	Tularaemia	nr
Helicobacter pylori	Gastritis, stomach ulcers	nr
Legionella pneumophila	Legionnaires' disease	nr
Leptospira interrogans	Leptospirosis	nr
Mycobacterium tuberculosis complex	Tuberculosis	10^{4} MPN

Table 2 continued

Pathogen	Disease	Maximum concentration in sewage (100/ml)
Nocardia spp.	Nocardiosis	nr
Pseudomonas spp.		nr
**Salmonella* spp.	Salmonellosis, gastroenteritis	10^5 MPN
S. enterica paratyphi	Paratyphoid fever	nr
S. typhi	Typhoid fever	nr
**Shigella* spp.	Shigellosis (bacillary dysentery)	10^8 MPN
Vibrio spp.		
V. cholerae	Cholera, gastroenteritis	10^5 MPN
V. parahaemolyticus	Gastroenteritis	
Yersinia spp.	Yersiniosis, gastroenteritis	nr
Fungi		
Aspergillus fumigatus	"Farmer's lung"	nr
Candida spp	Candidiasis	nr
Helminths		
Ancylostoma duodenalis	Anaemia	10^2
Ascaris lumbricoides	Ascariasis	10^2
Echinococcus granulosus	Echinococosis	nr
Hymenolepsis nana	Hymenolepiasis	nr
Necator americanus	Anaemia	10^2
Schistomsoma spp.	Schistosomiasis	nr
Taenia spp.	Taeniasis	nr
Trichuris trichurus	Anaemia, diarrhoea	10

Compiled from Gerardi and Zimmerman (2005), Chahal et al. (2016), Garcia-Aljaro et al. (2018), Corpuz et al. (2020) and WHO (2022b). Pathogens considered to present a significant human health risk via wastewater are indicated with *.

GC – gene copies; MPN – most probable number; nr – not reported within these publications; PFU – plaque-forming units.

3.2 PATHOGENS IN STORMWATER

As discussed in Section 1.2.3, numerous studies have highlighted the ubiquitous issue of sewage contamination in stormwater systems (eg Sauer et al. 2011; Gasperi et al. 2012; Zgheib et al. 2012; Chong et al. 2013; Sidhu et al. 2013; Stea et al. 2015). Municipal wastewaters may therefore be a significant source of microbial pathogens in urban stormwater networks. Pathogens may also enter stormwater independently of wastewater networks, for example, as rainfall washes human or wild and domestic animal wastes (eg, dogs, cats, birds, rats) from lawns, footpaths, roofs and other areas into the stormwater network (Jiang et al. 2005; Staley et al. 2016; Garcia-Aljaro et al. 2018; Steele et al. 2018;

Müller et al. 2020; Monteiro et al. 2021). As the diversity and concentrations of pathogens present in faeces may differ between sources (eg, human, cattle, birds, dogs) (Soller et al. 2010, 2014), stormwater is expected to have a different pathogen profile to municipal wastewater, being dominated by animal rather than human waste (Garcia-Aljaro et al. 2018; Ahmed et al. 2019). The human health risk posed by pathogens in stormwater may therefore differ depending on the particular sources of faecal material present in stormwaters. In addition, the 'age' of faecal material (eg, whether freshly excreted or not) may also influence the level of risk, as pathogens tend to die off once they have been shed to the environment, although the rate of attenuation differs significantly between pathogens and with environmental conditions. Environmental pathogens associated with soils or surface waters may also wash into stormwater systems, but their presence in stormwater does not appear to be well-studied.

Multiple studies have reported the presence of faecal indicator organisms, faecal source tracking markers[16] and pathogenic microorganisms in stormwaters, waterways receiving significant stormwater volumes, and roof-harvested rainwaters[17] (eg Jiang et al. 2005; Noble et al. 2006; Rajal et al. 2007; Ahmed et al. 2008; Cizek et al. 2008; Sidhu et al. 2012, 2013; Ahmed et al. 2014; Dobrowsky et al. 2014; Staley et al. 2016; Steele et al. 2018; Ahmed et al. 2019; Denissen et al. 2021; Monteiro et al. 2021; Bouchali et al. 2022). A summary of the pathogens that have been reported in stormwaters is presented in Table 3 and demonstrates that a variety of human-specific, zoonotic and environmental pathogens may be present, including bacterial pathogens (*Campylobacter* spp.*, Salmonella* spp., *Legionella pneumophila* and enteropathogenic *E. coli*), protozoan pathogens (*Cryptosporidium* and *Giardia)* and enteric viruses *(*adenoviruses, enterovirus, norovirus and rotavirus). In general, however, quantitative data on the abundance of pathogens in stormwater runoff remains scarce, and is often based on samples collected from receiving waters rather than stormwaters *per se* (McBride et al. 2013; Ahmed et al. 2019; Monteiro et al. 2021). Pathogen concentrations in stormwater are, however, assumed to be low when compared with those in wastewater (Mallard 1980). Further, epidemiological studies have demonstrated mixed results regarding the possible increased risk of illness associated with exposure to stormwaters (UWRRC 2014). The human health risks associated with pathogens in stormwater are therefore less clearly understood than for municipal wastewaters.

[16] Faecal indicator organisms and faecal source tracking markers are microorganisms whose presence is used to identify the likely presence and potential sources of faecal contamination, but do not typically cause disease. Such assays are used as an alternative to direct pathogen assessment since they are more quickly and cheaply undertaken, and negate the need to analyse samples for an array of different pathogens.

[17] Roof-harvested rainwater can highlight the potential for pathogens to be washed from roofs and into the stormwater network.

Table 3: Occurrence and indicative maximum concentrations (where reported) of human pathogens that have been detected in urban stormwater, and the primary illness(s) (excluding sequelae) they cause.

Pathogen	Disease	Maximum concentration in stormwater (100 ml^{-1})
Viruses		
Adenoviruses	Respiratory illness, gastroenteritis	10^3 GC
*Human adenovirus 40/41	Gastroenteritis	10^2 GC
*Enteroviruses	Range of illnesses, including respiratory and gastrointestinal illness	10^3 GC
Poliovirus[a]	Poliomyelitis	nr
Coxsackievirus[a]	Respiratory illness, meningitis	nr
Echovirus[a]	Meningitis, encephalitis	nr
*Hepatitis A virus	Hepatitis	nr
*Noroviruses	Gastroenteritis	
Norovirus GI		10^2 GC
Norovirus GII		10^2 GC
*Rotaviruses	Gastroenteritis	nr
Protozoa		
**Cryptosporidium* spp.	Cryptosporidiosis	10^2 oocysts
C. hominis or parvum		1 oocyst
**Giardia* spp.	Giardiasis	0.3 cysts
Bacteria		
Aeromonas spp. *A. hydrophila*	Gastroenteritis, necrotising fasciitis	nr
**Campylobacter* spp.	Campylobacteriosis, gastroenteritis	10^3 GC
C. jejuni		10^2 GC
C. coli		10^2 GC
C. lari		10^2 GC
*Enteropathogenic *E. coli* (eg *E. coli* O157)	Gastroenteritis	10^4 GC
Listeria monocytogenes[b]	Listeriosis	nr
Mycobacterium tuberculosis[b]	Tuberculosis	nr
Pseudomonas aeruginosa	Respiratory, urinary tract and blood infections	nr
**Salmonella* spp.	Salmonellosis, food poisoning	10 MPN; 10^4 GC
Stenotrophamonas lamtophilia	Respiratory, urinary tract and blood infections	nr
Yersinia spp.[b]	Yersiniosis, gastroenteritis	nr

Compiled from Mallarge (1980), Ahmed et al. (2019), Denissen et al. (2021), Bouchali et al. (2022). GC – gene copies, nr – not reported within these references. Pathogens considered to present a significant human health risk via wastewater are indicated with *.

3.3 HEALTH EFFECTS OF PATHOGENS

As described above, a wide diversity of bacterial, viral and protozoan pathogens may be detected in wastewater and/or stormwater. However, the presence of some human pathogens in wastewater or stormwater likely presents a low risk to public health by virtue of their routes of transmission (eg, bloodborne or inhalation of aerosols) or high infectious dose. Similarly, opportunistic pathogens can cause serious illness but their effects are typically limited to individuals with predisposing conditions rather than the general public. Health outcomes caused by pathogens that are considered to have the greatest potential to cause infection in the general population following contact with wastewater, stormwater or their receiving environments are described below.

3.3.1 Viruses

Enteric viruses are amongst the most important of the pathogens that are found in wastewater and stormwater (Toze 1997): they are commonly shed at high levels by infected individuals (eg up to 10^{12} viral particles per gram of stool) (Fong and Lipp 2005; Hall 2012; UWRRC 2014), have very low infectious doses (Haas et al. 1993; Yezli et al. 2011; Teunis et al. 2020), and may persist in the environment for weeks to months (Lees 2000; Jiang et al. 2001). Enteric viruses have been linked to outbreaks of illness originating from sewage-contaminated drinking-water sources, recreational waters, and shellfish harvesting waters (Tang et al. 1991; Lees 2000; Fong and Lipp 2005).

Norovirus. Noroviruses (NoV) are a genetically diverse group that can be divided into ten genogroups (GI-GX)[18], of which GI, GII and GIV are known to infect humans (Ludwig-Begall et al. 2021). Human noroviruses are a major public health concern due to their high infectivity and environmental persistence (NZFSA 2017; Teunis et al. 2020), and are thought to be responsible for half of all outbreaks of gastroenteritis worldwide (Hall 2012). Following a brief incubation period (12-48 hours), clinical symptoms develop that include nausea, vomiting, diarrhoea, fever and abdominal pain[19] (NZFSA 2017; Ludwig-Begall et al. 2021). Illness persists for 12-60 hours after which most cases make a complete recovery (Lees 2000; WHO 2022b), although viral shedding may persist for several weeks (NZFSA 2017). Norovirus has a very low infectious dose, and <10 viral particles is thought to be sufficient to

[18] https://www.cdc.gov/norovirus/lab/virus-classification.html Accessed 3 May 2022
[19] https://www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/norovirus-vomiting-and-diarrhoea-bugs Accessed 3 May 2022

cause infection (Teunis et al. 2008, 2020; Hall 2012), although infectivity and pathogenicity is likely to differ between genogroups (Teunis et al. 2020).

Adenovirus. Adenoviruses (HAdV) are common human pathogens causing disease in the respiratory, gastrointestinal and urinary tracts, eyes, liver and/or adenoids (Fong and Lipp 2005). More than 100 HAdV types have been isolated and classified into 7 species (A-G) (Rafie et al. 2021). The two members of species F (types 40 and 41) are primarily associated with gastrointestinal tropism and are a major cause of diarrhoeal illness in children (Lees 2000; He and Jiang 2005; Rafie et al. 2021; WHO 2022b). Incubation periods vary between 3 and 10 days, and symptoms include vomiting, diarrhoea and abdominal pain, sometimes with fever. Disease is usually self-limiting, and may be less severe than other enteric viruses, however may show a prolonged course, with diarrhoea persisting for several weeks (Lees 2000). Asymptomatic infection is also common (Francy et al. 2011). High attack rates observed during outbreaks, however the role of contaminated water in transmission remains unclear (WHO 2022b).

Hepatitis A virus (HAV). Hepatitis A is characterised by self-limiting inflammation of the liver.[20] Following a protracted incubation period (up to 50 days), symptoms of HAV infection develop that may range from mild to debilitating, including flu-like symptoms, nausea and stomach pain, jaundice, fever and malaise, and usually persist for 1-2 weeks[21] (Lees 2000; Gerardi and Zimmerman 2005; Hofmeister et al. 2019; WHO 2022b). Some infected individuals, especially children, may remain asymptomatic (WHO 2022b). Most cases make a full recovery within six months, with fulminant hepatic failure occurring in less than 1% of cases (Hofmeister et al. 2019; WHO 2022b). In China, contamination of a significant shellfish-harvesting area with sewage resulted in approximately 300,000 cases of HAV infection (Tang et al. 1991). Vaccines developed for HAV are highly effective in preventing infection.

Rotavirus. There are at least seven species of rotavirus (A through G), with rotavirus A causing more than 90% of human rotavirus infections (Lees 2000; WHO 2022b). Rotavirus is most commonly identified in paediatric infections and represents a leading cause of severe diarrhoeal illness in infants and children worldwide[22] (Lees 2000; Payne and Parashar 2018; WHO 2022b). Clinical symptoms can range from mild, watery diarrhoea to severe diarrhoea with vomiting and fever, with dehydration and electrolyte imbalance being frequent

[20] https://www.who.int/news-room/fact-sheets/detail/hepatitis-a Accessed 28 April 2022
[21] https://www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/hepatitis Accessed 28 April 2022
[22] https://www.who.int/teams/health-product-policy-and-standards/standards-and-specifications/vaccines-quality/rotavirus Accessed 17 November 2022

complications. Symptoms usually present 1-3 days following infection, and last 3-8 days[23] (Payne and Parashar 2018). Rotavirus infection occurs in adults but tends to be mild or asymptomatic (Lees 2000; Payne and Parashar 2018). Data suggests that the virus can be shed at up to 10^{12} particles per gram faeces, and the infectious dose for rotavirus is <100 viral particles (Lees 2000; Payne and Parashar 2018). Vaccination has been effective in reducing the burden of rotavirus in children, although it remains high (WHO 2017; Payne and Parashar 2018). For example, in the year following the introduction of the RotaTeq® vaccine[24] in New Zealand, rotavirus-related hospitalisations in under-5 year olds declined 85% compared with the previous 5-year average, and rotavirus outbreaks were reduced over 93%.[25]

Enteroviruses. Enteroviruses are a large family of viruses that includes 69 distinct serotypes that can cause infection in humans, including polioviruses, groups A and B coxsackie viruses, echoviruses and numbered enteroviruses (Lees 2000; WHO 2022b). The range of enteroviruses cause a wide spectrum of illnesses, although in most instances they result in asymptomatic infection or mild illness, with symptoms including gastrointestinal distress, fever, running nose, cough, skin rashes, mouth sores, and body aches[26, 27] (Lees 2000). A small proportion of enterovirus infections are associated with serious disease such as severe respiratory illness, hand foot and mouth disease, myocarditis, pericarditis, aseptic meningitis, and poliovirus, or may play a role in the development of type 1 diabetes in children[28, 29] (Lees 2000; WHO 2022b).

3.3.2 Protozoa

In the developed world, the two protozoa most commonly associated with faecally-contaminated waters are *Giardia and Cryptosporidium* (UWRRC 2014). They are both associated with human and animal faeces, and produce (oo)cysts that are extremely resistant to environmental conditions and water treatment processes (WHO 2022b).

[23] https://www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/rotavirus Accessed 28 April 2022
[24] RotaTeq® was replaced by Rotatrix® on the National Immunisation Schedule in 2017. https://surv.esr.cri.nz/PDF_surveillance/Rotavirus/2016Rotavirus.pdf Accessed 28 April 2022.
[25] [25] https://www.health.govt.nz/our-work/immunisation-handbook-2020/18-rotavirus Accessed 28 April 2022.
[26] https://www.health.nsw.gov.au/Infectious/factsheets/Pages/enteroviruses.aspx Accessed 17 November 2022
[27] https://www.cdc.gov/non-polio-enterovirus/about/symptoms.html Accessed 17 November 2022
[28] https://www.health.nsw.gov.au/Infectious/factsheets/Pages/enteroviruses.aspx Accessed 17 November 2022
[29] https://www.cdc.gov/non-polio-enterovirus/about/symptoms.html Accessed 17 November 2022

Cryptosporidium. There are approximately 13 species of *Cryptosporidium*, of which *C. hominis* and *C. parvum* are of most significance to human health (Gerardi and Zimmerman 2005; WHO 2022b). *Cryptosporidium hominis* is almost exclusively found in human faeces, while *C. parvum* is commonly identified in cattle, as well as sheep, pigs, deer, mice and rats (UWRRC 2014; WHO 2022b). Infection with *Cryptosporidium* can cause cryptosporidiosis, an acute illness that includes self-limiting diarrhoea and abdominal pain, occasionally with nausea, vomiting and fever. Illness usually resolves within a week but cases lasting more than a month are reported (WHO 2022b)[30]. Incubation periods range between 1 and 12 days, averaging 7 days.[31] Data from human volunteers suggests that the infectious dose is as little as 10 oocysts (WHO 2022b). In 1993, an estimated 400,000 people in Milwaukee, USA became ill after the municipal drinking-water supply was contaminated with *C. parvum* (MacKenzie et al. 1994).

Giardia. Although there are multiple species of *Giardia*, human infection is usually caused by *G. intestinalis* (also known as *G. lamblia* or *G. duodenalis*) (WHO 2022b), which can be found in the faeces of humans, cattle, cats and dogs.[32] Incubation periods can vary from 3 to 25 days, with symptoms in both adults and children most commonly including diarrhoea, abdominal cramps, nausea and fatigue, and severe cases resulting in nutritional deficiencies due to intestingal malabsorption[33] (ESR 2018b; WHO 2022b). Known as giardiasis, illness is usually self-limiting, although chronic cases may persist for a year or more (WHO 2022b). Data from human volunteer studies suggests that the infectious dose is very low, and that ingestion of 1-10 cysts may present a meaningful risk of infection (ESR 2018b; WHO 2022b).

3.3.3 Bacteria

Campylobacter. *Campylobacter* is considered to be the most common cause of bacterial gastroenteritis in the world[34] and campylobacteriosis the most commonly notified disease in New Zealand (ESR 2021). A number of zoonotic species of *Campylobacter* are carried by cattle, sheep, pigs, cats, dogs and birds (especially poultry).[35] *Campylobacter jejuni* is most

[30] https://www.cdc.gov/parasites/crypto/illness.html Accessed 28 April 2022
[31] https://www.health.govt.nz/our-work/diseases-and-conditions/communicable-disease-control-manual/cryptosporidiosis Accessed 28 April 2022
[32] https://www.health.govt.nz/our-work/diseases-and-conditions/communicable-disease-control-manual/giardiasis Accessed 29 April 2022
[33] https://www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/food-and-water-borne-diseases/giardia Accessed 29 April 2022
[34] https://www.who.int/news-room/fact-sheets/detail/campylobacter Accessed 2 May 2022
[35] https://www.health.govt.nz/our-work/diseases-and-conditions/communicable-disease-control-manual/campylobacteriosis Accessed 2 May 2022

frequently isolated from infected individuals, although *C. coli* and *C. lari* are also associated with human illness.[36,] The infectious dose may be as low as <800-1,000 organisms (ESR 2018a; WHO 2022b). Following an incubation period of 1-4 days, illness is characterised by abdominal pain, diarrhoea (possibly bloody), vomiting, chills and fever. Infection is self-limited and resolves in 3-7 days. In some cases, *Campylobacter* infection can result in sequelae including reactive arthritis, Guillain-Barré syndrome, and inflammatory bowel disease (ESR 2018a; WHO 2022b). In 2016, contamination of the Havelock North water supply by *C. jejuni* following heavy rainfall resulted in the largest-ever reported outbreak of campylobacteriosis, with an estimated 6,260-8,320 cases (Gilpin et al. 2020).

Salmonella*. Salmonella* is a key cause of diarrhoeal disease globally, with a high burden of foodborne disease[37]. Domestic and wild animals and birds, as well as humans, are common hosts.[38] There are two species, with thousands of serotypes identified to date, all of which are expected to cause illness in humans[39]. Illness caused by *Salmonella* can cause four clinical conditions: gastroenteritis/salmonellosis, septicaemia, typhoid fever, and a carrier state in cases with previous infection (WHO 2022b). Salmonellosis is characterised by uncomplicated gastroenteritis lasting 2-7 days, and cases typically make a full recovery,[40] although rare complications can include bacteraemia and reactive arthritis (ESR 2018c). The infective dose for non-typhoidal *Salmonella* ranges from <10 to 10^9 colony-forming units, depending on serovar and mode of transmission (ESR 2018c). Typhoid fever caused by *Salmonella* Typhi can be a life-threatening infection and is largely restricted to developing countries and areas of poor sanitation (WHO 2022b).

STEC/*E. coli* O157. The majority of *E. coli* strains are harmless to people; however, a number of strains, such as shiga toxin-producing *E. coli* (STEC) are capable of causing severe illness.[41] There are approximately 200 strains of STEC, with serotype O157:H7 being most frequently reported in human illness (ESR 2018d). STEC is most commonly associated with cattle although sheep, goats, deer, pigs and some birds may also be carriers.[42] The infective dose for *E. coli* O157:H7 is estimated at 50 to several hundred cells (WHO 2017; ESR 2018d). The incubation period is 3 to 8 days, and symptoms range from mild, watery or bloody diarrhoea, abdominal cramps and vomiting[43] (ESR 2018d). Most people recover

[36] https://www.who.int/news-room/fact-sheets/detail/campylobacter Accessed 2 May 2022
[37] https://www.who.int/news-room/fact-sheets/detail/salmonella-(non-typhoidal) Accessed 4 May 2022
[38] https://www.health.govt.nz/our-work/diseases-and-conditions/communicable-disease-control-manual/salmonellosis Accessed 4 May 2022
[39] https://www.who.int/news-room/fact-sheets/detail/salmonella-(non-typhoidal) Accessed 4 May 2022
[40] https://www.who.int/news-room/fact-sheets/detail/salmonella-(non-typhoidal) Accessed 4 May 2022
[41] https://www.who.int/news-room/fact-sheets/detail/e-coli Accessed 4 May 2022
[42] https://www.who.int/news-room/fact-sheets/detail/e-coli Accessed 4 May 2022
[43] https://www.who.int/news-room/fact-sheets/detail/e-coli Accessed 4 May 2022

within ten days, however an estimated 10% of cases will go on to develop haemolytic uraemic syndrome (HUS), with a case-fatality rate of 3-5% (ESR 2018d) and up to 50% of survivors of HUS experiencing neurological or renal sequelae, including end-stage renal disease[44]. In Walkerton, Canada, contamination of the drinking-water supply by stormwaters contaminated with cattle faeces resulted in an outbreak of illness caused by *E. coli* O157 and *C. jejuni,* comprising more than 2,300 cases and 7 deaths (Hrudey et al. 2003; WHO 2022b)

Shigella. There are four species of *Shigella* that cause illness in humans: *S. dysenteriae, S. flexneri, S. boydii* and *S. sonnei. S. dysenteriae* can spread in epidemics and is associated with serious disease, while *S. boydii* and *S. sonnei* cause relatively mild illness (WHO 2005.) Shigellosis is characterised by acute diarrhoea (often bloody), fever and abdominal cramps, with high secondary attack rates amongst contacts. Incubation period may range between 12 hours and a week, although is usually 1-3 days, and illness lasts 5 to 7 days[45,46]. Rare complications may include reactive arthritis, blood stream infections, seizures, or HUS.[47] *Shigella* has a relatively low infectious dose compared with other bacteria, at 10-200 organisms[48] (ESR 2001; WHO 2005; WHO 2022b). An estimated 99% of *Shigella* infections occur in developing countries (WHO 2005) and the majority of cases notified in New Zealand are travel-related (ESR 2021).

3.4 REGULATIONS AND GUIDELINES

Because of the diversity of enteric pathogens that may be present in environmental waters (including wastewaters and stormwaters) and technical challenges associated with their detection, microbial indicator organisms are commonly used to infer the risk from enteric pathogens (Toze 1997; Field and Samadpour 2007; Harwood et al. 2014; UWRRC 2014). These indicator organisms (usually *E. coli,* enterococci or coliform bacteria) are present in high concentrations in the gastrointestinal tract of humans and warm-blooded animals, thus their presence in water highlights the likelihood of faecal contamination and possibility of associated pathogens. Assays for indicator organisms are relatively quick (<24 hours) and inexpensive, making them conducive to routine use in a range of settings, including

[44] https://www.who.int/news-room/fact-sheets/detail/e-coli Accessed 4 May 2022
[45] https://www.cdc.gov/shigella/symptoms.html Accessed 3 May 2022
[46] https://www.health.govt.nz/our-work/diseases-and-conditions/communicable-disease-control-manual/shigellosis Accessed 3 May 2022
[47] https://www.cdc.gov/shigella/symptoms.html. Accessed 3 May 2022.
[48] https://www.health.govt.nz/our-work/diseases-and-conditions/communicable-disease-control-manual/shigellosis Accessed 3 May 2022.

monitoring the quality of drinking-waters and wastewater effluents. However, there are limitations in using faecal indicators as a proxy to indicate human health risk: in particular, the presence and concentrations of indicators does not correlate well with all pathogens (especially viruses), and they are unable to identify the source of faecal contamination (ie, human or animal) (Field and Samadpour 2007; Korajkic et al 2018). When concentrations of indicator organisms suggest contamination has occurred, faecal source tracking (FST) tools can be used to help attribute a faecal source(s) (Field and Samadpour 2007; Harwood et al 2014).

Pertinent limits and guidelines for exposure to microbial pathogens and indicators are shown in Table 4. In contrast to the chemical contaminants, microbial contaminants are considered in the context of acute exposure events only, and the likelihood that a single exposure results in infection and/or subsequent illness.

Table 4: Microbial standards and guidelines for drinking-water and recreational waters.

	Microorganism					
	Total coliforms	***E. coli* (/100ml)**	**Enterococci (/100ml)**	**Enteric viruses**	***Cryptosporidium***	***Giardia lamblia***
Drinking water						
Maximum Allowable Value, DWSNZ[49]		<1			<1 infectious (oo)cyst per 100 L[a]	
Maximum Contaminant Limit, US EPA[50]	<5% samples[b]	<1		TT[c]	TT[c]	TT[c]
EU Drinking Water Directive[51]		0	0			
Recreational water						
WHO[52]			≤40[d]			
NZ MfE and MoH[53]		≤260[e]	≤140[f]			
US EPA[54]		100 GM[g] 320 STV	30 GM[g] 110 STV			

[a] MAV is for total pathogenic protozoa. Methods for enumerating pathogenic protozoa are becoming less expensive and more reliable, but they are not available for routine use. The referee method cannot determine species, viability or infectivity of any detected *Cryptosporidium/Giardia* (oo)cysts, and so results ae to be reported as verified (oo)cysts.

[b] No more than 5% of samples within a month are positive for total coliforms. A TC positive sample must then be analysed for *E. coli.*

[c] TT is a required treatment process intended to reduce the level of a contaminant. Use of surface water or groundwater under influence of surface water requires disinfection and filtration to remove contaminants to the following levels: *Giardia* 99.9% removal/inactivation, viruses 99.99% removal/activation. For *Cryptosporidium*, unfiltered systems must include *Cryptosporidium* in watershed control provisions.

[d] Corresponds to the 95th percentile value, with an average probability of <1 case GI in every 100 exposures. Applies to both marine and freshwaters.

[e] For fresh waters. Under 'green surveillance mode,' no single sample should exceed 260 *E. coli*/100ml. For long-term grading of a recreation site, the highest quality band requires a 95th percentile of <130 *E. coli*/100ml.

[f] For marine waters. Under 'green' surveillance mode, no single sample should exceed 140 enterococci /100 mL. For long-term grading of a recreation site, the highest quality band requires a 95th percentile of <40/100ml.

[g] GM – geometric mean. STV – statistical threshold value. The STV approximates the 90th percentile. Guidelines calculated based on estimated illness risk of 32 cases per 1,000 primary recreators.

[49] Water Services (Drinking Water Standards for New Zealand) Regulations 2022. https://www.legislation.govt.nz/regulation/public/2022/0168/latest/whole.html
[50] https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations
[51] European Union Drinking Water Directive 2020/2184.
[52] WHO. (2021a.) *Guidelines on Recreational Water Quality. Volume 1: Coastal and Fresh Waters.* Geneva, Switzerland: World Health Organisation (WHO). 138p.
[53] Ministry for the Environment and Ministry of Health. (2003). Microbiological Water Quality Guidelines for Marine and Freshwater Recreational Areas.
[54] https://www.epa.gov/sites/default/files/2015-10/documents/rwqc2012.pdf

4. HEAVY METALS

Heavy metals are metallic or metalloid elements that have a high atomic weight and a density at least five times greater than water (Tchounwou et al. 2012; Balali-Mood et al. 2021). They are naturally-occurring in the earth's crust, and are therefore present in the environment, usually at trace concentrations (ppb to ppm), as a result of natural processes including rock and soil erosion and volcanic activity (Tchounwou et al. 2012; Jaishankar et al. 2014). However, environmental contamination and human exposure often results from anthropogenic activities, including mining and metal processing/smelting, fossil fuel combustion, nuclear power production, plastics and textiles manufacturing, buildings and infrastructure, wood preservation, pulp and paper manufacturing, agricultural applications, vehicular transport, and discharge of domestic effluents. An overview of key sources and uses of priority heavy metals is presented in Table 5.

Some heavy metals, including copper, chromium, iron, magnesium, selenium and zinc, are required by plants and animals for various biochemical and physiological functions (eg as important constituents of key enzymes), and inadequate intake of these 'essential nutrients' is associated with deficiency syndromes (WHO 1996; Tchounwou et al 2012). Other metals, including aluminium, cadmium, gold, lead, mercury, and nickel do not have an established biological role, and are therefore considered non-essential (WHO 1996; Tchounwou et al 2012). The toxicity of different heavy metals ranges from relatively harmless unless in large amounts or certain forms, to highly toxic at low levels, depending on the specific metal and its chemical species, phase association (ie dissolved or solid), dose and route of exposure, and the age, genetics, and nutritional status of the exposed individual (Goyer et al 2004; Tchounwou et al 2012; McDonald et al. 2022). Even for essential nutrients, there can be a narrow range between beneficial and toxic effects. Heavy metals are not biodegradable, meaning they remain in the environment indefinitely and accumulate with continued input. Further, certain forms are highly soluble in the aquatic environment and are easily absorbed by plants and animals (Kinuthia et al 2020); once they enter the food chain or contaminate drinking water supplies, they can accumulate within the body. This combination of potential toxicity, longevity in the environment and ability to bioaccumulate means the contamination of terrestrial and aquatic environments with heavy metals is a significant environmental concern, with consequences for public health. Further, some heavy metals have been classified as known or probable carcinogens by the US EPA and IARC (International Agency for Research on Cancer) (Tchounwou et al 2012).

Among the metals that are frequently considered to be priority or hazardous pollutants (eg, by the WHO, under the European Water Framework Directive and subsequent legislation) are arsenic, cadmium, chromium, copper, mercury, nickel, lead and zinc (Tchounwou et al 2012; Chashinto et al. 2015; Mannetjie et al. 2018).

Table 5: Significant uses and anthropogenic sources of key heavy metals in the environment.

Arsenic	A ubiquitous element, detected at low concentrations in nearly all natural matrices. Natural sources include volcanic activity and soil erosion. A grey metalloid, with intermediate properties of metals and non-metals. May be present in elemental, organic or inorganic forms. Anthropogenic uses leading to environmental pollution include metal-processing industries and alloys; leather and tanning; wood preservatives; pesticides; pharmaceuticals; manufacture of glass, pigments and paper; ammunition; electronic semiconductors; phosphate fertiliser and other agricultural products. Use in many of these applications is declining following recognition of its toxicity.
Cadmium	Widely distributed at trace levels in the environment; occurs as a minor component of zinc ores. Used in a variety of industrial applications, including manufacturing of batteries, pigments, plasticisers, textiles, electronics, chemicals, ceramics, photographic components, steel coatings and solders, and in metal smelting and finishing. Industrial effluents are a key source of cadmium release to the environment. Also released to atmosphere by metal smelting, fossil fuel combustion, and incineration of municipal solid waste, and to soils and water through phosphate fertilisers.
Chromium	Present in Earth's crust in various oxidation states, mostly trivalent Cr(III); elemental chromium doesn't occur naturally. Enters the environment from a range of natural and anthropogenic sources – anthropogenic emissions are largely the toxic hexavalent chromium (VI), which is subsequently reduced to Cr(III). Naturally present in petroleum and coal and released by their combustion. Widely used in numerous industries, including as chemical catalysts, production of stainless steel, metallurgy, electroplating, paint and pigment production, tanning, wood preservation, oil well drilling, and pulp and paper production.
Copper	A reddish metal that occurs naturally in soils, sediments, water and low levels in air. Properties including durability, malleability, thermal and electrical conductivity and antimicrobial properties afford it a range of applications including electrical wiring and devices, cookware, metal alloys including brass and bronze, animal feeds, fertilisers, wood preservatives, coins, and personal care products.
Lead	A naturally-occurring, bluish-grey metal with a high density, low melting point and relative inertness to oxidation. Activities including fossil fuel combustion, mining and manufacturing contribute to environmental pollution. Applications include lead-acid batteries, ammunition, metal products (solder, pipes, construction materials). Historically also used in other products including petrol additives, paints, batteries, ceramics, cosmetics, plumbing materials, although many of these applications have been phased out.

Table 5 continued.

Nickel	Silvery-white metal, ubiquitous in soils, oil and water at trace levels. Key sources to the environment Used in the production of stainless steel and other alloys with high corrosion and temperature resistance (especially vehicle parts, production machinery, tools, electrical equipment); in jewellery; currencies, household appliances. Nickel compounds are also used as chemical reaction catalysts, pigments and in batteries. Key sources of environmental release are combustion of fossil fuels, incineration of solid waste and sewage sludge, mining, steel production and electroplating.
Mercury	A transition element that exists in nature in three forms (elemental, inorganic and organic). At room temperature, elemental mercury is a silver liquid with a high vapor pressure. Mercury and mercury compounds have been used in a range of industrial and residential products, including fluorescent lighting, electrical switches, pigments, wood preservatives, personal care products, dental amalgams, thermometers and batteries. Key industrial sources include paper production, electric utilities, metal sectors (mining, primary and fabricated metals) and dentistry. Industrial demand for mercury has declined since the 1980s following bans due to its toxicity.
Zinc	Zinc is one of the most common trace metals in the Earth's crust. Most zinc enter the environment as a result of anthropogenic activity, including mining and purification of lead or cadmium ores, burning of coal and incineration of solid wastes. In pure form is a blueish grey metal with many industrial uses, particularly galvanising of steel and iron to prevent corrosion, and production of brass and bronze alloys. May also be present in compounds used to produce pigments, ceramics, rubber, automotive compounds, in wood preservation, textile manufacturing and personal care products.

Data compiled from Makepeace et al. (1995), Tchounwou et al. (2012) and Balali-Mood et al. (2021).

4.1 HEAVY METALS IN WASTEWATER

Municipal wastewaters are known to be a major source of heavy metal pollution of waters and soils (Üstün et al. 2009). The specific metals and their concentrations in wastewater are highly variable depending on the relative contribution of domestic, commercial, industrial and stormwater contributions to the overall wastewater stream (Üstün et al. 2009; Cashinto et al. 2015). Sources of metals in domestic (ie, household) wastewater may include the water supply itself, faeces (reflecting dietary intake and pharmaceutical use), detergents and washing powders, personal care products (toothpastes, shampoos, dental amalgam), household dusts, leaching or corrosion of plumbing systems, cleaning of metal cookware, pigments in paints and cosmetics, pesticides, and various other household chemicals (Moriyama et al. 1989; Sörme and Lagerkvist 2002; Cashinto et al. 2015; Drozdova et al. 2018). Industrial effluents are a key source of heavy metals where they are discharged to municipal wastewater networks as trade wastes (reviewed by Eaton 2022), and stormwaters

may contribute to heavy metal loads in wastewater through inflow or infiltration (Cashinto et al. 2015; Sakson et al. 2018).

An array of different heavy metals has been detected in municipal wastewaters, with published data indicating that the metal composition of wastewater can be complex and variable both between catchments and within a catchment through time (Üstün et al. 2009). Metals may be present in suspended, dissolved or complexed forms, and phase distribution may differ between metal species and with the characteristics of the wastewater (Karvelas et al. 2003; Buzier et al. 2006; Cashinto et al. 2015); Carletti et al. (2008) reported that heavy metals in municipal wastewater tended to be particle-associated, while those in commercial and industrial effluents were more likely to be dissolved. Metals of primary concern in wastewater include arsenic, cadmium, chromium, copper, iron, mercury, nickel, lead and zinc, and much of the literature has focused on various subsets of these (Table 6; Chipasa et al. 2001; Karvelas et al. 2003; Buzier et al. 2006; Carletti et al. 20008; Teijon et al. 2010; Chashinto et al. 2015). Other elements that have been detected in sewage and WWTP influent include aluminium, antimony, barium, boron, cobalt, iron, lithium, manganese, molybdenum, rubidium, selenium, silver, thallium, tin, titanium, uranium and vanadium (Choubert et al. 2011; Joshua et al. 2017). Several studies have reported the ubiquitous nature of many of these metals, particularly iron and zinc, with many metal species being detected in 80-100% of samples in which they are analysed (Karvelas et al. 2003; Carletti et al. 2008; Choubert et al. 2011), although some such as cadmium, chromium and mercury are detected less frequently (Chipasa 2001; Teijon et al. 2011). Similarly, while concentrations of individual metal species may be highly variable, ranging from several µg/L to several mg/L, cadmium, chromium and mercury tend to have the lowest concentrations, and iron, zinc and aluminium the highest (Table 6). However, the nature of land use in the catchment can have a significant impact on the metals present in wastewater (Rule et al. 2006a); for example, Üstün et al. (2009) reported that in several Turkish WWTPs, chromium was among the most prevalent heavy metals, attributable to textile, leather and metal industries within the catchments.

Wastewater treatment plants are not designed to remove metal contaminants, although some metals will be removed through their association with particulate matter and partitioning to the solid phase of treatment systems, so that sewage sludge becomes a temporary sink (Carletti et al. 2008; Cashinto et al. 2015; Du et al. 2020). Overall, approximately 80-90% of heavy metals may accumulate in sludge (Agoro et al. 2020), although this may vary widely between technologies used in different countries and contexts, and some metal species have much lower removal efficiencies (eg arsenic and cobalt may be less than 20%; Choubert et al. 2011; Du et al. 2020).

Table 6: Concentrations of selected heavy metals (µg/L) in untreated municipal wastewater reported in international studies.

Country	Al	As	Cd	Cr	Cu	Fe	Hg	Mn	Ni	Pb	Zn	Reference
France			0.6	9	65	650			12	18		Buzier et al. (2006)^
Italy	786±46%	4*	<LOD	8.1±11%	9.9±56%	515±29%	1.5±58%			8*	348±31%	Carletti et al. (2008)#
Italy	1,940±8%	<LOD	8.7±13%	56.4±24%	9.8±44%	361±10%	0.7±8%			8.6*	1,233±20%	Carletti et al. (2008) #
Poland			20		100					50	450	Chipasa (2003)
France	1,300 Max. 3,400	1.0 Max. 1.5	0.2 Max. 0.4	15 Max. 70	75 Max. 110	600 Max. 1,500			20 Max. 90	8 Max. 15	200 Max. 400	Choubert et al. (2011)
China		6.2±4.8 Max. 95	15±9 Max. 78	170±64 Max. 650			0.67±1.5 Max. 38			160±100 Max. 940		Du et al. (2020)
UK		12	1.7	35	300		2.5		40	600	290	Goldstone et al. (1990a, 1990b, 1990c)
India		1.12±0.66 Max. 2.22		3.5±1.3 Max. 5.5	14.2±13.9 Max. 38	235±56 Max. 298			4.1±1.8 Max. 6.5	0.18±0.09 Max. 0.31	19.8±4.3 Max. 26.9	Joshua et al. (2017)
Greece			3.3±1.1	40±12	79±35	480±87		67±12	770±200	39±9.4	470±140	Karvelas et al. (2003)
Spain			5 Max. 5				0.37 Max. 0.37		47.5 Max. 107.6	2.5 Max. 17.6		Teijon et al. (2010)
Turkey	1,891±872 Max. 3,753		19±40 137	1,086±509 2,120	60±43 Max. 179	1,975±712 3,580		126±33 Max. 217	100±41 Max. 202	84±100 Max. 385	533±209 Max. 982	Üstün et al. (2009)

Data are shown as mean values, with standard deviation and maximum concentration shown where that data was available. LOD – limit of detection.
*only one sample above LOD; ^ combined sewer network; # data shown as mean ± variation coefficient; ~WWTPs receiving municipal or municipal and hospital effluent.

4.2 HEAVY METALS IN STORMWATER

Heavy metals are ubiquitous in urban stormwater worldwide; they are among the most commonly-reported stormwater pollutants and are consistently identified as priority pollutants (Birch et al. 2004; Zgheib et al. 2012; McDonald et al. 2022). In particular, vehicular dust and debris deposited onto roads and carparks (eg, from wear of brake pads and tyres, vehicular exhaust) is a significant source of heavy metals in stormwaters, as is the leaching and/or corrosion of metals used in building materials including galvanised steel and iron roofing, architectural metal claddings or treated timbers (Makepeace et al. 1995; Brown and Peake 2006; Göbel et al. 2007; Ma et al. 2016; Müller et al. 2020; McDonald et al. 2022). Other potential sources of heavy metals in stormwater include soil erosion, household chemicals and pesticides, and atmospheric deposition following combustion of fossil fuels and municipal solid wastes (Makepeace et al. 1995; Göbel et al. 2007; Müller et al. 2020). Industrial runoff may be a significant source where the appropriate controls are not in place to contain and treat these (Brown and Peake 2006; McDonald et al. 2022), while contamination by municipal wastewater – itself a source of heavy metals – is likely to be of lesser importance.

Amongst the diversity of heavy metals identified in urban stormwaters, copper, zinc and lead are often detected in 100% of samples collected, irrespective of the nature of the catchment from which samples have been collected (eg Göbel et al. 2007; Gasperi et al. 2012; Zgheib et al. 2012; Ma et al. 2016; Table 7). Other metals including chromium, iron, nickel, cobalt, arsenic, silver, cadmium, aluminium, manganese, mercury, vanadium, platinum and barium have also been detected in urban stormwaters at lower concentrations and/or frequency (Makepeace et al. 1995; Bertrand-Krajewski et al. 2011; Masoner et al. 2019; Ma et al. 2016).

The concentrations of heavy metals in stormwater runoff can vary by several orders of magnitude (Ingvertsen et al. 2011). Copper, zinc and lead tend to exhibit the highest concentrations; for example, in summarising the findings of three large datasets, Ingvertsen et al. (2007) noted median concentrations ranging from 15 to 2,600 µg/L, 103 to 6,000 µg/L and 10 to 344 µg/L, respectively. In contrast, median concentrations of cadmium, chromium and nickel were reported to range from 0.7 to 4.2 µg/L, 4 to 15 µg/L, and 4 to 45 µg/L, respectively. Concentrations may vary depending on the land use within a catchment, the surface onto which rainwater falls, and the nature and extent of contamination in the catchment area (Ingvertsen et al. 2011; Huber et al. 2016; Müller et al. 2020). Concentrations of heavy metals in stormwater are often higher in catchments with industrial and commercial activity compared with residential or green space (Rule et al. 2006a; Ma et

al. 2016) and where there are high densities of buildings with metal roofing materials (Brown and Peake 2006; Göbel et al. 2007; Sakson et al. 2018), as well as in runoff from roadways with high traffic volumes (Göbel et al. 2007; Ma et al. 2016; Sakson et al. 2018).

Temporal variation of heavy metal concentrations is also reported, likely reflecting the influence of both climatic factors on contaminant accumulation and release (eg dry deposition rates, antecedence dry period, season, rainfall volume and intensity) and sampling strategy (eg, flow proportional, time proportional, first flush or mixed), with a first flush phenomena usually observed (Rule et al., 2006a; Huber et al. 2016; Sakson et al. 2018). Some heavy metals may also show historical trends based on their reduced use in certain applications and subsequent release to the environment. The most notable example of this is the significant reduction of lead in urban stormwater in recent decades following cessation of its use as an anti-knock additive in petrol (Huber et al. 2016).

In a human health risk assessment based on metal concentrations in road sweepings in Australia, Ma et al. (2016) determined that although several metals would likely exceed guidelines for recreational and/or potable water use, they were not considered to present a significant health risk. However, they noted that the combined presence of multiple heavy metals – which is more typical of stormwater samples – could be detrimental to human health.

Table 7: Concentrations of selected heavy metals (µg/L) in urban stormwater reported in international studies.

Country	Al	As	Cd	Cr	Cu	Fe	Hg	Mn	Ni	Pb	Zn	Reference
US	28±19.5 Max. 111 100%	0.84±0.5 Max. 3.2 100%	0.041±0.1 Max. 0.37 98%	1.1±1.1 Max. 4.7 90%	8.4±10.7 Max. 68 100%	70.6+69.7 Max. 325 96%	0.039±0.0 Max. 0.180 100%	38.0±57.9 Max. 205 100	1.4±2.1 Max. 15 98%	0.53±0.5 Max. 2.1 100%	27±33.0 Max. 189 92%	Masoner et al. (2019)
France				4.5* Max. 45 31%	55* Max. 220 100%					27* Max. 129 92%	270* Max. 520 100%	Zgheib et al. (2012)
UK			0.2±0.20 Max. 0.67 100%	3.1±2.54 Max. 9.43 92%	34.7±56.6 Max. 206 100%		0.5±0.19 Max. 0.82 42%		3.9±2.58 Max. 8.06 100%	10.4±23.7 Max. 85.2 100%	82.5±125.5 Max. 445 100%	Rule et al. (2006)
France				Max. 20	Max. 134					Max. 175	Max. 1,137	Gasperi et al. (2012)[#]
Poland			Max. 0.6		Max. 95					Max. 126	Max. 1,060	Sakson et al. (2018)
New Zealand					37* Max. 165					11* Max 212	29* Max 659	Trowsdale and Simcock (2011)
New Zealand		2.4* Max. 3.2	0.29* Max. 0.47	2.9* Max. 5.8	30* Max. 823	3.5* Max. 3.7		189* Max. 219	2.3* Max. 3.2	21* Max. 4,100]	895* Max. 18,549	Brown (2002)
Australia				3±1	27±9	1,960±870		122±8	5±4	38±17	32±11	Birch et al. (2004)
Multiple	Max. 16,000	Max. 210	Max. 13,730	Max. 2,300	Max. 1,410	Max. 440,000	Max. 0.67	Max. 3,800	Max. 49,000	Max. 26,000	Max. 22,000	Makepeace et al. (1995)
Multiple			Max. 13.0	Max. 50	Max. 3,416				Max. 70	Max. 525	Max. 4,880	Goebel et al. (2007)

Data are shown as mean values, with standard deviation, maximum concentration and detection frequency shown where that data was available.

* Denotes median value. [#] samples were collected from a combined stormwater/wastewater network. LOD – limit of detection.

4.3 HEALTH EFFECTS OF HEAVY METALS

Heavy metal toxicity and carcinogenicity involves numerous mechanistic aspects and cellular targets, some of which are not clearly understood, although each metal has unique physicochemical properties that contribute to its specific toxicology (see reviews by Tchounwou et al 2012; Jaishankar et al 2014; Balali-Mood et al 2021). Heavy metals can adversely affect multiple cellular components, including cell membranes, mitochondria, DNA and enzymes, resulting in damage to the central nervous system, cardiovascular system, lungs, liver, kidneys and blood, with long-term exposures associated with endocrine and reproductive system dysfunction, degenerative muscular and neurological conditions, and increased cancer risk.

4.3.1 Arsenic

Arsenic is one of the WHO's ten chemicals of major public health concern.[55] It exists in two main forms: inorganic and organic. Inorganic arsenic is highly toxic, while organic forms are considerably less harmful (Tchounwou et al. 2012; WHO 2019a). Acute exposure to inorganic arsenic can result in vomiting, abdominal cramps, diarrhoea, tingling and numbness in extremities and muscle cramps, with high doses associated with persistent gastrointestinal complications, disturbance of cardiovascular and nervous system function, bone marrow suppression, hepatomegaly and melanosis (IPCS 2001b; ATSDR 2007; WHO 2019a). Effects of chronic arsenic poisoning can include skin lesions and hyperkeratosis, cardiovascular disease (including hypertension, peripheral vascular disease, coronary heart disease and stroke), damage to the kidneys, liver, nerves and blood, gastrointestinal disease, diabetes, spontaneous abortion and pre-term birth, and impaired cognitive development in children (IPCS 2001b; ATSDR 2007; EFSA 2009; WHO 2019a). Dermal contact may cause skin irritation (ATSDR 2007).

Inorganic arsenic is also a potent carcinogen, and chronic oral exposures have been causally associated with cancers of the skin, bladder and lungs, with limited evidence to suggest it may also cause cancer of the kidneys, liver and prostrate (IPCS 2001b; IARC 2012; WHO 2019a). The IARC have classified arsenic and inorganic arsenic compounds as Group 1 carcinogens (carcinogenic to humans) (IARC 2012).

There remains considerable uncertainty around the dose response to arsenic at low intakes (WHO 2019a; Tsuji et al. 2021). Adverse health outcomes have been reported in association

[55] https://www.who.int/news-room/fact-sheets/detail/arsenic. Accessed 16 March 2022

with drinking-water concentrations exceeding 50-100 µg/L (WHO 2019a). In 2010, the Joint Food and Agriculture Organisation of the United Nations (FAO)/WHO Expert Committee of Food Additives (JEFCA) withdrew their provisional tolerable weekly intake (PTWI) for arsenic, and determined no new PTWI could be determined (WHO 2019a).

4.3.2 Cadmium

Cadmium is highly toxic, even at low levels of exposure, and is one of the WHO's ten chemicals of public health concern[56]. An estimated 1-10% of ingested cadmium will be absorbed through the digestive tract, although this may be higher in individuals with certain nutrient deficiencies (IPCS 1992; ATSDR 2012a). The effects of acute cadmium exposure include rapid onset and severe nausea, vomiting, abdominal pain, vertigo and convulsions; severe cases may also experience gastrointestinal, pulmonary, hepatic and/or renal injury, although most will make a full recovery (IPCS 1992; Tchounwou et al. 2012). The kidney is the critical target organ following chronic exposure to cadmium, where it accumulates with a half-life of 10-35 years, leading to renal tubular dysfunction and nephropathy (IPCS 1992; ATSDR 2012a; WHO 2019b). Some cadmium may also be stored in the liver (ATSDR 2012a). High cadmium intake can also lead to disturbances in calcium metabolism, resulting in the formation of kidney stones and bone conditions including osteomalacia and osteoporosis (ATSDR 2012a; WHO 2019b). Inhalation has been associated with acute pneumotisis, pulmonary oedema and development of chronic obstructive pulmonary disease (IPCS 1992; WHO 2019b). Animals studies also suggest reproductive and developmental toxicity, hepatic effects, and immunological effects, but more data is required (ATSDR 2012a).

The IARC determined cadmium and cadmium compounds can cause cancer of the lung following inhalation exposure, and that there is limited evidence that cadmium may cause cancers of the kidney and prostrate; cadmium has therefore been classified as a Group 1 carcinogen (carcinogenic to humans) (IARC 2012; WHO 2019b). However, much of the data describes occupationally-exposed cohorts, presumably with inhalation exposure, and the carcinogenicity of cadmium from low levels of environmental exposure remains unclear.

[56] https://www.who.int/news-room/photo-story/photo-story-detail/10-chemicals-of-public-health-concern

4.3.3 Chromium

The toxicity and health effects of chromium differ significantly depending on the valence state, of which chromium III and chromium VI predominate, and the route of exposure (WHO 2020a). Although the available data relate mainly to total chromium, Cr (III) is poorly absorbed and therefore relatively non-toxic, while chromium VI readily penetrates cell membranes to interact with intracellular components, and is therefore highly toxic (IPCS 1988; ATSDR 2012b; WHO 2020a). Gastrointestinal absorption of Cr (III) and Cr (VI) are estimated at <1% and 7% respectively (IPCS 1988; IARC 2012; WHO 2020a). Chromium that is absorbed into the body can be found in nearly all body tissues, but concentrates in the kidney, liver and bone (ATSDR 2012b; IARC 2012).

Symptoms of chromium exposure tend to be associated with site of uptake/point of contact (eg respiratory symptoms for inhalation, gastrointestinal for oral, skin irritation for dermal), although dermal, oral or inhalation exposure may all cause allergic sensitisation in some individuals (WHO 2020a). Acute, oral exposures can cause severe gastrointestinal illness, respiratory and liver injury, acute nephritis and cardiovascular collapse (IPCS 1988; WHO 2020a). In human volunteers, a single dose of up to 4-5 mg Cr (III) or Cr (VI) did not yield adverse effects (WHO 2020a). No information on adverse health outcomes of chronic Cr(III) ingestion are reported, while chronic ingestion of Cr (VI) may cause adverse effects on the liver, kidney, gastrointestinal tract and reproductive and immune systems and possibly the blood, and may also exacerbate dermatitis in sensitised individuals[57] (ATSDR 2012b; WHO 2020a). Dermal exposure to chromium compounds may cause skin irritation, ulcers and eczema (IPCS 1988), with inhalation associated with irritation and ulceration of the respiratory tract, rhinitis, asthma, and in severe cases, liver and kidney necrosis and poisoning of the blood-forming organs (IPCS 1988).

The IARC have classified Cr (VI) as carcinogenic to humans, causing lung cancer in humans following inhalation (Group 1). Oral exposure to chromium IV in animal studies is associated with cancer of the oral cavity and gastrointestinal tract, but human data is lacking (IARC 2012). Cr (III) has not been shown to be carcinogenic in oral toxicity studies (IARC 2012).

4.3.4 Lead

Lead is one of the WHO's top ten chemicals of public health concern.[58] The toxicity of lead has been known for over 2000 years (ATSDR 2020). Absorbed lead accumulates primarily in

[57] https://www.epa.gov/sites/default/files/2016-09/documents/chromium-compounds.pdf
[58] https://www.who.int/news-room/photo-story/photo-story-detail/10-chemicals-of-public-health-concern

the bone, as well as the kidney, liver and brain. Lead exposure has adverse effects on multiple body systems, however the developing nervous system is the most vulnerable system to the effects of lead poisoning (Tchounwou et al. 2012; WHO 2019d, 2022b).

Lead is classically considered a chronic or cumulative toxin, with acute effects usually only observed following exposure to very high doses, including nausea, vomiting, abdominal pain, renal damage, hypertension and neurological effects (malaise, drowsiness, irritability, headaches, encephalopathy) (ATSDR 2020; WHO 2019d). Chronic exposure to lead can cause reduced neurological and cognitive function (including impaired learning and memory, attention deficit, headaches, visual and hearing loss, tremor, ataxia), psychiatric symptoms (irritability, anxiety, confusion), kidney dysfunction (proteinuria, reduced anion and glucose transport, tubular necrosis), cardiovascular effects (increased risks of hypertension, heart disease and stroke), haematological disturbance (reduced haemoglobin), immune toxicity (leading to inflammation and autoimmunity), gastrointestinal disorder and abdominal colic, peripheral neuropathy and reproductive toxicity (reduced sperm count and viability, miscarriage and stillbirth) (IPCS 1977; Tchounwou et al. 2012; ATSDR 2020; WHO 2019d, 2022b). Children and infants are especially vulnerable to adverse effects of lead exposure, and exposure during pregnancy is of significant concern (Tchounwou et al. 2012; WHO 2019d). The most critical effect of lead exposure in young children is on the developing nervous system, with even low levels of exposure causing decrements in IQ and behaviour in the absence of obvious symptoms, and increasing exposure resulting in impaired development and intellectual disability (WHO 2022b). No exposure threshold for the adverse effects of lead has been determined and no tolerable exposure limits can be derived. The New Zealand Ministry of Health policy is that there is no safe level of lead for humans (Ministry of Health 2021b).

There is limited evidence that chronic occupational exposure to lead may contribute to the development of cancer, although results are inconsistent (ATSDR 2020); the IARC has classified inorganic lead compounds as Group 2A carcinogens (probably carcinogenic to humans) (IARC 2006). Organic lead compounds are not classifiable due to inadequate evidence of carcinogenicity (IARC 2006).

4.3.5 Mercury

Mercury is one of the WHO's top ten chemicals of major public health concern. It is found in three main forms – elemental, organic and inorganic – each with its own toxicity profile (Tchounwou et al. 2012; WHO 2021c; ATSDR 2022). Once absorbed into the body, mercury

has a very low excretion rate, accumulating primarily in the kidneys, liver and neurological tissues (Tchounwou et al. 2012).

Organic mercury is most commonly encountered as methylmercury, formed when mercury in water and soils is methylated by environmental bacteria (ATSDR 2022). It is highly bioaccumulative in aquatic biota and readily absorbed across the gastrointestinal tract when these are consumed (estimated up to 95%; Ministry of Health 2021a). Methylmercury is a powerful neurotoxin, causing parasthesia, headaches, dysarthria, vision and hearing impairment, muscle weakness, tremors, and loss of coordination (Ministry of Health 2021a; ATSDR 2022). There is also evidence that oral exposure may cause renal, cardiovascular, reproductive, and developmental effects (ATSDR 2022). Although dermal absorption of organic mercury is not well characterised, it may be significant (Ministry of Health 2021a).

Inorganic salts of mercury is corrosive to the skin, eyes and gastrointestinal tract (WHO 2021c). Approximately 10-30% of the ingested dose is absorbed through the gastrointestinal tract, causing profuse vomiting and diarrhoea, neurological disturbance (irritability, memory loss), muscle weakness, tremor, dermatitis, hypertension, and hypovolemic shock (Ministry of Health 2021a; WHO 2021c; ATSDR 2022). In particular, the kidney is the critical organ following ingestion of inorganic mercury (IPCS 1991b), and chronic exposure through drinking-water has been associated with kidney damage and renal failure.[59]

Exposure to elemental mercury is primarily through inhalation of vapours, resulting in harmful effects on the respiratory, neurological, immune and renal systems (Ministry of Health 2021a; WHO 2021c; ATSDR 2022). It is relatively non-toxic through oral and dermal exposure, with almost no absorption through the gastrointestinal tract or skin (Ministry of Health 2021a; WHO 2021c).

Children are especially vulnerable to the effects of mercury, especially methylmercury, which can readily cross the placenta and cause neurodevelopmental problems in the developing foetus, including intellectual disability, seizures, vision and hearing loss, delayed development and language disorders (IPCS 1990; Ministry of Health 2021a; WHO 2021c). There is no conclusive evidence linking mercury exposure to cancer in humans (WHO 2021c), however data from animal studies have led the IARC to classify methylmercury compounds as Group 2B (possibly carcinogenic to humans). Metallic and inorganic mercury compounds are not classifiable as to their carcinogenicity (IARC 1993).

[59] https://www.epa.gov/mercury/health-effects-exposures-mercury Accessed 22 November 2021.

4.3.6 Nickel

The most common harmful heath effect associated with nickel exposure is allergic reaction; nickel and its salts are potent skin irritants, and dermal exposure can result in contact dermatitis and vesicular eczema (IPCS 1991a; ATSDR 2005a). An estimated 10-20% of the population is sensitive to nickel (ATSDR 2005a). Nickel is thought to be poorly absorbed through the gastrointestinal tract, with nickel that is absorbed concentrating within the kidneys (ATSDR 2005a). Adverse health effects of nickel ingestion include vomiting, nausea, diarrhoea, headaches, giddiness, reduced red blood cell counts and transient nephrotoxicity, with cardiac arrest in severe cases (IPCS 1991a; ATSDR 2005a). Ingestion may also exacerbate skin irritation and dermatitis in sensitised people (IPCS 1991a; ATSDR 2005a; WHO 2022b); individuals who are not hypersensitive must consume large amounts to experience adverse effects (ATSDR 2005a). Data on chronic oral toxicity of nickel is based largely on animal studies, which suggest adverse effects on the lungs, stomach, liver, kidneys and immune and reproductive systems (ATSDR 2005a; WHO 2022b). The most serious harmful health effects (reduced lung function and respiratory cancers) associated with nickel occur in people with inhalation exposure in occupational settings (ATSDR 2005a).

The IARC have classified nickel compounds as Group 1 carcinogens (carcinogenic to humans) based on sufficient evidence that that they can cause cancer of the lung, nasal cavity and sinuses, and metallic nickel as a Group 2B carcinogen (possibly carcinogenic to humans) (IARC 2012; WHO 2022b). However, human studies have focused on inhalation exposure in occupational settings, and data on both the oral carcinogenicity of nickel almost completely lacking (WHO 2022b).

4.3.7 Zinc

Compared with other heavy metals, zinc is relatively harmless and only exposure to very high doses have toxic effects, making zinc intoxication a rare event (Plum et al. 2010); deficiency is thought to be as important a health issue as consuming too much (ATSDR 2005b). Recommended dietary allowances are 8-11 mg/day for women and men, respectively, and acute ingestion at 10-15 times these levels can cause nausea, vomiting and diarrhoea (ATSDR 2005b). Chronic ingestion (15-50 mg/day) may interfere with copper absorption and homeostasis, causing deficiency and anaemia, with reversible leukocyte dysfunction and reduced high density lipoprotein (HDL) cholesterol observed following pharmacological intake of zinc (IPCS 2001a; ATSDR 2005b).

Zinc is not generally associated with dermal effects, although zinc chloride can cause severe skin irritation (ATSDR 2005b). The IARC has determined that zinc is not classifiable as to its carcinogenicity.

4.4 REGULATIONS AND GUIDELINES

Pertinent regulations and guidelines regarding exposure to priority heavy metals are summarised in Table 8.

Table 8: Recommended oral exposure limits for selected heavy metals.

	Arsenic	Cadmium	Chromium III	Chromium VI	Lead	Mercury	Nickel	Zinc
Provisional Tolerable Weekly Intake, JECFA[60] (µg/kg bw/day)	[withdrawn]	25* (monthly intake)			[withdrawn]	1.6 (methylmercury) 4 (inorganic mercury)		300-1,000 (daily intake)
Reference Dose, US EPA[61] (µg/kg bw/day)	0.3 (inorganic arsenic)	0.5 (in water) 1.0 (in food)	1,500	3		0.3 (inorganic mercury)	20	300
Minimum Risk Level, ATSDR[62] (µg/kg bw/day)	5.0 (acute) 0.3 (chronic)	0.5 0.1		5.0 (int.) 0.1 (chronic)		inorganic salts 2 (acute) 0.01 (int.)		300 (int.) 300 (chronic)
Drinking-water Guideline, WHO[63] (µg/L)	10*	3	50* (total chromium)		10*	6 inorganic	70	
Drinking-water Maximum Allowable Value, DWSNZ[64] (µg/L)	10	4	50* (total chromium)		10	7 inorganic	80	1,500^
Drinking-water Maximum Contaminant Limit, US EPA[65] (µg/L)	10	5	100 (total chromium)		15	2 inorganic		

US EPA Reference Doses and ATSDR Minimum Risk Levels are for oral exposures.
***** Guidelines value is designated as provisional (often on the basis of achievability by available treatment methods, analytical methods and toxicology).
^ Guideline value is based on taste/aesthetic considerations rather than health risk. int. – intermediate term exposure
A blank space indicates that a limit has not been established.

[60] https://apps.who.int/food-additives-contaminants-jecfa-database/ Accessed 12 April 2022
[61] https://iris.epa.gov/ChemicalLanding/&substance_nmbr=278. Accessed 12 April 2022
[62] https://wwwn.cdc.gov/TSP/MRLS/mrlslisting.aspx. Accessed 12 April 2022
[63] WHO (2022). *Guidelines for Drinking-water Quality: Fourth Edition Incorporating the First and Second Addenda*.
[64] https://www.legislation.govt.nz/regulation/public/2022/0168/latest/whole.html Accessed 5 December 2022
[65] https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations Accessed 12 April 2022

5. PER- AND POLYFLUOROALKYL SUBSTANCES

Per- and polyfluoroalkyl substances are a broad family of synthetic, highly fluorinated, aliphatic compounds. The strength of the carbon-fluorine bond confers a high thermal and chemical stability, and the combination of different hydrophobic, lipophobic and hydrophilic moieties give PFAS unique surface-active properties (Buck et al. 2011; Lenka et al. 2021). As such, these compounds are extremely resistant to degradation, are water- and oil-repellent and friction-resistant. These properties have resulted in PFAS being manufactured for a vast range of industrial and consumer uses, including water- and stain-resistant fabrics and carpets, protective coatings on paper and packaging, cosmetics and personal care products, surfactants, non-stick cooking utensils, paints and varnishes, fire-fighting foams, and in electroplating, aviation and oil-production industries (Wang et al. 2017; Phong Vo et al. 2020; Lenka et al. 2021). Their extensive use, solubility in water and high persistence have led to their ubiquitous presence in the environment, with PFAS having been detected in environmental (lakes, rivers, groundwater, coastal environments, soils, air) and biological (human serum and breast milk, plants, wildlife) samples around the world (Ateia et al. 2019; Coggan et al. 2019; Barisci and Suri 2021; Brase et al. 2021; Cookson and Detwiler 2022). PFAS compounds bioaccumulate and biomagnify within the food chain (Rumsby and Manning 2018).

There are many different classes of PFAS compounds, comprising more than 4,000 known substances (Buck et al. 2011; Cookson and Detwiler 2022). However, most studies have focused on only a small number of perfluoroalkyl acids (PFAAs), especially perfluorocarboxylic acids (PFCAs) and perfluorosulfonic acids (PFSAs), and specifically the compounds perfluorooctanoic acid (PFOA) perfluorooctane sulfonate (PFOS). Concerns regarding the potential environmental and health impacts of PFAS arose in the early 2000s, resulting in regulation of the production and use of traditional 'long-chain' PFAS[66] (Buck et al. 2011). For example, PFOA and PFOS (and their salts and related compounds) have been registered under Annexes A and B, respectively, of the Stockholm Convention on Persistent Organic Pollutants. In addition, perfluorohexane sulfonic acid (PFHxS) and its salts and

[66] "Long-chain" PFAS are typically defined as PFSAs with ≥6 carbon atoms in their fluoroalkyl chain, and PFCAs with ≥7 carbon atoms (Buck et al. 2011).

related compounds are currently under consideration for addition to Annex A of the Convention.[67]

Despite significant reductions in the usage of many long-chain PFAS in developed countries, the long half-lives of these compounds (eg >41 years for PFOS, >92 years for PFOA) means they continue to be reported in environmental samples (Lenka et al. 2021; Barisci and Suri 2021; Brase et al. 2021). In addition, increasing regulation has resulted in a shift towards the production of 'short-chain and ultra-short chain' PFAS [68] (eg, perfluorobutane sulfonic acid (PFBS), perfluorobutanoic acid (PFBA)) and other fluorinated alternatives (eg, hexafluoropropylene oxide dimer acid ('GenX'), 6:2 polyfluoroalkyl ether sulfonate ('F-53B')) to replace restricted compounds (Wang et al. 2017; Ateia et al. 2019; Jeong et al. 2022). There is little information available regarding the environmental fate and health risks associated with these emerging PFAS; however, available data shows that these compounds are also widely detected in the environment, and may exhibit similar persistence, toxicity and potential to bioaccumulate as their long-chain counterparts (Wang et al. 2017; Brendel et al. 2018; Ateia et al. 2019; Brase et al. 2021; Lenka et al. 2021; Jeong et al. 2022).

In addition to direct sources of PFAS production and discharge, some polyfluoroalkyl substances have the potential to act as precursors and be transformed (biotically or abiotically) into more stable perfluoroalkyl end products, including restricted compounds (Buck et al. 2011; Phong Vo et al. 2020). For example, some fluorotelomers degrade into PFCAs such as PFOA and perfluorohexanoic acid (PFHxA), and perfluorooctane sulfonamides can degrade to PFSAs such as PFOS (Buck et al. 2011; Barisci and Suri 2021; Cookson and Detweiler 2022). As early as 2007, the Organisation for Economic Co-operation and Development (OECD) compiled a list of 615 PFAS and related compounds that had the potential to break down into PFCAs (IOMC 2007). Thus, when assessing and managing PFAS, these precursor compounds must also be considered as relevant sources and managed accordingly (Buck et al. 2011).

67 http://www.pops.int/TheConvention/ThePOPs/ChemicalsProposedforListing/tabid/2510/Default.aspx Accessed 29 June 2022

68 "Short chain" PFAS are typically defined as PFSAs with ≤5 fully fluorinated carbon atoms, and PFCAs with ≤6 carbon atoms (Buck et al. 2011). Ultra-short chain may be used by some authors to describe PFAS having 1-2 fully perfluorinated carbon atoms (eg Ateia et al. 2019).

5.1 PER- AND POLYFLUOROALKYL SUBSTANCES IN WASTEWATER

Per and poly-fluoroalkyl substances are ubiquitous in municipal wastewaters, especially where the network receives trade wastes and/or landfill leachates (Phong Vo et al. 2020; Barisci and Suri 2021; Lenka et al. 2021; Thompson et al. 2022). However, PFAS are also detected in wastewaters where there are no industrial inputs, including in septic tanks (Thompson et al. 2022). Suspected sources of PFAS in domestic wastewaters include the degradation of polyfluorinated microfibres in waterproof clothing during laundry, human excretion following exposure (eg via dietary intake), household dusts, and the use of PFAS-containing consumer products such as personal care products, non-stick cookware and cleaning products (Phong Vo et al. 2020; Lenka et al. 2022; Thompson et al. 2022). In some instances, a portion of the PFAS in wastewater can be attributed to PFAS in the community's tap water (Thompson et al. 2022). Total PFAS concentrations in wastewater may vary widely, ranging from tens of ng/L to more than 3,000 ng/L (Jeong et al. 2022; Nguyen et al. 2022). Concentrations of PFAS tend to be significantly lower in domestic wastewaters than industrial effluents or municipal networks receiving trade wastes (Xiao et al. 2012a; Gallen et al. 2018; Coggan et al. 2019; Nguyen et al. 2022). For example, Yu et al. (2009) reported PFOA and PFOS concentrations were up to 22 and 15 times greater, respectively, at a Singaporean WWTP receiving 60% industrial and 40% domestic wastewaters, compared with another plant receiving 95% domestic wastewater.

A range of conventional, short-chain and alternative PFAS are detected in WWTP influents at concentrations up to several hundred ng/L for individual compounds (Bossi et al. 2009; Murikami et al. 2009; Pan et al. 2016; Gallen et al. 2018; Coggan et al. 2019; Lorenzo et al. 2019; Jeong et al. 2022; Lenka et al. 2022; Nguyen et al. 2022; Table 9). The profiles of PFAS detected may differ significantly between treatment plants (Jeong et al. 2022; Nguyen et al. 2022). Commonly detected compounds include PFOS, PFOA, PFBS, PFBA, PFHxA, PFHxS, perfluoropentanoic acid (PFPeA), perfluorodecanoic acid (PFDA), perfluorononanoic acid (PFNA), 8:2 fluorotelomer sulfonic acid (8:2 FTS) and 6:2 fluorotelomer sulfonic acid (6:2 FTS). Despite the phase-out of long-chain PFAS, PFOA and PFOS are often reported as being the dominant compounds (eg, Sinclair and Kannan 2006; Bossi et al. 2009; Xiao et al. 2012a; Murikami et al. 2009; Gallen et al. 2018; Lorenzo et al. 2019), although several authors have also reported a shift towards short-chain alternatives including PFBA, PFBS, PFHxS, PFHxA (Zhang et al. 2013; Cookson and Detwiler 2022; Jeong et al. 2022; Lenka et al. 2022).

Per and poly-fluoroalkyl substances may present a unique challenge to water managers, as not only are the compounds present in influents poorly removed by conventional treatment

processes (Pan et al. 2016; Phong Vo et al. 2020; Barisci and Suri 2021), the transformation of precursor compounds may yield additional persistent PFAS, including PFOA. As a result, WWTP effluents often contain elevated concentrations of certain PFAS compounds (Bossi et al. 2008; Murakami et al. 2009; Xiao et al. 2012a; Zhang et al. 2013; Coggan et al. 2019; Thompson et al. 2022); for example, Gallen et al. (2018) reported that in a study of 14 Australian WWTPs, total PFAS in the effluents from 8 plants were, on average, 10 times higher than for corresponding influents.

Table 9. Concentrations of selected PFAS (ng/L) in untreated municipal wastewater reported in international studies.

Country	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFDA	PFNA	PFBS	PFHxS	PFOS	Reference
Spain	4.8# Max. 20.9 31%	2.1# Max. 27.0 8%	1.8# Max. 23.3 8%	1.9# Max. 7.5 31%	3.4# Max. 5.9 100%	nd	nd	nd	6.9# Max. 51.8 38%	11.1# Max. 63.1 54%	Lorenzo et al. (2019)
USA					135# Max. 202	34# Max. 47	6# Max. 11			6# Max. 10	Sinclair and Kannan (2006)
Japan				4.7*	22.5 Max. 41	<1 Max. 1.7	29 Max. 70			20.5 Max. 336	Murakami et al. (2009)
Belgium	18.5 Max. 108 100%	27.5 Max. 295 100%	7.0 Max. 185 94%	6.2 Max. 155 94%	7.25 Max. 2,726 100%	0.90 Max. 8.3 94%	0.58 Max. 8.6 81%	7.15 Max. 23 100%	3.35 Max. 7.6 94%	6.3 Max. 101 100%	Jeong et al. (2022)
Australia	5.35 Max. 52.1 100%	<LOQ Max. 47.3 86%	8.65 Max. 33.5 100%	2.80 Max. 10.4 100%	4.35 Max. 40.5 100%		<LOQ Max. 2.60 91%	2.55 Max. 33.3 100%	2.30 Max. 142 91%	6.96 Max. 129 97%	Coggan et al. (2019)
Australia			9.5±15# 71%	2.5±3.3# 79%	4.8±6.9# 50%	0.36±0.68# 29%	0.64±2.0# 14%		20±54# 86%	17±35# 86%	Gallen et al. (2018)
Australia		4.1 Max. 31 91%	7.1 Max. 119 100%	3.1 Max. 17 96%	5.6 Max. 33 99%	3.2 Max. 6.3 35%	1.8 Max. 3.3 14%	6.5 Max. 87 99%	5.9 Max. 509 91%	7.7 Max. 137 92%	Nguyen et al. (2022)
Denmark					8.7 Max. 23.5 72%	nd	1.7 Max. 8.4 82%		4.8 Max. 32.8 72%	3.3 Max. 10.1 91%	Bossi et al. (2009)
China#	0.95±0.08 0.45±0.08	1.76±0.09 2.10±0.09	1.48±0.04 1.03±0.18	0.73±0.03 0.51±0.01	3.73±0.20 3.04±0.09	0.38±0.08 0.30±0.10	1.74±0.19 1.29±0.21	1.29±0.14 14.4±0.83	nd nd	7.50±0.22 6.45±0.79	Pan et al. (2016)
NZ#	nd nd	nd 1.4±0.3	6.9±0.2 2.3±0.7	1.3±0.1 0.6±0.0	4.0±0.2 1.7±0.4	0.7±0.1 0.3±0.0	0.6±0.2 0.2±0.2	7.1±0.0 2.1±0.1	1.6±0.2 0.8±0.2	7.7±1.1 1.7±0.2	Lenka et al. (2022)

Data are median values unless otherwise indicated, with maximum concentration and detection frequency shown below, where that data was available. Where two sets of data are shown together, the study had reported data for individual WWTPs separately. nd – not detected. A blank space means a study did not assess the compound.

# indicates data is presented as a mean value, with standard deviation where that information was available. * only one sample for which data was available.

5.2 PER- AND POLYFLUOROALKYL SUBSTANCES IN STORMWATER

Compared with point source discharges such as WWTPs, there is less data available for PFAS originating from non-point sources (Xiao et al. 2012b). Nonetheless, available data show that stormwaters can be a source of PFAS (Murakami et al. 2009; Page et al. 2019) (Table 10). As for many other stormwater contaminants, the diversity and concentrations of PFAS in stormwaters are influenced by the nature of the catchment and the activities occurring within it. Street dusts and other urban particulates and debris have been shown to contain PFAS (Murakami and Takada 2008; Pramanik et al. 2020). In addition, rainfall itself has also been suggested to be a source of PFAS, especially PFOA, in stormwaters (Kim and Kannan 2007; Murakami et al. 2009; Xiao et al. 2012b). Total PFAS concentrations in stormwater tend to be reported in the range of tens (Houtz and Sedlak 2012; Perkola and Sainio 2013; Codling et al. 2020) to low hundreds of ng/L (Nielsen et al. 2010; Xiao et al. 2012b). PFAS concentrations are typically higher in highly trafficked commercial or industrial areas than residential catchments (Kim and Kannan 2007; Xiao et al. 2012b), and increase with increasing duration of antecedent dry period (Murakami et al. 2009).

A variety of legacy and emerging PFAS and precursor compounds have been detected in stormwaters from various residential, light commercial and industrial catchments, including PFOA, PFOS, PFNA, PFDA, PFBS, PFHxA, PFHxS, perfluoroundecanoic acid (PFUA), perfluoroheptanoic acid (PFHpA), perfluoropentanoic acid (PFPeA), perfluoroundecanoic acid (PFUnDA), perfluorododecanoic (PFDoDA), perfluorooctanesulfonamide (FOSA), N-ethyl perfluorooctane sulfonamido acetic acid (N-EtFOSSA), 8:2 FTS and 6:2 FTS (Kim and Kannan 2007; Murakami and Takada 2008; Murakami et al. 2009; Nielsen et al. 2010; Houtz and Sedlak 2012; Xiao et al. 2012b; Perkola and Sainio 2013; Codling et al. 2020). Many studies report that stormwater samples tend to be dominated by PFOA and/or PFOS (Kim and Kannan 2007; Nielsen et al. 2010; Houtz and Sedlak 2012; Xiao et al. 2012b; Perkola and Sainio 2013).

Table 10. Concentrations of selected PFAS (ng/L) in urban stormwater reported in international studies.

Country	PFOA	PFOS	PFDA	PFNA	PFHpA	PFHxS	PFUnDA	PFHxA	6:2 FtS	8:2 FtS	Reference
US	3.8 Max. 29.3	0.81 Max. 14.6	0.46 Max. 8.39	0.71 Max. 5.9	1.13 Max. 6.44	0.35 Max. 13.5	<LOQ Max. 1.99		1.22 Max. 21.3	<LOQ Max. 5.84	Kim and Kannan (2007)
US	Max. 306	Max. 155.8	Max. 10.6	Max. 10.7	Max. 6.8		Max. 2.9				Xiao et al. (2012b)
US	7.3	15	2*	2*	3*	2*		4.5			Houtz and Sedlak (2012)
Japan	90 Max. 174	5.9 Max. 50	23 Max. 77	24 Max. 70			7.0 Max. 45				Murakami et al. (2009)
Finland	4.3# Max. 5.1	8.2# Max. 9.9	<0.5# Max. 0.6					10# Max. 17			Perkola and Sainio (2013)
Denmark	Max. 67	Max. 419	Max. 5.4	Max. 8.6	Max. 46.6	Max. 58.3	Max. <3.3	Max. 181			Nielsen et al. (2010)
Sweden	48	6.9	20								Kaj et al. (2011)

Data are median values unless otherwise indicated, with the maximum value indicated below this where that data was available.

# indicates date presented is a mean value; * indicates concentration estimated from figures provided; nd – not detected; LOQ – limit of quantification.

5.3 HEALTH EFFECTS OF PER- AND POLYFLUOROALKYL SUBSTANCES

Despite recently increased attention to PFAS from regulatory authorities, the human health risks associated with PFAS remain unclear (enHealth 2019). The main concern is the potential for harm due to the period of time that PFAS may be stored in the body: PFAS binds to proteins, and may accumulate in the blood, liver, kidneys and muscle (Rumsby and Manning 2018). The compounds are not metabolised, although some may transform to more stable PFAS. The estimated elimination half-lives for different PFAS range from days to years (EFSA 2020; ATSDR 2021).[69]

The toxicity of PFOS and PFOA have been evaluated in a large number of human and animal studies, with significantly less data available for other legacy and emerging PFAS (EFSA 2020; ATSDR 2021). Animal studies suggest hepatic, immune, endocrine, reproductive and developmental effects. However, the relevance of these studies in determining human health effects is uncertain (enHealth 2019), due to significant differences in elimination half-lives (eg, hours in rodents versus years in humans), and some data suggesting mechanisms of toxicity that may be less relevant to humans (ATSDR 2021). Data from human epidemiological studies has suggested associations between PFAS exposure and adverse health outcomes including altered liver function, increased blood cholesterol, pregnancy-induced hypertension, immunotoxicity, and developmental effects including decreased birth weight; however, the data are inconsistent, effects are generally small, and no causal relationships have been established for these outcomes (enHealth 2019; NHMRC 2019; EFSA 2020; ATSDR 2021).

The IARC (2017) have classified PFOA as possibly carcinogenic to humans (ie, group 2B), and the US EPA have classified PFOA and PFOS are potentially carcinogenic to humans, with increased testicular and kidney cancers being observed in highly exposed individuals (ATSDR 2021).[70]

5.4 REGULATIONS AND GUIDELINES

Pertinent international regulations and guidelines regarding exposure to PFAS are summarised in Table 11. The uncertainty regarding the adverse health effects of PFAS is reflected in the wide range of health-based guidance values. For example, in June 2022, the

[69] For example, the estimated elimination half-life is 72 hours for PFBA, 28 days for PFBS, 2-10 years for PFOA and 3-27 years for PFOS (EFSA 2020; ATSDR 2021).
[70] http://www.pops.int/TheConvention/ThePOPs/AllPOPs/tabid/2509/Default.aspx Accessed 29 June 2022.

US EPA announced new chronic reference doses (RfDs) for GenX and PFBS, and draft updated RfDs for PFOS and PFOA; these revised values were 2-4 orders of magnitude lower than those previous set for PFOS and PFOA in 2016.[71]

In 2018, Dawson et al. (2018) concluded that following discussions with officials in Australia and Europe, no jurisdiction was identified where regulatory limits for PFAS in influent, effluent or biosolids from WWTPs had been set. This does however appear to be a space that environmental agencies are currently reviewing.[72, 73, 74 75]

Table 11. Recommended oral exposure limits for PFAS.

	PFOA	PFOS	PFHxS	PFNA	PFBS	GenX
Tolerable Weekly Intake, EFSA[76] (ng/kg bw/wk)	4.4 (note, this is a group TWI for PFOA, PFOS, PFNA and PFHxS)					
Tolerable Daily Intake, FSANZ[77] (ng/kg bw/day)	160	20	*			
Reference Dose,*# US EPA[78] (ng/kg/day)	0.0015 (chronic)	0.0079 (chronic)			3 (chronic)	300 (chronic)
Minimum Risk Level, ATSDR[79] (ng/kg bw/day)	3 (int.)	2 (int.)	20 (int.)	3 (int.)		

US EPA Reference Doses and ATSDR Minimum Risk Levels are for oral exposures. * For PFHxS, there was insufficient evidence to justify establishing a TDI; however, as a precaution, the TDI for PFOS should also be used for PFHxS. Thus, the sum of both PFOS and PFHxS should be compared with the TDI for PFOS. # Draft RfD values int. = intermediate exposure.

[71] https://www.epa.gov/system/files/documents/2022-06/technical-factsheet-four-PFAS.pdf Accessed 29 June 2022
[72] https://www.epa.gov/newsreleases/new-interim-strategy-will-address-pfas-through-certain-epa-issued-wastewater-permits Accessed 29 June 2022
[73] https://www.dcceew.gov.au/sites/default/files/documents/pfas-nemp-2.pdf Accessed 29 June 2022
[74] https://www.michigan.gov/pfasresponse/investigations/wastewater Accessed 29 June 2022
[75] https://www.dcceew.gov.au/sites/default/files/documents/pfas-nemp-2.pdf Accessed 12 July 2022.
[7676] https://www.efsa.europa.eu/en/news/pfas-food-efsa-assesses-risks-and-sets-tolerable-intake Accessed 29 June 2022
[77] https://www.foodstandards.gov.au/consumer/chemicals/Pages/Perfluorinated-compounds.aspx Accessed 29 June 2022
[78] https://www.epa.gov/system/files/documents/2022-06/technical-factsheet-four-PFAS.pdf Accessed 29 June 2022
[79] https://wwwn.cdc.gov/TSP/MRLS/mrlsListing.aspx Accessed 29 June 2022

6. PESTICIDES

Chemical pesticides are substances that are used to deter, incapacitate or kill pests including weeds, invertebrates (insects, mites and arachnids), fungi, algae and rodents. Over 1,000 different pesticides are used around the world for various purposes, primarily in agriculture to protect crops and in public health to control vectors of disease (eg mosquitos).[80,81] Other uses of pesticides include in forestry, maintenance of public and private gardens and green spaces, preventing biofouling of infrastructure, and maintenance of pools and aquaria (WHO 1990, 2019c; Nicolopoulou-Stamati et al. 2016).

Pesticides may be classified according to their target organism (eg herbicide, insecticide, fungicide etc), or their chemical structure (eg organochlorine, organophosphate, etc). The main classes of pesticides, with common examples, are shown in Table 12. Some classes of pesticide exhibit high lipophilicity, bioaccumulation, and/or high environmental persistence, and because they are designed to disturb biological activities, are often associated with toxic effects on environmental and/or human health (Jayaraj et al. 2016; WHO 2019c). As such, a large number of pesticides have been subject to restriction or prohibition by international convention (eg Stockholm and Rotterdam Conventions) or national regulatory agencies (eg US EPA, European Commission; see Appendix A) (Campo et al. 2013). It is commonly considered that human exposure to these priority compounds, mainly organochlorine and organophosphate insecticides, is low since they are no longer being manufactured or widely used, especially in developed countries (WHO 2020b; however, many of these pesticides and their degradation products can still be detected in the environment as a result of historic use (Masoner et al. 2019). Nonetheless, the benefits afforded by pesticides, including increased food security and the management of vector-borne disease, have continued to drive the development of new pesticides (Bonner and Alavanja 2017), with a shift towards substances with faster degradation rates, reduced environmental persistence and/or reduced non-target toxicity, including carbamates, neonicotinoids and pyrethroids (Carpenter et al. 2016; Cressey 2018; Kalyabina et al. 2021). However, some of these compounds may still feature characteristics of concern, such as the ability to migrate over long distances, or moderate toxicity despite a short half-life. Further, the widespread and continuous use of

[80] https://www.who.int/news-room/questions-and-answers/item/chemical-safety-pesticides Accessed 12 May 2022.
[81] https://ec.europa.eu/food/plant/pesticides/eu-pesticides-database/start/screen/active-substances Accessed 20 December 2022

certain pesticides means they exhibit a pseudo-persistence, being frequently reintroduced into the environment (Kalyabina et al. 2021).

Whilst this section focuses on pesticides based on their active ingredients, it is important to note that most pesticide preparations also contain carrier substances, stabilisers, solvents, surfactants and other additives, known as ‘inert ingredients’ or adjuvants (WHO 1990; Mesnage et al. 2019; Kalyabina et al. 2021). These additional ingredients are not required to be disclosed on product labels and in some jurisdictions may not be subject to the same toxicity assessment processes as active ingredients (Mesnage et al. 2019), even though some are known to exert their own adverse health effects (WHO 1990; Kalyabina et al. 2021). For example, certain commercial formulations of glyphosate, carbaryl, bifenthrin, malathion, imidacloprid and tebuconazole have been shown to exhibit greater ecological toxicity compared with active ingredients alone (Mesnage et al. 2019; Kalyabina et al. 2021).

Table 12. Overview of the main chemical classes of pesticides, noting key characteristics and representative compounds. Characteristics are intended as a generalisation only; within each class, differences may exist based on the unique chemical structure of individual compounds.

Chemical class	Characteristics	Representative compounds
Organochlorines	Chlorinated compounds used extensively between 1940s and 1970s until many were banned in developed countries due to extreme environmental persistence. Nine were original inclusions in the Stockholm Convention POP (persistent organic pollutant) list. Exert effects through multiple targets in the central nervous system, including sodium and calcium channels and transporters, and GABA neurotransmission. High environmental persistence: half-life in the environment may range from several weeks (methoxychlor, pentachlorophenol) to more than ten years (DDT, endrin, chlordane). Typically highly lipophilic and resistant to metabolism; persist and bioaccumulate, with long-term storage in adipose tissues.	Aldrin* # Chlordane* # DDT* # Dieldrin* # Dicofol* Endosulfan* # Heptachlor* # Hexachlorobenzene Lindane*# Methoxychlor Mirex* Toxaphene* #
Organophosphates	Broad spectrum of insecticides, largely esters of phosphoric acid. Exert effects through irreversible inhibition of acetylcholinesterase enzyme, which is essential for nerve function. Slightly soluble in water. Degrade rapidly in the environment by hydrolysis following exposure to air or light, although small amounts are detected in food and water.	Chlorpyrifos Dichlorvos Malathion Parathion Dimethoate Fenthion Dimefox
Carbamates	Organic compounds derived from carbamic acid, largely used as insecticides, but may also be herbicides, nemacides and fungicides. Mechanism of action by reversible inactivation of acetylcholinesterase. Acute toxicity can range from low to very high. Break down in the environment within weeks to months.	Aldicarb# Carbofuran# Propoxur Carbaryl Dimethan Vernolate Disulfiram Molinate
Pyrethroids and pyrethrins	Pyrethrins and pyrethroids are naturally-occurring insecticides isolated from *Chrysanthemum* flowers and synthetic analogues, respectively. Pyrethroids are often more toxic to insects than to mammals, and last longer in the environment. Affect voltage-gated sodium channels in nerve cells and lead to paralysis of target organism. Hydrophobic, adsorb strongly to soils. Readily degraded by UV light and microorganisms over days to weeks. Comparatively low mammalian toxicity and fast biodegradation capacity.	Permethrin Cyfluthrin Bifenthrin Fenvalerate Deltamethrin Pyrethrin Dimethrin Allethrin Cypermethrin
Triazines	Broad-spectrum herbicides; among the most widely used herbicides globally (especially atrazine). Mechanism of action is via inhibition of electron transfer in photosystem II in chloroplasts of broadleaf and grassy weeds, thereby inhibiting photosynthesis. Classified as persistent organic compounds as they resist chemical and biological degradation.	Atrazine Simazine Propazine Terbutryn Simetryn

Table 12 continued.

Chemical class	Characteristics	Representative compounds
Neonicotinoids	Relatively new, extensively used, suggested to be a low-risk alternative. Bind irreversibly to nicotinic acetylcholine receptors (nAChRs), causing paralysis and death of target organism. Different receptor structures in mammals and insects means binding is stronger in insect neuron than in mammals, thus more toxic to insects. Water-soluble, breakdown slowly in the environment when exposed to sunlight or microorganisms. Imidacloprid (most widely used insecticide in the world since 1999) has been banned in the EU for outdoor use, and restricted in several US states).	Imidacloprid Thiacloprid Clothianidin Thiamethoxam
Phenylamides/ carbanilates	Predominantly herbicides. Inhibit photosystem II in chloroplast and therefore photosynthesis. Degradation in the environment is slow, occurring via UV photodegradation or presence of acidic or alkaline conditions. The most common, diuron, is among the 10 most-used pesticides in the US.	Diuron Isoproturon Fenuron Carbetamide
Phenylamides/ acylanalides	Commonly used as fungicides, inhibit certain polymerase system enzymes and impact mitosis and cell division. Have been reported to enter the food chain and be present in higher organisms.	Alachlor[#] Propanil Propachlor Solane
Azoles	Used as fungicides in agriculture and in biocidal material protection. Some compounds may be used in pharmaceuticals. Persistent in soils and water due to stability towards hydrolytic, photolytic and biological degradation.	Propiconazole Tebuconazole Cyproconazole Carbendazim
Phenoxy alkonates	Widely used group of herbicides. Two families with different mechanisms of action: one mimics the growth hormone indoleacetic acid to cause uncontrolled growth, the other inhibits the plant acetylCoA-carboxylase enzyme. Nearly all are degraded by microorganisms.	2,4-D Mecoprop Dichloroprop
Other	Glyphosate is a broad-spectrum herbicide that works by inhibiting the production of essential aromatic amino acids in plants. Hundreds of different formulations are known.	Glyphosate

Compiled from Jayaraj et al. (2016), Nicolopoulou-Stamati et al. (2016), WHO (2020) and Rani et al. (2021). *Subject to the Stockholm Convention, meaning that production and use has been prohibited or severely restricted. [#] Subject to the Rotterdam Convention on prior informed consent regarding the trade of hazard chemicals.

6.1 PESTICIDES IN WASTEWATER

Compared with other emerging contaminants groups, there is relatively little information available on the presence of pesticides in municipal wastewaters, as pesticides are typically considered to be of agricultural or horticultural origin rather than urban origin, and therefore to have greater relevance to run-off and diffuse sources of contamination than to wastewater (Morasch et al. 2010; Köck-Schulmeyer et al. 2013; Loos et al. 2013). However, the available data shows that municipal wastewater can be a source of pesticide release to the environment, and consistent inputs during dry weather and in separated networks highlight the potential for 'down-the-drain' pathways (Weston et al. 2013; Sutton et al. 2019b; Xie et al. 2021). Pesticides may enter domestic wastewaters through use patterns including bathing of people and pets (eg, using pet shampoos, tick and flea treatments or lice shampoos), indoor pest control (eg, carpet and upholstery treatments, indoor space sprays), laundry (eg of impregnated fabrics for gear/apparel or clothing contaminated with pesticide products), washing of application equipment, improper disposal, or maintenance of swimming pools, public fountains and aquaria (Sutton et al. 2019b; Xie et al. 2021). Nurseries and greenhouses may also contribute where these are connected to the municipal network (Weston et al. 2013; Xie et al. 2021). Widespread application of pesticides in urban outdoor environments may also contribute to significant pesticide loads in urban stormwaters, which may enter the wastewater system through inflow or infiltration, or following dry-weather flows associated with power-washing and related activities (Weston et al. 2013; Sutton et al. 2019b).

Monitoring data for pesticides in wastewater is relatively sparse and ad-hoc; throughout the literature, a wide diversity of candidate compounds have been screened for, but there does not appear to be a core group of analytes that are consistently included in studies, and often studies will focus on a smaller number of compounds of the same class (eg Kahle et al. 2008; Sutton et al. 2019b). A large number of different pesticide classes have been detected in municipal wastewater, including organophosphates, carbamates, azoles, triazines, pyrethroids, ureas, phenoxy alkonates and acylanilides (Table 13). In particular, commonly reported compounds (and/or their derivatives) include diuron, mecoprop, 2,4-dichlorophenoxyacetic acid (2,4-D), diazinon, atrazine, terbutylazine, isoproturon, simazine, simetryn, propiconazole, permethrin and glyphosate (Katosoyiannis and Samara 2004; Terzic et al. 2008; Morasch et al. 2010; Singer et al. 2010; Teijon et al. 2010; Campo et al. 2013; Köck-Schulmeyer et al. 2013; Loos et al. 2013; Weston et al. 2013; Korgmaa et al. 2020). Restricted organochlorine pesticides (OCs) appear to be seldom tested for; they are reported in several studies carried out prior to or shortly after ratification of the Stockholm Convention (eg Katsoyiannis and Samara 2004), however other studies have report testing

for, but not detecting OCs or other priority pesticide in wastewater (Rule et al. 2006b; Korgmaa et al. 2020). Many other pesticides have not yet been examined for their presence in wastewater (Sutton et al. 2019b). Differences in the pesticide profile from different WWTPs are reported, and usually reflect land use and management practices within the respective catchments, especially where there is stormwater influence (Campo et al. 2013; Köck-Schulmeyer et al. 2013). Some pesticides may be frequently detected (up to 80-100%) within a wastewater network or study, and not detected at all in another (Table 13).

Concentrations of pesticides in wastewater are highly variable, ranging from <0.1 ng/L to several µg/L depending on the specific pesticide and catchment characteristics, although in most instances are less than 100 ng/L (Margot et al. 2015; Sutton et al. 2019b). Higher concentrations are often associated with wastewaters from combined stormwater networks and/or receiving industrial inputs (Terzic et al. 2008; Singer et al. 2010; Stamatis et al. 2010; Xie et al. 2021). Seasonal patterns may also be evident in some catchments and/or for some pesticides (Terzic et al. 2008; Stamatis et al. 2010; Weston et al. 2013; Margot et al. 2015); for example, in a survey of Estonian WWTPs, Korgmaa et al. (2020) reported the detection of diuron and glyphosate in samples collected during summer and autumn, consistent with the primary application and use period for these pesticides.

The removal of pesticides from WWTPs is highly variable due to the diversity in their chemical structures and properties; some compounds show low removal rates (25-30% for atrazine, mecoprop, glyphosate), while others show relatively high removal rates (80-90% for many organochlorine pesticides and pyrethroids) (Morasch et al. 2010; Weston et al. 2030; Margot et al. 2015). On average however, poor removal efficiencies (<50%) are often reported (Martin Ruel et al. 2010; Stamatis et al. 2010; Köck-Schulmeyer et al. 2013; Margot et al. 2015).

Table 13. Concentration of pesticides (ng/L) in untreated municipal wastewaters reported in international studies.

	Country	Detection frequency %	Concentration (ng/L)			Reference
			Median	Mean	Max	
2,4-D	Spain	33	32.1	88.5	442	Kock-Schulmeyer et al. (2010)
	Estonia	0			<LOD	Korgmaa et al. (2020)
	EU-wide	72	11.9	27.1	357	Loos et al. (2013)
Acetochlor	Spain	9	28.9		35.2	Campo et al. (2013)
	Spain	0	<LOD		<LOD	Campo et al. (2013)
α-HCH	Spain	14		0.2	0.2	Teijon et al. (2010)
	Greece	94	33	39	120	Katosoyiannis and Samara (2004)
	Estonia	6	7	7	10	Korgmaa et al. (2020)
Alachlor	Spain	4	2.59		2.59	Kock-Schulmeyer et al. (2013)
	Spain	0			<LOD	Teijon et al. (2010)
	Spain	0			<LOD	Campo et al. (2013)
	Estonia	0			<LOD	Korgmaa et al. 2020
Aldrin	Greece	35	<LOD	10±25	102	Katosoyiannis and Samara (2004)
	France			<2		Martin Ruel et al. (2011)
AMPA	Estonia	28	530	1,080	3640	Korgmaa et al 2020
Atrazine	Spain	75		6.7	9	Teijon et al. (2010)
	Switzerland			30±10		Singer et al. (2010)
	Spain	17	1.23	1.24	1.74	Kock-Schulmeyer et al. (2013)
	China	100		104±5	122	Wang et al. (2022)
	Balkans	38		3.3	28,000*	Terzic et al. (2008)
	Switzerland	71		52±29		Morasch et al. (2010)
	Spain	47		12.8	27.4	Campo et al. (2013)
	Spain	13		20.9	36.9	Campo et al. (2013)
	France			10±0		Martin Ruel et al. (2011)
	EU-wide	68	2.2	4.2	36.6	Loos et al. (2013)
	Estonia	0			<LOD	Korgmaa et al. (2020)
β-HCH (lindane)	Spain	85.7		1.57	2.6	Teijon et al. (2010)
	Greece	29	<LOD	26±71	290	Katosoyiannis and Samara (2004)
	Estonia	8	13	16	34	Korgmaa et al. (2020)
Bifenthrin	US	100	24.7	22.8	39.3	Weston et al. (2013)
	Estonia	0			<LOD	Korgmaa et al. (2020)
Carbaryl	EU-wide	9	<3	1.6	81.4	Loos et al. (2013)
Carbendazim	Switzerland			110±30		Singer et al. (2010)
	Switzerland	29		110±60		Morasch et al. (2010)
	Spain	47		269	2,821	Campo et al. 2013)
Carbofuran	Spain	27		9.0	42.1	Campo et al. (2013)
	Spain	6		3.6	4.5	Campo et al. (2013)
Clofenvinphos	Spain	80		52.6	268	Campo et al. (2013
	Spain	44		18.1	78	Campo et al. (2013
	France			<2		Martin Ruel et al. (2011)
	Estonia	0			<LOD	Korgmaa et al. (2020)
Chlorpyriphos	Spain	90		19.1	164	Campo et al. (2013)
	Spain	72		14.1	109	Campo et al. (2013)
	US	83	17	23.1	81.9	Weston et al. (2013)
	Estonia	0			<LOD	Korgmaa et al (2020)
Cyfluthrin	US	8	12.2		12.2	Weston et al. (2013)
	Estonia	0			<LOD	Korgmaa et al. (2020)
Cyhalothrin	US	100	16.3	16.8	31.1	Weston et al. (2013)
Cypermethrin	US	100	28.2	31.2	44.4	Weston et al. (2013)
Cyproconazole	Greece			313	1,735	Stamatis et al. (2010)
DDD	Greece	53	3.2	22±34	130	Katosoyiannis and Samara (2004)
DDT	Greece	29	<LOD	6.9±17	59	Katosoyiannis and Samara (2004)

Table 13 continued.

	Country	Detection frequency %	Concentration (ng/L)			Reference
			Median	Mean	Max	
Desethylatrazine	Spain	13	2.59	24.1	67.9	Kock-Schulmeyer et al. (2013)
	Switzerland			40±6		Singer et al. (2010)
δ-HCH	Spain	100		3.54	6.2	Teijon et al. (2010)
Diazinon	Spain	96	53.6		684	Kock-Schulmeyer et al. (2013)
	Switzerland			60±10	1,130	Singer et al. (2010)
	Switzerland	43		32±31		Morasch et al. (2010)
	Spain	97		73.9	316	Campo et al. (2013)
	Spain	84		15.9	75.3	Campo et al. (2013)
	EU-wide	71	4.1	21.4	391	Loos et al. (2013)
Dichlorofenthion	Spain	53.3		15.0	34.9	Campo et al. (2013)
	Spain	0		<LOD	<LOD	Campo et al. (2013)
Dieldrin	Greece	94	23	27±20	82	Katosoyiannis and Samara (2004)
	France			10		Martin Ruel et al. (2011)
Dimethoate	Spain	25	1.86	4	10.9	Kock-Schulmeyer et al. (2013)
	Balkans	4	<LOD		80	Terzic et al. (2008)
	Spain	93		101	621	Campo et al. (2013)
	Spain	50		88.1	640	Campo et al. (2013)
Diuron	Spain	100		324	512	Teijon et al. (2008)
	Spain	88	42.2	93	452	Kock-Schulmeyer et al. (2013)
	Switzerland			60±30		Singer et al. (2010)
	Switzerland	0		<LOD		Morasch et al. (2010)
	Spain	77		322	2,526	Campo et al. (2013)
	Spain	81		159	1,218	Campo et al. 2013)
	France			170±110		Martin Ruel et al. (2011)
	Estonia	6	61	61	89	Korgmaa et al. (2020)
	EU-wide	77	11.6	61.7	1,426	Loos et al. (2013)
Endosulfan	Greece	47	<LOD	51±95	347	Katosoyiannis and Samara (2004)
	France			<2		Martin Ruel et al. (2011)
Endrin	Greece	6	<LOD	1.8±7.2	30	Katosoyiannis and Samara (2004)
	France			<2		Martin Ruel et al. (2011)
Fenthion sulfone	Spain	33		16.0	35.3	Campo et al. (2013)
	Spain	3		13.2	13.2	Campo et al. (2013)
Glyphosate	Estonia	31	410	421	970	Korgmaa et al.(2020)
Heptachlor	Greece	71	18	46±62	230	Katosoyiannis and Samara (2004)
	Estonia	0			<LOD	Korgmaa et al. (2020)
Hexachlorobenzene	Spain	71		2.0		Teijon et al. (2010)
	Greece	71	22	20±16	51	Katosoyiannis and Samara (2004)
	Estonia	0			<LOD	Korgmaa et al. (2020)
Hexachlorocyclohexane	France		60			Martin Ruel et al. (2011)
Hexythiazox	Spain	90		5	15.7	Campo et al. (2013)
	Spain	13		1.8	2.0	Campo et al. (2013)
Imazalil	Spain	80		292	2,121	Campo et al. (2013)
	Spain	69		35.9	229	Campo et al. (2013)
Imidacloprid	Spain	67		3.4	6.8	Campo et al. (2013)
	Spain	59		34.4	166	Campo et al. (2013)
Irgarol	Switzerland	29		6±0		Morasch et al. (2010)
	Switzerland			10±4		Singer et al. (2010)
Isoproturonhaha	Spain	0			<LOD	Kock-Schulmeyer et al. (2013)
	Switzerland			90±100		Singer et al. (2010)
	Switzerland	14		70		Morasch et al. (2010)
	Spain	77		15.3	102	Campo et al. (2013)
	Spain	56		10.5	34.2	Campo et al. (2013)
	France			<2		Martin Ruel et al. (2011)
	Estonia	3	3	3	32	Korgmaa et al. (2020)
	EU-wide	51	0.4	10.1	270	Loos et al. (2013)

Table 13 continued.

	Country	Detection frequency %	Concentration (ng/L)			Reference
			Median	Mean	Max	
Malathion	Spain	0			<LOD	Kock-Schulmeyer et al. (2013)
	Spain	3			848	Campo et al. (2013)
	Spain	0			<LOD	Campo et al. (2013)
Mecoprop	Spain	25	52.9	106	391	Kock-Schulmeyer et al. (2013)
	Switzerland			870±590		Singer et al. (2010)
	Switzerland	100		170±170		Morasch et al. (2010)
	EU-wide	72	17.2	127	2,209	Loos et al. (2013)
Methiocarb	Spain	0				Kock-Schulmeyer et al. (2013)
	Spain	20		4.7	5.7	Campo et al. (2013)
	Spain	31		14.9	105	Campo et al. (2013)
Metolachlor	Spain	0			<LOD	Kock-Schulmeyer et al. (2013)
	Spain	33		129	313	Campo et al. (2013)
	Spain	16		18.1	42.6	Campo et al. (2013)
	EU-wide	29	<3	12.4	38.0	Loos et al. (2013)
Molinate	Spain	0			<LOD	Kock-Schulmeyer et al. (2013)
	Spain	10		12.5	19.2	Campo et al. (2013)
	Spain	0			<LOD	Campo et al. (2013)
Permethrin	US	100	306	294	449	Weston et al. 2013
	Estonia	36	20	25	50	Korgmaa et al. (2020)
Propanil	Spain	33	4.02	9.0	35.9	Kock-Schulmeyer et al. (2013)
	Spain	10		14.6	49.8	Campo et al. (2013)
	Spain	0			<LOD	Campo et al. (2013)
Propazine	China	0		<LOD	<LOD	Wang et al. (2022)
	Spain	27		43	277	Campo et al. (2013)
	Spain	9		3.7	5.7	Campo et al. (2013)
Propiconazole	Switzerland	100	10	11.7±6.7	17	Kahle et al. (2008)
	Balkans	0			<80	Terzic et al. (2008)
	Switzerland	100		86±22		Morasch et al. (2010)
	Estonia	50	16	251	2,300	Korgmaa et al. (2020)
Simazine	Balkans	8		0.3	500	Terzic et al. (2008)
	Spain	75		16.3	28	Teijon et al. (2010)
	Spain	29	1.61	7.3		Kock-Schulmeyer et al. (2013)
	China	0		<LOD	<LOD	Wang et al. (2022)
	Spain	7		5.0	5	Campo et al. (2013)
	Spain	22		20.3	37.8	Campo et al. (2013)
	France			5±3		Martin Ruel et al. (2010)
	EU-wide	28	<5	26.3	689	Loos et al. (2013)
Tebuconazole	Greece			564	1,893	Samatis et al. (2010)
	Switzerland	90	2.0	2.4±2.3	8	Kahle et al. (2008)
	Balkans	0			<80	Terzic et al. (2008)
	Spain	34		23.0	261	Campo et al. (2013)
Terbutryn	Switzerland			70±20		Singer et al. (2010)
	China	0		<LOD	<LOD	Wang et al. (2022)
	Balkans	8		0.1	150	Terzic et al. (2008)
	Estonia	11	42	41	59	Korgmaa et al. (2020)
	Switzerland	43		110		Morasch et al. (2010)
	Spain	74		23	183	Campo et al. (2013)
	Spain	84		18	73.5	Campo et al. (2013)

Table 13 continued.

	Country	Detection frequency %	Concentration (ng/L)			Reference
			Median	Mean	Max	
Terbutylazine	Spain	46	8.8	20.6	71.3	Kock-Schulmeyer et al. (2013)
	China	0		<LOD	<LOD	Wang et al. (2022)
	Balkans	4			100	Terzic et al. (2008)
	Switzerland			20±3		Singer et al. (2010)
	Spain	50		12.4	35.5	Campo et al. (2013)
	EU-wide	67	4.7	90.6	2,411	Loos et al. (2013)
Thiabendazole	Spain	91		40.6	505	Campo et al. (2013)
γ-HCH	Spain	0				Teijon et al. (2010)
	Greece	6	<LOD	1.4±5.9	25	Katosoyiannis and Samara (2004)
	Estonia	3	<LOD		2	Korgmaa et al. (2020)

2,4-D – 2,4-dichlorophenoxyacetic acid; α-HCH – α-hexachlorocyclohexane; AMPA – α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid; β-HCH – β-hexachlorocyclohexane; DDT – dichlorodiphenyltrichloroethane; DDD – dichlorodiphenyldichloroethane; δ-HCH – δ-hexachlorocyclohexane; γ-HCH – hexachlorocyclohexane

6.2 PESTICIDES IN STORMWATER

Pesticides are commonly detected in stormwaters and runoff, which are well-recognised pathways to transport pesticides to surface waters (Xie et al. 2021). In a survey of stormwater across the United States of America, Masoner et al. (2019) reported that of 738 organic chemicals screened for, pesticides were the most commonly detected group of contaminants, accounting for 35% of all detections. The pesticide concentrations in stormwater are often higher than in wastewaters (Gasperi et al 2012; Zgheib et al 2012). The widespread application of pesticides in the urban environment means their sources and pathways to stormwaters are diverse, including application to lawns, parks, berms and road embankments, sports fields, green spaces and cemeteries for plant management and insect and weed control; application to building materials, facades and other structures to prevent moss and algae growth or root penetration; to paths and paved areas to prevent moss or lichen growth; and application to a range of surfaces to control insect or vermin populations (Domagalski et al. 1996; Bollman et al. 2012; Margot et al. 2015; Mūller et al. 2020). Other pesticides may be transported as aerosols from agricultural operations, and be deposited onto various surfaces (Domagalski et al. 1996; Vialle et al. 2013; Burant et al. 2018). During rain events, pesticides are readily washed and/or leach from these surfaces into the stormwater network: for example, Huang et al. (2013) estimated some 43% of herbicides applied along highways to control roadside vegetation is lost to runoff. Blanchoud et al.

(2007) noted that while the amount of pesticides used in rural areas is greater than in urban areas, the extent of impervious surfaces in urban areas facilitates significant transfer of pesticides to stormwater, while Wittmer et al. (2010) concluded that in mixed-use catchments, pesticide inputs to surface water from urban sources are as important as those from agricultural sources.

A significant diversity of different pesticide compounds is reported in stormwaters (Table 14); for example, Botta et al. (2012) detected 49 different pesticide compounds in stormwaters in France, while Vialle et al. (2013) detected 39, Rippy et al. (2017) 19, and Carpenter et al. (2016) 18. Several studies have reported that pesticide profiles in urban stormwaters are dominated by herbicides (Gasperi et al. 2012; Zgheib et al. 2012), reflecting an estimated 85% of all pesticide use in urban areas being herbicides (Blanchoud et al. 2007). Among the most commonly reported pesticides in stormwater are glyphosate and its derivative aminomethylphosphonic acid (AMPA), diuron, carbendazim, atrazine, imidacloprid, metolachlor, isoproturon, carbaryl, mecoprop, 2-methyl-4-chlorophenoxyacetic acid (MCPA), amitrole, oryzalin, 2,4-dichlorophenoxyacetic acid (2,4-D), fipronil, cyfuthrin, simazine, bifenthrin, aminotriazole, terbutryn, permethtin, triclopyr and dinitro-ortho-cresol (DNOC) (Botta et al. 2012; Zgheib et al 2012; Vialle et al. 2013; Bollmann et al. 2014; Gasperi et al. 2014; Carpenter et al. 2016; Rippy et al. 2017; Burant et al. 2018; Masoner et al. 2019). Importantly, the detection of restricted or banned pesticides including aldrin, dieldrin, chlordane and pentachlorophenol demonstrates the potential for the historic application of pesticides to act as a continued source that may pose a risk to environmental or human health (Gasperi et al. 2012; Zgheib et al. 2012; Masoner et al. 2019). The concentrations of detected pesticide compounds are hugely variable and may range from several ng/L to 100 µg/L (Gasperi et al. 2012; Vialle et al. 2013; Masoner et al. 2019); for example, Rippy et al. (2017) and Masoner et al. (2019) reported that the concentrations of detected compounds in catchments across Australia and the US spanned three and five orders of magnitude, respectively. In many instances however, concentrations are typically less than 100 ng/L.

The pesticide profile of stormwaters has been shown to be highly variable between catchments, with the presence of certain pesticides generally reflecting specific land uses within the catchment, and at a larger scale, regional differences in the pesticide needs and/or regulation (Wittmer et al. 2010; Vialle et al. 2013; Carpenter et al. 2016; Rippy et al. 2017; Burant et al, 2018). For example, Vialle et al. (2013) noted differences between the pesticides detected in roof-harvested rainwater in rural and suburban catchments in France, while Carpenter et al. (2016) observed differences between residential, mixed use and commercial catchments in the United States. Burant et al. (2018) noted that while the insecticide fipronil is widely used in California to control ants and termites and is therefore

frequently detected in storm and surface waters, it was not detected in Wisconsin stormwaters, where these insects are not of great concern. Similarly, seasonal trends in the presence of specific pesticides and/or their concentrations may be observed, associated with temporal variation in their application (Wittmer et al. 2010; Botta et al, 2012; Vialle et al. 2013). For example, Vialle et al. (2013) reported that roof-harvested rainwater samples collected during summer were dominated by pesticides commonly used in vineyards, while spring samples were dominated by crop-associated pesticides, and autumn and winter samples by pesticides associated with winter crops. Wittmer et al. (2010) reported the concentration of mecoprop, a pesticide used in both bitumen roofing and applied to lawns, reached concentrations up to 32 µg/L between May and September, compared with <0.1 µg/L during October and November.

Table 14. Concentrations of pesticides (ng/L) in urban stormwaters reported in international studies.

	Country	Detection frequency %	Concentration (ng/L)			Reference
			Median	Mean	Max	
2,4-D	US	100	475		2,743	Burant et al. (2018)
	US	100	470		2,867	Burant et al. (2018)
	Australia		6–2,008			Rippy et al. (2017)
	France	42	20		40	Vialle et al. (2013)
	US	31	79	88.3	235	Masoner et al. (2019)
2,4-MCPA	France	71	20		310	Botta et al. (2012)
	France	46	10		660	Botta et al. (2012)
	France	42	10		180	Botta et al. (2012)
	France	50	20		30	Vialle et al. (2013)
Acetochlor	US	27	89.9	141.7	342	Masoner et al. (2019)
	France	42	50		80	Vialle et al. (2013)
Aldrin	France	7	<20		40	Zgheib et al. (2012)
Aminotriazole	France	80	130		3250	Zgheib et al. (2012)
	France	100			460	Gasperi et al. (2012)*
	France	100	220		870	Botta et al. (2012)
	France	100	110		1,900	Botta et al. (2012)
	France	67	60		1,770	Botta et al. (2012)
AMPA	France	100			1,600	Gasperi et al. (2012)*
	France	93	640		9,370	Zgheib et al. (2012)
	France			824±7,074		Gasperi et al. (2014)
	France	58	300		900	Vialle et al. (2013)
Atrazine	US	82	9.2		273	Burant et al. (2013)
	US	100	87			Burant et al. (2013)
	US	0	<2.3			Carpenter et al. (2016)
	Australia		<10-624			Rippy et al. (2017)
	US	58	11.7	39	419	Masoner et al. (2019)
Bifenthrin	US	100	17.3		29.7	Weston et al. (2009)
	US	80	32		120	Carpenter et al. (2016)
	US	16	10.9	18.1	77	Masoner et al. (2019)
Boscalid	US	20	<2.8		8.6	Carpenter et al. (2016)
	Us	16	22.3	35.7	129.6	Masoner et al. (2019)
	France	33	35		60	Vialle et al. (2013)
Carbaryl	US	40	<6.5		50	Carpenter et al. (2016)
	Australia		<10			Rippy et al. (2017)
	US	22	62.6	68.5	114.2	Masoner et al. (2019)
	France	8	20		20	Vialle et al. (2013)
Carbendazim	US	9			6.1	Burant et al. (2018)
	US	69	3.3		54	Burant et al. (2018)
	France			213±1,355		Gasperi et al. (2014)
	US	94	701	1,614	9,577	Masoner et al. (2019)
	France	50	20		20	Vialle et al. (2013)
	Denmark	100	45		306	Bollman et al. (2014)
Carbofuran	US	0	<3.1		<3.1	Carpenter et al. (2016)
Chlordane, cis	US	82	1.1	2.49	14	Masoner et al. (2019)
Chlordane, trans	US	76	0.78	1.98	12	Masoner et al. (2019)
Chlorfenvinphos	France	7	<50		120	Zgheib et al. (2012)
Chlorpyrifos	US	0	<2.1			Carpenter et al. (2016)
	US	57	0.65	1.51	9.8	Masoner et al. (2019)
Chlorotoluron	France	70.8	20		70	Botta et al. (2012)
	France	79.2	70		690	Botta et al. (2012)
	France	66.6	50		530	Botta et al. (2012)
	France	8	70		70	Vialle et al. (2013)

Table 14 continued.

	Country	Detection frequency %	Concentration (ng/L)			Reference
			Median	Mean	Max	
Cyfluthrin	US	0	<5.2		<5.2	Carpenter et al. (2016)
	US	100	8.7		22.6	Weston et al. (2009)
	US	78	0.65	1.51	9.8	Masoner et al. (2019)
Cypermethrin	US	88	8.5		25.9	Weston et al. (2009)
DDT	US	0	<4		<4	Carpenter et al. (2016)
Deisopropylatrazine	US	82	7.2		96	Burant et al. (2018)
	US	92	13		144	Burant et al. (2018)
Deltamethrin	US	25	<LOD		3.5	Weston et al. (2009)
DEA	US	73	7.4		110	Burant et al. (2018)
	US	85	14		177	Burant et al. (2018)
	France	20	<30		30	Zgheib et al. (2012)
	Australia		<10-36			Rippy et al. (2017)
Diazinon	US	0	<0.9		<0.9	Carpenter et al. (2016)
Dieldrin	France	27	<20		200	Zgheib et al. (2012)
	France	100			980	Gasperi et al. (2012)*
	US	76	0.77	2.77	18	Masoner et al. (2019)
Diuron	US	91	4.8		11	Burant et al. (2018)
	US	100	7.7		14	Burant et al. (2018)
	France	100	370		1,750	Zgheib et al. (2012)
	France	100			500	Gasperi et al. (2012)*
	France	100	200		580	Botta et al. (2012)
	France	100	140		3,100	Botta et al. (2012)
	France	91.3	100		230	Botta et al. (2012)
	Denmark		7			Bollman et al. (2014)
	Australia		<10-895			Rippy et al. (2017)
	US	86	51		1,787	Masoner et al. (2019)
DNOC	France	75	80		410	Vialle et al. (2013)
Endrin	France	7	<20		410	Zgheib et al. (2012)
Esfenvalerate	US	20	<3.9		6.2	Carpenter et al. (2016)
	US	0	nd		nd	Weston et al. (2009)
Febuconazole	US	20	<5.2		7.2	Carpenter et al. (2016)
	US	2	6.7		6.7	Masoner et al. (2019)
Fipronil	US	60	6.1		59	Carpenter et al. (2016)
	US	78	16.7	24.3	139	Masoner et al. (2019)
Fluroxypyr	Australia		<10--51			Rippy et al. (2017)
Flurtamone	France	17	20		20	Vialle et al. (2013)
Flusilazole	US	20	<4.5		6.3	Carpenter et al. (2016)
Glyphosate	France	93	1,110		232,000	Zgheib et al. (2012)
	France	100			1,200	Gasperi et al. (2012)*
	France			337±806		Gasperi et al. (2014)
	France	83	500		6,000	Vialle et al. (2013)
	France	100	620		6,600	Botta et al. (2012)
	France	87.5	430		2,600	Botta et al. (2012)
	France	66.6	830		310	Botta et al. (2012)
Hexachlorobenzene	US	22	0.7	0.84	1.7	Masoner et al. (2019)
Hexaxinone	Australia		<10-38			Rippy et al. (2017)
	US	4	30.6	30.6	49.2	Masoner et al. (2019)
Imidacloprid	US	100	13		428	Burant et al. (2018)
	US	100	11		72	Burant et al. (2018)
	US	86	23.4	48.2	234	Masoner et al. (2019)

Table 14 continued.

	Country	Detection frequency %	Concentration (ng/L)			Reference
			Median	Mean	Max	
Isoproturon	France	60	30		140	Zgheib et al. (2012)
	France	100			40	Gasperi et al. (2014)
	France	50	10		250	Botta et al. (2012)
	France	70.8	20		160	Botta et al. (2012)
	France	41.6	10		100	Botta et al. (2012)
	Denmark		2			Bollman et al. (2014)
	France			88±929		Gasperi et al. (2014)
	France	17	50		50	Vialle et al. (2013)
MCPA	US	91	27		52.7	Burant et al. (2018)
	US	62	7.3		662	Burant et al. (2018)
	Australia		6-748			Rippy et al. (2017)
	France	71	20		310	Botta et al. (2012)
	France	46	10		660	Botta et al. (2012)
	France	42	10		180	Botta et al. (2012)
Mecoprop	France	50	20		30	Vialle et al. (2013)
Metaldehyde	France	60	60		580	Zgheib et al. (2012)
	France	33	145		240	Vialle et al. (2013)
Metolachlor	US	10	<28		34	Burant et al. 2018)
	US	69	39		388	Burant et al. 2018)
	Australia		<10			Rippy et al. (2017)
	US	60	6		13	Carpenter et al. (2016)
	US	52	20	39.2	234	Masoner et al. (2019)
	France	58	160		680	Vialle et al. (2013)
Nonachlor	US	61	0.42	0.47	1.9	Masoner et al. (2019)
Oryzalin	US	100	13		318	Burant et al. (2018)
	US	100	155		1,186	Burant et al. (2018)
	US	12	35.5	36		Masoner et al. (2019)
Pentchlorophenol	US	78	435	635.3	3,500	Masoner et al. (2019)
Permethrin	US	0	<3.4		<3.4	Carpenter et al. (2016)
	US	88	16.8		66.1	Weston et al. (2009)
	US	2	30.6		30.6	Masoner et al. (2019)
Propiconazole	US	30	85.8	85.6	182	Masoner et al. (2019)
Propoxure	Australia		<10-20			Rippy et al. (2017)
Simazine	France	33	<10		150	Zgheib et al. (2012)
	US	18			6	Burant et al. (2018)
	US	54	3.6		23	Burant et al. (2018)
	US	0	<5		<5	Carpenter et al. (2016)
	Australia		8-79			Rippy et al. (2017)
	US	34	38.6	132	794	Masoner et al. (2019)
Tebuconazole	US	0	<3.7		<3.7	Carpenter et al. (2016)
	US	2	81.7	81.7	81.7	Masoner et al. (2019)
	France	42	65		70	Vialle et al. (2013)
Terbutryn	Denmark	100	52		1,840	Bollman et al. (2014)
	Australia		<10-45			Rippy et al. (2017)
Thiabendazole	US	28	8.25	108.9	1,157	Masoner et al. (2019)
Triclopyr	Australia		<10-310			Rippy et al. (2017)
Zoxamide	US	40	<3.5		28	Carpenter et al. (2016)

2,4-D – 2,4-Dichlorophenoxyacetic acid; AMPA – α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid; DDT – Dichlorodiphenyltrichloroethane; DEA - *des-ethyl atrazine;* DNOC - 4,6-Dinitro-o-cresol; MCPA – 2-methyl-4-chlorophenoxyacetic acid.

6.3 HEALTH EFFECTS OF PESTICIDES

Pesticides encompass a wide variety of chemical compounds that exhibit considerable differences in their mode of action, uptake by the body, metabolism and elimination, and toxicity to humans (WHO 1990). The adverse health effects that may result from exposure to pesticides are therefore highly variable depending on the specific compound and its formulation, as well as the dose and route of exposure, and factors including the overall health and nutritional status of the individual (WHO 1990, 2019c; Nicolopoulou-Stamati et al. 2016). It is beyond the scope of this chapter to document the health implications of exposure to individual pesticides; however, an overview of the potential health outcomes of the main classes of pesticides is shown in Table 15.

Depending on the specific pesticide and its chemical properties, as well as the route of exposure, pesticides may be absorbed through the skin, mouth, gastrointestinal tract, eyes and respiratory system (Kalyabina et al. 2021). Effects of acute exposure in humans have been documented through studies of occupational, accidental or deliberate (eg suicide) exposure, and may include central and peripheral neurotoxicity (headaches, confusion, disturbed vision, tremors, convulsions and coma), gastrointestinal symptoms (nausea, vomiting, diarrhoea), respiratory distress, irritation of skin and mucous membranes (skin rash, eye irritation), and reduced blood clotting (WHO 2019c; Kalyabina et al. 2021; Syafudrin et al. 2021). Chronic exposure to some pesticides may result in neurological disorder, endocrine disorder, reproductive dysfunction, immunotoxicity, developmental effects, cardiovascular effects, asthma, dermatitis, and injury to the liver and kidneys (WHO 1990; Jayaraj et al. 2016; WHO 2019c; Syafudrin et al. 2021; Kalyabina et al. 2021). However, the health effects of chronic exposure to many other pesticides, especially of exposure to low (ie non-occupational) doses, remains unclear or contentious due to a lack of epidemiological data (WHO 1990; Bonner and Alavania 2017). The IARC has classified as number of pesticides, mainly organochlorines, as known, possible or probable human carcinogens (Table 16).

Children are often considered to be at greater risk from pesticides, as their behaviour (eg playing in soils, hand-to-mouth behaviours) can lead to greater exposure. It is also purported that they maybe more sensitive to the effects of exposure due to their smaller size and hence greater relative exposure, differences in metabolism, and their still-developing organs[82] (WHO 2019c).

[82] https://www.epa.gov/sites/default/files/2015-12/documents/pest-impact-hsstaff.pdf Accessed 23 February 2023

Table 15. Summary of potential health effects of exposure to common classes of pesticides.

Organochlorines	Neurotoxic. Acute exposure may cause headaches, confusion, dizziness, slurred speech, nausea, vomiting, tremors, muscle weakness, sweating, paraesthesia, and in severe cases, respiratory failure. Can also cause irritation of the eyes and respiratory tract, Chronic exposure may be associated with endocrine disruption, liver and kidney damage, reproductive effects, neuromuscular and metabolic disorders.
Organophosphates	Inhibit acetylcholinesterase (AChE); acute exposure can result in headaches, vomiting, bradycardia, muscle weakness or paralysis, seizures and respiratory failure. Chronic exposure may be associated with fatigue, delayed neuropathy, behavioural change, cardiovascular disease, disrupted cellular metabolism, effects on male reproductive systems and dementia.
Pyrethroids	Comparatively low toxicity in mammals. Rapidly metabolised and excreted in urine. Act on voltage-gated sodium channels of nerve cells; acute exposure may cause numbness, itching, burning, dizziness, headaches, vomiting and nausea. Significant poisoning can cause shortness of breath, tremors and paralysis that rapidly resolve.
Carbamates	Acute toxicity of different carbamate pesticides ranges from virtually non-toxic to highly toxic (aldicarb, carbofuran). Inhibit AchE, but with lower activity than organophosphates. Acute exposure can include dizziness, nausea, diarrhoea, sweating, blurred vision. Dermal toxicity is generally low, causing slight to moderate skin or eye irritation. Chronic exposure may be associated with male reproductive disorder, and effects on the liver and kidney, and on cellular and metabolic function. Health effects from low doses are uncertain.
Triazines	Acute exposure can cause nausea and vomiting, with irritation of the mouth/skin/respiratory tract depending on route of exposure. Triazines are generally considered to be of low toxicity to humans. Chronic exposure of animals to high doses has been associated with potential reproductive- and immunotoxicity, disruption of endocrine function and cellular metabolism, and liver, kidney and heart damage.
Carbanilates	Relatively slight acute toxicity; moderately irritating to eyes. Chronic exposure may cause mild anaemia, bone marrow and haematological changes, and increased body and organ mass in animals.
Phenyl amides	Generally considered to have low acute toxicity. May be absorbed through skin, causing dermatitis. Chronic exposure in animals has shown hepatotoxicity, ocular/uveal degeneration and hemosiderosis
Azoles	Possible endocrine disruption, effects on reproductive development and liver function.

Table 15 continued.

Phenoxy alkonates	Relatively low toxicity to mammals. Acute exposures may cause tachycardia, vomiting, leukocytosis, liver and kidney distress, and at high levels, neurological effects. Little data on chronic exposure; may cause haematological alterations, effects on renal and hepatic systems, endocrine and hormonal disruption, developmental effects.
Neonicotinoids	Acute exposure to some compounds may cause neurological symptoms including uncoordinated movement, agitation, aggression and drowsiness, as well as hypotension and skin irritation. Possible link to oxidative stress, metabolic changes, liver and thyroid function and developmental effects.

Data compiled from Jayaraj et al. (2016), Nicolopoulou-Stamati et al. (2016) and IPCS Environmental Health Criteria monographs[83] and ATSDR Toxicological Profiles[84] for pesticide classes and/or representative compounds within each class.

Table 16. IARC classifications of pesticides.[85]

IARC classification	Pesticides
Class 1 – Carcinogenic to humans	γ-Hexacyclohexane (Lindane) Pentachlorophenol 1,2-Dichloropropane
Class 2a – Probably carcinogenic to humans	Dichlorodiphenyltrichloroethane (DDT) Dieldrin Diazinon Glyphosate Malathion
Class 2b – Possibly carcinogenic to humans	Chlordane Heptachlor Hexaclorobenzene Mirex Toxaphene Parathion 2,4 dichlorophenoxyacetic acid (2,4-DT)

[83] https://inchem.org/pages/ehc.html
[84] https://www.atsdr.cdc.gov/toxprofiledocs/index.html
[85] https://monographs.iarc.who.int/list-of-classifications

6.4 REGULATIONS AND GUIDELINES

Pertinent international regulations and guidelines regarding exposure to selected pesticides are summarised in Table 17. Compounds were included if at least one of the following guidelines was identified: an ATSDR minimum risk level (MRL) for oral exposure, a US EPA reference dose (RfD) for oral exposure, a WHO guideline value for drinking-water, or a US EPA maximum contaminant limit (MCL) for drinking water. In addition, the Joint FAO/WHO Meeting on Pesticide Residues (JMPR) has evaluated a large number of pesticides and established acceptable daily intakes (ADIs) and acute reference doses (ARfD) for many of these; the list of compounds is extensive, therefore JMPR ADIs or ARfDs have been included in Table 17 only where one of the other values was also identified. The full list of exposure guideline values established by JMPR has been reproduced in Appendix B.

Table 17. Recommended oral exposure limits for selected pesticides.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
1,2-dichloropropane	300 (acute) 70 (int)				40	
2,4-D	200 (int) 200 (chronic)	10	0.01		30	70
2,4,5-T		10	0.03 (<0.01 mg TCDD/kg)		9	
Acetochlor		20	0.01	1		
Acrylonitrile	100 (acute) 40 (chronic)					
Alachlor		10			20	2
Aldicarb		1	0.003	0.003	10	
Aldrin	2 (acute) 0.04 (chronic)	0.03	0.0001[a] (P)		0.03[a]	
Amitraz		2.5	0.01	0.01		
Atrazine	10 (acute) 3 (int)	35	0.02[b]	0.1 [b]	100[c]	3
Benomyl		50	0.1			
Bentazon		30	0.09	0.5	500*	
Bromomethane		1.4	1.0[d]			
Captan		130	0.1 (W)	0.3		
Carbaryl		100	0.008	0.2	50*	
Carbofuran		5	0.001	0.001	7	40
Carbosulfan		10	0.01	0.02		
Chlordane	1 (acute) 0.6 (chronic)	0.5	0.0005 (P)		0.2	2
Chlorfenvinphos	2 (acute) 0.7 (chronic)		0.0005			
Chlorobenzilate		20	0.02			
Chlorothalonil		15	0.02	0.6		
Chlorotoluron					30	
Chlorpropham		20~	0.05	0.5		

Table 17 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Chlopyrifos	3 (acute) 1 (chronic)		0.01	0.1	30	
Cyanazine					0.6	
Cyhalothrin	10 (acute) 10 (int)	5~	0.02	0.02		
Cypermethrin	20 (acute)	10~	0.02	0.04		
Cyromazine		7.5~	0.06	0.1		
DDT	0.5 (acute) 0.5 (chronic)	0.5	0.01 (P)		1.0[e]	
Demeton-S-methyl		0.005	0.0003			
Diazinon	6 (acute) 0.7 (chronic)		0.003	0.03		
Dicamba		20	0.3	0.5		
Dichlorprop					100	
Dichlorvos	4 (acute) 0.5 (chronic)	0.5	0.004	0.1	20*	
Dicofol			0.002	0.2	10*	
Dieldrin	0.1 (int) 0.05 (chronic)	0.05			0.03[a]	
Diflubenzuron		20	0.02			
Dimethipin		20~	0.02	0.2		
Dimethoate		0.2~	0.002	0.02	6	
Diquat		2.2	0.006	0.8	30*	20
Disulfoton	0.3 (acute) 0.06 (chronic)	0.05	0.0003	0.003		
Diuron		2				
Dodine		4~	0.1	0.2		
Endosulfan	7 (acute) 5 (chronic)	6	0.006	0.02	20*	
Endrin	0.6 (acute) 0.3 (chronic)	0.3	0.0002 (P)		0.6	2
Ethion	2 (acute) 0.4 (chronic)	0.5	0.002			
Fenamiphos		0.25	0.0008	0.003		
Fenitrothion			0.006	0.04	8*	
Fenoprop					9	
Flutolanil		60~	0.09			
Folpet		100~	0.1 (W)	0.2		
Fosetyl-aluminium		3,000	10			
Glufosinate ammonium		0.4~	0.01[f]	0.01[f]		
Glyphosate	1,000 (acute) 1,000 (chronic)	100	1.0[g]		900*[#]	700
Haloxyfop		0.05	0.0007	0.08		
Heptachlor	0.6 (acute) 0.1 (chronic)	0.5	0.0001 (P)		0.03*	0.4
Hexachlorobenzene	8 (acute) 0.07 (chronic)	0.8			1*	1
Hexachlorocyclo-hexane α	2 (int) 0.9 (chronic)					
Hexachlorocyclo-hexane β	80 (acute) 0.6 (int)					
Hexachlorocyclo-hexane γ (lindane)	3 (acute) 0.008 (chronic)	0.3	0.005	0.06		
Hydrogen cyanide		0.6	0.05			

Table 17 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Imazalil		13~	0.03[h]	0.05[h]		
Iprodione		40	0.06			
Isoproturon					9	
Malathion	20 (acute) 20 (chronic)	20	0.3	2	900*	
Maneb		5	0.03			
MCPA		0.5	0.1	0.6	700*	
Mecoprop					10	
Metalaxyl and metalaxyl-m		60	0.08			
Methamidophos		0.05	0.004	0.01		
Methidathion		1~	0.001	0.01		
Methomyl		25	0.02	0.02		
Methoxychlor	5 (int)	5	0.1		20	40
Methyl parathion	7 (int) 3 (chronic)	0.25			9*	
Metolachlor		150			10	
Mirex	0.3 (chronic)	0.2				
Molinate		2			6	
N,N-diethyl-meta-toluamide (DEET)	1 (int)					
Norflurazon		40	0.005	0.3		
Oxamyl		25	0.009[i]	0.009[i]		200
Paclobutrazol		13	0.1			
Paraquat		4.5	0.005	0.006		
Parathion	9 (int)		0.004	0.01	10*	
Pendimethalin		40~	0.1	1.0	20	
Pentachlorophenol	5 (acute) 5 (chronic)	5			9	1
Permethrin	300 (acute) 200 (chronic)	50	0.05	1.5	300*	
Phosmet		20	0.01	0.2		
Pirimiphos-methyl		10~	0.03	0.2		
Prochloraz		9	0.01	0.1		
Propargite		20~	0.01			
Propanil		5				
Propazine		20				
Propham		20				
Propiconazole		13~	0.07	0.3		
Propylene oxide		30	0.04	0.04		
Resmethrin		30	0.03[j]			
Simazine		5			2	4
Terbuthylazine					7	
Terbutryn		1				
Thiophanate-methyl		80~	0.09	1.0		
Thiram		5~	0.01			
Toxaphene	5 (acute) 2 (chronic)					
Trifluralin		7.5			20	
Vinclozolin		2.5~	0.01			
Zineb		50	0.03[k]			

[footnotes over-page]

* Formal guideline value not established, however a ‘health-based value’ has been determined to provide guidance when there is a reason for local concern. ^ Combined for aldrin and dieldrin. ˜ The EPA announced in 2004 that chemicals used as pesticides would not be reassessed by the IRIS Program. This entry is an archived value whose presence in the IRIS database was preserved at the request of the EPA; values were archived in 2016.

P – provisional tolerable daily intake. W – established for women of childbearing age; unnecessary for the general population unless a second value is specified.

a. Total for combined aldrin and dieldrin.

b. Group ADI and ARfD for atrazine, deethyl-atrazine, deisopropyl-atrazine and diaminochlorotriazine.

c. Group limit for atrazine and its chloro-s-triazine metabolites.

d. As bromide ion.

e. Combined for DDT and its metabolites.

f. ADI and ARfD apply to glufosinate-ammonium, n-aceteyl-gluphosinate and 3-

g. Sum of glyphosate and AMPA.

h. ADI and ARfD also apply to metabolites R061000 and R014821

i. ADI and ARfD apply to metabolites IN-A2213, IN-QKT34, IND2708 and IN-N009.

j. Estimated as bioresmethrin

k. Group ADI with maneb, metiram and zineb

7. POLYCYCLIC AROMATIC HYDROCARBONS

Polycyclic aromatic hydrocarbons (PAH) are a large group (>400) of organic compounds containing two or more fused benzene rings (Mojiri et al. 2019; WHO 2021b). They are ubiquitous environmental pollutants, generated primarily through the incomplete combustion of organic materials, and are therefore commonly associated with emissions from vehicle exhaust, power generation (eg by burning coal), industrial processes, waste incineration, domestic heating, volcanic activity and forest fires. They may also be associated with fuel and oil spills, coal tars, bitumen, as intermediaries in the production of some plastics, photographic products and pesticides, and are produced in food through some cooking processes (EFSA 2008; Jelic et al. 2015; Mojiri et al. 2019; WHO 2021b). PAH produced through combustion processes are often referred to as 'pyrogenic' and are typically higher molecular weight (commonly 4-6 ring) compounds, while those present as constituents of crude oils and their derivatives are referred to as 'petrogenic,' and are mostly low molecular weight compounds (Mojiri et al. 2019; Rocha and Palma 2019). A third group, known as 'biogenic' PAH, are formed by plants, algae and microorganisms, or during the slow chemical transformation of organic matter (Mojiri et al. 2019; Rocha and Palma 2019).

The physicochemical properties of different PAH compounds differ based on their size and structure: in general, PAH are lipophilic and show low solubility in water, with solubility decreasing with increasing molecular weight (IPCS 1998; SCF 2002; Balmer et al. 2019; Mojiri et al. 2019). Low molecular weight PAH (ie, two- and three-ring compounds) tend to be semi-volatile, although most PAH tend to adsorb onto organic particulate matter; compounds with ≥5 rings are predominantly associated with particulates (SCF 2002; Mojiri et al. 2019).

PAH are extensively detected in air, surface and groundwater, plants, sediments and soils (Mojiri et al. 2019). They are chemically stable and poorly degraded by hydrolysis, but are susceptible to oxidation and photodegradation (IPCS 1998; SCF 2002). Estimated half-lives range from hours to days in air, to months to years in soils (SCF 2002). Most organisms have a high potential to biotransform PAH, meaning parent compounds are metabolised and excreted rather than accumulated within the body, although this process is much slower in invertebrates (IPCS 1998; SCF 2002).

In 1976, the US EPA established a list of 16 PAH compounds to assess risks to human health from drinking-water, with compounds selected based on their presence in the

environment, toxicity, and availability of analytical standards (Andersson and Achten 2015). These 16 PAH, listed in Table 18, are now widely considered as 'priority PAH.' However, Andersson and Achten (2015) suggest this list needs to be updated, as other compounds (eg dibenzopyrenes, substituted PAHs and alkylated derivatives) are also known to occur in the environment, and may have similar or greater toxicities than those on the EPA list. More recently, reviews by the European Commission Scientific Committee on Food (SCF) (SCF 2002) and JECFA (JECFA 2006) identified a number of PAH as priority contaminants in food due to their potential genotoxicity and carcinogenicity in humans, half of which were common to the EPA list (Table 19). A subsequent assessment by EFSA determined that the sum of 8 PAH compounds (referred to as PAH8)[86] for which data on both oral carcinogenicity and occurrence in food exists could be used as indicators of the occurrence of PAH during dietary exposure assessments (Table 33) (EFSA 2008). Nonetheless, the EPA list of 16 PAH has become something of a *de facto* standard for environmental monitoring (Balmer et al. 2019), and much of the scientific literature regarding environmental matrices have focused on these compounds (Ingvertsen et al. 2011; Mojiri et al. 2019).

As most PAHs are not present in isolation but as complex mixtures of many different PAH compounds (IPCS 1998), their concentrations are commonly reported as the sum of the analysed congeners – often $\sum_{16}$PAH, for the 16 compounds listed on the EPA list (Jelic et al. 2015).

Table 18. List of the US EPA 16 priority PAH compounds, with their common abbreviation and number of aromatic rings also shown.

Compound	No. of rings	Compound	No. of rings
Naphthalene (NAP)	2	Benz[*a*]anthracene (BaA)	4
Acenaphthylene (ACY)	3	Chrysene (CRY)	4
Acenaphthene (ACE)	3	Benzo[*b*]fluoranthene (BbF)	5
Fluorene (FLU)	3	Benzo[*k*]fluoranthene (BkF)	5
Phenanthrene (PHE)	3	Benzo[*a*]pyrene (BaP)	5
Anthracene (ANT)	3	Dibenz[*a,h*]anthracene (DahA)	5
Fluoranthene (FLT)	4	Benzo[*g,h,i*]perylene (BghiP)	6
Pyrene (PYR)	4	Indeno([1,2,3-*cd*]pyrene (IP)	6

[86] The sum of a subset of 4 of these compounds (ie PAH4) was also found to be suitable as an indicator of PAH in food; PAH did not provided much added value when compared to PAH4 (EFSA 2008).

Table 19. List of the PAH identified as priority contaminants in foods by the EU SCF, JECFA and EFSA.

EU SCF (2002)	JECFA (2006)	EFSA (2008)
benz[*a*]anthracene*	benz[*a*]anthracene*	benzo[*a*]pyrene[#]
benzo[*b*]fluoranthene*	benzo[*b*]fluoranthene*	benz[*a*]anthracene[#]
benzo[*j*]fluoranthene	benzo[*j*]fluoranthene	benzo[*b*]fluoranthene[#]
benzo[*k*]fluoranthene*	benzo[*k*]fluoranthene*	benzo[*k*]fluoranthene
benzo[*g,h,i*]perylene*	benzo[*a*]pyrene*	benzo[*g,h,i*]perylene
benzo[*a*]pyrene*	chrysene*	chrysene[#]
chrysene*	dibenz[*a,h*]anthracene*	dibenz[*a,h*]anthracene
cyclopenta[*cd*]pyrene	dibenzo[*a,e*]pyrene	
dibenz[*a,h*]anthracene*	dibenzo[*a,h*]pyrene	
dibenzo[*a,*e]pyrene	dibenzo[*a,i*]pyrene	
dibenzo[*a,h*]pyrene	dibenzo[*a,l*]pyrene	
dibenzo[*a,i*]pyrene	indeno[1,2,3-*cd*]pyrene*	
dibenzo[*a,l*]pyrene	5-methylchrysene	
indeno[1,2,3-*cd*]pyrene*	benzo[*c*]fluorene	
5-methylchrysene		

* Denotes compound is common to the US EPA list of priority PAH. [#]PAH4 compounds. The EFSA (2008) review determined that a sub-set of four PAH could be used as indicators (ie, PAH4), providing almost the same level of information as PAH8.

7.1 POLYCYCLIC AROMATIC HYDROCARBONS IN WASTEWATER

Polycyclic aromatic hydrocarbons are common contaminants in municipal wastewaters, with total PAH concentrations ranging from several hundred ng/L to more than 10 µg/L (Pham and Proulx 1997; Busetti et al. 2006; Vogelsang et al. 2006; Fatone et al. 2011; Tian et al. 2012; Wang et al. 2013; Ozaki et al. 2015, 2019; Table 33). Concentrations of PAH tend to be higher in wastewaters where networks also receive inputs from industrial sources (Alhafez et al. 2012; Jelic et al. 2015; Mezzonnottee et al. 2015), since industrial effluents may contain up to several hundred µg/L PAH (Syafiuddin and Boopathy 2020). At the higher end of PAH concentrations, Blanchard et al. (2004) reported that combined industrial and domestic wastewaters and stormwaters entering the Seine Aval WWTP plant in Paris contained $\sum_{16}$PAH up to 28 µg/L.

Individual PAH compounds may be detected at the ng/L to low µg/L range (Table 20). Several studies report the detection of all 16 US EPA priority PAHs from wastewater samples (Pham and Proulx 1997; Manoli and Samara 1999), while others report the detection of only several compounds (Liu et al. 2017). In general, low molecular weight PAH (eg, naphthalene, phenanthrene, fluoranthene) are detected most frequently and in the highest concentrations (Blanchard et al. 2004; Fatone et al. 2011; Mezzonnette et al. 2011; Tian et al. 2012); several studies have reported two and three-ringed PAH account for 60-90% of the total PAH content of treatment plant influents (Blanchard et al. 2004; Vogelsang et al. 2006; Mezzannotte et al. 2012; Tian et al. 2012). In particular, naphthalene is commonly reported at the highest concentrations, likely due to its use in various bathroom products, cleaners, deodorants, and insecticides (eg mothballs) (Fatone et al. 2011; Mezzannottee et al. 2012; Tian et al. 2012; Wang et al. 2013; Liu et al. 2017). Among high molecular weight PAH compounds, chrysene, benzo[a]pyrene and indeno[1,2,3-cd]pyrene are most commonly detected (Pham and Proulx 1997; Blanchard et al. 2004; Tian et al. 2012; Wang et al. 2013; Jelic et al. 2015). Qiao et al. (2014) and Syafiuddin and Boopathy (2020) also highlight the presence of substituted PAHs (SPAHs) in municipal wastewaters, resulting from direct discharge and/or transformation of parent compounds. Concentrations of various classes of SPAHs, including nitrated PAHs, oxygenated PAHs and methylated PAHs, may range from several to several hundred ng/L.

Because of their lipophilic nature, PAH – especially higher molecular weight compounds – preferentially partition onto particulate matter of wastewaters. The fraction of PAH sorbed to particulates has been reported to range from 63-95% in various WWTP influents (Blanchard et al. 2004; Busetti et al. 2006; Fatone et al. 2011; Qiao et al. 2014). Thus, whilst most WWTPs are not specifically designed for the degradation of PAHs, they may be concentrated within sludge, with some low-molecular weight PAH also removed via air-stripping and volatisation (Zhang et al. 2019). However, removal rates can be highly variable between different compounds, treatment plants and processes (Manoli and Samara 2008; Mezzannotte et al. 2012; Qiao et al. 2014). For example, Vogelsang et al. (2006) reported that $\sum_{16}$PAH removal ranged from 0-12% at one treatment plant, to 94-100% at another, and that further, removal of different groups of PAH compounds (eg, 2- and 3-ringed versus 4-, 5- and 6-ringed) differed within a plant.

Table 20. Concentrations of selected PAH (ng/L) in untreated municipal wastewater reported in international studies.

	∑16 PAH	NAP	ACY	ACE	PHE	FLU	ANT	PYR	BaA	CHR	BaP	
Norway	620±340 Max. 1,340 100%	240±150 Max. 560 100%									10±7 Max. 28 85%	Vogelsang et al. (2006)
Italy		80±6	46±4	138±13	399±35	149±14	342±27	406±32	335±30	218±13	297±26	Busetti et al. (2006)
China	5,758±2,238	1,437±645	123±27	48±14	323±138	106±49	37±10	121±89	64±15	132±66	1,384±466	Wang et al. (2013)
Italy	760±570	240±230 95%	20±10 32%	140±100 56%	80±60 85%	90±70 71%	10±10 22%	30±40 44%	20±10 42%	20±20 43%	10±10 22%	Fatone et al. (2011)
Greece		2,800*			1,000*	500*	70*	220*	40*	60*	20*	Manoli and Samara (2008)
China	1,147.5 1,156.9	206±18 971±74	92.7±5 2.0±0.09	5.3±0.18 27.1±2.0	17.6±1 83.4±6.0	230.5±15 42.0±2	106.9±11 3.8±0.21	238.2±18 4.6±0.22	2.5±0.11 4.1±0.19	3.4±0.21 8.3±0.29	nd nd	Tian et al. (2012)[#]
France	27,783*					77.4* Max. 400					21.4* Max. 104	Blanchard et al. (2004)
China	657±81	435±68	24±6	41±9		55±8		28±8	48±3	23±2		Liu et al. (2017)
Romania		27.2*		6.4*	<LOD*	<LOD*	1.6*	<LOD*	6.0*	30.8*	<LOD*	Alhafez et al. (2012)

Naphthalene (NAP), acenaphthylene (ACY), acenaphthene (ACE), fluorene (FLU), phenanthrene (PHE), anthracene (ANT), fluoranthene (FLT), pyrene (PYR), benzo(a)anthracene (BaA), chrysene (CHR), benzo(a)pyrene (BaP). Data are shown as mean (± standard deviation, where available) unless otherwise indicated, with the maximum concentration and detection frequency indicated where this data was available.
nd – not detected. *data shown as median. [#] data was presented separately for two different sampling events.

7.2 POLYCYCLIC AROMATIC HYDROCARBONS IN STORMWATER

Urban stormwaters are commonly contaminated with a wide variety of PAH from a range of sources. Atmospheric deposition of pyrogenic PAH associated with vehicle exhausts, solid waste incineration, power generation, and industrial emissions onto roads, other paved surfaces and roofs is a major source of PAH to urban stormwaters (Stout et al. 2004; Ingversten et al. 2011). Additional sources of PAH to stormwaters include the erosion or abrasion of asphalts, bitumen and tyres; leaching from coal tars used in roofing products or parking lot sealants; leaching from creosote-impregnated timbers; and vehicle drip loss and spilt oil and petroleum (Watts et al. 2010; Ingvertsen et al. 2011; Lorenzi et al. 2011; Mahler et al. 2012). In addition to PAH accumulated on surfaces, rainfall itself may be a source of PAH, as it washes both soluble and particle-associated compounds from the atmosphere (Olivella et al. 2006; Zhang et al. 2008). The majority of PAH in stormwaters tend to be associated with suspended particulate matter, with the dissolved concentrations typically being low (Herngren et al. 2010; Gasperi et al. 2012; Kennedy et al. 2016). PAH are therefore readily mobilised during the first flush; for example, Stein et al. (2006) estimated up to 60% of PAH load was discharged in the first 20% of stormwater volume during a series of studies in Los Angeles.

Total concentrations of PAH in stormwater typically range from tens of ng/L to low µg/L (Ngabe et al. 2000; Menzie et al. 2002; Stout et al. 2004; Hwang and Foster 2006; Stein et al. 2006; Zhang et al. 2008; Ingvertsen et al. 2011; Gasperi et al. 2012; Zgheib et al. 2012; Al-Mashagbeh et al. 2021), although higher concentrations are occasionally reported, with Masoner et al. (2019) reporting up to 180 µg/L in a survey of stormwaters across the United States of America, and Watts et al. (2010) reporting concentrations up to 5,890 µg/L in stormwater collected from freshly-sealed parking lots. Concentrations tend to be higher in samples from industrial or commercial catchments, or highly trafficked areas (Brown and Peake 2006; Stein et al. 2006; Herngren et al. 2010; Al-Mashagbeh et al. 2021). Total PAH load may also increase during winter due to increased use of residential heating and cold vehicle engines (Rocher et al. 2004), and with increasing antecedence dry period (Stein et al. 2006).

The profile of PAH in urban stormwaters and road dusts is often dominated by high-molecular weight (eg 4-6 ring) compounds, consistent with pyrogenic sources. Among the most commonly reported PAH in stormwater are phenanthrene, fluoranthene, pyrene, chrysene, benzo(a)pyrene and benzo(b)fluoranthene (Ngabe et al. 2000; Menzie et al. 2002; Rocher et al. 2004; Stout et al. 2004; Hwang and Foster 2006; Stein et al. 2006; Brown and Peake 2006; Ingversten et al. 2011; Lorenzi et al. 2011; Zgheib et al. 2012). Indicative

concentrations for these compounds are shown in Table 21. However, several authors have reported detecting all 16 priority PAH in stormwater samples and suspended sediments (Brown and Peake 2006; Gasperi et al. 2012; Zgheib et al. 2012); for example, Masoner et al. (2019) reported that in stormwater samples collected across the United States, all 16 priority PAH were detected, of which six (fluoranthene, pyrene, phenanthrene, benzo[b]fluoranthene, chrysene and benzo[a]pyrene) had maximum concentrations exceeding 10 µg/L.

Table 21. Concentrations of selected PAH (ng/L) in urban stormwater reported in international studies.

	∑16 PAH	NAP	PHE	FLU	ANT	FLA	PYR	CHR	BbF	BghiP	IP	BaP	
USA	Max 180,000	50 Max 821 61%	994 Max 28,800 86%	110 Max 1,190 53%	165 Max 5,860 61%	1,590 Max 36,700 90%	1,250 Max 29,100 90%	1,255 Max 14,700 82%	1,825 Max 18,500 82%	704 Max 4,970 71%	854 Max 6,230 67%	766 Max 13,500 80%	Masoner et al. (2019)
USA	1,510-12,500	18-59	26-338	8.8-152	5.4-119	87-1,380	66-774	49-519	58-733	15-548	37-505	28-466	Hwang and Foster (2006)
France	1,327 Max 6,477	82 Max 490 100%	140 Max 726 100%	28 Max 106 100%	23 Max 104 100%	134 Max 945 100%	177 3,254 100%	104 Max 655 100%	134 Max 656 100%	100 Max 569 100%	80 Max 354 100%	66 Max 315 100%	Zgheib et al. (2012)
China	548[d] 3,872[p]	38.5[d] 66.0[p]	113[d] 411[p]	25.8[d] 40.2[p]	24.3[d] 45.1[p]	137[d] 904[p]	86.2[d] 632[p]	29.6[d] 441[p]	15.1[d] 202[p]	1.7[d] 83.8[p]	2.0[d] 45.1[p]	4.5[d] 174[p]	Zhang et al. (2008)
Jordan	2,220	10 Max 20	60 Max 230	110 Max 360	190 Max 530	240 Max 530	130 Max 330	40 Max 90	130 Max 290	210 Max 790	500 Max 1,480	410 Max 1,200	Al-Mashaqbeh et al. (2021)

Naphthalene (NAP), fluorene (FLU), phenanthrene (PHE), anthracene (ANT), fluoranthene (FLT), pyrene (PYR), benzo(a)anthracene (BaA), chrysene (CHR), benzo[b]benzo(a)pyrene (BaP). Data are shown as median unless otherwise indicated, with the maximum concentration and detection frequency indicated where this data was available. nd – not detected. *data shown as mean (± standard deviation, where available). d – dissolved phase, p – particulate phase.

7.3 HEALTH EFFECTS OF POLYCYCLIC AROMATIC HYDROCARBONS

The health effects of PAH exposure depend on the specific PAH(s), the length, dose and route of exposure, and overall health of the individual (Mallah et al. 2022). However, little information on human exposure to single PAH compounds exists; the complex profile of PAH in the environment means people are typically exposed to mixtures (ATSDR 1995; IPCS 1998), with data for individual compounds limited to accidental exposures (eg, naphthalene poisoning in children) and several volunteer studies assessing dermal exposure. In addition, epidemiological studies are often in occupational settings, focus on inhalation exposure, and are largely limited to carcinogenic potential (IPCS 1998; SCF 2002; WHO 2022). There is almost no published data on the human health effects of oral exposure to PAH (SCF 2002; WHO 2021b).

PAH are absorbed across the lung, gut and skin of mammals, and are widely distributed throughout the body, although they tend to concentrate in lipid-rich organs including the kidneys and liver and in adipose tissue (ATSDR 1995). The metabolism of PAH is complex, with parent compounds converted to chemically reactive intermediates and derivatives including phenols, diols and nitro-PAH; in many instances, it is these metabolites rather than the parent compound that are harmful (ATSDR 1995; IPCS 1998; WHO 2010a). Metabolites are typically excreted via urine and faeces within several days (ATSDR 1995; WHO 2010a).

PAH are generally considered to have a low degree of acute toxicity to humans (WHO 2010a)[87]. Several PAH, including anthracene, benzo[a]pyrene, fluoranthene and phenanthrene have been shown to induce skin irritation and sensitisation (WHO 2010a), and eye irritation, nausea and vomiting are reported in acute occupational exposure (Mallah et al. 2022). Accidental ingestion of naphthalene has been associated with acute haemolytic anaemia (IPCS 1998). Chronic health effects may include cardiovascular and respiratory disease, cataracts, low birth weights, kidney and liver damage (WHO 2010a; Mallah et al. 2022).[88,89] Animal and cell culture studies have indicated that several PAH are genotoxic and can cause adverse effects on immune, hematopoietic, cardiovascular and reproductive systems (ATSDR 1995; SCF 2002; WHO 2010a). The most significant endpoint for PAH toxicity is cancer; increased incidence of lung, skin and bladder cancers may be associated

[87] https://www.atsdr.cdc.gov/csem/polycyclic-aromatic-hydrocarbons/health_effects.html Accessed 10 May 2022

[88] https://www.sahealth.sa.gov.au/wps/wcm/connect/public+content/sa+health+internet/conditions/chemicals+and+contaminants/polycyclic+aromatic+hydrocarbons+pahs Accessed 10 May 2022

[89] https://www.atsdr.cdc.gov/csem/polycyclic-aromatic-hydrocarbons/health_effects.html Accessed 10 May 2022

with occupational exposure to PAH (WHO 2010a).[90] The IARC has classified one PAH as a human carcinogen (benzo[*a*]pyrene), one as a probable human carcinogen (dibenz[*a,h*]anthracene) and six as possible human carcinogens (chrysene, benzo[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, indeno[1,2,3-*cd*]pyrene and naphthalene)[91].

7.4 REGULATIONS AND GUIDELINES

Pertinent guidelines and exposure limits relating to PAH are detailed in Table 22. Due to their suspected genotoxicity and carcinogenicity, tolerable intakes of PAH have not been set by JECFA,[92] EFSA or FSANZ.[93] A benchmark dose (lower confidence limit, $BMDL_{10}$) equivalent to 100 µg of benzo[a]pyrene per kg body weight per day was derived by JECFA for PAH in food, based on a study of carcinogenicity of orally-administered PAH mixtures in mice (JECFA 2006).

[90] https://www.atsdr.cdc.gov/csem/polycyclic-aromatic-hydrocarbons/health_effects.html accessed 10 ay 2022
[91] https://monographs.iarc.who.int/list-of-classifications Accessed 10 May 2022

[92] https://apps.who.int/food-additives-contaminants-jecfa-database/Home/Chemical/1155 Accessed 21 July 2022
[93] https://www.foodstandards.gov.au/science/surveillance/documents/PAH%20Survey%20for%20website.pdf Accessed 11 July 2022

Table 22. Recommended oral exposure limits for PAH.

	NAP	ACE	FLU	ANT	FLT	PYR	B[*a*]P
Reference Dose, US EPA[94] (µg/kg/day)	20	60	40	300	40	30	0.3
Minimum Risk Level, ATSDR[95] (µg/kg bw/day)	600 (acute) 600 (int.)	600 (int.)	0.4 (int.)	10,000 (int.)	400 (int.)		
Drinking-water Guideline, WHO[96] (µg/L)							0.7
Drinking-water Maximum Contaminant Limit, US EPA[97] (µg/L)							0.2

US EPA Reference doses and ATSDR Minimum Risk Levels are for oral exposures NAP - naphthalene; ACE – acenaphthene; FLU – fluorene; ANT – anthracene; FLT – fluoranthene; PYR – pyrene; B[a]P – benzo(a)pyrene. int. – intermediate duration exposure.

[94] https://iris.epa.gov/AtoZ/?list_type=alpha Accessed 14 July 2022
[95] https://wwwn.cdc.gov/TSP/MRLS/mrlsListing.aspx Accessed 14 July 2022
[96] WHO (2022). Guidelines for Drinking-water Quality: Fourth Edition Incorporating the First and Second Addenda.
[97] https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations Accessed 14 July 2022

8. PHARMACEUTICALS AND PERSONAL CARE PRODUCTS

Pharmaceuticals are a large and diverse group of synthetic or natural chemicals that can be found in prescription, over-the-counter and veterinary medicines, that are designed to diagnose, treat or prevent various health conditions. Some definitions may extend to include illicit drugs and 'lifestyle stimulants' including caffeine and nicotine. Pharmaceuticals contain active ingredients that are designed to interact with various biological receptors and systems, or to be toxic to various organisms (eg bacteria, viruses, fungi, parasites) (Khetan and Collins 2007; WHO 2012; Adeleye et al. 2022). Personal care products and cosmetics are products that are applied topically to the body (eg, skin, hair, nails and oral cavity). These include soaps and shampoos, moisturisers and lotions, toothpastes, sunscreens, antiperspirant deodorants, fragrances, insect repellents, sanitisers, make-up, and hair dyes (Daughton and Ternes 1999).

Pharmaceuticals and personal care products (PPCPs) are one of the largest groups of emerging contaminants (Reyes et al. 2021). They are widely used and often in large amounts, and are increasingly being reported as environmental contaminants. Trace levels of PPCPs (ng/L to tens of µg/L range) have been widely reported in wastewater, surface water, groundwater and drinking water (Kolpin et al. 2002; Boyd et al. 2003, 2004; Kasprzyk-Hordern et al. 2009; WHO 2012; aus der Beek et al. 2016; Adeleye et al. 2022). For example, in 1999 a national US study found PPCP-related compounds in at least 40% of 139 streams sampled (Kolpin et al. 2002), and several reviews have reported more than 580 unique compounds being detected in environmental water samples (surface water, drinking water, groundwater, wastewater) globally (aus der Beek et al. 2016; Reyes et al. 2021). Whilst some of these compounds undergo relatively rapid attenuation in the environment, others (eg clofibric acid, x-ray contast, musks) are highly persistent (Daughton and Ternes 1999; Khetan and Collin 2007). Nonetheless, the ubiquitous use of many PPCPs has resulted in a continuous discharge of these compounds to the environment, meaning that even compounds showing rapid environmental degradation may exhibit a "pseudo-persistence" (Daughton and Ternes 1999; WHO 2012; Sengar and Vijayanandan 2022). Together with the fact that pharmaceuticals in particular are intended to cause biological effects, concerns have been raised regarding the potential ecological and human health impacts of unintended exposure to these compounds (Kumar et al. 2010). However, whilst significant efforts have focused on assessing the environmental and ecological impacts of

biologically active PPCPs in the environment (see reviews by Khetan and Collins 2007; Orias and Perrodin 2013), the human health risks remain less clear.

Table 23 lists some of the major PPCP functional classes and common examples of specific compounds. Note that compounds that are primarily associated with non-PPCP consumer or industrial uses, such as BPA, phthalates and nonylphenols have been included in Chapter 8.

Table 23. Major classes of PPCPs and representative compounds.

Compound group	Function	Example compounds
Pharmaceuticals		
Analgesics and anti-inflammatory	Reduce pain and inflammation	Acetaminophen (paracetamol), naproxen, acetylsalicylic acid (aspirin), ketoprofen, ibuprofen, diclofenac, codeine
Antibiotics	Kill bacteria	Trimethoprim, erythromycin, sulfamethoxazole, chloramphenicol, amoxycillin, ofloxacin, chlortetracycline, oxytetracycline, streptomycin, ciprofloxacin, penicillin, flumequine, ampicillin, doxycycline
Anticonvulsants	Treat epilepsy, mood disorder and nerve pain	Carbamazepine, primidone, dilantin, phenobarbital, gabapentin
Antihistamine	Reduce severity and symptoms of allergic reaction	Cetirizine, diphenylhydramine, fexofenadine hydroxyzine, loratadine, ranitidine
Beta-blockers	Inhibit adrenaline and noradrenaline	Metoprolol, propranolol, nadolol, timolol, sotalol, atenolol
Cancer therapies	Control or kill neoplastic cells	Cyclophosphamide, ifosphamide, epirubicin, methotrexate, tamoxifen
Lipid regulators	Regulate cholesterol and triglycerides in blood	Clorfibric acid, bezafibrate, fenofibrate, gemfibrozil, simvastatin
Psychiatric drugs	Improve mood, relieve anxiety or psychoses	Fluoxetine, diazepam, imipramine, meprobamate, oxazepam, thioridazne
Steroids and hormones	Regulate metabolism; control sexual development and function; homeostasis	17-α-ethynylestradiol; 17-β-estradiol, estrone, estriol, testosterone
X-ray contrasts	Enhance x-ray imaging	Ipromide, ipramidol, diatrizoate
Other		Furosemide, salbutamol
Personal care products		
Antiseptics/ disinfectants	Kill unwanted germs and parasites	Triclosan, triclocarban, chloroprene, 2-phenylphenol, 4-chloroxylenol
Fragrances	Create pleasant odour	Galaxolide, tonalide, musk xylene, musk ketone
Insect repellents	Repel insects	N,N-diethyl-m-toluamide (DEET)
Parabens and phenols	Preservatives, prevent microbial growth or undesirable chemical changes	2-phenoxyethanol, methyl paraben, ethyl 4-hydroxybenzoate, propyl 4-hydroxybenzoate, butyl 4-hydroxybenzoate
UV filters	Protect skin or other products from sunburn and UV damage	Benzophenone, methylbenzylidene, octocrylene, ethylhexyl methoxycinnamate

Adapted from Nikolaou et al. (2007), Wang and Wang (2016) and Adeleye et al. (2022).

8.1 PHARMACEUTICALS AND PERSONAL CARE PRODUCTS IN WASTEWATER

Municipal wastewaters are a significant source environmental contamination with pharmaceuticals and personal care products (Nikolaou et al. 2007; WHO 2012). Following ingestion, pharmaceuticals (as parent compounds and/or metabolites[98]) are excreted in the urine and/or faeces, and therefore into the wastewater network (Khetan and Collins 2007; Reyes et al. 2021); patient excretion is considered the primary source of pharmaceuticals in wastewater, and subsequently, the environment (Adeleye et al. 2022). Improper disposal of unwanted or expired pharmaceuticals may also occur (eg, pouring down the sink, flushing down the toilet), but is considered to be a minor pathway (Castiglioni et al. 2006; Khetan and Collins 2007; Reyes et al. 2021). Effluents from pharmaceutical manufacturing, hospitals, veterinary clinics and aged care facilities may also be important locally, although many of these facilities will undertake a degree of wastewater treatment prior to discharge to the municipal network (aus der Beek et al. 2016; Frascaroli et al. 2021; Adeleye et al. 2022). Personal care products applied topically are readily washed into the network during showering or bathing (Reyes et al. 2021). Several reviews provide further information on the presence and fate of PPCPs in wastewater and environmental waters (Nickolaou et al. 2007; aud der Beek et al. 2016; Wang and Wang 2016; Tran et al. 2019; Reyes et al. 2021; Adeleye et al. 2022).

Because of the diversity of PPCPs, only a small percentage of all available compounds have been assessed for their presence in wastewater; a summary of the most prevalent compounds is shown in Table 24. Among those most commonly reported are pharmaceuticals with the highest consumption rates including analgesics, antibiotics, psychiatric drugs, blood lipid regulators and beta-blockers, especially acetaminophen, diclofenac, ibuprofen, naproxen, carbamazepine, gemfibrozil, sulfamethoxazole, ciprofloxacin and atenolol. The stimulants caffeine and nicotine are also prevalent where these are included in study definitions. Pharmaceuticals used to treat less prevalent conditions (eg cancer therapies) or that are consumed at lower doses (eg contraceptive hormones) tend to be detected less frequently and at much lower concentrations. Reported concentrations for various PPCPs in municipal wastewater range from <1 ng/L to more tens of µg/L, and in rare instances up to 100 µg/L (Castiglioni et al. 2006; Terzic et al. 2008; Kasprzyk-Hordern et al. 2009; Morasch et al. 2010; Martin Ruel et al. 2011; Margot et al. 2015; Yu et al. 2013; Korgmaa et al. 2020; Reyes et al. 2021).

[98] Depending on the specific compound, up to 95% of the administered dose can be excreted unmetabolized (Castiglioni et al. 2006).

Geographic variation in the presence of PPCPs is driven by regional patterns in the use of different pharmaceuticals and/or personal care products by the connected population (Terzic et al. 2008), as well as catchment-specific factors including population size and density, infrastructure condition, and climatic conditions (Tran et al. 2019; dos Santos et al. 2021). Localised seasonal variation in PPCP concentrations may also be observed: for example, pharmaceuticals used in the management of chronic conditions are likely to be comparable throughout the year, while wastewater concentrations of several antibiotics, analgesics and blood lipid regulators are reported to peak in winter, associated with increased prevalence of seasonal cold/flu and related ailments, and increased blood lipid content (Castiglioni et al. 2006; Yu et al. 2013). Roberts et al. (2016) reported an increased in the concentration of over-the-counter antihistamines, reflecting increased prevalence of seasonal allergic rhinitis (ie hayfever).

Wastewater treatment plants are not designed to treat or remove PPCP compounds and a substantial amount pass through to receiving environments (Luo et al. 2014; Margot et al. 2015; Reyes et al. 2021; Adeleye et al. 2022). However, PPCPs may be removed to varying degrees depending on various physicochemical properties of the specific compound, treatment processes (eg primary, secondary, tertiary, and specific technologies), operational parameters including retention time, and characteristics of the wastewater including pH and temperature (Castiglioni et al. 2006; Kasprzyk-Hordern et al. 2009; Sun et al. 2014; Wang and Wang 2016; Adeleye et al. 2022; Sengar and Vijayanandan 2022). Several reviews have highlighted that removal efficiencies for different PPCP-related compounds may range from 0 to 100%, with compounds including erythromycin and carbamazepine being poorly removed, and others such as ibuprofen and acetaminophen being almost completely removed (Castiglioni et al. 2006; WHO 2012; Luo et al. 2014; Margot et al. 2015; Wang and Wang 2016; Reyes et al. 2021). In addition, negative removal may also be observed, attributed to the transformation of conjugates to parent compounds, desorption from sludge and release of PPCPs from faecal matter as it is degraded by microorganisms (Wang and Wang 2016; Reyes et al, 2021).

Table 24. Concentrations of PPCPs (ng/L) in untreated municipal wastewater reported in international studies.

Compound	Detection frequency (%)	Concentration (ng/L)			Country	Reference
		Mean	Median	Maximum		
Analgesics/ anti-inflammatory						
Acetaminophen	100		77,7721	128,790	Singapore	Tran et al. (2019)
		7,460		10,234	South Korea	Behera et al. (2011)
		9,900		11,400	Spain	Radjenovic et al. (2009)
	100	194,748			UK	Kasprzyk-Hordern et al. (2009)
		380			France	Martin Ruel et al. (2011)
Acetylsalicylic acid	100		1,577	5,448	UK	Kasprzyk-Hordern et al. (2009)
			180		France	Martin Ruel et al. (2011)
Buprenorphine	6		<LOD	259	EU-wide	Loos et al. (2013)
Codeine	100	8,637		32,298	UK	Kasprzyk-Hordern et al. (2009)
	98		20.9	826	EU-wide	Loos et al. (2013)
	100	531		1,552	Spain	Teijon et al. (2010)
Diclofenac	100	164.5		1,161	UK	Kasprzyk-Hordern et al. (2009)
	89		43.3	174	EU-wide	Loos et al. (2013)
		1,320		1,600	Spain	Radjenovic et al. (2009)
	100		294	1,380	Singapore	Tran et al. (2019)
	100	1,868		4,549	Spain	Teijon et al. (2010)
		630			France	Martin Ruel et al. (2011)
		131		243	South Korea	Behera et al. (2011)
	100	859		4,200	Balkans	Terzic et al. (2008)
Fentanyl	6	0.1		1.6	EU-wide	Loos et al. (2013)
Ibuprofen	100		1,988	6,328	UK	Kasprzyk-Hordern et al. (2009)
	0		<LOQ	<LOQ	US	Boyd et al. (2003)
	57		7	2,219	EU-wide	Loos et al. (2013)
		21,700		31,300	Spain	Radjenivic et al. (2009)
	25		<LOQ	55,975	Singapore	Tran et al. (2019)
	25	24		24	Spain	Teijon et al. (2010)
		2,500			France	Martin Ruel et al. (2011)
		2,265		2,853	South Korea	Behera et al. (2011)
	96		3,200	11,900	Balkans	Terzic et al. (2008)
Indomethacin		875		1,000	Spain	Radjenovic et al. (2009)
	25		<LOQ	499	Singapore	Tran et al. (2019)
	13		177	240	Balkans	Terzic et al. (2008)
Ketoprofen	81	75.5		346	UK	Kasprzyk-Hordern et al. (2009)
	48		0	1,653	EU-wide	Loos et al. (2013)
		1,080		1,200	Spain	Radjenovic et al. (2009)
		640			France	Martin Ruel et al. (2011)
		202		286	South Korea	Behera et a. (2011)
	79	561		1,520	Balkans	Terzic et al. (2008)
Mefenamic acid	88	106.5		1,269	UK	Kasprzyk-Hordern et al. (2009)
		1,070		1,200	Spain	Radjenovic et al. (2009)
		121		328	South Korea	Behera et al. (2011)
	38	53		120	Balkans	Terzic et al. (2008)
Naproxen	100	1,005		3,504	UK	Kasprzyk-Hordern et al. (2009)
	100		935	106	US	Boyd et al. (2003)
	66		8.2	958	EU-wide	Loos et al. (2013)
		463		670	Spain	Radjenovic et al. (2009)
	100		2,670	13,676	Singapore	Tran et al. (2019)
	75	421		688	Spain	Teijon et al. (2010)
		60			France	Martin Ruel et al. (2011)
		2,584		5,033	South Korea	Behera et al. (2011)
	92		335	1,550	Balkans	Terzic et al. (2008)

Table 24 continued.

Compound	Detection frequency (%)	Concentration (ng/L)			Country	Reference
		Mean	Median	Max		
Salicylic acid	100	9,256		32,082	UK	Kasprzyk-Hordern et al. (2009)
	100		7,631	74,467	Singapore	Tran et al. (2019)
Tramadol	100	42,619		89,026	UK	Kasprzyk-Hordern et al. (2009)
	100		218	1,166	EU-wide	Loos et al. (2013)
Antibiotics						
Azithromycin	100	456		1,140	Balkans	Terzic et al. (2008)
Chloramphenicol	66.5	131		452	UK	Kasprzyk-Hordern et al. (2009)
Ciprofloxacin	88	405		2,610	Balkans	Terzic et al. (2008)
	90		82.1	264	EU-wide	Loos et al. (2013)
Enrofloxacin	33	12		18	Balkans	Terzic et al. (2008)
Erythromycin		820		2,700	Spain	Radjenovic et al. (2009)
	100	2,069		10,025	UK	Kasprzyk-Hordern et al. (2009)
	100		134	420	Balkans	Terzic et al. (2008)
Norfloxacin	96	976		2,940	Balkans	Terzic et al. (2008)
Ofloxacin	0		<LOQ	<LOQ	Balkans	Terzic et al. (2008)
	100	357		464	Spain	Teijon et al. (2010)
		10,500		31,700	Spain	Radjenovic et al. (2009)
Sulfamethoxazole	84	72		274	UK	Kasprzyk-Hordern et al. (2009)
		93		1,300	Spain	Radjenovic et al. (2009)
	100	332		451	Spain	Teijon et al. (2010)
		180			France	Martin Ruel et al. (2011)
		120		216	South Korea	Bahera et al. (2011)
	100	1,180		11,600	Balkans	Terzic et al. (2008)
	83		164.1	1,691	EU-wide	Loos et al. (2013)
Sulfapyridine	100	2,942		12,397	UK	Kasprzyk-Hordern et al. (2009)
	92		339	931	Balkans	Terzic et al. (2008)
Trimethoprim	100	2,559	178	6,796	UK	Kasprzyk-Hordern et al. (2009)
	93			800	EU-wide	Loos et al. (2013)
		204		430	Spain	Radjenovic et al. (2009)
		205		277	South Korea	Behera et al. (2011)
	100	781		2,550	Balkans	Terzic et al. (2008)
Anticonvulsant						
Carbamazepine	100	1,322		3,110	UK	Kasprzyk-Hordern et al. (2009)
		156		220	Spain	Radjenovic et al. (2009)
		640			France	Martin Ruel et al. (2011)
		72		127	South Korea	Behera et al. (2011)
	100		368	913	Singapore	Tran et al. (2019)
	100	250		284	Spain	Teijon et al. (2010)
	100	419		1,550	Balkans	Terzic et al. (2008)
	90		752	4,609	EU-wide	Loos et al. (2013)
Gabapentin	100	16,480		37,426	UK	Kasprzyk-Hordern et al. (2009)
	100		4,616	15,359	Singapore	Tran et al. (2019)
Antidepressants and other psychiatric drugs						
Alprazolam	8		0	33	EU-wide	Loos et al. (2013)
		10			France	Martin Ruel et al. (2011)
Amitriptyline	100	1,670		6,711	UK	Kasprzyk-Hordern et al. (2009)
	2	0.3		14.6	EU-wide	Loos et al. (2013)
		30			Balkans	Terzic et al. (2008)
Bupropion	91		0.6	4.9	EU-wide	Loos et al. (2013)
Fluoxetine	0		<LOQ	<LOQ	US	Boyd et al. (2003)
	22		<LOQ	21.5	EU-wide	Loos et al. (2013)
		53		2,300	Spain	Radjenovic et al. (2009)
		10			France	Martin Ruel et al. (2011)

Table 24 continued.

Compound	Detection frequency (%)	Concentration (ng/L)			Country	Reference
		Mean	Median	Max		
Oxazepam	90		64.3	1,766	EU-wide	Loos et al. (2013)
Risperidone	100		3.3	85.8	EU-wide	Loos et al. (2013)
Sulpiride	100		376	791	Balkans	Terzic et al. (2008)
Venlafazine	99		97	548	EU-wide	Loos et al. (2013)
Antidiabetics						
Glibenclamide	2		<LOQ	28	EU-wide	Loos et al. (2013)
		9,890		15,900	Spain	Radjenovic et al. (2009)
Repaglinide	98		2.1	12.3	EU-wide	Loos et al. (2013)
Antihistamines						
Diphenylhydramine	98		4.9	142	EU-wide	Loos et al. (2013)
Famotidine		80		140	Spain	Radjenovic et al. (2009)
	13	59		120	Balkans	Terzic et al. (2008)
Fexofenadine	80		58.8	1,287	EU-wide	Loos et al. (2013)
Ranitidine	80	3,397		11,664	UK	Kasprzyk-Hordern et al. (2009)
	42		<LOQ	43.6	EU-wide	Loos et al. (2013)
			347	540	Spain	Radjenovic et al. (2009)
	13		253	758	Balkans	Terzic et al. (2008)
Beta blocker						
Atenolol	100	13,568		33,106	UK	Kasprzyk-Hordern et al. (2009)
		2,000		2,800	Spain	Radjenovic et al. (2001)
	100		2,363	9,267	Singapore	Tran et al. (2019)
	100		1,445	536	Spain	Teijon et al. (2010)
			390		France	Martin Ruel et al. (2011)
			7,801	11,239	South Korea	Behera et al. (2011)
	96		1,880	7,560	Balkans	Terzic et al. (2008)
Bisoprolol	97		15.7	423	EU-wide	Loos et al. (2013)
		70			France	Martin-Ruel et al. (2011)
Metoprolol	100	845		146	UK	Kasprzyk-Hordern et al. (2009)
		39		63	Spain	Radjenovic et al. (2009)
		30			France	Martin Ruel et al. (2011)
		4		6	South Korea	Behera et al. (2011)
	21	953		4,683	Balkans	Terzic et al. (2008)
Sotalol		509		850	Spain	Radjenovic et al. (2009)
		55			France	Martin Ruel et al. (2011)
	54	221		1,080	Balkans	Terzic et al. (2008)
Propanolol	100	598		1,962	UK	Kasprzyk-Hordern et al. (2009)
		160			France	Martin Ruel et al. (2011)
	21	132		255	Balkans	Terzic et al. (2008)
		292		1,130	Spain	Radjenovic et al. (2009)
Lipid regulator						
Bezafibrate		14,900		29,800	Spain	Radjenovic et al. (2009)
	100	510		1,391	UK	Kasprzyk-Hordern et al. (2009)
	63		3.5	343	EU-wide	Loos et al. (2013)
	29		65	260	Balkans	Terzic et al. (2008)
Clofibric acid	35	10		57	IK	Kasprzyk-Hordern et al. 2009)
	0		<LOD	<LOD	US	Boyd et al. (2003)
	26		5.3	127	EU-wide	Loos et al. (2013)
	0		<LOD		Singapore	Tran et al. (2019)
		28		65	South Korea	Behera et al. (2011)
	29	57		110	Balkans	Terzic et al. (2008)

Table 24 continued.

Compound	Detection frequency (%)	Concentration (ng/L)			Country	Reference
		Mean	Median	Max		
Gemfibrozil		222		318	South Korea	Behera et al. (2011)
	60		4.9	3,619	EU-wide	Loos et al. (2013)
		3,080		5,900	Spain	Radjenovic et al. (2009)
	100		252	415	Singapore	Tran et al. (2019)
	100	1,736		5,714	Spain	Teijon et al. (2010)
		1,500			France	Martin Ruel et al. (2011)
	96	377		1,700	Balkans	Terzic et al. (2008)
Fenofibrate	7	1.1		25.7	EU-wide	Loos et a. (2013)
Simvastatin	15	115		798	UK	Kasprzyk-Hordern et al. (2009)
Diuretics						
Furosemide	100	2,133		6,022	UK	Kasprzyk-Hordern et al. (2009)
	100		1,021	1,914	Spain	Teijon et al. (2010)
Hydrochlorothiazide	100	5.79		9,511	Spain	Teijon et al. (2010)
		2,740		4,800	Spain	Radjenovic et al. (2009)
Illicit drugs						
Amphetamine	100	2,753		12,020	UK	Kasprzyk-Hordern et al. (2009)
Cocaine	100	364		1,837	UK	Kasprzyk-Hordern et al. (2009)
Hormones and steroids						
Estradiol (E2)		4		4	Spain	Behera et al (2011)
Estriol (E3)	0		<LOQ	<LOQ	Singapore	Tran et al. (2019)
		415		802	South Korea	Behera et al. (2011)
	0		<LOQ	<LOQ	France	Martin Ruel et al. (2011)
Estrone (E1)	0		<LOQ	<LOQ	US	Boyd et al. (2003)
			<LOQ	<LOQ	Singapore	Tran et al. (2019)
		47		70	South Korea	Behera et al. (2011)
		30			France	Martin Ruel et al. (2011)
Stimulants						
Caffeine	93		34.6	3,002	EU-wide	Loos et al. (2013)
	100		35,261	178,354	Singapore	Tran et al. (2019)
	100	327		609	Spain	Trijon et al. (2010)
		660			France	Martin Ruel et al. (2011)
		2,349		3,217	South Korea	Behera et al. (2011)
Nicotine	100	895		3,249	Spain	Teijon et a. (2010)
X-ray contrasts						
Amidotrizoic acid	47	619		8,400	EU-wide	Loos et al. (2013)
Gadolinium	100		58.4	789	UK	Loos et al. (2013)
Iohexol	18		<LOQ	7,0007	EU-wide	Loos et al. (2013)
	100		27,416	132,244	Singapore	Tran et al. (2019)
Iopamidol	15		<LOQ	6,100	EU-wide	Loos et al. (2013)
	100		2,055	45,611	Singapore	Tran et al. (2019)
Antiseptics/disinfectants						
Chlorophene	100	73.5		258	UK	Kasprzyk-Hordern et al. (2009)
	0		<0.1	<0.1	US	Boyd et al. (2003)
p-benzylphenol	48	246		1,111	UK	Kasprzyk-Hordern et al. (2009)
Triclocarban	100		340	6,150	Singapore	Tran et al. (2019)
Triclosan	93	250		463	UK	Kasprzyk-Hordern et al. (2009)
	100	1,000		21	US	Boyd et al. (2003)
	41	42,619		4,259	EU-wide	Loos et al. (2013)
	100		218	3,259	Singapore	Tran et al. (2019)
				785	South Korea	Behera et al. (2011)

Table 24 continued.

Compound	Detection frequency (%)	Concentration (ng/L)			Country	Reference
		Mean	Median	Max		
Fragrances						
Aberonne	94	2,800		16,500	Balkans	Terzic et al. (2008)
Acetyl cedrene	94	1,600		13,900	Balkans	Terzic et al. (2008)
Galaxolide	75 100 100	4,839 630 4,300		9,669 2,670 13,000	Spain Balkans Austria	Teijon et al. (2010) Terzic et al. (2008) Clara et al. (2011)
Musk xylene	89	170		560	Balkans	Terzic et al. (2008)
Tonalide	94 100	250 1,000		860 1,800	Balkans Austria	Terzic et al. (2008) Clara et al. (2011)
Insect repellent						
DEET	100 100 67	 840	196 1,140	15,800 3,328 6,900	EU-wide Singapore Balkans	Loos et al. (2013) Tran et al. (2019) Terzic et al. (2008)
Icaridin	13	1,300		2,200	Balkans	Terzic et al. (2008)
Preservatives						
Benzylparaben		0.07		0.26	China	Li et al. (2015)
Butylparaben	79	386.5 27.9		1,595 35.5	UK China	Kasprzyk-Hordern et al. (2009) Li et al. (2015)
Ethyl 3,5-dichloro4-hydroxybenzoate			30	49.4	China	Li et al. (2015)
Ethylparaben	100	1,295 140		3,312 220	UK China	Kasprzyk-Hordern et al. (2009) Li et al. (2015)
Methyl 3,5-dichloro-4-hydroxybenzoate		22.9		730	China	Li et al. (2015)
Methylparaben	100	7,210 567		30,688 1,002	UK China	Kasprzyk-Hordern et al. (2009) Li et al. (2015)
p-hydroxybenzoic acid (common metabolite of parabens)		590		1,660	China	Li et al (2018)
Propylparaben	96		1,844 438	8,286 605	UK China	Kasprzyk-Hordern et al. (2009) Li et al. 2015)
UV filters						
Benzophenone-1	100	196		700	UK	Kasprzyk-Hordern et al. (2009)
Benzophenone-2	100	123.5		403	UK	Kasprzyk-Hordern et al. (2009)
Benzophenone-3	61 56	916.5	 90	3,975 2,617	UK Singapore	Kasprzyk-Hordern et al. (2009) Tran et al. (2019)
Benzophenone-4	100		3,874.5	13,248	UK	Kasprzyk-Hordern et al. (2009)
Other						
Cimetidine	100	2,835		13,057	UK	Kasprzyk-Hordern et al. (2009)
Crotamiton	100		49.6	235	Singapore	Tran et al. 2019)
Diltiazem	100 79	1,164	 6.4	5,258 64.4	UK EU-wide	Kasprzyk-Hordern et al. (2009) Loos et al. (2013)
Fluconizole	98		67.5	598	EU-wide	Loos et al. (2013)
Orphenadrine	85		0.5	46.7	EU-wide	Loos et al. (2013)
Salbutamol	100	44.6 10		321	UK France	Kasprzyk-Hordern et al. (2009) Martin-Ruel et al. (2011)
Terbutaline	76	 5	0.9	4.7	EU-wide France	Loos et al. (2013) Martin Ruel et al. (2011)
Valsartan	100	1,038		5,388	UK	Kasprzyk-Hordern et al. (2009)
Zolpidem	58		0.6	42.6	EU-wide	Loos et al. (2013)

8.2 PHARMACEUTICALS AND PERSONAL CARE PRODUCTS IN STORMWATER

Compared with wastewater and receiving waters, there are relatively few studies that have assessed the presence of PPCPs in stormwater and runoff, presumably because stormwater is considered to be a relatively minor source of these compounds compared with municipal wastewater. However, available studies have documented the presence of numerous PPCP-related compounds in urban and agricultural stormwaters (Tables 25, 26). It is assumed that these compounds enter separated stormwater networks either through contamination with sewage (eg through cross-connections or leaking infrastructure) (Boyd et al. 2004; Liu et al. 2019a; Masoner et al. 2019; Tran et al. 2019), or through runoff from land where effluents or biosolids may have been applied (Khetan and Collins 2007). In addition, Ferry et al. (2018) detected several PPCPs, including ciprofloxacin, cocaine, DEET, enrofloxacin, naproxen and sulfamethoxazole in rain and/or snow samples in Minnesota; although the source of these compounds to the atmosphere is unknown, possible sources could include biosolids or effluents applied to land or the evaporation or aerosolization of wastewaters. Litter may contribute to presence of some compounds such as nicotine in stormwaters (eg discarded cigarette butts) (Fairbairn et al. 2018).

The specific compounds detected in urban stormwaters and their concentrations may vary substantially over time, with the frequency and intensity of rainfall, and between catchments depending on land use and the condition of the stormwater and wastewater networks (Liu et al. 2019a; Tran et al. 2019). Among the compounds most frequently detected are acetaminophen, caffeine, carbamazepine, crotamiton, gemfibrozil, lidocaine, metformin, methenamine, DEET, naproxen, nicotine, oxybenzone, triclocarban and triclosan (Boyd et al. 2004; Fairbairn et al. 2018; Masoner et al. 2019; Tran et al. 2019). Concentrations range from below the limits of detection (low ng/L) to tens of µg/L, although most detections are in the ng/L range (Boyd et al. 2004; Fairbairn et al. 2018; Masoner et al. 2019; Tran et al. 2019). The concentration of PPCP-related compounds in stormwaters are typically low compared with raw sewage (Liu et al. 2019a), and lower in agricultural stormwaters compared with urban stormwaters (Tran et al. 2019).

Table 25. Concentration of PPCPs (ng/L) in urban stormwater reported in international studies.

Compound	Detection frequency (%)	Concentration (ng/L)			Country	Reference
		Mean	Median	Maximum		
Analgesic/ anti-inflammatory						
Acetaminophen	84	211	87.2	1,700	US	Masoner et al. (2019)
	69		23.9	2,110	US	Fairbairn et al. (2018)
	92		267	45,882	Singapore	Tran et al. (2019)
Ibuprofen	46		<2.6	674	US	Boyd et al. (2004)
	0		<6	<6	Singapore	Tran et al. (2019)
Lidocaine	69	13.9	5.8	242	US	Masoner et al. (2019)
	89		3.9	19.9	US	Fairbairn et al. (2018)
Methyl salicylate	55		28	664	US	Masoner et al. (2019)
Naproxen	86		7.7	18,300	US	Boyd et al. (2004)
	0		<2	3,890	US	Tran et al. (2019)
Salicylic acid	100		249	12,242	Singapore	Tran et al. (2019)
Tramadol	24		<15	36.8	US	Masoner et al. (2019)
	14		<LOQ	13.6	US	Fairbairn et al. (2018)
Antibiotics						
Methenamine	58		48.2	3,420	US	Masoner et al. (2019)
Sulfamethoxazole	24		<26	6,030	US	Masoner et al. (2019)
	3		<LOQ	31.6	US	Fairbairn et al. (2018)
Trimethoprim	12		<19	417	US	Masoner et al. (2019)
Antidepressant						
Bupropion	10		<18	10.8	US	Maosner et al. (2019)
	17		<LOQ	17	US	Fairbairn et al. (2019)
Antidiabetic						
Metformin	73		70.5	1,260	US	Masoner et al. (2019)
	64		14.9	247	US	Fairbairn et al. (2018)
Guanyl urea	27		<LOQ	2,190	US	Masoner et al. (2019)
Antihistamine						
Fexofenadine	10		<LOQ	101	US	Masoner et al. (2019)
Anticonvulsant						
Carbamazepine	100		2	108	Singapore	Tran et al. (2019)
	10		<11	38.6	US	Masoner et al. (2019)
Gabapentin	0		<0.8	<0.8	Singapore	Tran et al. (2019)
	24		<LOQ	2,190	US	Masoner et al. (2019)
Beta blocker						
Metopropolol	14		<27	84.1	US	Masoner et al. (2019)
Lipid regulator						
Gemfibrozil	98		2	30	Singapore	Tran et al. (2019)
Hormone						
cis-androsterone	24		<2	24.8	US	Masoner et al. (2019)
Stimulants						
Amphetamine	2		<8	38.6	US	Masoner et al. (2019)
	11		<LOQ	13.5	US	Fairbairn et al. (2018)

Table 25 continued.

Compound	Detection frequency (%)	Concentration (ng/L)			Country	Reference
		Mean	Median	Maximum		
Caffeine	100		1,186	69,500	Singapore	Tran et al. (2019)
	96		930	32,200	US	Masoner et al. (2019)
	92		207	1,710	US	Fairbairn et al. (2018)
Nicotine	98		776	18,300	US	Masoner et al. (2019)
	94		205	3,890	US	Fairbairn et al. (2018)
X-ray contrast						
Iohexol	33		<10	2,862	Singapore	Tran et al. (2019)
Iopamidol	33		<5	1,200	Singapore	Tran et al. (2019)
UV filters						
Benzophenone	67		70	740	US	Masoner et al. (2019)
Oxybenzone	77		25	404	Singapore	Tran et al. (2019)
Insect repellent						
N,N-diethyl-m-toluamide (DEET)	98		191	109,000	US	Masoner et al. (2019)
	100		119	972	Singapore	Tran et al. (2019)
	97		120	490	US	Fairbairn et al. (2018)
Antiseptics/disinfectant						
Triclocarban	100		10	6,374	Singapore	Tran et al. (2019)
Triclosan	79		5.1	29	US	Boyd et al. (2004)
	100		10	452	Singapore	Tran et al. (2019)
	8		<LOQ	165	US	Masoner et al. (2019)
	6		<LOQ	30	US	Fairbairn et al. (2019)
Fragrance						
Acetophenone	76		337	2,760	US	Masoner et al. (2019)
Camphor	63		61	924	US	Masoner et al. (2019)
	36		<LOQ	610	US	Fairbairn et al. (2019)
Indole	41		<LOQ	180	US	Masoner et al. (2019)
Other						
Crotamiton	100		3.0	49	Singapore	Tran et al. (2019)
Menthol	57		162	1,610	US	Masoner et al. (2019)
	67		75	1,340	US	Fairbairn et al. (2018)
Methocarbamol	20		<LOQ	300	US	Masoner et al. (2019)
	17		<LOQ	5,850	US	Fairbairn et al. (2018)
Pseudoephidrine + ephidrine	16		<11	57.4	US	Masoner et al. (2019)
	3		<LOQ	0.6	US	Fairbairn et al. (2018)

Table 26. PPCPs that have been detected in stormwaters with a frequency <10% or whose presence has been analysed but not detected.

PPCP compounds reported in stormwater with a frequency of <10%	2-Androstene-3,17-dione (hormone); Abacavir (antiviral); Acyclovir (antiviral); Atenolol (beta-blocker); Acetyl-hexamethyl-tetrahydro-naphthalene (AHTN) (musk fragrance); Carisoprodol (muscle relaxant); Cimetidine (H2 receptor antagonist); Codeine (analgesic/anti-inflammatory); Desvenlafaxine (antidepressant); Dextromethorphan (cough suppressant); Diclofenac (analgesic/anti-inflammatory); Estriol (hormone); Estrone (hormone); Fluconazole (antifungal); Ketoconazole (antifungal); Loratadine (antihistamine); Meprobamate (derivative, anxiolytic); Methotrexate (immunosuppressant); Morphine (analgesic/anti-inflammatory); Nadolol (beta-blocker); Omeprazole/esomeprazole (acid-reducer); Oxycodone (analgesic/anti-inflammatory); Ranitidine (H2 antagonist); Sitagliptin (anti-diabetic); Testosterone (hormone); Theophylline (anti-asthmatic, diuretic); Triamterene (diuretic); Venlafaxine (antidepressant)
PPCP compounds whose presence has been analysed in stormwater, but not detected	17β-estradiol (hormone); Clofibric acid (lipid regulator); Chlorophene (antiseptic); Cortisone (hormone); Corticosterone (hormone); Fenoprofen (analgesic/anti-inflammatory); Fluoxteine (antidepressant); Indomethacin (analgesic/anti-inflammatory); Octocrylene (UV filter); Sulpiride (antipsychotic)

Compiled from Boyd et al. (2004); Fairbairn et al. (2018); Masoner et al. (2019); Tran et al. (2019).

8.3 HEALTH EFFECTS OF PHARMACEUTICALS AND PERSONAL CARE PRODUCTS

The concentrations of pharmaceuticals in wastewaters and receiving environments are typically orders of magnitude less than those ingested directly by consumers to elicit pharmacological effects (Khetan and Collins 2007; WHO 2012; Cressey 2018); for example, the maximum reported concentration of paracetamol in Table 24 and 25 above for paracetamol is 482 mg/L compared with a maximum daily dose for adults of 3,000 mg, while ibuprofen was reported at up to 55 mg/L, compared with a maximum daily dose of 3,200 mg. However, pharmacological doses apply to situations of intentional ingestion in order to attain a specific benefit or therapeutic effect. Many pharmaceuticals are known to have the potential for adverse side effects, especially in sensitive individuals, or are contraindicated with other pharmaceutical compounds (Sengar and Vijayananadan 2022); thus, in situations of unintended consumption, the low levels of risk associated with these compounds should still be considered (Cressey 2018). Similarly, the concentrations of active compounds from personal care products that are reported in wastewater or stormwater are less than those present in products used for direct topical application, although such products are seldom intended for ingestion. Given the sheer number and diversity of compounds within the PPCP

group of contaminants[99], it is not possible to provide a detailed review of potential adverse health effects within the current document. Further, there is a paucity of data on the chronic effects of many PPCPs, especially those that are intended to achieve acute effects (Kumar et al. 2010); in many cases, the human health effects of exposure to low levels of PPCP-related compounds are unknown or unclear[100,101,102]. Purported effects of PPCPs may include endocrine-disrupting and hormonal effects, reproductive effects, haematological abnormalities, impaired kidney and liver function, skin irritation, teratogenicity, mutagenicity and carcinogenicity (Daughton and Ternes 1999; Cressey 2018).

Several studies have conducted human health risk assessments for PPCPs detected in surface and drinking waters, with almost all concluding that there are no appreciable risks to human health from the concentrations of PPCPs that have been reported (Webb et al. 2003; Schwab et al. 2005; de Jongh et al 2012; WHO 2012; de Jesus Gaffney et al. 2015; Cressey 2018; Chen et al. 2020; Sengar and Vijayanandan 2022). Several exceptions are noted: a review by dos Santos et al. (2021) determined that 17α-ethinylestradiol and17β-estradiol posed high and medium risks to human health, respectively, whilst Sengar and Vijayanandan (2020) reported concentrations of 11 different PPCPs in surface waters in Hyderabad, India that were sufficiently high as to present a potential human health risk, although their proximity to pharmaceutical manufacturing facilities means they are not representative of environmental concentrations elsewhere. Despite these findings, there remain significant knowledge gaps and uncertainties regarding the possible health risks associated with the presence of PPCPs in wastewater or stormwater and their subsequent discharge to the environment (Khetan and Collins 2007; Daughton 2008; Kumar et al. 2010; de Jongh et al. 2012; WHO 2012). These include:

- scarce information on adverse effects in humans resulting from chronic exposure to very low concentrations of PPCPs, especially for personal care products
- uncertainty associated with estimating appropriate exposure limits (eg, ADI) to be used in risk assessments
- the possible additive or synergistic effects of simultaneous exposure to multiple PPCPs (or other micropollutants and/or environmental stressors), especially for compounds with similar modes of action, as risk assessments have typically considered compounds separately

[99] For example, more than 3,000 pharmaceutical compounds are registered for use in the European Union.
[100] https://www.cdc.gov/biomonitoring/Triclosan_FactSheet.html
[101] https://www.cdc.gov/biomonitoring/Benzophenone-3_FactSheet.html
[102] https://www.cdc.gov/biomonitoring/Parabens_FactSheet.html

- the risks associated with exposure to metabolites or transformation products, which in some cases may be more toxic or bioactive than the parent compound
- the small but potential risk presented by bioaccumulation of certain compounds within the food chain
- the risks to sensitive individuals.

As Daughton (2008) notes, the absence of evidence for adverse human effects from low-level exposure to PPCPs does not eliminate cause for concern, as there are simply too many uncertainties needing to be addressed through further research.

8.3.1 Comment on antimicrobials and antimicrobial resistance

Perhaps the most immediate human health concern regarding PPCPs in wastewaters is related to the spread of antimicrobial resistance (AMR) in microbial pathogens, particularly bacteria and fungi (Kumar et al. 2010; WHO et al. 2020; Frascaroli et al. 2021; Sengar and Vijayanandan 2022). Antimicrobial resistance undermines the efficacy of antimicrobial therapies, prolonging morbidity and increasing mortality (Frascaroli et al. 2021). Selective pressures imposed on microorganisms promote the spread of AMR genes in environments where there is constant contact between microorganisms and antimicrobial compounds; thus, WWTPs are a primary source of AMR genes as novel pollutants (Frascaroli et al. 2021). Indeed, several antibiotics known to be responsible for the development of AMR, including erythromycin, clarithromycin, azithromycin and ciprofloxacin, were placed on the European Commission's watchlist under the Environmental Quality Standard Directive 2008/105/EC, and have been considered to present considerable risk in terms of their ubiquitous distribution, concentrations in wastewater and resistance to treatment or removal processes (Frascaroli et al. 2021). In addition, the presence of antibiotics and related compounds in urban stormwaters means stormwater may also contribute to the emergence and spread of AMR in the environment (Almaaki et al. 2019). However, as agreed with the Ministry of Health, the health risks presented by AMR are outside the scope of this review, and thus not discussed further.

8.4 REGULATIONS AND GUIDELINES

Pharmaceuticals are somewhat unique amongst the contaminants discussed in this report, as they are designed for intentional administration (eg, via ingestion, topical application) to people to elicit therapeutic effects. Pharmaceuticals are normally governed by stringent

regulatory processes and require rigorous pre-clinical and clinical studies to assess efficacy and safety before commercial production (WHO 2012). Health risks from pharmaceuticals in water are often assessed using the minimum therapeutic dose, however this is typically determined by controlled studies that may not account for sensitivities of sub-populations who would not normally be given the drug and/or may not reflect scenarios of chronic exposure. The lack of toxicological data available in the public domain means that it has not been possible to derive no observable adverse effects limits (NOAEL) or other health-based guidance values such as TDIs (WHO 2012). Exceptions occur for pharmaceuticals used in veterinary medicines that are relevant to food production, as traces of these compounds may be considered contaminants. Health-based guidance values have also been determined for a small number of compounds routinely used in personal care products. Pertinent international regulations and guidelines regarding exposure to selected PPCP-related compounds are summarised in Table 27; due to the sheer size of the PPCP classification, only those compounds reported in Tables 24 and 25 were included. Compounds have been included where at least one of the following guidelines was identified: an ATSDR minimum risk level (MRL) for oral exposure, a US EPA reference dose (RfD) for oral exposure, an ADI established by JMPR, a WHO guideline value for drinking-water, or a US EPA maximum contaminant limit (MCL) for drinking water.

Table 27. Recommended oral exposure limits for selected PPCP-related compounds.

	Minimum Risk Level, ATSDR[103] (µg/kg bw/day)	Reference Dose, US EPA[104] (µg/kg bw/day)	Acceptable Daily Intake, JECFA[105] (µg/kg bw/day)
N,N-Diethyl-meta-toluamide (DEET)	1,000 (int.)		10*
Hexachlorophene		0.3	
Enrofloxacin			2
Erythromycin			0.7
17β-estradiol			0.05
Methenamine			150
Methyl paraben			10,000 (ie 10 mg)
Ethyl paraben			

US EPA Reference Doses and ATSDR Minimum Risk Levels are for oral exposures.
int. – intermediate exposure duration *provisional tolerable daily intake (PTDI) established by the JMPR. ^group ADI for the ethyl and methyl esters of p-hydroxybenzoic acid, excluding propyl paraben. int. – intermediate exposure

[103] https://wwwn.cdc.gov/TSP/MRLS/mrlsListing.aspx Accessed 14 July 2022
[104] https://iris.epa.gov/AtoZ/?list_type=alpha Accessed 14 July 2022
[105] https://apps.who.int/food-additives-contaminants-jecfa-database/ Accessed 14 July 2022

9. ENDOCRINE-DISRUPTING COMPOUNDS

An endocrine-disrupting compound (EDC) may be defined as 'an exogenous agent that interferes with the synthesis, secretion, transport, metabolism, binding action, or elimination of natural blood-borne hormones that are present in the body and are responsible for homeostasis, reproduction, and developmental processes" (Diamanti-Kandarakis et al. 2009; Lauretta et al. 2019). Traditionally, EDCs were considered to exert effects primarily through interaction with nuclear hormone receptors, including estrogen, androgen, progesterone and thyroid receptors (Diamanti-Kandarakis et al. 2009; Giulivo et al. 2016). Indeed, most characterised EDCs act by binding to the estrogen receptor, either simulating the action of 17β-estradiol[106] to activate the receptor (agonist), or blocking the receptor so 17β-estradiol cannot access the receptor (antagonist), although estrogen-agonist EDCs usually have activity that is orders of magnitude less than 17β-estradiol. However, the actions of EDCs are now understood to be much broader, exerting effects via non-nuclear steroid hormones receptors (eg membrane estrogen receptors), nonsteroid receptors (eg dopamine or norepinephrine receptors), orphan receptors (eg aryl hydrocarbon receptor), and various enzymatic pathways involved in steroid biosynthesis and/or metabolism (Diamanti-Kandarakis 2009). Endocrine disruption may affect various body functions, depending on the pathway that is disrupted (Giulivo et al. 2016).

The informal group of compounds identified as EDCs is highly heterogeneous, and includes synthetic chemicals used in industrial solvents and lubricants, plastics, pesticides, pharmaceuticals, and a wide variety of other consumer products (Diamanti-Kandarakis et al. 2009). Various subgroups of EDCs may show a lack of structural similarity aside from a small molecular mass (<1,000 Daltons), although many have a halogen group substitution and a phenolic moiety that is thought to mimic natural steroid hormones and therefore enables their interaction with the endocrine system (Diamanti-Kandarakis et al. 2009). Some naturally-occurring chemicals such as phytoestrogens may also act as EDCs (Diamanti-Kandarakis et al. 2009).

Endocrine-disrupting compounds are ubiquitous in the environment, being detected in food, water, soils and biota. Many EDCs are highly lipophilic, accumulating in the adipose tissue of exposed organisms and potentially magnifying up the food chain (Diamanti-Kandarakis et al.

[106] 17β-estradiol is the major female sex hormone.

2009; Lauretta et al. 2019). Some EDCs, such as dioxins, are highly persistent; these compounds do not decay easily, and may not be metabolised or metabolised to compounds that are more toxic than the parent compound. As a result, some persistent EDCs continue to be detected in the environment decades after restrictions were imposed upon their use. Other EDCs, such as BPA and phthalates, are less persistent, however their widespread use confers a pseudo-persistence and continued potential for human exposure (Diamanti-Kandarakis et al. 2009; Giuliyo et al. 2016).

This section will provide an overview of the presence of four main groups of EDCs that have been identified in municipal wastewater and urban stormwater: BPA, dioxins, alkylphenols and phthalates. However, it is important to note that while these compounds have been grouped together as endocrine disruptors, reflecting their common grouping as such in the literature, this does not mean that their effects on human health are limited to endocrine disruption. It is also important to note that there are many compounds in addition to those included within this chapter that are also known to have endocrine-disrupting effects (Lauretta et al. 2019); some of those compounds, such as pesticides, pharmaceuticals, and even heavy metals, have been discussed elsewhere in this report.

9.1 BISPHENOL A

Bisphenol A is an organic industrial chemical with high production volumes, and is mainly used as a building block in the production of polycarbonate plastics and epoxy resins (WHO 2011). It has been widely used in a variety food contact materials, including food storage containers, plastic tableware, water bottles, baby bottles, and protective linings on canned foods and beverages and metals lids used on glass jars and bottles (WHO 2011; Cressey 2018). Small amounts of BPA can migrate from these products to foods and beverages, resulting in dietary exposure (WHO 2011; Cressey 2018). Additionally, BPA is used in the production of various electronic components, digital media (CDs and DVDs), construction materials, children's toys, paints and coatings, and thermal receipts (Cesen et al. 2018; Zhao et al. 2021).

9.1.1 Bisphenol A in wastewater

Bisphenol A can enter the municipal wastewater network due to human excretion of BPA that has been absorbed following dietary exposure. It has also been noted to be present in some toilet papers, adding to its levels in municipal wastewater (Höhne and Püttmann 2008).

Industrial effluents have also been noted to be a potentially substantial contributor to the level of BPA reaching municipal WWTPs (Santos et al. 2016).

Several studies have assessed BPA levels in wastewater and its concentration in influent wastewater has been noted to be highly variable, with concentrations in samples taken from two WWTPs in Frankfurt, Germany ranging from below the LOD to 9.1 and 12.2 µg/L respectively (average concentrations of 2.5 and 4.8 µg/L) (Höhne and Püttmann 2008). In contrast, influent wastewater samples from a sewage treatment plant in Greece were found to contain BPA concentrations ranging from 0.5 to 0.9 µg/L (Pothitou and Voutsa 2008), and concentrations in influent samples from WWTPs in Queensland, Australia ranged from below the LOD to 2.8 µg/L (Tan et al. 2007).

9.1.2 Bisphenol A in stormwater

Bisphenol A has been reported in stormwater samples from around the world, including China (Zhao et al. 2021), the UK (Wilkinson et al. 2016), France (Gasperi et al. 2014) and Australia (Gernjak et al. 2017). As noted above, BPA may be present in construction materials, such as phenolic resin insulation board used in construction of exterior walls (Zhao et al. 2021), providing a route for entry into stormwater via rainfall runoff. It may also be used in the manufacture of car bumpers, tires and brake fluid (Gasperi et al. 2014), providing additional routes for stormwater contamination.

High levels of BPA have been reported in stormwater, and some studies have noted that levels in stormwater were substantially higher than in WWTP effluents. For example, rainfall runoff from highly urbanised regions in China was found to contain up to 5.9 µg/L BPA, with the BPA mass load in stormwater more than ten times higher than that of effluents from municipal WWTPs assessed in the same study (Zhao et al. 2021). Similarly, BPA was found at concentrations over 2.4 µg/L in street runoff in the UK, 2.7 times higher than the highest level detected in sewage treatment works effluent in that study (Wilkinson et al. 2016). In Australia, BPA has been found at concentrations up to 2 µg/L in stormwater samples collected from across Australia during rainfall events (Gernjak et al. 2017).

9.1.3 Health effects of Bisphenol A

Although a large number of studies on the toxicity and hormonal activity of BPA in laboratory animals have been published, there are discrepancies in the outcomes of these studies with respect to the nature of the adverse effects observed and the levels at which they can occur (WHO 2011). BPA can have estrogenic activity, mimicking the female sex hormone 17β-

estradiol and binding to its receptor, potentially impairing reproductive capacity[107] (Rubin 2011; WHO 2011). Bisphenol A may also interact with receptors for thyroid hormones and the steroid hormone androgen (Rubin et al. 2011; Ma et al. 2019). Acute toxicity of BPA is low, however repeated-exposure animal studies are suggestive of effects on the liver, kidney and body weight at high doses (WHO 2011). In New Zealand, BPA has been classified by the Environmental Protection Authority as a chemical "suspected of damaging fertility or the unborn child"[108].

Several exposure guidelines have been set for BPA. In 2015, the TDI set by EFSA was reduced from 50 to 4 µg/kg body weight/day[109], and in December 2021 it was proposed that this limit be reduced further to 0.04 ng BPA/kg body weight/day[110]. The US EPA has set a reference dose of 20 µg/kg body weight/day.[111]

9.2 DIOXINS

The term dioxins is used to describe compounds belonging to two closely-related families – polychlorinated dibenzofurans (PCDFs) and polychlorinated dibenzo-*p*-dioxins (PCDDs) – as well as certain dioxin-like PCBs (WHO 2010b; Ministry of Health 2020). Although hundreds of PCDFs, PCDDs and dioxin-like PCBs exist, the WHO estimates that of 419 known dioxin-related compounds, approximately 30 are considered to have significant toxicity, of which 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD; often referred to as dioxin) is the most toxic.[112]

Dioxins are largely by-products of anthropogenic processes but can also result from natural processes such as volcanic activity and forest fires. For example, PCDDs and PCDFs are not created or used intentionally, but are a produced as by-products of the incineration of solid waste and fossil fuels, metal smelting, chlorine bleaching of pulp and paper products, and manufacturing of certain chemicals, including pesticides (WHO 2010b; Ministry of Health 2020). PCBs are manufactured products that were historically widely used as insulating and hydraulic fluids and as additives to paints, oils and caulks; they are no longer used or

[107] https://www.mpi.govt.nz/dmsdocument/25685-Bisphenol-A-Information-sheet#:~:text=SAFETY%20ASSESSMENTS&text=EFSA%20set%20a%20Tolerable%20Daily,kg%20body%20weight%2Fday). Accessed 10 May 2022
[108] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/B41EC9A7-D80E-41DF-A353-4AF0DCA80AC4 Accessed 14 December 2022
[109] https://www.efsa.europa.eu/en/topics/topic/bisphenol Accessed 9 November 2022
[110] https://www.efsa.europa.eu/en/news/bisphenol-efsa-draft-opinion-proposes-lowering-tolerable-daily-intake Accessed 2 May 2022
[111] https://iris.epa.gov/ChemicalLanding/&substance_nmbr=356
[112] https://www.who.int/news-room/fact-sheets/detail/dioxins-and-their-effects-on-human-health

produced[113], but their release to the environment continues through disposal of waste containing them (WHO 2010b). Dioxins are highly persistent and omnipresent in the environment, being detected in soils, sediments and food. They are listed in Annex C of the Stockholm Convention, meaning signatories must take steps to reduce their unintentional release[114], and the manufacture of PCBs is prohibited in many countries around the world. Dioxins may accumulate within the food chain, and once they enter the body are absorbed and stored in adipose tissues and the liver, where their estimated half-life is between 7 and 11 years (WHO 2010b; Ministry of Health 2020).

Dioxins are poorly soluble in water and generally exist in the environment as complex mixtures (WHO 2010b). Due to the large number of compounds that can contribute to 'dioxin toxicity,' each compound or congener is assigned a toxic equivalency factor (TEF), which is an indication of its toxicity relative to TCDD. The TEF of each congener can be multiplied by its concentration, and the total for each congener added together to give a total toxic equivalent (TEQ) determined as equivalents of TCDD (WHO 2010b; Ministry of Health 2020).

9.2.1 Dioxins in wastewater

Wastewater influents, treated effluents and sewage sludges around the world have been reported to contain PCDDs, PCDFs and PCBs (Rideout and Teschke 2004; Rossi et al. 2004; Clarke et al. 2008; Li et al. 2011; Sappington et al. 2015; Jing et al 2019). Potential sources include laundry waters involving contaminated textiles, bathing waters where compounds have transferred to skin, and sewage containing bleached toilet papers and faeces (reflecting human excretion) (Horstmann and McLachlan 1995; Rossi et al. 2004; Clarke et al. 2008). For example, an assessment of the presence of the 17 most toxic PCDDS and PCDFs in municipal WWTP effluents in Houston, Texas, identified concentrations for each congener ranging from 0.0012 to 12.1 pg/L and 0.0019 to 6.29 pg/L in the dissolved and suspended phases, respectively. Total (ie $\sum$PCDD/PCDF$_{17}$) concentrations were up to 4.9 and 126 pg/L (TEQ up to 0.1336 and 0.489 pg/L) for dissolved and suspended phases, respectively (Sappington et al. 2015). Loadings were greater in industrial effluents analysed in the same study, with $\sum$PCDD/PCDF$_{17}$ up to 13.2 and 683 pg/L in the dissolved and solid phases (Sappington et al. 2015); municipal wastewaters that receive trade effluents may therefore have higher concentrations of dioxins. Domestic

[113] https://www.cdc.gov/biomonitoring/DioxinLikeChemicals_FactSheet.html
[114] [114] http://www.pops.int/TheConvention/ThePOPs/AllPOPs/tabid/2509/Default.aspx Accessed 8 November 2021

wastewater from an apartment building in Germany was found to contain concentrations of PCDD/PCDF up to ten times higher than urban runoff samples, with washing machine effluents being a major source (Horstmann and McLachlan 1995). Another study assessed the presence of a range of PCBs in WWTP effluents and found an average mass of over 300 g total PCBs was discharged by the WWTP annually (excluding wet weather bypass discharge), despite production of PCBs being banned in the US since the 1970s (Jing et al. 2019).

During WWTP processes, dioxin-like compounds partition almost exclusively into sewage sludge due to their lipophilic nature (Clarke et al. 2008; Table 28), hence much of the literature focuses on their concentration in sludges. An international review of concentrations of PCDD and PCDF in municipal sewage sludges found concentrations ranging from 0.0005 to 186 pg TEQ/gram (Rideout and Teschke 2004). A recent report prepared for the Ministry for the Environment estimated that the total annual release of dioxins from WWTPs to water in New Zealand in 2020 was 0.01 g TEQ (Bingham 2022).

Table 28. Concentrations of dioxin congeners (pg/g dry weight) in sewage sludge from three WWTPs in Western Australia.
Reproduced from Clarke et al. (2008a).

	Beenyup								Subiaco								Woodman point							
	2002		2003		2005		2006		2002		2003		2005		2006		2002		2003		2005		2006	
2,3,7,8-TCDF	4.6	11.4	7.5	7.4	6	5.1	2.6	2.4	<3	<2	4.5	3.9	4.5	<1	<0.6	<1.6	<2.0	2.6	2.3	2.6	3	2.1	2.9	
2,3,7,8-TCDD	0.7	1	0.26	0.32	<0.6	0.85	<0.8	<1	9.3	<0.1	0.48	0.63	8.3	<0.4	<0.3	<0.5	<0.2	0.35	0.26	0.68	<1	<0.6	<0.7	
1,2,3,7,8-PeCDF	1.1	<6	1.7	1.6	<0.9	<0.6	<1	<0.7	0.7	<0.2	0.5	0.56	1.2	<0.3	<0.1	<0.9	2.1	1.1	1	1.1	<0.8	<0.9	<0.7	
2,3,4,7,8-PeCDF	1.7	13	1.8	1.6	1.4	1.4	<0.9	<2	<1	<0.7	0.87	0.93	1.2	0.69	<0.3	<0.8	3.2	1.1	1.2	1.4	1.4	<0.7	<1	
1,2,3,7,8-PeCDD	1.2	2.8	1.5	1.5	1.2	<1	<2	<2	<0.7	<0.4	1.3	1.2	<0.9	<0.8	0.56	0.9	2.3	1.4	1.1	2.2	1.9	<2	<2	
1,2,3,4,7,8-HxCDF	2.8	6.8	1.5	<3	2.2	2.6	<0.8	<0.8	<3	<3	0.27	<0.1	1.6	1.3	0.61	2	5.2	<2	<1	2.5	2.2	1.2	<1	
2,3,4,6,7,8-HxCDF	2	6.7	2	1.7	1.1	1.5	<0.9	<1	0.8	<0.4	0.775	0.87	0.75	<0.5	0.43	<0.6	4.4	1.8	1.8	1.5	1.6	1.3	<1	
1,2,3,6,7,8-HxCDF	2.7	8	<3	<3	2.2	2.9	<2	<2	1.2	1	<1	<1	1.5	1.3	<0.4	1.8	6.7	2.7	<2	2	2	<2	1.9	
1,2,3,7,8,9-HxCDF	<0.4	<3	<0.4	<0.3	<0.4	<0.3	<0.5	<0.8	<0.3	<0.2	<0.4	<0.1	<0.3	<0.2	<0.3	<0.3	0.7	<0.3	0.23	<0.3	<0.5	<0.4	<0.8	
1,2,3,4,7,8-HxCDD	<0.5	1.3	0.58	0.61	<0.5	<0.5	<0.8	<0.8	<0.2	<0.1	0.71	0.72	0.45	<0.4	<0.4	<0.3	<2	0.75	0.63	1.4	<0.7	<0.5	<1	
1,2,3,6,7,8-HxCDD	3	5.3	3.9	3.7	3.2	3.1	<2	2.5	2.6	1.8	4.5	4.8	5	5.4	3.7	3.2	13	4.2	4.3	4.6	4.3	2.9	3.1	
1,2,3,7,8,9-HxCDD	<0.5	1.33	<0.9	<0.7	<1	1.5	<0.8	<0.7	<1	<0.2	<1	<2	<2	<2	1.3	1.8	<2	<2	<3	3	3.8	1.2	<1	
1,2,3,4,6,7,8-HpCDF	72	92	110	99	71	72	47	44	30	29	43	43	35	32	14	68	99	99	99	58	54	36	40	
1,2,3,4,7,8,9-HpCDF	1.3	1.9	1.4	1.3	1.4	1.4	<0.6	<2	0.7	<0.5	<0.8	0.9	<0.6	0.82	<0.2	1.5	4.3	1.6	1.4	1.5	1.6	<1	<1	
1,2,3,4,6,7,8-HpCDD	87	104	88	85	82	85	62	52	60	65	110	110	150	160	63	83	188	100	100	86	81	71	78	
OCDF	333	379	340	330	240	250	170	150	137	137	170	170	60	78	22	321	389	330	320	170	180	130	130	
OCDD	882	1110	860	790	810	830	580	500	592	769	1270	1200	1000	1210	500	900	1160	999	990	740	780	700	710	
WHO_{98} TEQ ∑PCDD/Fs	**6.0**	**16.5**	**6.4**	**6.0**	**5.0**	**4.9**	**1.4**	**1.5**	**10.8**	**1.3**	**5.0**	**5.0**	**12.3**	**3.2**	**2.0**	**3.4**	**10.1**	**5.7**	**5.1**	**7.0**	**5.8**	**2.0**	**2.1**	
WHO_{05} TEQ ∑PCDD/Fs	**5.9**	**14.2**	**6.3**	**5.9**	**4.9**	**4.8**	**1.6**	**1.6**	**10.9**	**1.5**	**5.1**	**5.1**	**12.2**	**3.3**	**2.1**	**3.7**	**9.7**	**5.7**	**5.1**	**6.8**	**5.7**	**2.2**	**2.2**	
PCB 77	294	373	190	190	180	180	300	190	255	208	98	95	130	110	53	481	587	230	230	210	200	250	270	
PCB 81	14.9	40.3	8.4	8.4	7.9	8.5	13	8	25.1	8.43	7.4	6.6	11	5.1	2.1	15.9	23.4	9.9	9.5	9.4	8.3	8.2	8.3	
PCB 126	24.2	61	17	16	16	17	12	11	34.1	9.65	22	13	11	8.7	3.4	26.5	31.8	18	19	20	19	13	13	
PCB 169	<3	<10	2	2.3	2.2	1.9	<1	<1	<2	<2	2.3	1.4	<1	0.85	<0.2	3.35	<5	2.2	2.4	<2	2.4	<0.6	2	
PCB 105	1970	2100	860	820	1000	1030	680	580	1240	1290	810	770	680	680	250	1760	2250	1230	1300	1700	1780	670	750	
PCB 114	130	135	58	70	72	67	49	43	79	87.4	67	64	48	42	19	148	172	120	100	130	120	58	63	
PCB 118	3780	4710	2280	2110	2330	2400	1810	1490	2680	2600	2200	2280	1590	1410	660	3880	5040	3560	3190	4190	4010	1760	1800	
PCB 123	91	144	48	45	61	61	29	29	55.6	52.8	51	60	49	44	8	124	84.4	<100	73	99	97	29	28	
PCB 156	629	726	320	300	360	320	220	200	408	387	270	310	220	200	87	623	684	440	390	570	560	230	220	
PCB 157	119	161	100	72	80	89	49	45	80.2	<70	7	63	47	45	17	<100	161	75	110	140	150	51	49	
PCB 167	767	797	240	380	150	150	68	68	497	420	360	340	63	100	28	719	929	490	540	210	220	76	83	
PCB 189	35.5	31	16	15	19	20	16	<10	19.7	<20	<10	14	13	<10	<4	33.9	38.6	<20	24	23	27	<30	<20	
WHO_{98} TEQ ∑PCBs	**3.5**	**7.4**	**2.3**	**2.2**	**2.2**	**2.3**	**1.6**	**1.5**	**4.1**	**1.6**	**2.7**	**1.9**	**1.5**	**1.2**	**0.5**	**3.7**	**4.5**	**2.6**	**2.7**	**3.0**	**3.0**	**1.7**	**1.8**	
WHO_{05} TEQ ∑PCBs	**2.7**	**6.4**	**1.9**	**1.8**	**1.8**	**1.9**	**1.3**	**1.2**	**3.6**	**1.1**	**2.4**	**1.5**	**1.2**	**1.0**	**0.4**	**3.0**	**3.5**	**2.1**	**2.2**	**2.2**	**2.2**	**1.4**	**1.5**	
I-TEQ	**6.5**	**16.5**	**6.7**	**6.3**	**5.4**	**5.9**	**2.1**	**2.1**	**11.4**	**2.1**	**5.6**	**5.7**	**13.3**	**4.4**	**2.2**	**4.1**	**10.3**	**6.2**	**5.7**	**6.7**	**5.7**	**2.8**	**2.8**	
WHO_{98}TEQ (∑PCDD/Fs+∑PCBs)	**9.5**	**23.9**	**8.7**	**8.2**	**7.3**	**7.2**	**3.1**	**3.0**	**14.9**	**2.9**	**7.7**	**6.9**	**13.8**	**4.4**	**2.5**	**7.1**	**14.6**	**8.3**	**7.8**	**10.0**	**8.7**	**3.8**	**3.8**	
WHO_{05}TEQ (∑PCDD/Fs+∑PCBs)	**8.6**	**20.6**	**8.2**	**7.7**	**6.8**	**6.7**	**2.9**	**2.8**	**14.5**	**2.6**	**7.5**	**6.6**	**13.4**	**4.3**	**2.5**	**6.7**	**13.2**	**7.8**	**7.2**	**9.1**	**7.9**	**3.6**	**3.7**	
Total TCDD	13	66	16	14	9.7	8.4	<4	<6	18	13	48	50	32	22	5.8	14	28	19	18	26	20	14	16	
Total TCDF	28	219	41	39	33	32	23	19	19	14	26	23	120	20	10	22	19	27	24	21	19	20	22	
Total PeCDD	21	59	23	22	28	11	0.7	10	38	9.9	45	110	730	7710	7.3	5.6	45	19	19	15	28	11	<10	
Total PeCDF	12	119	31	27	23	18	8	9.7	11	10	17	18	30	16	140	10	29	23	22	21	22	9.4	12	
Total HxCDD	23	77	40	36	25	25	20	16	13	9.6	31	34	30	32	6.2	13	119	35	44	53	1	23	23	
Total HxCDF	20	59	38	29	21	23	15	17	10	5.1	15	14	11	10	21	14	43	29	28	21	20	18	17	
Total HpCDD	159	198	180	170	150	160	110	89	109	119	220	220	240	260	14	154	348	200	200	160	160	130	130	
Total HpCDF	117	136	170	150	100	110	66	58	49	48	36	38	49	49	110	111	152	160	150	89	84	54	55	
Total PCDD/Fs	**1610**	**2420**	**1740**	**1610**	**1440**	**1470**	**1000**	**870**	**996**	**1130**	**1880**	**1880**	**2300**	**2410**	**840**	**1560**	**2330**	**1840**	**1820**	**1320**	**1350**	**1110**	**1120**	

9.2.2 Dioxins in stormwater

Despite restrictions on the manufacture, use or release of dioxins to the environment, dioxins are often still present in stormwater due to their persistence in the environment, release from materials used prior to their prohibition, and their ongoing release by certain industrial, combustive, or natural processes. For example, PCBs were used as sealants and caulking in pre-1970s buildings and can be released in rainfall runoff (Rossi et al. 2004; Cao et al. 2019). These chemicals can also be released to stormwater from roads prepared using recycled construction and demolition waste (Cao et al. 2019). The application of sewage sludges containing high levels of various dioxins to agricultural soils may be a source in rural settings (Rossi et al. 2004). Atmospheric deposition of combustion-related particulates, and washout by rain itself are also sources of dioxins in stormwaters (Rossi et al. 2004; Cao et al. 2019).

As might be expected, the level of dioxins present in stormwater is influenced by land use within the watershed, and it has been noted that levels tend to be higher in stormwater from urban watersheds, particularly those containing industrial land, compared to watersheds dominated by open spaces or agricultural land (Gilbreath and McKee 2015). Indeed, a recent US study by Cao et al. (2019) found that PCB concentrations were almost twice as high in stormwater sediments from residential (average 35 ng/L) and dense urban areas (average 40 ng/L) compared to greenspace (average 18 ng/L). Average PCB concentrations were also found to be substantially higher in stormwater samples taken from highly dense urban areas in France (468 ng/L) compared with those taken from dense urban areas (259 ng/L) and residential areas (211 ng/L) (Zgheib et al. 2011).

Rain events have also been shown to play an important role in mobilisation of dioxins in stormwater, and it has been suggested that concentrations in urban stormwater may vary by one or two orders of magnitude between storm and low flow conditions (Gilbreath and McKee 2015). For example, samples taken from a stormwater conveyance in San Francisco were found to contain concentrations of up to 9.4 ng/L for PCBs and 0.2 ng/L for dioxins and furans during low flow conditions, and 109 ng/L for PCBs and 6.3 ng/L for dioxins and furans during storm flow conditions (Gilbreath and McKee 2015). Similarly, stormwater runoff into the Anacostia River in the US was found to contain up to 80-fold higher levels of PCBs under storm flow compared to base flow (Hwang and Foster, 2008). It is likely that the elevated PCB levels during storm conditions are due to mobilisation of particulate matter as PCBs are known to be sorbed on to particulate matter (Cao et al. 2019). As such, sedimentation or filtration methods which remove particulate matter from stormwater could be used to reduce PCB levels in stormwater, thereby reducing the level discharged to receiving waters (Hwang and Foster 2008; Cao et al. 2019).

In addition to storm runoff, snowmelt runoff has also been identified as a potentially important contributor of PCDDs/PCDFs into stormwater due to the rapid release of contaminants accumulated in the mass of melting snow and ice (Urbaniak et al. 2016).

9.2.3 Health effects of dioxins

The health risks associated with exposure to dioxins were recently assessed in a report prepared by the Ministry of Health (Ministry of Health 2020). This report notes that the health effects of dioxins are not completely understood, but that dioxins can affect the growth and development of cells in ways that have potential to result in a broad range of adverse health effects (Ministry of Health 2020).

Short-term exposure to dioxins can result in skin lesions and altered liver function; chloracne is the most unequivocal toxicity outcome although it only occurs only after high exposures (resulting in serum levels > 20,000 pg/g fat) (WHO 2010b). Longer-term environmental exposure may cause a range of toxicity, including immunotoxicity, developmental and neurodevelopmental effects, and effects on thyroid and steroid hormones and reproductive function (WHO 2010b; EFSA 2018). A range of conditions have been associated with exposure to TCDD, including Hodgkin's and non-Hodgkin's lymphoma, soft tissue sarcoma, chronic lymphatic leukaemia, hypertension and monoclonal gammopathy. There is limited or suggestive evidence for an association with type 2 diabetes, respiratory cancers, bladder cancer, early onset peripheral neuropathy, porphyria cutanea tarda, AL amyloidosis, Parkinson's disease, hypothyroidism, stroke and ischemic heart disease (NASEM 2018; Ministry of Health 2020). In addition, TCDD, 2,3,4,7,8-pentachlorodibenzofuran and several dioxin-like PCBs (PCBs 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189) have been classified by the IARC as Group 1 carcinogens.[115]

Several authorities have set recommended exposure limits for dioxins, including WHO, US EPA, EFSA and New Zealand Ministry of Health. These limits are summarised in Table 29.

[115] https://monographs.iarc.who.int/list-of-classifications/

Table 29. Recommended exposure limits for dioxins.

Provisional Tolerable Monthly Intake, JECFA[116] (pg/kg bw/day)	Minimum Risk Level, ATSDR[117] (pg/kg bw/day)	Reference Dose, US EPA [118] (pg/kg bw/day)	Tolerable Weekly Intake, EFSA[119] (pg/kg bw/week)	Tolerable Monthly Intake, NZ MoH[120] (pg/kg bw/month)
70	200 (acute) 1 (chronic) (for TCDD)	0.7 (for TCDD)	2	30

US EPA Reference Doses and ATSDR Minimum Risk Levels are for oral exposures.

9.3 ALKYLPHENOLS

Alkylphenols are organic compounds produced by alkylation of phenol. Nonylphenol (NP) and octylphenol (OP) are used to create alkylphenol ethoxylate (APEO) surfactants by reacting them with ethylene oxide (Ying et al. 2002). These APEOs consist of an alkylphenol and ethoxylate moiety which helps them to disperse grease and dirt from soiled surfaces (Ying et al. 2002). Alkylphenol ethoxylates are therefore widely used as surfactants, detergents, solubilisers, foaming agents, emulsifiers and wetting agents, as well as antistatic and curing agents, in a variety of industries including the cleaning, paper, textile, plastics, pesticide, leather, petroleum recovery and metal industries (Ying et al. 2002; Priac et al. 2017; Crini et al. 2021). Nonylphenol is the most commercially important alkylphenol, followed by octylphenol, with approximately 80% of APEOs produced being NPEOs and the remaining 20% being OPEOs (Priac et al. 2017).

Alkylphenols are ubiquitous environmental contaminants, being detected in rivers and other freshwater systems, coastal and marine environments, sediments, soils, sewage and sludge, and in the atmosphere (Crini et al. 2021). As a result of their widespread presence in the environment, persistent nature and high potential toxicity, alkylphenols and their derivatives have been increasingly subject to regulation and other initiatives to reduce or eliminate their use. For example, both NP and OP have been classified as a priority hazardous substance

[116] https://cdn.who.int/media/docs/default-source/food-safety/dioxins.pdf?sfvrsn=4bcd5f4d_1 Accessed 10 November 2022
[117] https://wwwn.cdc.gov/TSP/MRLS/mrlsListing.aspx Accessed 10 November 2022
[118] https://iris.epa.gov/ChemicalLanding/&substance_nmbr=1024 Accessed 10 November 2022
[119] https://www.efsa.europa.eu/en/press/news/dioxins-and-related-pcbs-tolerable-intake-level-updated Accessed 9 November 2021
[120] Ministry of Health (2020).

and priority substance, respectively, under the European Water Framework Directive. (2013/39/CE) (Crini et al. 2021).

From a structural perspective, NP and OP are not single chemical structures, but exist numerous isomers: for example, technical nonylphenol consists of a mixture of 211 possible isomers (Crini et al. 2021). Alkylphenols are hydrophobic and preferentially associate with suspended solids (Crini et al. 2021).

9.3.1 Alkylphenols in wastewater

Several studies have suggested that the widespread presence of NP in aquatic environments is mainly due to their discharge from WWTPs (Priac et al. 2017; Crini et al. 2021). Indeed, a review by Ying et al. (2002) noted that APEOs were commonly found in WWTP effluents. Of added concern is the observation that APEOs present in WWTPs degrade to form more persistent compounds including shorter-chain APEOs (eg, NP1EO, NP2EO, NP3EO) and parent alkyphenols (eg, NP and OP) (Ying et al. 2002). Nonylphenol has been noted to typically be present in µg/L concentrations in WWTP effluents, but concentrations up to 343 µg/L have been reported (reviewed by Priac et al. (2017) and Ying et al. (2002)). Concentrations of a range of different APs and APEOs found in influent wastewater around the world are summarised in Table 30. A general trend can be seen that levels have been decreasing since 2000, which likely reflects that many of these chemicals have been classified as hazardous substances and strict legislation regarding their usage has been imposed[121,122] (Crini et al. 2021). However, these substances are still found in the environment (Crini et al. 2021) reflecting, at least in part, their persistent nature. The levels of OP and NPs have also been quantified in different types of greywater from French households (Deshayes et al. 2017), with greywater from the bathroom sink, and floor cleaning contained the highest OP concentrations, whilst greywater from floor cleaning and the washing machine contained the highest NP concentrations (Table 31). Effluents from several industries have also been found to contain nonylphenols (Eaton 2022), and where these are discharged to the municipal network this will add to the load reaching the WWTP.

Removal of APEOs from wastewater appears to be highly variable, with Chokwe et al. (2017) noting that removal efficiencies vary from 9 to 94%, depending on the type of treatment and location.

[121] https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-management-nonylphenol-and-nonylphenol-ethoxylates Accessed 15 December 2022
[122] https://www.canada.ca/en/environment-climate-change/services/management-toxic-substances/list-canadian-environmental-protection-act/nonylphenol-ethoxylates.html Accessed 15 December 2022

Table 30. Concentration of alkylphenols (ng/L) in untreated municipal wastewater reported in international literature.

Alkylphenol		Country	Average concentration (ng/L)	Reference
Tech. 4-nonylphenol*	Tech. 4-NP	Germany	2,004 – 2,736[#]	Höhne and Püttmann (2008)
4-nonylphenol	4-NP	Greece	1,574 ± 1,063 (dissolved phase) 160 ± 108 (particulate phase)	Pothitou and Voutsa (2008)
4-*n*-nonylphenol	4-*n*-NP	Greece	230	Stasinakis et al. (2008)
Nonylphenol	NP	Italy	11,000 – 13,000[@]	Di Corcia et al. (1994)
		Spain	131,000	Solé et al. (2000)
		Serbia	4.9	Čelić et al. (2020)
		Hong Kong	646 – 2,235 wet season 907 – 1,468 dry season	Xu et al. (2014)^
		Japan	3,109	Nakada et al. (2006)
4-Nonylphenol monoethoxylate	NP1EO	Greece	2,224 ± 1,772 (dissolved phase) 152 ± 94 (particulate phase)	Pothitou and Voutsa (2008)
		Greece	5,760	Stasinakis et al. (2008)
4-Nonylphenol diethoxylate	NP2EO	Greece	1,479 ± 1,093 (dissolved phase) 92 ± 65 (particulate phase)	Pothitou and Voutsa (2008)
		Greece	3,990	Stasinakis et al. (2008)
Nonylphenol ethoxylates	NPEOs	USA	24,363	Lara-Martín et al. (2014)
		Italy	195,000 – 208,000[@]	Di Corcia et al. (1994)
		Spain	33,000	Solé et al. (2000)
Nonylphenol ethoxycarboxylates	NPEC	USA	5,465	Lara-Martín et al. (2014)
		Spain	8,000	Solé et al. (2000)
Octylphenol	OP	Serbia	1.9	Čelić et al. (2020)
		Japan	937	Nakada et al. (2006)

Table 30 continued.

Alkylphenol		Country	Average concentration (ng/L)	Reference
4-*t*-octylphenol	*t*-4-OP	Germany	363 – 745[#]	Höhne and Püttmann (2008)
4-*t*-octylphenol	*t*-4-OP	Greece	94 ± 157 (dissolved phase) 1.2 ± 0.61 (particulate phase)	Pothitou and Voutsa (2008)
4-*n*-octylphenol	*n*-4-OP	Greece	n.d. (dissolved phase) 0.45 ± 0.32 (particulate phase)	Pothitou and Voutsa (2008)
4-Octylphenol monoethoxylate	OP1EO	Greece	33.9 ± 21.0 (dissolved phase) 2.51 ± 1.53 (particulate phase)	Pothitou and Voutsa (2008)
4-Octylphenol diethoxylate	OP2EO	Greece	402 ± 355 (dissolved phase) 0.70 ± 0.54 (particulate phase)	Pothitou and Voutsa (2008)

*Tech. 4-nonylphenol is a complex mixture of different isomers (Höhne and Püttmann 2008). [#]Range represents the averages for two different WWTPs. Solé et al. (2000) data restricted to WWTP not receiving industrial contributions. ^Range represents the averages for three different WWTPs. [@] Range of averages from three different sample preparation methods.

Table 31. Concentration of alkylphenols in different French household greywater sources. Data from Deshayes et al. (2017).

Alkylphenol	Shower	Washing machine	Dishwasher	Manual dishwashing	Bathroom sink	Floor cleaning
OP	<LOQ – 1,560	<LOQ – 1,330	<LOQ – 1,710	<LOQ – 1,850	<LOQ – 5,140	<LOQ – 2,880
NP	490 – 5,800	490 – 25,800	300 – 3,770	220 – 1,530	220 – 7,030	350 – 29,300

Concentration range (ng/L) for dissolved + particulate phase.

9.3.2 Alkylphenols in stormwater

Several studies have identified various alkylphenols in stormwater (Table 32). A recent study assessed a variety of construction materials and automotive supplies that come into contact with rain for leaching of alkylphenols and their ethoxylates as summarised in Table 33 (Lamprea et al. 2018). The authors found that APs were ubiquitous in leachate from the various materials which included PVC, drainage materials, concrete, SBS-modified bitumen, polycarbonate, automotive bodies and tyres. The most commonly leached alkylphenol was NP, which was found in at concentrations from 1–10 ng/g in leachate from SBS-modified bitumen, automotive body samples, PVC and some concretes, whilst OP was only found at concentrations between 1-10 ng/g in leachate from tyres (Lamprea et al. 2018). It has previously been noted that both NP and OP are expected to be in stormwater due to their presence in building materials, paints, concrete, automotive parts and asphalt (Gasperi et al. 2014).

Several studies have noted that detergents used in car washes and domestically for cleaning private vehicles may be an important source of APEOs to stormwater (Björklund et al. 2009; Rule et al. 2006; Zhao et al. 2021). In a study assessing the presence of NPs in rainfall runoff in the UK, it was noted that the highest NP concentration was seen at the beginning of a rain event, which the authors suggest was reflective of the association of NPs with particulate matter (Rule et al. 2006).

Table 32. Concentration of alkylphenols (ng/L) in urban stormwater reported in international literature.

Alkylphenol		Country	Concentration range (ng/L)	Detection frequency (%)	Reference
4-*n*-nonylphenol	4-n-NP (straight chain)	Sweden	n.d.	0	Björklund et al. (2009)
Iso-nonylphenol	Iso-NP (branched)	Sweden	240 – 1,200	63	Björklund et al. (2009)
Iso-nonylphenol	4-NP	Sweden	270 - 1,100	-	Kalmykova et al. (2013)^
4-nonylphenol	4-NP (branched)	Sweden	100 – 500	57	Björklund et al. (2009)
Nonylphenol	NP	France	359 (mean)	>80	Gasperi et al. (2014)
		Australia	<10 – >2,500	46	Gernjak et al. (2017)
		China	18.7 ± 2.28 – 219 ± 28.1 (water) 21.3 ± 2.58 – 408 ± 21.1 (SPM)	100 100	Zhao et al. (2021)*
Nonylphenols	NPs	France	100 – 9,170	100	Zgheib et al. (2011)
Nonylphenol ethoxylates	NPEOs	United Kingdom	Up to 400,000 +	100	Rule et al. (2006)
Nonylphenol monoethoxylate	NP1EO	Sweden	1,100	13	Björklund et al. (2009)
		France	347 (mean)	>80	Gasperi et al. (2014)
		Sweden	921 - 1,160	-	Kalmykova et al. (2013)^
Nonylphenol diethoxylate	NP2EO	Sweden	2,000	13	Björklund et al. (2009)
		France	164 (mean)	>80	Gasperi et al. (2014)
		Sweden	695 - 2,380	-	Kalmykova et al. (2013)^
Nonylphenol triethoxylate	NP3EO	Sweden	2,200	13	Björklund et al. (2009)
		Sweden	441 - 2,740	-	Kalmykova et al. (2013)^
Nonylphenol tetraethoxylate	NP4EO	Sweden	900 – 2,100	25	Björklund et al. (2009)
		Sweden	172 - 1,530	-	Kalmykova et al. (2013)^

Table 32 continued.

Alkylphenol		Country	Concentration range (ng/L)	Detection frequency (%)	Reference
Nonylphenol pentaethoxylate	NP5EO	Sweden	n.d.	0	Björklund et al. (2009)
		Sweden	<100 - 1,250	-	Kalmykova et al. (2013)^
Nonylphenol hexaethoxylate	NP6EO	Sweden	n.d.	0	Björklund et al. (2009)
		Sweden	<100 - 451	-	Kalmykova et al. (2013)^
Nonylphenol acetic acid	NP1EC	France	466 (mean)	>80	Gasperi et al. (2014)
4-*t*-octylphenol	4-OP	France	61 (mean)	>80	Gasperi et al. (2014)
		France	<LOQ – 260	86	Zgheib et al. (2011)
		China	2.8 ± 0.21 – 50.3 ± 7.63 (water) 0.67 ± 0.07 – 121 ± 11.7 (SPM)	56 100	Zhao et al. (2021)*
		Australia	<10 – 4,900	63	Gernjak et al. (2017)
		Sweden	110 – 820	-	Kalmykova et al. (2013)^
Octylphenol monoethoxylate	OP1EO	France	23 (mean)	62	Gasperi et al. (2014)
		Sweden	<10 - 42	-	Kalmykova et al. (2013)^
octylphenol diethoxylate	OP2EO	France	10 (mean)	>80	Gasperi et al. (2014)
		Sweden	<10	-	Kalmykova et al. (2013)^
octylphenol triethoxylate	OP3EO	Sweden	<10	-	Kalmykova et al. (2013)^
octylphenol tetraethoxylate	OP4EO	Sweden	<10	-	Kalmykova et al. (2013)^
4-*t*-butylphenol	4-*t*-BP	France	<LOQ – 200	86	Zgheib et al. (2011)
		Sweden	82 – 480	-	Kalmykova et al. (2013)^
4-*t*-pentylphenol	4-*t*-PP	Sweden	<10	-	Kalmykova et al. (2013)^

SPM, suspended particulate matter. *Only samples taken from street runoff were included. ^Only including stormwater from urban areas not from the waste-sorting sites.

Table 33. Concentrations of APs and APEOs (ng/L) in leachate from construction materials and automotive supplies. Adapted from Lamprea et al. (2018).

Materials	Type	4-NP	NP1EO	NP2EO	NP1EC	OP	OP1EO	OP2EO
Blanks (mean)		30	41	40	3	5	< LQ	2
PVC	New gutter 1	420	78	85	11	17	< LQ	5
	New gutter 2	220	19	58	9	12	< LQ	1
	New gutter 3	660	500	390	180	68	12	14
	Old gutter 1	220	30	47	16	6	< LQ	2
	Old gutter 2	310	20	69	21	11	< LQ	2
	New roller blind	150	28	43	15	10	6	3
	Old roller blind	250	23	35	16	7	< LQ	2
Concrete	Sidewalk 2	680	99	180	2300	14	< LQ	7
	Sidewalk 4	390	52	57	210	26	9	7
Polycarbonate	Panel	61	110	63	4	4	< LQ	2
Drainage	Layer in polypropylene	170	29	34	3	9	< LQ	2
	Layer in polystyrene	46	24	44	7	5	< LQ	2
	Filtering geotextile	88	23	37	9	8	< LQ	2
SBS	Sealing membrane 1	690	11	12	190	28	< LQ	3
	Sealing membrane 2	800	24	22	27	5	< LQ	1
Tire	Car 1	790	92	38	21	170	< LQ	< LQ
	Car 2	200	< LQ	14	< LQ	900	< LQ	< LQ
Steel	Lacquered	32	30	24	3	6	< LQ	1
Bodywork	Door	110	33	60	17	16	40	58
	Roof	100	68	47	7	16	< LQ	2
	Hood	37	180	200	110	14	8	17

9.3.3 Health effects of alkylphenols

The exact human health hazard posed by alkylphenols is an area requiring further investigation. However, a previous report prepared for the Ministry of Health assessed available information on the potential health risks posed by NP and NPEOs, and noted that the main health concerns associated with these chemicals is due to structural similarity to the female sex hormone estrogen (17β-estradiol) (Cressey 2018). The California Environmental Protection Agency (CEPA) assessed the toxicity of NP and found sufficient evidence that it causes reproductive effects in laboratory animals but that there was limited information on possible human reproductive effects (CEPA 2009). They also noted that there was some evidence for effects on the nervous and immune systems based on animal studies, but no evidence of carcinogenicity (CEPA 2009).

Several studies have noted that the products of APEO degradation are more toxic than their parent substances (reviewed by Ying et al. (2002)). Both NPEOs and OPEOs are unstable in

the environment and are known to break down into more stable and persistent compounds, with NPEOs breaking down to the more toxic and persistent NP (Priac et al. 2017).

In the EU, NPs and NPEOs have been placed on the EU Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Candidate list of substances of very high concern for Authorisation[123,124], which "aims to ensure that substances of very high concern (SVHCs) are progressively replaced by less dangerous substances or technologies where technically and economically feasible alternatives are available"[125]. Information on the potential human health effects of APs/APEOs listed by the European Chemicals Agency (ECHA) is summarised in Table 34. Nonylphenol, 4-NP and NPEOs are all classified as endocrine-disrupting, and NP is suspected to be toxic to reproduction. In contrast, OP and OPEOs were not noted to be classified as endocrine disrupting or as substances of very high concern on the REACH candidate list for Authorisation. In New Zealand, the EPA has classified both NP and 4-NP as "harmful if swallowed" and they are noted to causes severe skin burns and serious eye damage[126],[127].

Recommended exposure limits have been set for NP and NPEOs by the Danish Veterinary and Food Administration (DVFA), with TDIs of 5 µg/kg bw/day, and 13 µg/kg bw/day, respectively (Nielsen et al. 2000).

[123] https://echa.europa.eu/candidate-list-table/-/dislist/details/0b0236e1807db370 Accessed 16 December 2022
[124] https://echa.europa.eu/candidate-list-table/-/dislist/details/0b0236e1807df0ea Accessed 16 December 2022
[125] https://echa.europa.eu/substances-of-very-high-concern-identification-explained Accessed 16 December 2022
[126] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/59DF1719-D788-4EF1-9260-1551B559DA6B Accessed 15 December 2022
[127] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/F94128D9-B89D-4974-93FA-01BF21BDF38B Accessed 15 December 2022

Table 34. Health effects of selected alkylphenols, as reported by the European Chemicals Agency.

Alkylphenol		Properties of concern	Additional information	Reference
Nonylphenol	NP	• Suspected to be toxic to reproduction • Endocrine disrupting	• Substance of very high concern included in the candidate list for authorisation. • Some uses restricted under ANNEX XVII of REACH	https://echa.europa.eu/substance-information/-/substanceinfo/100.042.414
Nonylphenol, ethoxylated	NPEO	• Endocrine disrupting • Majority of submitters to ECHA agree this substance is persistent, bioaccumulative and toxic	• Substance of very high concern included in the candidate list for authorisation. • Requires authorisation before it is used under Annex XIV of REACH. • Some uses restricted under ANNEX XVII of REACH.	https://echa.europa.eu/substance-information/-/substanceinfo/100.105.533
4-nonylphenol (branched and linear)	4-NP	• Endocrine disrupting	• Substance of very high concern included in the candidate list for authorisation.	https://echa.europa.eu/substance-information/-/substanceinfo/100.239.149
Octylphenol	OP	• Causes serious eye damage and skin irritation		https://echa.europa.eu/substance-information/-/substanceinfo/100.060.634
Octylphenol, ethoxylated	OPEO	• Causes serious eye damage and skin irritation; harmful if swallowed		https://echa.europa.eu/substance-information/-/substanceinfo/100.190.682

9.4 PHTHALATES

Phthalates are a diverse group of lipophilic chemicals, often referred to as phthalate esters (PEs) or phthalic acid esters (PAEs) (Huang et al. 2021). These chemicals are commonly employed as plasticisers in polymer production, most notably in production of polyvinyl chloride (PVC) (reviewed by Huang et al. (2021) and US Environmental Protection Agency (2013)). Phthalates have been, and in some cases are still used, in production of a diverse range of products including home furnishings, wall and floor coverings, building materials, food packaging, children's toys, cosmetics and personal care products, and medical supplies (reviewed by Huang et al. (2021) and US Environmental Protection Agency (2013)). As these chemicals are not strongly bound to these products they can leach out into the environment (reviewed by US Environmental Protection Agency (2013)). Once in the environment, these chemicals tend to adsorb onto particulate matter (Clara et al. 2010).

One of the most common phthalates is di(2-ethylhexyl) phthalate (DEHP), which has been estimated to account for around 80% of the phthalates produced in China (Meng et al. 2014) and around a third of those produced in the EU (Huang et al. 2008).

9.4.1 Phthalates in wastewater

A wide variety of different phthalates have been detected in untreated municipal wastewater, as summarised in Table 35. As noted above, phthalates may leach from final products as they are not chemically bonded to the polymer (Fromme et al. 2002). As such, there is a variety of potential sources to municipal wastewater due to the use of phthalates in a wide range of consumer products. The levels of various phthalates have also been quantified in different types of greywater from French households (Deshayes et al. 2017). This study revealed that greywater from washing machines and showering contained the highest amounts of phthalates compared with other domestic inputs such as dishwashing or the bathroom sink. Phthalates have also been identified in industrial effluents, as discussed in Eaton (2022); thus, where industrial effluents are discharged to the municipal wastewater network this may add to the final load of phthalates reaching WWTPs.

The removal of phthalates by WWTPs appears highly variable, depending on the phthalate and type of WWTP (Nas et al. 2022). Additionally, a Turkish study found negative removal efficiencies for benzyl butyl phthalate (BBP), DEHP and di-*n*-nonyl phthalate (DNOP), which the authors proposed could be due to release of phthalates from microplastics during treatment or desorption from the treatment sludge (Nas et al. 2022).

Table 35. Concentration of phthalates (ng/L) in untreated municipal wastewater reported in international literature.

Phthalate		Country	Mean concentration (ng/L)	Reference
Benzyl butyl phthalate	BBP	France	1,120 ± 540	Dargnat et al. (2009)
		Australia	62.1 ± 19.5 – 4,100 ± 5590	Tan et al. (2007)
		Denmark	390 ± 300	Fauser et al. (2003)
		China	3,660	Gao et al. (2014)
		Turkey	117	Nas et al. (2022)
		Austria	950	Clara et al. (2010)
Dibutyl phthalate	DBP	France	1,100 ± 370	Dargnat et al. (2009)
		Australia	173 ± 202 – 804 ± 474	Tan et al. (2007)
		Denmark	1,030	Fauser et al. (2003)
		China	14,340	Gao et al. (2014)
		Austria	2,200	Clara et al. (2010)
Di(2-ethylhexyl) phthalate	DEHP	France	22,460 ± 13,220	Dargnat et al. (2009)
		Australia	312 ± 103 – 2,240 ± 1,380	Tan et al. (2007)
		Denmark	35,400 ± 10,600	Fauser et al. (2003)
		China	16,860	Gao et al. (2014)
		Turkey	4393	Nas et al. (2022)
		Austria	18,000	Clara et al. (2010)
		Finland	209,000	Marttinen et al. (2003)
Diethyl phthalate	DEP	France	7,710 ± 5,210	Dargnat et al. (2009)
		Australia	1,080 ± 74.5 – 8,080 ± 4,340	Tan et al. (2007)
		China	8,070	Gao et al. (2014)
		Austria	4,100	Clara et al. (2010)
Dimethyl phthalate	DMP	France	820 ± 1,130	Dargnat et al. (2009)
		China	4,930	Gao et al. (2014)
		Austria	950	Clara et al. (2010)
Di-*n*-nonyl phthalate	DNP	Denmark	440 ± 170	Fauser et al. (2003)
Di-*n*-octyl phthalate	DNOP	France	100 ± 160	Dargnat et al. (2009)
		Denmark	570 ± 190	Fauser et al. (2003)
		China	8,080	Gao et al. (2014)
		Austria	490	Clara et al. (2010)
		Turkey	213	Nas et al. (2022)
Di-*n*-pentyl phthalate	DPP	Denmark	70 ± 50	Fauser et al. (2003)

Values from Fauser et al. (2003) represent 8-day mean concentrations. Values for Tan et al. (2007) represent a range for the mean concentrations for grab samples taken from five different WWTPs.

9.4.2 Phthalates in stormwater

Several studies from around the world have found a range of phthalates in stormwater, as summarised in Table 36. A variety of potential sources of phthalates to stormwater have been identified including road and traffic associated sources such as tyre wear, car care products, road paint, bitumen/asphalt, fuels, oils and lubricants (Markiewicz et al. 2017), and runoff from building surface materials (Müller et al. 2019).

A study of 15 different phthalates in six major stormwater drains in Hong Kong found phthalate levels were higher during the wet season compared to the dry season (2520 ± 2050 ng/L and 947 ± 904 ng/L), respectively, and identified spatial variation in phthalate levels which the authors speculate was due to differences in land use and intensity of human activity in the different catchments (Cao et al. 2022). The authors also noted that the levels of phthalates detected in the stormwater samples were one order of magnitude higher than those reported by Wu et al. (2017) for effluents from three WWTPs in Hong Kong. In contrast, Clara et al. (2010) found that phthalate concentrations were generally higher in effluents taken from 17 Austrian municipal WWTPs than in two road runoff samples assessed in the same study.

Table 36. Concentration of phthalates (ng/L) in urban stormwater reported in international literature.

Phthalate		Country	Concentration range (ng/L)	Detection frequency (%)	Reference
Benzyl butyl phthalate	BBP	USA	430 – 16,700	30	Masoner et al. (2019)
		Sweden	150	8	Björklund et al. (2009)
		Austria	14 – 330	63	Clara et al. (2010)
		Hong Kong	21	6	Cao et al. (2022)
		Iran	1,050 – 3,480	100	Hajiouni et al. (2022)
Bis(2-butoxyethyl) phthalate	DBEP	Hong Kong	696	56	Cao et al. (2022)
Dibutyl phthalate	DBP	USA	200 – 4,100	38	Masoner et al. (2019)
		Sweden	110 – 450	31	Björklund et al. (2009)
		Hong Kong	22.9 – 1,220	100	Cao et al. (2022)
		Austria	79 – 270	63	Clara et al. (2010)
		Iran	2,390 – 6,670	100	Hajiouni et al. (2022)
Dicyclohexyl phthalate	DCHP	Hong Kong	8.44	19	Cao et al. (2022)
Di(2-ethylhexyl) phthalate	DEHP	USA	220 – 19,000	52	Masoner et al. (2019)
		Sweden	1,400 – 5,000	23	Björklund et al. (2009)
		Hong Kong	77.7 – 1,740	100	Cao et al. (2022)
		France	3,000 – 58,000	100	Zgheib et al. (2011)
		Austria	450 – 24,000	100	Clara et al. (2010)
		United Kingdom	~700 – 1,400	100	Rule et al. (2006)
		Iran	80 –91,070	100	Hajiouni et al. (2022)
		Germany	14,000*	-	Wicke et al. (2021)

Table 36 continued

Phthalate		Country	Concentration range (ng/L)	Detection frequency (%)	Reference
Diethyl phthalate	DEP	USA	50 – 2,300	74	Masoner et al. (2019)
		Sweden	100 – 390	62	Björklund et al. (2009)
		Hong Kong	13.2 – 78,500	100	Cao et al. (2022)
		Austria	<20 – 270	100	Clara et al. (2010)
		Iran	500 – 2,030	100	Hajiouni et al. (2022)
Diisobutyl phthalate	DIBP	Hong Kong	17.5 – 344	100	Cao et al. (2022)
Diisodecyl phthalate	DIDP	Sweden	580 – 17,000	69	Björklund et al. (2009)
		Austria	530 – 9,900	38	Clara et al. (2010)
Diisononyl phthalate	DINP	Sweden	350 – 85,000	69	Björklund et al. (2009)
		Austria	220 – 23,000	100	Clara et al, (2010)
Diisodecyl phthalate and diisononyl phthalate	DIDP + DINP	Germany	130,000*	-	Wicke et al. (2021)
Diisopentyl phthalate	DIPP	Hong Kong	67.6	33	Cao et al. (2022)
Dimethyl phthalate	DMP	USA	82 – 1,940	22	Masoner et al. (2019)
		Sweden	130 – 230	23	Björklund et al. (2009)
		Hong Kong	3.24 – 1,640	100	Cao et al. (2022)
		Austria	<10 – 79	75	Clara et al. (2010)
		Iran	1,120 – 36,200	100	Hajiouni et al. (2022)
Di-*n*-octyl phthalate	DNOP	USA	270 – 3,760	8	Masoner et al. (2019)
		Sweden	160	8	Björklund et al. (2009)
		Austria	10 – 530	88	Clara et al. (2010)
		Iran	620 – 4,800	100	Hajiouni et al. (2022)
Di-*n*-pentyl phthalate	DPP	Hong Kong	1.62	6	Cao et al. (2022)

*Maximum concentrations detected.

9.4.3 Health effects of phthalates

Several phthalates reported to be present in wastewater or stormwater are known to have potential effects on human health, particularly reproduction. In the EU, several phthalates are included in the REACH candidate list for Authorisation. Information on the potential human health effects of the phthalates identified in wastewater or stormwater studies cited in this report from the ECHA is summarised in Table 37.

Of the 14 phthalates reported in wastewater or stormwater, seven are noted by the ECHA as toxic to reproductive systems and five are noted as endocrine-disrupting. Several of these phthalates have also been classified as hazardous by the New Zealand Environmental Protection Authority (Table 38). Additionally, DEHP, DBP, BBP, DIBP, DINP, DIDP and DNOP were previously evaluated in a health risk assessment prepared by ESR for the Ministry of Health (Ashworth and Chappell 2015). This assessment noted that DEHP, DBP, BBP and DIBP had “reproductive or developmental (antiandrogenic) concerns” and DINP, DNOP and DIDP had “hepatoxicity concerns” (Ashworth and Chappell 2015).

Recommended exposure limits have been set for a number of these phthalates by the US EPA, ATSDR and EFSA (Table 39). The EFSA set a group TDI of 50 µg/kg bw/day for DBP, BBP, DEHP and DINP due to similar reproductive effects, with DEHP as the index compound as “it has the most robust underlying toxicological dataset” [128]. A separate TDI of 0.15 mg/kg bw/day was set for DIDP due to liver effects[129]. The EFSA is also currently re-evaluating the risks posed by phthalates due to migration from food contact materials, expanding their assessment to include other phthalates, as they note that phthalates that have previously been assessed are largely being replaced by other plasticisers[130].

[128] https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2019.5838 Accessed 14 December 2022
[129] https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2019.5838 Accessed 14 December 2022
[130] https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/sp.efsa.2022.EN-7660 Accessed 14 December 2022

Table 37. Health effects of selected phthalates as reported by the European Chemicals Agency.

Phthalate		Properties of concern	Additional information	Reference
Benzyl butyl phthalate	BBP	• Toxic to reproduction • Endocrine disrupting	• Substance of very high concern included in the candidate list for authorisation. • Authorisation required before it is used under ANNEX XIV of REACH. • Some uses restricted under ANNEX XVII of REACH	https://echa.europa.eu/substance-information/-/substanceinfo/100.001.475
Dibutyl phthalate	DBP	• Toxic to reproduction • Endocrine disrupting • Under assessment as persistent, toxic and bioaccumulative	• Substance of very high concern included in the candidate list for authorisation. • Authorisation required before it is used under ANNEX XIV of REACH. • Some uses restricted under ANNEX XVII of REACH	https://echa.europa.eu/substance-information/-/substanceinfo/100.001.416
Diisobutyl phthalate	DIBP	• Toxic to reproduction • Endocrine disrupting	• Substance of very high concern included in the candidate list for authorisation. • Authorisation required before it is used under ANNEX XIV of REACH. • Some uses restricted under ANNEX XVII of REACH	https://echa.europa.eu/substance-information/-/substanceinfo/100.001.412

Table 37 continued.

Phthalate		Properties of Concern	Additional Information	Reference
Diisopentyl phthalate	DIPP	• Toxic to reproduction	• Substance of very high concern included in the candidate list for authorisation. • Authorisation required before it is used under ANNEX XIV of REACH.	https://echa.europa.eu/substance-information/-/substanceinfo/100.009.172
Diethyl phthalate	DEP	• Under assessment as endocrine disrupting		https://echa.europa.eu/substance-information/-/substanceinfo/100.001.409
Di(2-ethylhexyl) phthalate	DEHP	• Toxic to reproduction • Endocrine disrupting	• Substance of very high concern included in the candidate list for authorisation. • Authorisation required before it is used under ANNEX XIV of REACH. • Some uses restricted under ANNEX XVII of REACH	https://echa.europa.eu/substance-information/-/substanceinfo/100.003.829
Dimethyl phthalate	DMP	• No hazards classified		https://echa.europa.eu/substance-information/-/substanceinfo/100.004.557
Diisononyl phthalate	DINP	• No hazards classified		https://echa.europa.eu/substance-information/-/substanceinfo/100.044.602
Dicyclohexyl phthalate	DCHP	• Toxic to reproduction • Endocrine disrupting • Skin sensitising	• Substance of very high concern included in the candidate list for authorisation.	https://echa.europa.eu/substance-information/-/substanceinfo/100.001.405

Table 37 continued.

Phthalate		Properties of Concern	Additional Information	Reference
Di-*n*-octyl phthalate	DNOP	• No hazards classified	• Some uses of this substance are restricted under Annex XVII of REACH.	https://echa.europa.eu/nl/substance-information/-/substanceinfo/100.003.832
Di-*n*-pentyl phthalate	DPP	• Toxic to reproduction	• Substance of very high concern included in the candidate list for authorisation. • Authorisation required before it is used under ANNEX XIV of REACH.	https://echa.europa.eu/nl/substance-information/-/substanceinfo/100.004.563
Di-*n*-nonyl phthalate	DNP	• Harmful if swallowed		https://echa.europa.eu/nl/substance-information/-/substanceinfo/100.001.418

DBEP and DIDP were not identified in the ECHA database.

Table 38. Classification of phthalates by the New Zealand Environmental Protection Authority.

Phthalate		Classification
Di-*n*-butyl phthalate	DBP	May damage fertility or the unborn child; Serious eye irritation[131]
Diisobutyl phthalate	DIBP	Suspected of damaging fertility or the unborn child[132]
Diethyl phthalate	DEP	Acute toxicity – harmful if inhaled or swallowed[133]
Dimethyl phthalate	DMP	Acute toxicity – harmful if inhaled or swallowed[134]
Di(2-ethylhexyl) phthalate	DEHP	May damage fertility or the unborn child; May cause damage to organs through prolonged or repeated exposure: hepatotoxicity, renal toxicity, effects on blood and hematopoietic system[135]
Di-*n*-nonyl phthalate	DNP	Acute toxicity - harmful if swallowed[136]
Diisononyl phthalate	DINP	Skin and serious eye irritation[137]

Table 39. Recommended exposure limits for phthalates.

Phthalate		Minimum Risk Level, ATSDR[138] (µg/kg/ bw/day)	Reference Dose, US EPA (µg/kg/ bw/day)[139]	Tolerable Daily Intake, EFSA[140] (µg/kg/ bw/day)
Butyl benzyl phthalate	BBP		200	50 (total for BBP, DBP, DEHP, DINP)
Di-*n*-butyl phthalate	DBP	500 (acute)	100	
Di(2-ethylhexyl) phthalate	DEHP	3 (acute) 0.1 (int.)	20	
Diisononyl phthalate	DINP		Assessment suspended/ discontinued	
Diethyl phthalate	DEP	7,000 (acute) 6,000 (int.)	800	
Di-*n*-octylphthalate	DNOP	3,000 (acute) 400 (int.)		
Diisodecyl phthalate	DIDP			150

US EPA Reference Doses and ATSDR Minimum Risk Levels are for oral exposures.
int. – intermediate exposure duration.

[131] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/F1CAD33D-608F-4C22-BCCF-98BA3726559D Accessed 14 December 2022
[132] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/1D671A8C-C16B-4F05-8BB5-0336C055D877 Accessed 14 December 2022
[133] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/BC2FC065-E748-45A6-9B79-A5F7C95F2944 Accessed 14 December 2022
[134] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/A558777F-6FC7-4867-A2AE-73D671405527 Accessed 14 December 2022
[135] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/4AAB262A-4C27-4B20-8C25-F3C1C34CDFC2 Accessed 14 December 2022
[136] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/2AE76A87-CA6D-491F-AE20-4384A3F2CD67 Accessed 14 December 2022
[137] https://www.epa.govt.nz/database-search/chemical-classification-and-information-database-ccid/view/132C1010-D310-41A4-AB24-5CDA345F160F Accessed 14 December 2022
[138] https://wwwn.cdc.gov/TSP/MRLS/mrlsListing.aspx Accessed 14 December 2022
[139] https://iris.epa.gov/AtoZ/?list_type=alpha Accessed 14 December 2022
[140] https://www.foodpackagingforum.org/news/efsa-updated-risk-assessment-of-five-phthalates Accessed 14 December 2022

10. BROMINATED FLAME RETARDANTS

Brominated flame retardants (BFRs) are organobromine compounds that have an inhibitory effect on combustion chemistry, and are added to a wide variety of products, both industrial and consumer, to make them less flammable.[141] They are commonly used in plastics, textiles, epoxy resins, electrical equipment and consumer electronics (eg wiring casings, connectors, printed circuit boards), thermal insulation, building materials, children's toys, furniture and upholstery, and polystyrene foams (Daso et al. 2010; Schlabach et al. 2011; Feiteiro et al. 2021). Many different BFRs are produced synthetically, with widely varying chemical properties. The five main classes of BFRS[142] are:

- *Polybrominated diphenyl ethers (PBDEs).* There are 209 possible congeners, in mono- through deca- homologues. This is the most extensively studied group of BFRs.
- *Polybrominated biphenyls (PBBs).* Also has 209 possible congeners. Similar in structure to PCBs. However, these have been rarely used since the 1970s.
- *Hexabromocyclododecane (HBCD).* There are 16 possible stereoisomers, of which α-, β-, and γ-HBCD are most prevalent.
- *Tetrabromobisphenol A (TBBPA),* and other phenols.
- Other brominated flame retardants (eg phthalic acid derivatives).

Brominated flame retardants may be classified as either additive or reactive. Additive BFRs like PBDEs and HBCD are not chemically bonded to the polymeric materials and are therefore more likely to diffuse or leach out of the product over its lifetime when compared with reactive BFRs like TBBPA that are covalently bonded to product polymers (Daso et al. 2010; Schlabach et al. 2011; Feiteiro et al. 2021). Brominated flame retardants are commonly applied to consumer products in the form of commercial mixtures, each of which is dominated by certain congeners or isomers. For example, PBDEs were used in three key commercial formulations, known as PentaBDE, OctaBDE and DecaBDE[143]; PentaBDE was dominated by the congeners BDE-47 (38-42%) and BDE-99 (45-49%), but also contained a

[141] https://www.efsa.europa.eu/en/topics/topic/brominated-flame-retardants Accessed 1 March 2023
[142] https://www.efsa.europa.eu/en/topics/topic/brominated-flame-retardants Accessed 1 March 2023
[143] Note that the use of a capital letter denotes reference to the commercial formulation, while the use of lower case describes the isomers of that homologue. For example, pentaBDE refers to isomers of pentabromodiphenyl ether (PBDE congenrs 82-127), where Penta-BDE refers to the commercial mixture dominated by pentaBDE congeners.

number of other tri-, tetra-, penta- and hexa-BDE congeners at lower or trace concentrations (Sharkey et al. 2020).

Some BFRs are highly stable and persistent in the environment, and together with their widespread use, means they are commonly detected in air, fresh surface waters, coastal marine waters, sediments, soils, wildlife and humans around the world (EFSA 2011a; US EPA 2020; Feiteiro et al. 2021). They are generally hydrophobic and lipophilic, binding to soils and other particulates and some have been shown to bioaccumulate and biomagnify (EFSA 2011a). Some have been shown to be toxic to both humans and the environment. As a result, BFRs have been subject to significant scrutiny by regulatory agencies and the use of many has been prohibited or heavily restricted (Sharkey et al. 2020). For example, five BFRs (or groups of BFRs) have been listed under Annex A of the Stockholm Convention: the PBB hexabromobiphenyl (HBB); HBCD; commercial Penta-BDE; commercial Octa-BDE; and commercial Deca-BDE[144] (Sharkey et al. 2020). Tetrabromodiphenol A and HBCD are listed in the Basel Convention, and PBBs are heavily restricted or prohibited from manufacture and use in the United States, EU and China, among other jurisdictions. However, the extensive historic use of these compounds means that concerns remain as to their potential effects on public health due to their presence in existing products (either still in use or disposed of as waste) and in the environment.[145]

As a result of restrictions on the use of PBFRs mentioned above, an increasing number of alternative flame retardant compounds are utilised to ensure that consumer products continue to comply with fire safety standards, including both existing ("emerging") and novel compounds (Schalbach et al. 2011; EFSA 2012; Wang et al. 2020). Examples of these alternative compounds include 1,2-bis(2,4,6-tribromophenoxy)ethane (BTBPE), decabromodiphenyl ethane (DBDPE), 2,4,6-tribromophenyl allyl ether (ATE), tetrabromo-o-chlorotoluene (TBCT), pentabromobenzyl acrylate (PBBA), and bis(2-ethylhexyl)-3,4,5,6-tetrabromo-phthalate (TBPH). Although little is known about their environmental fate and toxicity, given their common properties with regulated BFRs and considering that most are additive flame retardants, similar environmental fates may be expected (Schalbach et al. 2011). Indeed, some novel BFRs are already being widely detected in the environment (Wang et al. 2020), and a number have been identified as being highly persistent and/or

[144] The Stockholm Convention refers to specific chemical substances related to these BFRs based on their Chemical Abstracts Service (CAS) registry number in addition to some non-specific references, namely "… other hexa- and heptabromodiphenyl ethers present in commercial Octa-BDE" and "… other tetra- and pentabromodiphenyl ethers present in commercial Penta-BDE" (Sharkey et al. 2020).
[145] https://www.efsa.europa.eu/en/topics/topic/brominated-flame-retardants Accessed 1 March 2023.

having significant genotoxic, carcinogenic or bioaccumulative potential (EFSA 2012; Wang et al. 2020).

10.1 BROMINATED FLAME RETARDANTS IN WASTEWATER

Municipal wastewaters are well-recognised as key sources of BFRs to the environment (Peng et al. 2012). Sources of BFRs into wastewater include household dusts, human excretion (following inhalation or ingestion of dust, or possibly dermal exposure), or laundering of textiles that have been treated with flame retardants (Daso et al. 2010; EFSA 2011a; Schreder and La Guardia 2014; ATSDR 2017; Feiteiro et al. 2021). Effluents from manufacturing and/or recycling facilities and landfill leachates may also be important sources to networks receiving these inputs (Daso et al. 2010; Kim et al. 2013).

Polybrominated diphenyl ethers are the most well-studied group of BFRs, and a large number of different congeners have been detected in municipal wastewater (Table 40). For example, Raye and Ikonomou (2005) reported that 46 different PBDE congeners were detected in at least 30% of the samples collected from a Canadian WWTP, and Kim et al. (2013) similarly reported detecting 31 PBDE congeners had detection frequencies of at least 35% across 20 Canadian WWTP. Congeners BDE-47, -99, -100, -153 and -209 are most frequently detected, with -47, -99 and -209 in particular reported to dominate wastewater PBDE loads, likely reflecting their dominance in commercial mixtures (North 2004; Raye and Ikonmou 2005; Vogelsang et al. 2006; Hope et al. 2012; Peng et al. 2012; Kim et al. 2013; Wang et al. 2013). For example, Peng et al. (2012) reported BDEs -47, -99 and -209 accounted for 87.6 to 99.5% of the total concentration of PBDEs assayed. The relative abundance of different congeners typically reflects local usage patterns (eg current use or historic use in products still in circulation); for example, studies from Asia often report the dominance of BDE209, reflecting the primary use of Deca-BDE (Wang et al. 2013), while studies from North America typically report the dominance of BDEs -47 and 99, consistent with greater usage of Penta-BDE (Song et al. 2006; Peng et al. 2012).[146] Total PBDE concentrations in raw wastewaters range from pg/L or low n/gL (Vogelsang et al. 2006; Kim and Oh 2018) to several µg/L (Peng et al. 2009, 2012; Wang et al. 2013).

Hexabromocyclododecane (predominantly α-, β-, and γ-HBCD isomers) have also been detected in municipal wastewaters, as have TBBPA and related metabolites, with

[146] Note that wastewater concentrations reflected local usage patterns at the time of study. Additional regulatory measures implemented since studies were undertaken, such as the addition of Deca-BDE to Annex A of the Stockholm Convention, may have led to changes in usage patterns.

concentrations typically in the low ng/L range (Potvin et al. 2012; Ichihara et al. 2014; de Guzman 2016; Kim and Oh 2018). In addition, a number of novel and emerging BFRs have been detected in raw wastewater or in sewage sludges (indicative of their presence in wastewater), including DBDPE, BTBPE, ATE, bis(2-ethylhexyl)-2,3,4,5-tetrabromophthalate (TBPH), 2-ethylhexyl-2,3,4,5-tetrabromobenzoate (TBB), 1,2-dibromo-4-(1,2 dibromoethyl)cyclohexane (TBECH) and tetrabromobisphenol A bis(2-hydroxyethyl) ether (TBBPA-DBPE) (Covaci et al. 2011; Gorga et al. 2013; Nyholm et al. 2013). Data on the concentrations and fates of these compounds is limited, but concentrations largely appear to be <10 ng/L (Nyholm et al. 2013; Margot et al. 2015).

The presence of PBBs in wastewater has received little attention, possibly due to their prohibition in some markets in the 1970s and the assumption that products in which they would have been used will now have reached the end of their life and therefore been landfilled, recycled or otherwise disposed of. In 2003, a study of environmental samples from the Netherlands that included WWTP influents, effluents and sludges did not detect any of the analysed PBB congeners (-15, -49, -52, -101, -153, -169, -209) at detection limits between 0.1 and 1.0 µg/kg dry weight[147] (de Boer et al. 2003). However, Daso et al. (2012) reported the detection of the PBB congener BB-153 at concentrations up to 18.4ng/L in wastewater effluents from a WWTP in South Africa, and Oberg et al. (2002) and Clarke et al. (2010) reported its detection in sewage sludges in Sweden[148] and Australia, respectively, highlighting the potential for PBBs to be present in wastewaters.

Conventional wastewater treatment processes are typically effective in removing hydrophobic contaminants through sedimentation of suspended solids; overall elimination of BFRs into sewage sludges therefore tends to be high, and reduction of overall PBF loads from influent to effluent is commonly reported in the range of 90% (Raye and Ikonmou 2005; Hale et al. 2006; Song et al. 2006; Potvin et al. 2012; Wang et al. 2013; Ichihara et al. 2014). Some variation appears to exist for individual congeners, with Wang et al. (2013) reporting removal rates between 30 and 100%, with lower brominated PBDEs being removed at a lower rate than higher brominated PBDEs.

[147] Wastewaters filtrates and the particulate matter analysed.
[148] This detection may have been due to analytical interference.

Table 40. Concentration of selected BFRs (ng/L) in untreated municipal wastewater reported in international studies.

	Detection frequency (%)	Concentration ng/L			Country	Reference
		mean	median	max		
PBDEs						
BDE7	3		0.16	0.19	US	Hope et al. (2012)
		0.016±0.0078	7.8		US	North (2004)
BDE8		0.0042±0.001	1		US	North (2004)
BDE15	16		0.18	1.95	US	Hope et al. (2012)
		0.0079±0.0054	5.4		US	North (2004)
BDE17		0.192±0.014	14		US	North (2004)
	64		0.21	1.64	US	Hope et al. (2012)
BDE25		0.0099±0.00045	0.45		US	North (2004)
BDE28		0.266±0.011	11		US	North (2004)
	100	0.1±0.03			Norway	Nyholm et al. (2013)
	100	0.2±0.1			Norway	Nyholm et al. (2013)
	100	0.5±0.6			Norway	Nyholm et al. (2013)
			<0.5		UK	Gardner et al. (2012)
		0.3941			Korea	Kim and Oh (2018)
		0.023±0.015			China	Wang et al. (2013)
		1.3±1.0			Canada	Song et al. (2006)
	75		0.26	1.79	US	Hope et al. (2012)
BDE35		0.0046±0.00006	0.06		US	North (2004)
BDE47		10.467±0.212	212		US	North (2004)
	100	20±12			Norway	Nyholm et al. (2013)
	100	21±29			Norway	Nyholm et al. (2013)
	100	105±152			Norway	Nyholm et al. (2013)
			0.7		UK	Gardner et al. 2012
		8.8±3.7	8.5		UK	Gardner et al. 2013
		0.172±0.049			China	Wang et al. (2013)
		3.48		11.063	China	Peng et al. (2012)
		102±83			Canada	Song et al. 2006
	44		8.1	22.4	US	Hope et al. (2012)
BDE49	18	18	0.52	3.51	US	Hope et al. (2012)
		0.266±0.018	18		US	North (2004)
BDE66		0.217±0.00071	0.71		US	North (2004)
	54		0.22	0.9	US	Hope et al. (2012)
BDE71	44		0.22	1.37	US	Hope et al. (2012)
		0.043±0.001	0.99		US	North (2004)
BDE75		0.018±0.00325	3.25		US	North (2004)
BDE85		0.352±0.013	13		US	North (2004)
	100	0.4±0.6			Norway	Nyholm et al. (2013)
	100	0.8±1.1			Norway	Nyholm et al. (2013)
	100	5±7			Norway	Nyholm et al. (2013)

Table 40 continued.

	Detection frequency (%)	Concentration ng/L			Country	Reference
		mean	median	max		
BDE99		1.639±0.867			China	Wang et al. (2013)
		3.404		10.488	China	Peng et al. (2012)
		121±93			Canada	Song et al. (2006)
	26		9.04	17.2	US	Hope et al. (2012)
		11.2±0.2	200		US	North (2004)
	100	11±6			Norway	Nyholm et al. (2013)
	100	23±32			Norway	Nyholm et al. (2013)
	100	137±201			Norway	Nyholm et al. (2013)
			0.6		UK	Gardner et al. (2012)
		10.5±4.9	10.7		UK	Gardner et al. (2013)
		0.317			Korea	Kim and Oh (2018)
BDE100		1.983±0.042	42		US	North (2004)
	12		2.5	3.4	US	Hope et al. (2012)
	100	2.6±1.5			Norway	Nyholm et al. (2013)
	100	4.3±6.4			Norway	Nyholm et al. (2013)
	100	29±44			Norway	Nyholm et al. (2013)
			<0.5		UK	Gardner et al. (2012)
		0.25			Korea	Kim and Oh (2018)
		0.242±0.149			China	Wang et al. 2013
		19±16			Canada	Song et al. (2006)
BDE119		0.014±0.0049	4.9		US	North (2004)
	1		0.22	0.22	US	Hope et al. (2012)
BDE126	6		0.18	0.42	US	Hope et al. (2012)
BDE138		0.096±0.014	14		US	North (2004)
	16		0.13	0.52	US	Hope et al. (2012)
		1±0.7			Canada	Song et al. (2006)
BDE139	16		0.13	0.33	US	Hope et al. (2012)
BDE140		0.031±0.0043	4.3		US	North (2004)
BDE153		0.259±0.196			China	Wang et al. (2013)
		11+9			Canada	Song et al. (2006)
		0.983±0.074	74		US	North (2004)
	100	2.2±1			Norway	Nyholm et al. (2013)
	100	1.9±2.7			Norway	Nyholm et al. (2013)
	100	20±29			Norway	Nyholm et al. (2013)
			<0.5		UK	Gardner et al. (2012)
		0.3171			Korea	Kim and Oh (2018)
BDE154		0.148±0.572			China	Wang et al. (2013)
		7.6±6.1			Canada	Song et al. (2006)
		0.776±0.045	45		US	North (2004)
			<0.5		UK	Gardner et al. (2012)
		0.1585			Korea	Kim and Oh (2018)

Table 40 continued

	Detection frequency (%)	Concentration ng/L			Country	Reference
		mean	median	max		
BDE155		0.073±0.0007	0.7		US	North (2004)
BDE183		0.085±0.064			China	Wang et al. (2013)
		1.7±1.2			Canada	Song et al. (2006)
		0.08±0.026	26		US	North (2004)
	1		0.3	0.3	US	Hope et al. (2012)
	33	0.1			Norway	Nyholm et al. (2013)
	67	0.2±0.06			Norway	Nyholm et al. (2013)
	33	0.6			Norway	Nyholm et al. (2013)
		0.22929			Korea	Kim and Oh (2018)
BDE190		0.0039±0.00019	0.19		US	North (2004)
BDE196	1		0.54	0.54	US	Hope et al. (2012)
		0.5986			Korea	Kim and Oh (2018)
BDE197		0.38336			Korea	Kim and Oh (2018)
BDE203	2	2	0.37	0.49	US	Hope et al. (2012)
BDE206		0.041±0.014	14		US	North (2004)
	12		0.56	1.67	US	Hope et al. (2012)
BDE207		0.095±0.083	83		US	North (2004)
	1		0.92	0.92	US	Hope et al. (2012)
BDE208		0.051±0.042	42		US	North (2004)
	3		0.55	0.72	US	Hope et al. (2012)
BDE209		1.73±0.652	652		US	North (2004)
	5		17.2	51	US	Hope et al. (2012)
	100	105±23			Norway	Nyholm et al. (2013)
	100	41±12			Norway	Nyholm et al. (2013)
	100	61±66			Norway	Nyholm et al. (2013)
		550.2		2,412	China	Peng et al. (2012)
		186±79.3			China	Wang et al. (2013)
		1.1521			Korea	Kim and Oh (2018)
BDE247		0.3363			Korea	Kim and Oh (2018)
$\sum_{27}$PBDE		7.568			Korea	Kim and Oh (2018)
∑PBDE		261±207			Canada	Song et al. (2006)
∑PBDE		29.023±1.49	1,490		US	North (2004)
$\sum_{8}$PBDE		265±210			Canada	Song et al. (2006)
∑PBDE		8.28±1.34		9.3	Norway	Vogelsang et al. (2006)
$\sum_{8}$PBDE		188.6±81.2			China	Wang et al. (2013)
$\sum$PBDE$_{(tri\ to\ hepta)}$		10.775		32.9	China	Peng et al. (2012)
∑5OH-BDEs		0.0752			Korea	Kim and Oh (2018)
∑8MeO-BDEs		0.1414			Korea	Kim and Oh (2018)

Table 40 continued.

	Detection frequency (%)	Concentration ng/L			Country	Reference
		mean	median	max		
HBCD						
α-HBC	83	6±10.3	2.3	39	Japan	Ichihara et al. (2014)
		0.858			Korea	Kim and Oh (2018)
β-HBCD	75	4.3±10.7	1.4	40	Japan	Ichihara et al. (2014)
		0.4439			Korea	Kim and Oh (2018)
γ-HBCD	100	67.5±83.6	4	320	Japan	Ichihara et al. (2014)
		1.759			Korea	Kim and Oh (2018)
δ-HBCD	0	<2	<2	<2	Japan	Ichihara et al. (2014)
ε-HBCD	0	<1	<1	<1	Japan	Ichihara et al. (2014)
∑HBCD		77.5±103.8	45	400	Japan	Ichihara et al. (2014)
		3.062			Korea	Kim and Oh (2018)
		1.2			China	De Guzman (2016)
TBBPA						
TBBPA	100	22.3±8.3	25	29	Canada	Potvin et al. (2012)
		2.499			Korea	Kim and Oh (2018)
Novel/emerging BFRs						
TBBPA-AE	0	nd			Norway	Nyholm et al. (2013)
TBBPA-DBPE	33	18			Norway	Nyholm et al. (2013)
TBECH	67	5.3±0.5			Norway	Nyholm et al. (2013)
	100	3.5±1.4			Norway	Nyholm et al. (2013)
	33	1.2			Norway	Nyholm et al. (2013)
DBDPE	67	5.1±5.6			Norway	Nyholm et al. (2013)

10.2 BROMINATED FLAME RETARDANTS IN STORMWATER

Although stormwaters are well-recognised as important pathways for the transport of urban contaminants, especially those that exhibit a strong tendency to adsorb to particulates such as higher brominated PBDEs (Sutton et al. 2019), there is relatively little data available in the literature describing the presence of BFRs in urban stormwater. Anticipated sources for BFRs to stormwaters include atmospheric deposition, inappropriate disposal of consumer products (especially electronic wastes), dust and debris from vehicles, and leaching from construction and building materials (Oram et al. 2008; Daso et al. 2010; Gasperi et al. 2022). Runoff from soils conditioned with sewage sludges may also be relevant in rural areas (Daso et al. 2010).

Much of the available data focuses on PBDEs, especially BDE-47 and BDE-209, although other congeners including BDE-28, -47, -66, -85, -99, -100, -138, -153, -154, and -183 are reported with lower prevalence (Table 41) (Gilbreath et al. 2012; Gasperi et al. 2014, 2022; Remberger et al. 2014; Sutton et al. 2014, 2019; Masoner et al. 2019). Reported concentrations range from tens of pg/L to tens of µg/L: Sutton et al. (2014) reported total PBDEs of 35 to 830 pg/L in California, while in France, Gasperi et al. (2014) reported that concentrations for BDE-209 alone ranged from 23 to 251 ng/L, accounting for 90% of the overall PBDE load, with other congeners present in the 0.5-3 ng/L range. Remberger et al. (2014) also reported the dominance of BDE-209 in stormwater from a heavily trafficked area of Sweden, at up to 31 µg/L compared with several ng/L to several µg/L for other PBDE congeners. Conversely, Birch et al. (2011) and Wick et al (2021) did not detect PBDEs in urban stormwaters from Copenhagen or Berlin, respectively.

In addition to PBDEs, HBCD isomers, TBBPA and a range of emerging contaminants including TBP-AE, BTPBE, DBDPE, EH-TBB, HBB, PBT, and BEHTBP are also reported in stormwaters, again mostly in the 0.2-3 ng/L range (Remberger et al. 2014; Vorkamp et al. 2014; Sutton et al. 2019; Gasperi et al. 2022).

Table 41. Concentration of selected BFRs (ng/L) in urban stormwater reported in international studies.

	Detection frequency %	Concentration (ng/L)			Country	Reference
		Mean ± SD	Median	Max		
PBDEs						
BDE28	75-100				France	Gasperi et al. (2014)
	50	0.40±0.23	0.40	0.6	US	Sutton et al. (2019)
	79		0.05		France	Gasperi et al. (2022)
BDE47	>80				France	Gasperi et al. (2014)
	6.1	19	18	25	US	Masoner et al. 2019
	100	12.66±8.96	10.20	33	US	Sutton et al. (2019)
	100		19	100	Sweden	Remberger et al. (2014)
				20	US	Gibreath (2012)
				26.5	US	Oram et al. (2008)
	23			18	US	Moreace (2012)
	100		0.2		France	Gasperi et al. (2022)
BDE49	87.5	3.59±3.71	1.70	9.7	US	Sutton et al. (2019)
BDE66	14.3	0.42	0.43	0.54	US	Masoner et al. 2019
	62.5	1.24±1.01	0.90	3	US	Sutton et al. (2019)
	27			0.45	US	Moreace (2012)
BDE85	8.2	1.06	0.96	2	US	Masoner et al. 2019
	0			<0.2	US	Sutton et al. (2019)
	27			0.1	US	Moreace (2012)
BDE99	50-100				France	Gasperi et al. (2014)
	16.3	15.75	8	43	US	Masoner et al. 2019
	100	10.54±7.72	6.95	27	US	Sutton et al. (2019)
	100		28	230	Sweden	Remberger et al. (2014)
	38			22	US	Moreace (2012)
	100		0.3		France	Gasperi et al. (2022)
BDE100	75-100				France	Gasperi et al. (2014)
	12.2	3.22	2.8	6.7	US	Masoner et al. 2019
	87.5	3.76±3.80	2.70	12	US	Sutton et al. (2019)
	100		7.8	33	Sweden	Remberger et al. (2014)
	31			4.2	US	Moreace (2012)
	93		0.1		France	Gasperi et al. (2022)
BDE138	2.0	0.52	0.52	0.52	US	Masoner et al. 2019
	0			<0.2	US	Sutton et al. (2019)
	13			0.19	US	Moreace (2012)
BDE153	50-75				France	Gasperi et al. (2014)
	22.4	0.96	0.53	3.4	US	Masoner et al. 2019
	62.5	2.94±1.64	2.90	5	US	Sutton et al. (2019)
	50			2.6	US	Moreace (2012)
	79		0.1		France	Gasperi et al. (2022)

Table 41 continued.

	Detection frequency %	Concentration (ng/L)			Country	Reference
		Mean ± SD	Median	Max		
BDE154	0-38				France	Gasperi et al. (2014)
	14.3	1.04	0.55	2.6	US	Masoner et al. 2019
	75	4.33±4.28	2.75	13	US	Sutton et al. (2019)
	40			1.8	US	Moreace (2012)
BDE154	71		0.1		France	Gasperi et al. (2022)
BDE183	50-100				France	Gasperi et al. (2014)
	2.0	0.23	0.23	0.23	US	Masoner et al. 2019
	0			<0.3	US	Sutton et al. (2019)
	13			3.1	US	Moreace (2012)
BDE196	62.5	9.92±16.91	2.00	40	US	Sutton et al. (2019)
BDE197	62.5	0.78±0.54	0.60	1.5	US	Sutton et al. (2019)
BDE201	50	1.00±0.49	0.90	1.6	US	Sutton et al. (2019)
BDE202	87.5	2.79±1.79	2.40	6.1	US	Sutton et al. (2019)
BDE203	50	1.03±0.53	0.90	1.7	US	Sutton et al. (2019)
BDE206	75	2.82±2.76	1.70	7.8	US	Sutton et al. (2019)
BDE207	87.5	2.61±3.28	0.90	9.2	US	Sutton et al. (2019)
BDE208	75	2.30±2.62	1.20	7.1	US	Sutton et al. (2019)
BDE209	>80	25-90			France	Gasperi et al. (2014)
	87.5	64.9±52.2	56.00	160	US	Sutton et al. (2019)
	100		290	31,000	Sweden	Remberger et al. (2014)
				240	US	Gibreath (2012)
				119	US	Oram et al. (2008)
	93		20		France	Gasperi et al. (2022)
∑$PBDE_9$		23-91			France	Gasperi et al. (2014)
∑PBDE	37	6.9±5.7	5	23	US	Lubliner (2009)
∑PBDE				430	US	Gibreath (2012)
∑PBDE	100	108±55	100.00	180	US	Sutton et al. (2019)
HBCD						
α-HBCD	37.5	5.27±8.43	0.50	15	US	Sutton et al. (2019)
	91		1.4		France	Gasperi et al. (2022)
B-HBCD	12.5	0.90	0.90	0.9	US	Sutton et al. (2019)
	91		0.3		France	Gasperi et al. (2022)
γ-HBCD	25	0.75±0.78	0.75	1.3	US	Sutton et al. (2019)
	91		1.1		France	Gasperi et al. (2022)
∑HBCD	91		2.9		France	Gasperi et al. (2022)
TBBPA						
TBBPA	91		0.5		France	Gasperi et al. (2022)

Table 41 continued.

	Detection frequency %	Concentration (ng/L)			Country	Reference
		Mean ± SD	Median	Max		
Novel/emerging BFRs						
BEHTBP	87.5	3.47±3.90	2.60	12	US	Sutton et al. (2019)
BTPBE	50	0.98±0.56	0.75	1.8	US	Sutton et al. (2019)
DBDPE	0			0	US	Sutton et al. (2019)
	100		1300	1,500	Sweden	Remberger et al. (2014)
EH-TBB	87.5	8.13±4.80	7.90	15	US	Sutton et al. (2019)
HBB	100		14.00	22	Sweden	Remberger et al. (2014)
PBEB	67		7.70	91	Sweden	Remberger et al. (2014)
PBT	33		<2	2.2	Sweden	Remberger et al. (2014)
	6			0.13	US	Moreace (2012)
TBECH	0		<2	<2	Sweden	Remberger et al. (2014)
TBP-AE	0			0	US	Sutton et al. (2019)

10.3 HEALTH EFFECTS OF BROMINATED FLAME RETARDANTS

10.3.1 Polybrominated diphenyl ethers

The human health effects from PBDEs at low environmental exposures are largely unknown[149]; the majority of information regarding their toxicity is derived from animal studies, with several recent studies evaluating possible associations between PBDE concentrations in human tissues with health outcomes (ATSDR 2017). PBDEs are absorbed through the gut and lung into the bloodstream, with lower brominated congeners more likely to be absorbed into the bloodstream (ATSDR 2017). Once in the body, PBDEs may be partially metabolised, and parent compounds and metabolites are excreted predominantly in faeces with a small amount in urine. Excretion half-lives appear to increase with decreasing bromination of the congener, with estimates ranging from 15 days for BDE-209 to over 1,000 days for some lower brominated PBDEs (EFSA 2011a; ATSDR 2017). Compounds may therefore be stored in the body for several years, predominantly in adipose tissues (EFSA 2011a; ATSDR 2017). Because of differences in the absorption and storage of deca-BDE in the body, it is expected to be less toxic than lower brominated PBDEs (ATSDR 2017).

In animal studies, ingestion of PBDEs has been observed to result in neurobehavioural changes, reproductive toxicity, and effects on the thyroid, liver, pancreas, and nervous,

[149] https://www.cdc.gov/biomonitoring/PBDEs_FactSheet.html Accessed 22 March 2023

endocrine and immune systems (EFSA 2011a; ATSDR 2017); effects were often observed for both lower and higher brominated PBDEs, but at higher doses for higher brominated compounds (ie decaBDE) (ATSDR 2017). The developing nervous system appears to be one the organs most vulnerable to PBDE-induced toxicity (EFSA 2011a).

Data from human studies is also suggestive of an association between PBDE exposure and neurodevelopmental effects, including impaired cognitive development, decreased attention and impaired motor skills (ATSDR 2017). Potential effects on the reproductive, endocrine and immune systems have been identified, including (sub-)clinical hyperthyroidism; however, evidence for these end points is limited and the data is inconsistent (EFSA 2011a; ATSDR 2017). The developing fetus and young children appear to be more susceptible to effects of PBDE exposure (ATSDR 2017).

IARC have determined that the PBDE group is not classifiable as to its carcinogenicity due to inadequate evidence of carcinogenicity in humans and limited or inadequate evidence in experimental animals (ATSDR 2017).

10.3.2 Polybrominated biphenyls

PBBs are absorbed across the gastrointestinal tract to a significant extent (up to 90%) and accumulate in lipid-rich tissues including adipose tissue, adrenals and liver, as well as being detected in the brain, muscle and skin (ATSDR 2004; EFSA 2010). Estimated half-lives depend on the congener and tissue concerned; BB-153 is estimated at between 9 and 69 weeks for different tissues, and epidemiological data indicate the mean serum half-life for PBBs in humans varies between 10 and 30 years (EFSA 2010). Excretion of PBBs is mainly via faeces, however milk is an important route in lactating mammals (EFSA 2010). Most toxicity studies involve technical mixtures rather than individual congeners (EFSA 2010).

Most of what is known about the human health effects of PBBs comes from studies of Michigan residents who ate contaminated animal products for several months (IPCS 1994; ATSDR 2004). Some residents reported nausea, loss of appetite, abdominal pain, joint pain, fatigue and weakness, but it could not be established that PBB exposure was the cause of these issues; no definite changes in liver or immune function were observed (ATSDR 2004). There is evidence that PBBs can also cause skin disorders including acene and hair loss and potentially effects on the thyroid gland (IPCS 1994; ATSDR 2004; EFSA 2010). Other reported effects in humans include neurodevelopmental effects in exposed children, and immunological and reproductive issues, but data is limited and equivocal (ATSDR 2004; EFSA 2010) Very little is known about the human health outcomes associated with chronic

exposure to low levels of PBBs, whether through oral, inhalation or dermal contact (ATSDR 2004).

In animal studies, ingestion of PBBs has been associated with hepatic effects including liver enlargement, hepatocellular hypertrophy, and enzyme induction; changes in thyroid hormone homeostasis and effects on the thyroid gland; effect on other endocrine systems including changes in sex and adrenal cortex hormones; immune system effects including thymic atrophy and altered antibody production; developmental neurotoxicity and behavioural effects; embryonic and developmental effects including structural abnormalities and growth retardation; weight loss/wasting syndromes, skin disorders, ocular irritation and effects on the kidneys (IPCS 1994; ATSDR 2004; EFSA 2010). A limited number of animal studies involving dermal exposure have observed adverse hepatic, dermal and ocular effects (ATSDR 2004). Based on the adverse effects observed for multiple endpoints in animals, the possibility of similar harms in humans cannot be refuted (ATSDR 2004).

Animal studies have shown evidence of carcinogenicity, and the IARC has classified PBBs as Group 2A (probably carcinogenic to humans) (IARC 2016).

10.3.3 Hexabromocyclododecane

There is very limited data available regarding the human health effects of exposure to HBCD. In human studies, the estimated half-life for the sum of α-, β-, and γ-HBCD isomers is 64 days. Epidemiological studies have assessed a number of health effects of human HBCD exposure, but none have reached statistical significance for any observed outcomes or been able to replicate findings at subsequent follow-up points (EFSA 2021). These studies are limited by their small sample size, varying methodological quality, and heterogeneity in the assessed populations, exposure estimation and endpoints assessed (US EPA 2020; EFSA 2021).

Animal studies have shown that HBCD isomers are rapidly and extensively absorbed across the gastrointestinal tract, and considering the presence of HBCD in human samples (eg, breast milk, blood, cord blood), intestinal absorption is also expected in humans (US EPA 2020; EFSA 2021). Dermal absorption is possible but likely of a much lower magnitude (US EPA 2020). HBCD is subsequently distributed through a range of tissues including adipose tissue, muscle, liver, skin and brain (EFSA 2021). Oral toxicity studies have demonstrated some evidence of changes in thyroid function, changes in liver weight, female reproductive toxicity, immune system effects, developmental effects including reduced pup weight and viability, and neurodevelopmental effects including delayed neurodevelopmental milestones

and reduced locomotor activity (US EPA 2020; EFSA 2021). Acute overt toxicity (ie, lethality) is very low (US EPA 2020).

The limited human database on HBCD exposure means that in recent assessments of HBCD, the derivation of health-based guideline values has been considered inappropriate (US EPA 2020; EFSA 2021). HBCD has not been assessed by the IARC.

10.3.4 Tetrabromobisphenol A

The health effects of TBBPA in humans and animals remains somewhat unclear and contradictory. There is essentially no data on human exposure to TBBPA and/or its derivatives available in the literature, and less still on TBBPA derivatives (IPCS 1995; EFSA 2011b; Zhou et al. 2020).

Limited toxicokinetic data suggests that TBBPA is absorbed extensively across the gastrointestinal tract of rodents, whereafter it is distributed throughout the body and rapidly metabolised and excreted in faeces, with no significant retention or bioaccumulation of TBBPA or its metabolites within tissues (EFSA 2011b; IARC 2018). One human study showed TBBPA was also absorbed and rapidly metabolised in healthy human volunteers administered a single oral dose (NTP 2014). Detection of TBBPA in human and rat samples do however show the potential for cross-placental transfer, and detection in human breast milk highlights the potential for exposure during nursing (EFSA 2011b; IARC 2018). Elimination half-lives are estimated at 2-3 days in humans (EFSA 2011b). Dermal exposure did not result in skin sensitisation in human volunteers (IPCS 1995).

Animal studies have reported variable results as to the adverse effects of exposure to TBBPA. Acute oral toxicity is low (IPCS 1995; EFSA 2011b; NTP 2014). The thyroid hormone system seems to be a primary target where adverse effects are reported (EFSA 2011b; Feiteiro et al. 2021). Other potential effects may include changes in haematological parameters, body weight and immune function, hepatotoxicity, nephrotoxicity, and reproductive, developmental and neurobehavioral effects (EFSA 2011b; NTP 2014; Zhou et al. 2020; Feiteiro et al. 2021). However, these effects are reported inconsistently, with many studies reporting no effects across numerous end points, or that effects are only observed at excessive concentrations that are unlikely to be biologically relevant (Kacew and Hayes 2020; Zhou et al. 2020). Cell models have shown that TBBPA can affect a range of cellular systems and structures, predominantly through mechanisms relating to oxidative stress (Zhou et al. 2020).

Assessment by the IARC determined that animal studies provide sufficient evidence for carcinogenicity of TBBPA in animals, and have therefore classified it as Class 2A (probably carcinogenic to humans) (IARC 2018).

10.4 REGULATIONS AND GUIDELINES

Pertinent international regulations and guidelines regarding exposure to BFRs are documented in Table 42.

Table 42. Recommended oral exposure limits for BFRs.

	Provisional Tolerable Weekly Intake, JECFA[150] (µg/kg bw/day)	Reference Dose, US EPA[151] (µg/kg bw/day)	Minimum Risk Level, ATSDR[152] (µg/kg bw/day)
PBDEs (lower brominated)	Unable to be set	-	0.06 (acute) 0.003 (int.)
BDE47	-	0.1	-
BDE99	-	0.1	-
BDE153	-	0.4	-
HBCD	-	7	-
PBB	-	-	10 (acute)

US EPA Reference Doses and ATSDR Minimum Risk Levels are for oral exposures.
int. – intermediate exposure duration.

[150] https://apps.who.int/food-additives-contaminants-jecfa-database/
[151] https://iris.epa.gov/AtoZ/?list_type=alpha
[152] https://wwwn.cdc.gov/TSP/MRLS/mrlslisting.aspx.

11. MICROPLASTICS

Nanoplastics and microplastics (NMP) are heterogeneous mixtures of plastic particles and fibres, characterised by a wide range of different polymers, shapes, sizes and colours (Rochman et al. 2019; Gautam and Cressey 2022; WHO 2022a). Microplastics are typically considered to be particles that are smaller than 5 mm in length or diameter, while nanoplastics are commonly defined as particles smaller than 100 nm[153] (Mohana et al. 2021; Singh et al. 2022).

Microplastics may be classified as primary or secondary microplastics: primary microplastics are those that originate directly from manufactured products (eg pre-production pellets used in plastic manufacturing, microfibres in clothing), while secondary microplastics are those that are formed due to the breakdown of larger plastic fragments by physical, chemical or biological processes (Rochman et al. 2019; Singh et al. 2022). The breakdown of microplastics may in turn yield smaller nanoplastics (Singh et al. 2022). Particles may be described as fragments, fibres, spheroids, granules, pellets, flakes or beads (Gautam and Cressey 2022). The most common polymers in NMPs are polyethylene (PE), polyethylene terephthalate (PET), polyamide (PA), polypropylene (PP), polystyrene (PS), polyvinyl alcohol (PVA) and polyvinyl chloride (PVC). As well as being contaminants in their own right, NMPs frequently contain various chemical additives, including plasticisers, flame retardants, heavy metals and pesticides (Rochman et al. 2019).

Both microplastics and nanoplastics are ubiquitous in the environment, being detected in air, fresh and marine waters, drinking-waters, food and beverages, soils and biota (Singh et al. 2022; WHO 2022a), indicating possible risks to environmental and human health associated with exposure to both the particles themselves and to chemical and biological agents vectored by NMP (WHO 2022a). Further, as NMPs degrade, their physical and chemical properties change, leading to changes in their potential impacts on the environment and their potential for biological uptake (Singh et al. 2022; WHO 2022a). Indeed, microplastics have been referred to as “perhaps one of the most challenging contaminants created by humankind” (Arias-Andres and Rojas-Jimenez 2022).

[153] The classification of nanoplastics has not been officially acknowledged, thus different definitions are also used (Mohana et al. 2021). For example, in their recent review, the WHO (2022) included particles <1,000nm in this group.

While there is literature available for nanoplastics, until recently it has been difficult to accurately detect and identify nanoplastics in the environment (Singh et al. 2022), and the relationship between particle characteristics (including shape, size, surface chemistry) and toxicity are largely unknown (Gautam and Cressey 2022). This section will therefore focus primarily on microplastics.

11.1 MICROPLASTICS IN WASTEWATER

Effluents from WWTPs have been identified as a potentially significant route for microplastics pollution into the environment, with a recent New Zealand study estimating that 2.4×10^5 microplastic particles are discharged daily to the coastal receiving environment in the effluents of three WWTPs in Canterbury (Ruffell et al. 2021). Numerous studies from around the world have also identified microplastics in the influent arriving at WWTPs, including in Sweden (Fältström et al. 2021), Finland (Talvitie et al. 2015; Lares et al. 2018), Scotland (Murphy et al. 2016; Blair et al. 2019), Spain (Bayo et al. 2020), Hong Kong (Cao et al. 2020), Italy (Magni et al. 2019), the USA (Carr et al. 2016; Conley et al. 2019), Canada (Gies et al. 2018), Denmark (Simon et al. 2018), the Netherlands (Leslie et al. 2017) and New Zealand (Ruffell 2019). Of additional concern is the observation that WWTPs may break down plastics and microplastics into nanoplastics (Pramanik et al. 2021; Mohana et al. 2021).

Important sources of microplastics to wastewater include microplastic fibres released during washing of synthetic fabrics (discussed in Prata 2018) and microbeads present in wash-off personal care products (eg, toothpaste, cosmetics) (banned in New Zealand since 2018[154]) (Carr et al. 2016; Napper et al. 2015). Where industrial effluents are discharged to the municipal wastewater network, they may also contribute to the load of microplastics reaching the WWTP (discussed in Eaton (2022)). However, washing of synthetic fabrics has been suggested to be the most significant source of microplastics into wastewater (Sundt et al. 2014). Several studies have assessed microplastic fibre release during domestic laundry, with one study finding that wastewater produced by a domestic washing machine after washing a single polyester garment can contain more than 1,900 microplastic fibres, with all garments tested releasing more than 100 fibres per litre of effluent (Browne et al. 2011). Another study conducted in France found concentrations ranging from 8,850 to 35,500 fibres/L in washing machine effluents, although this was from standard wash loads with multiple garments including a range of fabric types and did not distinguish between plastic

[154] https://environment.govt.nz/acts-and-regulations/regulations/microbeads-regulations/ Accessed 8 November 2022

and natural fibres (Dris et al. 2018). It has been noted that the amount of fibres released during washing varies depending on factors such as fabric type, washing temperature and time, water hardness, and type of detergent or softener used (De Falco et al. 2018).

11.2 MICROPLASTICS IN STORMWATER

Stormwater also presents a potentially important route for microplastics pollution into aquatic environments (Horton et al. 2017; Piñon-Colin et al. 2020; Werbowski et al. 2021). Sources of microplastics pollution into the stormwater network may include runoff from construction/ building materials and artificial turfs, breakdown of plastic litter, and road dust-associated microplastics derived from tyre and road surface wear (eg, polymer-modified bitumen, polymers used in road paint markings) (Vogelsang et al. 2018; Monira et al. 2022). Road dust in particular has been noted to be a major source of microplastics pollution into marine environments (Roychand and Pramanik 2020; Monira et al. 2021). It has been estimated that 42% of microplastics exported to sea by European rivers are tyre and road wear-associated particles (Siegfried et al. 2017).

Several studies have investigated the presence of microplastics in road dust in countries such as Vietnam, Japan, Nepal (Yukioka et al. 2020), Australia (Roychand and Pramanik 2020; Su et al. 2020; O'Brien et al. 2021; Monira et al. 2022), Iran (Dehghani et al. 2017) and India (Patchaiyappan et al. 2021). In one Australian study, roadside "microplastic hotspots" were noted to be close to areas of intensive urban land use, and microplastics levels were found to be proportional to regional population size (Su et al. 2020). A separate Australian study noted that road dust microplastic concentration displayed a linear relationship to the volume of vehicles passing the sampling site, suggesting microplastic pollution increases with increased traffic load (O'Brien et al. 2021). Although, it has been noted that road dust particles are smaller in areas of high traffic volume (Abbasi et al. 2017), so the higher microplastics abundance may also be due to increased fragmentation of plastic particles by the high traffic load into smaller particles (Yukioka et al. 2020).

During periods of rainfall, road dust associated microplastics may be washed off the road surface (Vogelsang et al. 2018; Su et al. 2020) and enter the stormwater network (Pramanik et al. 2020; Roychand and Pramanik 2020; Monira et al. 2022). A recent study conducted in Gothenburg, Sweden assessed the presence of tyre and bitumen microplastic particles in road dust, stormwater, and sweepsand and washwater generated during street sweeping, and found that stormwater contained substantial amounts (1,500 – 6,000 particles/L) of small tyre and bitumen microplastic particles (≥ 20 µm) (Järlskog et al. 2020). The authors noted

that weekly sweeping may be effective at removing ‘considerable amounts’ of tyre and bitumen microplastic particles preventing them being transported into the stormwater network (Järlskog et al. 2020).

Several other studies have also assessed the presence of microplastics in stormwater or rain runoff. For example, a recent study in Tijuana, Mexico found relatively high levels of microplastics in rainfall runoff from industrial (191 particles/L) and residential areas (158 particles/L), although it was noted that potential discard of domestic laundry wastewaters and industrial wastewaters to the streets may have led to increased microplastics levels in the residential samples (Piñon-Colin et al. 2020). However, these high microplastics levels were suggested to be due, at least in part, to Tijuana being a semiarid region with a long inter-rain dry period over which pollution builds up before being washed off during rainy periods, and lack of road cleaning which allows larger pieces of plastic build up and eventually break down into smaller pieces on the road (Piñon-Colin et al. 2020). This study also noted that higher rainfall events led to higher microplastic abundance in the runoff. Two recent Australian studies both conducted in Melbourne identified fibers as the dominant form of microplastic present in stormwater samples (Pramanik et al. 2020; Monira et al. 2022). Monira et al. (2022) also identified that microplastic concentrations were higher in samples taken from industrial sites (26 particles/L) compared to residential sites (17 particles/L). Fibres were also identified as the dominant microplastic form in stormwater samples taken in Canada, representing over 40% of microplastics present, with tyre and road wear particles accounting for a further 22% of the particles (Grbić et al. 2020). Stormwater samples taken in San Francisco were found to contain microplastic concentrations ranging from 1 to 25 particles/L, and were noted to be ‘much higher’ than concentrations found in effluent from a WWTP discharged within the study area (Werbowski et al. 2021).

Microplastics have also been identified in stormwater retention ponds in Denmark, with ponds collecting runoff draining from industrial or commercial areas having higher microplastics levels (averages of 8,300 and 22,900 particles/m^3 respectively) than those collecting runoff draining from highway or residential areas (averages of 500 and 900 particles/m^3 respectively) (Liu et al. 2019b).

In addition to the microplastics sources discussed above, discarded cigarette butts may also contribute to environmental microplastics pollution due to the plasticized additives present in the cellulose acetate fibre strands that make up the filters, which can be released as microfibres in the environment (Belzagui et al. 2021) and may be washed into the stormwater network during periods of rainfall. Cellulose acetate has been identified in stormwater samples taken in Canada (Grbić et al. 2020).

11.3 HEALTH EFFECTS OF MICROPLASTICS

Assessment of human exposure to microplastics (and nanoplastics) requires characterisation of several parameters, as summarised in Figure 3: particle size and shape, polymer composition, chemical additives, particle density and surface activity are all important in understanding the potential effects on human health (WHO 2022a). The heterogeneity of microplastics therefore significantly complicates human health assessments, and is exacerbated by inconsistencies in analytical capacities and reporting by different research groups (WHO 2022a).

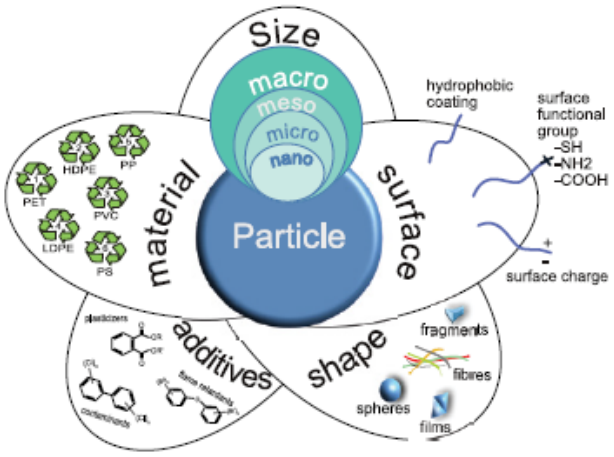

Figure 3. Attributes of micro- and nano-plastics to be considered in assessing both exposure and hazard. Reproduced from WHO (2022).

Several recent reviews have evaluated the current knowledge on potential human health effects of microplastics exposure (Rahman et al. 2021; Bhuyan 2022; Gautam and Cressey 2022; Sangkham et al. 2022; WHO 2022a). Some of the potential health effects of microplastics exposure based on animal studies and *in vitro* and *in vivo* cell culture/cell line studies are summarised in Figure 4. These include cyto-, geno-, neuro- and reproductive-toxicity, inflammation, and energy and metabolic disruption (Sangkham et al. 2022). Of particular concern is the possibility that microplastics may act as vectors for various chemical and microbial contaminants with which they are associated into the human body, followed by potential translocation of these microplastics to sites around the body (Rahman et al. 2021; Sangkham et al. 2022). Indeed, the probability of uptake into the body increases with decreasing size (WHO 2022a), and microplastics have been detected in human blood (Leslie et al. 2022), placenta (Ragusa et al. 2021) and cirrhotic liver tissue (Horvatits et al. 2022).

However, these reviews note that further research is needed to fully understand any health hazard posed by microplastics. For example, the WHO (2022a) noted that:

> *"Generally, the characterisation and quantification of exposure to NMPs and the associated human health effects are incomplete and insufficient for an assessment of risk, although the potential effects of NMPs on human health should continue to be monitored. As more data become available to better understand the mechanisms of action and subsequent effects, it may be possible to characterise and quantify human health risk in the future."*

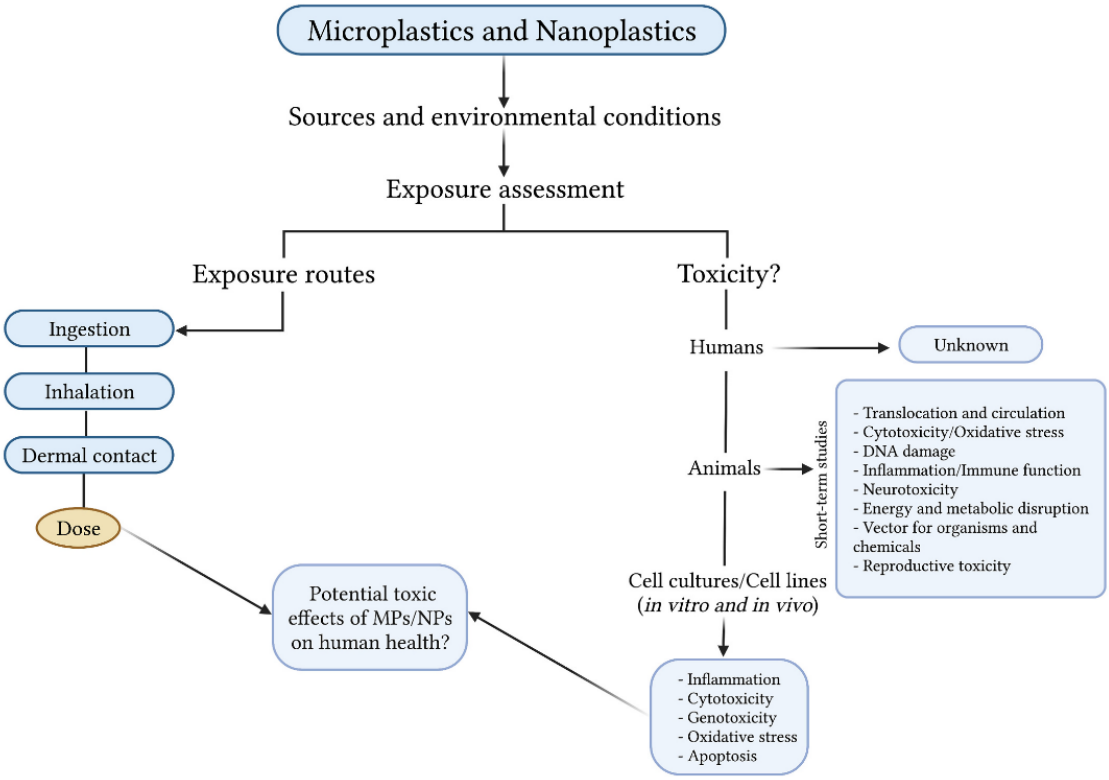

Figure 4. Potential human health effects of microplastics exposure. Reproduced from Sangkham et al. (2022).

11.4 REGULATIONS AND GUIDELINES

Because microplastics are an emerging contaminant and our understanding of the possible health effects of exposure is limited, no regulations or guideline concerning exposure limits have been developed (Gautam and Cressey 2022).

CONCLUSIONS

Municipal wastewater and urban stormwater contain complex mixtures of organic matter, suspended solids, nutrients, debris, and microbiological and chemical contaminants. A large number of these microbiological and chemical contaminants are either known, or likely to be, human health hazards. Such contaminants include pathogenic bacteria, viruses, protozoa or helminths; heavy metals and metalloids including arsenic and mercury; perfluoroalkyl substances including PFOA, PFOS and new-generation alternatives; PAHs; pesticides; BFRs; prescription pharmaceuticals; bioactive compounds associated with a variety of personal care products; endocrine-disrupting compounds including plasticisers such as BPA and various phthalates, and surfactants such as nonylphenol; and microplastics. The presence of “conventional” contaminants, such as microorganisms or heavy metals, in wastewater and/or stormwater has been well-characterised, with a wealth of international data collected over several decades. Other contaminants discussed in this report are considered “emerging contaminants,” and their presence in wastewater and/or stormwater is not as well characterised. This includes contaminants that have likely been present in wastewater and/or stormwater for some time but have only recently been recognised as a possible health hazard (eg pharmaceuticals), or novel compounds developed in response to the regulation of recalcitrant or toxic legacy contaminants (eg new-generation PFAS and pesticides). A large number of these emerging contaminants have been detected in wastewater and/or stormwater, and the number continues to grow as analytical methods advance and additional compounds are assessed. Many of the contaminants included in this review have been detected in both wastewater and stormwater, likely due to their ubiquitous use in a range of industrial, commercial, infrastructural and consumer applications and products, as well as the frequency with which there is interaction between wastewater and stormwater networks, even where these have been designed as separate systems.

The presence and concentrations of specific contaminants in wastewater and stormwater is highly dependent on characteristics of the catchment (eg land use, population size and density, consumption and behaviour patterns, presence of specific industries and associated trade waste or stormwater management practices), characteristics of the network (eg combined or separated network, general condition or integrity, cross connections, potential for inflow/infiltration), and climatic factors (eg rainfall frequency and intensity, ambient temperature, seasonal influence). The presence and concentration of contaminants in stormwaters may also show significant temporal variation throughout a storm event,

associated with ‘first-flush’ phenomena. Where contaminants have been detected in wastewater or stormwater, their concentrations may range from a few ng/L through to several hundred µg/L, and tend to be higher in networks or catchments trade wastes or industrial run-off. A higher concentration of one contaminant relative to another will not necessarily imply a greater health risk, since the relative toxicity of different contaminants can differ by orders of magnitude.

The human health effects of exposure to some of the contaminants identified in this report are well characterised, while the effects of exposure to others remain unclear. Health outcomes for exposure to a given contaminant are further influenced by the dose, duration and route of exposure, and the overall health of the individual. Acute oral exposures may cause effects ranging from relatively mild gastrointestinal and neurological symptoms (eg nausea, vomiting, diarrhoea, dizziness and irritability) to severe complications including renal failure, cardiorespiratory effects, coma and death. Health outcomes associated with chronic exposure to lower doses are more subtle and tend to be associated with long-term complications that may include developmental disorder, intellectual impairment, or renal, hepatic, gastrointestinal, haematological, reproductive or immune dysfunction. In addition, several of the contaminants identified in this report are known human carcinogens, including as a result of oral exposure. For many conventional contaminants (eg heavy metals), the health effects of human exposure are well documented for both acute and chronic outcomes, and sufficient dose response data exists to allow determination of various health-based exposure limits and guidance values (eg ADI, TDI, MRL). For some emerging contaminants (eg pharmaceuticals), the results of acute exposure may be known, but the health outcomes associated with low-level, chronic exposure are not well understood; for other emerging contaminants such as microplastics, there is growing concern about the effects of exposure, although there is little data currently available. Human HBGVs were included alongside health effects information where such data was readily available; however for many contaminants, especially emerging contaminants, there was little consolidated information available. It is likely that for many of these contaminants, there is insufficient data available to support the setting of HBGVs, although it is also possible that these do exists for some contaminants but are dispersed in the literature and/or regulations, and were unable to be identified and collated within time and resource constraints of the current report.

A further significant knowledge gap regarding the potential human health effects of exposure to the contaminants discussed in this review is the potential for additive, synergistic or antagonistic effects of exposure to multiple contaminants. Much of the health effects data that is available is based on exposure to the contaminant of interest in isolation, yet that does not reflect the reality of contaminant profiles in either wastewater or stormwater.

Rather, a huge diversity of contaminants will be present at the same time, some with similar mechanisms of toxicity to each other, and others having varying mechanisms of action. The potential effects of exposure to such mixtures, in particular chronic exposure to low levels of mixed contaminants, remains an area of considerable uncertainty.

It was initially intended that information on health-based discharge limits from key international jurisdictions (eg, Australia, US, EU, Canada) would also be included in the review. However, it became apparent that the various regulatory frameworks of different jurisdictions were diverse and complex, there was little consolidated information available, and the derivation of such limits (ie, whether health-based or ecosystem-based) was not obvious. Further, relatively few emerging contaminants are regulated. As agreed with the Ministry of Health, it was therefore not possible to compile this information within the time and resource constraints of this review. Future work could focus on identifying this information for specific or priority contaminants of concern.

This review also considered a Māori perspective on contaminants in wastewater and stormwater. In traditional Māori society, waste management was highly prescriptive, with protocols based in the principles of tapu and noa, and the protection of the mauri and mana of wai. The scale and nature of contemporary wastewater and stormwater systems do not permit the practice of relevant tikanga, and compliance with regulatory water quality standards is insufficient to ensure cultural safety, as intrinsic tapu may remain. Further, the presence of contemporary contaminants presents additional concerns for which new tikanga may be required. For Māori, concerns regarding the discharge of wastewater and stormwater to the environment extend beyond potential physical health concerns, and include adverse impacts on cultural, spiritual and social wellbeing that may result from the loss of access to wahi tapu, wai māori and mahinga kai, or the degradation of the environment for which they are kaitiaki.

The aim of this review was to provide a broad overview of contaminants of concern for human health that are reported in municipal wastewater and/or urban stormwater, based on international scientific and grey literature. The focus on untreated wastewaters and stormwaters allows for understanding of the contaminants that may be present in these matrices in the first instance, and may be released to the environment in situations of unintended discharge (eg spills, overflows), illegal discharge, or inadequate treatment (either operational failure or where a contaminant is not removed by the treatment process(es) employed). Although the review covers a wide range of contaminant types, it is acknowledged that many other contaminants that may be present in wastewater and/or stormwater that have been omitted, either as a result of time and resource constraints or a

lack of available data at this time. As the composition of both wastewater and stormwater are highly dependent on the characteristics of the catchment, network and climate, understanding the relevance of these contaminants to the New Zealand context will require further work to assess their presence in wastewater and stormwater (either through the review of additional literature, consent applications etc or directly through sampling). Additional work to understand the removal of relevant contaminants by treatment processes commonly used in New Zealand WWTPs, and consideration of the route and magnitude of exposure (eg through hazard or risk assessment) would further help characterise the potential risks to public health.

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APPENDIX A: US EPA AND EUROPEAN COMMISSION PRIORITY POLLUTANT LISTS

US EPA PRIORITY POLLUTANT LIST

United States Environmental Protection Agency

Priority Pollutant List

Priority Pollutants are a set of chemical pollutants we regulate, and for which we have developed analytical test methods. The current list of 126 Priority Pollutants, shown below, can also be found at 40 CFR Part 423, Appendix A.

These are not the only pollutants regulated in Clean Water Act programs. The list is an important starting point for EPA to consider, for example, in developing national discharge standards (such as Effluent Guidelines) or in national permitting programs (such as NPDES).

1. Acenaphthene
2. Acrolein
3. Acrylonitrile
4. Benzene
5. Benzidine
6. Carbon tetrachloride
7. Chlorobenzene
8. 1,2,4-trichlorobenzene
9. Hexachlorobenzene
10. 1,2-dichloroethane
11. 1,1,1-trichloreothane
12. Hexachloroethane
13. 1,1-dichloroethane
14. 1,1,2-trichloroethane
15. 1,1,2,2-tetrachloroethane
16. Chloroethane
17. (Removed)
18. Bis(2-chloroethyl) ether
19. 2-chloroethyl vinyl ethers
20. 2-chloronaphthalene
21. 2,4,6-trichlorophenol
22. Parachlorometa cresol
23. Chloroform
24. 2-chlorophenol
25. 1,2-dichlorobenzene
26. 1,3-dichlorobenzene
27. 1,4-dichlorobenzene
28. 3,3-dichlorobenzidine
29. 1,1-dichloroethylene
30. 1,2-trans-dichloroethylene
31. 2,4-dichlorophenol
32. 1,2-dichloropropane
33. 1,3-dichloropropylene
34. 2,4-dimethylphenol
35. 2,4-dinitrotoluene
36. 2,6-dinitrotoluene
37. 1,2-diphenylhydrazine
38. Ethylbenzene
39. Fluoranthene
40. 4-chlorophenyl phenyl ether
41. 4-bromophenyl phenyl ether
42. Bis(2-chloroisopropyl) ether
43. Bis(2-chloroethoxy) methane
44. Methylene chloride
45. Methyl chloride
46. Methyl bromide
47. Bromoform
48. Dichlorobromomethane
49. (Removed)
50. (Removed)
51. Chlorodibromomethane
52. Hexachlorobutadiene
53. Hexachlorocyclopentadiene
54. Isophorone
55. Naphthalene
56. Nitrobenzene
57. 2-nitrophenol
58. 4-nitrophenol
59. 2,4-dinitrophenol
60. 4,6-dinitro-o-cresol
61. N-nitrosodimethylamine
62. N-nitrosodiphenylamine
63. N-nitrosodi-n-propylamine
64. Pentachlorophenol
65. Phenol
66. Bis(2-ethylhexyl) phthalate
67. Butyl benzyl phthalate
68. Di-N-Butyl Phthalate

69. Di-n-octyl phthalate
70. Diethyl Phthalate
71. Dimethyl phthalate
72. Benzo(a) anthracene
73. Benzo(a) pyrene
74. Benzo(b) fluoranthene
75. Benzo(k) fluoranthene
76. Chrysene
77. Acenaphthylene
78. Anthracene
79. Benzo(ghi) perylene
80. Fluorene
81. Phenanthrene
82. Dibenzo(,h) anthracene
83. Indeno (1,2,3-cd) pyrene
84. Pyrene
85. Tetrachloroethylene
86. Toluene
87. Trichloroethylene
88. Vinyl chloride
89. Aldrin
90. Dieldrin
91. Chlordane
92. 4,4-DDT
93. 4,4-DDE
94. 4,4-DDD
95. Alpha-endosulfan
96. Beta-endosulfan
97. Endosulfan sulfate
98. Endrin
99. Endrin aldehyde
100. Heptachlor
101. Heptachlor epoxide
102. Alpha-BHC
103. Beta-BHC
104. Gamma-BHC
105. Delta-BHC
106. PCB-1242 (Arochlor 1242)
107. PCB-1254 (Arochlor 1254)
108. PCB-1221 (Arochlor 1221)
109. PCB-1232 (Arochlor 1232)
110. PCB-1248 (Arochlor 1248)
111. PCB-1260 (Arochlor 1260)
112. PCB-1016 (Arochlor 1016)
113. Toxaphene
114. Antimony
115. Arsenic
116. Asbestos
117. Beryllium
118. Cadmium
119. Chromium
120. Copper
121. Cyanide, Total
122. Lead
123. Mercury
124. Nickel
125. Selenium
126. Silver
127. Thallium
128. Zinc
129. 2,3,7,8-TCDD

EUROPEAN UNION PRIORITY POLLUTANTS

Several pieces of European Union legislation contain provisions aimed at protecting surface waters from chemical pollution,[155] including:

- The Water Framework Directive 2000/60/EC (the 'WFD') requires members to identify and regulate the pollutants of most concern (called priority substances and listed in Annex X of the WFD[156]), and to identify substances of national or local concern (ie, river basin-specific pollutants). Measures must be taken to reduce the emmission, discharge and loss of priority pollutants, and to phase out the most harmful (referred to as priority hazardous substances).
- The Environmental Quality Standards Directive 2008/105/EC (the 'EQSD') sets out environmental quality standards (EQSs) for priority pollutants and a number of other pollutants in surface water, that Member States must comply with.
- Under Directive 2013/39/EU, priority substances must be reviewed every six years, with a watchlist mechanism established that requires members to monitor substances of potential concern for up to four years to determine whether they present a risk, and support future prioritisation exercise. The most recent watch list is published in the Annex of the Commission Implementing Decision (EU) 2022/1307 of 22 July 2022.

[155] https://ec.europa.eu/environment/water/water-dangersub/index.htm. Accessed 8 July 2022
[156] The list of priority substances in Annex X of the WFD was superseded by Annex II of the EQSD, and again in 2013 by Annex I to Directive 2013/39/EU.

Annex X of Water Framework Directive 2000/60/EC

LIST OF PRIORITY SUBSTANCES IN THE FIELD OF WATER POLICY

Number	CAS number (1)	EU number (2)	Name of priority substance (3)	Identified as priority hazardous substance
(1)	15972-60-8	240-110-8	Alachlor	
(2)	120-12-7	204-371-1	Anthracene	X
(3)	1912-24-9	217-617-8	Atrazine	
(4)	71-43-2	200-753-7	Benzene	
(5)	not applicable	not applicable	Brominated diphenylethers	X (4)
(6)	7440-43-9	231-152-8	Cadmium and its compounds	X
(7)	85535-84-8	287-476-5	Chloroalkanes, C_{10-13}	X
(8)	470-90-6	207-432-0	Chlorfenvinphos	
(9)	2921-88-2	220-864-4	Chlorpyrifos (Chlorpyrifos-ethyl)	
(10)	107-06-2	203-458-1	1,2-dichloroethane	
(11)	75-09-2	200-838-9	Dichloromethane	
(12)	117-81-7	204-211-0	Di(2-ethylhexyl)phthalate (DEHP)	X
(13)	330-54-1	206-354-4	Diuron	
(14)	115-29-7	204-079-4	Endosulfan	X
(15)	206-44-0	205-912-4	Fluoranthene	
(16)	118-74-1	204-273-9	Hexachlorobenzene	X
(17)	87-68-3	201-765-5	Hexachlorobutadiene	X
(18)	608-73-1	210-168-9	Hexachlorocyclohexane	X
(19)	34123-59-6	251-835-4	Isoproturon	
(20)	7439-92-1	231-100-4	Lead and its compounds	
(21)	7439-97-6	231-106-7	Mercury and its compounds	X
(22)	91-20-3	202-049-5	Naphthalene	
(23)	7440-02-0	231-111-4	Nickel and its compounds	
(24)	not applicable	not applicable	Nonylphenols	X (5)
(25)	not applicable	not applicable	Octylphenols (6)	
(26)	608-93-5	210-172-0	Pentachlorobenzene	X
(27)	87-86-5	201-778-6	Pentachlorophenol	
(28)	not applicable	not applicable	Polyaromatic hydrocarbons (PAH) (7)	X
(29)	122-34-9	204-535-2	Simazine	
(30)	not applicable	not applicable	Tributyltin compounds	X (8)

Number	CAS number (1)	EU number (2)	Name of priority substance (3)	Identified as priority hazardous substance
(31)	12002-48-1	234-413-4	Trichlorobenzenes	
(32)	67-66-3	200-663-8	Trichloromethane (chloroform)	
(33)	1582-09-8	216-428-8	Trifluralin	X
(34)	115-32-2	204-082-0	Dicofol	X
(35)	1763-23-1	217-179-8	Perfluorooctane sulfonic acid and its derivatives (PFOS)	X
(36)	124495-18-7	not applicable	Quinoxyfen	X
(37)	not applicable	not applicable	Dioxins and dioxin-like compounds	X (9)
(38)	74070-46-5	277-704-1	Aclonifen	
(39)	42576-02-3	255-894-7	Bifenox	
(40)	28159-98-0	248-872-3	Cybutryne	
(41)	52315-07-8	257-842-9	Cypermethrin (10)	
(42)	62-73-7	200-547-7	Dichlorvos	
(43)	not applicable	not applicable	Hexabromocyclododecanes (HBCDD)	X (11)
(44)	76-44-8/ 1024-57-3	200-962-3/ 213-831-0	Heptachlor and heptachlor epoxide	X
(45)	886-50-0	212-950-5	Terbutryn	

(1) CAS: Chemical Abstracts Service.

(2) EU-number: European Inventory of Existing Commercial Substances (EINECS) or European List of Notified Chemical Substances (ELINCS).

(3) Where groups of substances have been selected, unless explicitly noted, typical individual representatives are defined in the context of the setting of environmental quality standards.

(4) Only Tetra, Penta, Hexa and Heptabromodiphenylether (CAS -numbers 40088-47-9, 32534-81-9, 36483-60-0, 68928-80-3, respectively).

(5) Nonylphenol (CAS 25154-52-3, EU 246-672-0) including isomers 4-nonylphenol (CAS 104-40-5, EU 203-199-4) and 4-nonylphenol (branched) (CAS 84852-15-3, EU 284-325-5).

(6) Octylphenol (CAS 1806-26-4, EU 217-302-5) including isomer 4-(1,1',3,3'-tetramethylbutyl)-phenol (CAS 140-66-9, EU 205-426-2).

(7) Including benzo(a)pyrene (CAS 50-32-8, EU 200-028-5), benzo(b)fluoranthene (CAS 205-99-2, EU 205-911-9), benzo(g,h,i)perylene (CAS 191-24-2, EU 205-883-8), benzo(k)fluoranthene (CAS 207-08-9, EU 205-916-6), indeno(1,2,3-cd)pyrene (CAS 193-39-5, EU 205-893-2) and excluding anthracene, fluoranthene and naphthalene, which are listed separately.

(8) Including tributyltin-cation (CAS 36643-28-4).

(9) This refers to the following compounds:
7 polychlorinated dibenzo-p-dioxins (PCDDs): 2,3,7,8-T4CDD (CAS 1746-01-6), 1,2,3,7,8-P5CDD (CAS 40321-76-4), 1,2,3,4,7,8-H6CDD (CAS 39227-28-6), 1,2,3,6,7,8-H6CDD (CAS 57653-85-7), 1,2,3,7,8,9-H6CDD (CAS 19408-74-3), 1,2,3,4,6,7,8-H7CDD (CAS 35822-46-9), 1,2,3,4,6,7,8,9-O8CDD (CAS 3268-87-9)
10 polychlorinated dibenzofurans (PCDFs): 2,3,7,8-T4CDF (CAS 51207-31-9), 1,2,3,7,8-P5CDF (CAS 57117-41-6), 2,3,4,7,8-P5CDF (CAS 57117-31-4), 1,2,3,4,7,8-H6CDF (CAS 70648-26-9), 1,2,3,6,7,8-H6CDF (CAS 57117-44-9), 1,2,3,7,8,9-H6CDF (CAS 72918-21-9), 2,3,4,6,7,8-H6CDF (CAS 60851-34-5), 1,2,3,4,6,7,8-H7CDF (CAS 67562-39-4), 1,2,3,4,7,8,9-H7CDF (CAS 55673-89-7), 1,2,3,4,6,7,8,9-O8CDF (CAS 39001-02-0)
12 dioxin-like polychlorinated biphenyls (PCB-DL): 3,3',4,4'-T4CB (PCB 77, CAS 32598-13-3), 3,3',4',5-T4CB (PCB 81, CAS 70362-50-4), 2,3,3',4,4'-P5CB (PCB 105, CAS 32598-14-4), 2,3,4,4',5-P5CB (PCB 114, CAS 74472-37-0), 2,3',4,4',5-P5CB (PCB 118, CAS 31508-00-6), 2,3',4,4',5'-P5CB (PCB 123, CAS 65510-44-3), 3,3',4,4',5-P5CB (PCB 126, CAS 57465-28-8), 2,3,3',4,4',5-H6CB (PCB 156, CAS 38380-08-4), 2,3,3',4,4',5'-H6CB (PCB 157, CAS 69782-90-7), 2,3',4,4',5,5'-H6CB (PCB 167, CAS 52663-72-6), 3,3',4,4',5,5'-H6CB (PCB 169, CAS 32774-16-6), 2,3,3',4,4',5,5'-H7CB (PCB 189, CAS 39635-31-9).

(10) CAS 52315-07-8 refers to an isomer mixture of cypermethrin, alpha-cypermethrin (CAS 67375-30-8), beta-cypermethrin (CAS 65731-84-2), theta-cypermethrin (CAS 71697-59-1) and zeta-cypermethrin (52315-07-8).

(11) This refers to 1,3,5,7,9,11-Hexabromocyclododecane (CAS 25637-99-4), 1,2,5,6,9,10- Hexabromocyclododecane (CAS 3194-55-6), α-Hexabromocyclododecane (CAS 134237-50-6), β-Hexabromocyclododecane (CAS 134237-51-7) and γ- Hexabromocyclododecane (CAS 134237-52-8).'.

Annex I of Environmental Quality Standards Directive 2008/105/EC

ENVIRONMENTAL QUALITY STANDARDS FOR PRIORITY SUBSTANCES AND CERTAIN OTHER POLLUTANTS

PART A: ENVIRONMENTAL QUALITY STANDARDS (EQS)

AA: annual average.

MAC: maximum allowable concentration.

Unit: [µg/l] for columns (4) to (7)

[µg/kg wet weight] for column (8)

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
No	Name of substance	CAS number (1)	AA-EQS (2) Inland surface waters (3)	AA-EQS (2) Other surface waters	MAC-EQS (4) Inland surface waters (3)	MAC-EQS (4) Other surface waters	EQS Biota (12)
(1)	Alachlor	15972-60-8	0,3	0,3	0,7	0,7	
(2)	Anthracene	120-12-7	0,1	0,1	0,1	0,1	
(3)	Atrazine	1912-24-9	0,6	0,6	2,0	2,0	
(4)	Benzene	71-43-2	10	8	50	50	
(5)	Brominated diphenyl-ethers (5)	32534-81-9			0,14	0,014	0,0085
(6)	Cadmium and its compounds (depending on water hardness classes) (6)	7440-43-9	≤ 0,08 (Class 1) 0,08 (Class 2) 0,09 (Class 3) 0,15 (Class 4) 0,25 (Class 5)	0,2	≤ 0,45 (Class 1) 0,45 (Class 2) 0,6 (Class 3) 0,9 (Class 4) 1,5 (Class 5)	≤ 0,45 (Class 1) 0,45 (Class 2) 0,6 (Class 3) 0,9 (Class 4) 1,5 (Class 5)	
(6a)	Carbon-tetrachloride (7)	56-23-5	12	12	not applicable	not applicable	
(7)	C10-13 Chloroalkanes (8)	85535-84-8	0,4	0,4	1,4	1,4	
(8)	Chlorfenvinphos	470-90-6	0,1	0,1	0,3	0,3	
(9)	Chlorpyrifos (Chlorpyrifos-ethyl)	2921-88-2	0,03	0,03	0,1	0,1	
(9a)	Cyclodiene pesticides: Aldrin (7) Dieldrin (7) Endrin (7) Isodrin (7)	309-00-2 60-57-1 72-20-8 465-73-6	Σ = 0,01	Σ = 0,005	not applicable	not applicable	

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
No	Name of substance	CAS number (1)	AA-EQS (2) Inland surface waters (3)	AA-EQS (2) Other surface waters	MAC-EQS (4) Inland surface waters (3)	MAC-EQS (4) Other surface waters	EQS Biota (12)
(9b)	DDT total (7), (9)	not applicable	0,025	0,025	not applicable	not applicable	
	para-para-DDT (7)	50-29-3	0,01	0,01	not applicable	not applicable	
(10)	1,2-Dichloroe-thane	107-06-2	10	10	not applicable	not applicable	
(11)	Dichlorome-thane	75-09-2	20	20	not applicable	not applicable	
(12)	Di(2-ethylhexyl)-phthalate (DEHP)	117-81-7	1,3	1,3	not applicable	not applicable	
(13)	Diuron	330-54-1	0,2	0,2	1,8	1,8	
(14)	Endosulfan	115-29-7	0,005	0,0005	0,01	0,004	
(15)	Fluoranthene	206-44-0	0,0063	0,0063	0,12	0,12	30
(16)	Hexachloro-benzene	118-74-1			0,05	0,05	10
(17)	Hexachloro-butadiene	87-68-3			0,6	0,6	55
(18)	Hexachloro-cyclohexane	608-73-1	0,02	0,002	0,04	0,02	
(19)	Isoproturon	34123-59-6	0,3	0,3	1,0	1,0	
(20)	Lead and its compounds	7439-92-1	1,2 (13)	1,3	14	14	
(21)	Mercury and its compounds	7439-97-6			0,07	0,07	20
(22)	Naphthalene	91-20-3	2	2	130	130	
(23)	Nickel and its compounds	7440-02-0	4 (13)	8,6	34	34	
(24)	Nonylphenols (4-Nonylphenol)	84852-15-3	0,3	0,3	2,0	2,0	
(25)	Octylphenols ((4-(1,1′,3,3′-tetramethyl-butyl)-phenol))	140-66-9	0,1	0,01	not applicable	not applicable	
(26)	Pentachloro-benzene	608-93-5	0,007	0,0007	not applicable	not applicable	

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
No	Name of substance	CAS number (1)	AA-EQS (2) Inland surface waters (3)	AA-EQS (2) Other surface waters	MAC-EQS (4) Inland surface waters (3)	MAC-EQS (4) Other surface waters	EQS Biota (12)
(27)	Pentachloro-phenol	87-86-5	0,4	0,4	1	1	
(28)	Polyaromatic hydrocarbons (PAH) (11)	not applicable	not applicable	not applicable	not applicable	not applicable	
	Benzo(a)pyrene	50-32-8	$1,7 \times 10^{-4}$	$1,7 \times 10^{-4}$	0,27	0,027	5
	Benzo(b)fluor-anthene	205-99-2	see footnote 11	see footnote 11	0,017	0,017	see footnote 11
	Benzo(k)fluor-anthene	207-08-9	see footnote 11	see footnote 11	0,017	0,017	see footnote 11
	Benzo(g,h,i)-perylene	191-24-2	see footnote 11	see footnote 11	$8,2 \times 10^{-3}$	$8,2 \times 10^{-4}$	see footnote 11
	Indeno(1,2,3-cd)-pyrene	193-39-5	see footnote 11	see footnote 11	not applicable	not applicable	see footnote 11
(29)	Simazine	122-34-9	1	1	4	4	
(29a)	Tetrachloro-ethylene (7)	127-18-4	10	10	not applicable	not applicable	
(29b)	Trichloro-ethylene (7)	79-01-6	10	10	not applicable	not applicable	
(30)	Tributyltin compounds (Tributyltin-cation)	36643-28-4	0,0002	0,0002	0,0015	0,0015	
(31)	Trichloro-benzenes	12002-48-1	0,4	0,4	not applicable	not applicable	
(32)	Trichloro-methane	67-66-3	2,5	2,5	not applicable	not applicable	
(33)	Trifluralin	1582-09-8	0,03	0,03	not applicable	not applicable	
(34)	Dicofol	115-32-2	$1,3 \times 10^{-3}$	$3,2 \times 10^{-5}$	not appli-cable (10)	not appli-cable (10)	33
(35)	Perfluorooctane sulfonic acid and its derivatives (PFOS)	1763-23-1	$6,5 \times 10^{-4}$	$1,3 \times 10^{-4}$	36	7,2	9,1
(36)	Quinoxyfen	124495-18-7	0,15	0,015	2,7	0,54	

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
No	Name of substance	CAS number (1)	AA-EQS (2) Inland surface waters (3)	AA-EQS (2) Other surface waters	MAC-EQS (4) Inland surface waters (3)	MAC-EQS (4) Other surface waters	EQS Biota (12)
(37)	Dioxins and dioxin-like compounds	See footnote 10 in Annex X to Directive 2000/60/EC			not applicable	not applicable	Sum of PCDD+PCDF+ PCB-DL 0,0065 µg.kg^{-1} TEQ (14)
(38)	Aclonifen	74070-46-5	0,12	0,012	0,12	0,012	
(39)	Bifenox	42576-02-3	0,012	0,0012	0,04	0,004	
(40)	Cybutryne	28159-98-0	0,0025	0,0025	0,016	0,016	
(41)	Cypermethrin	52315-07-8	8×10^{-5}	8×10^{-6}	6×10^{-4}	6×10^{-5}	
(42)	Dichlorvos	62-73-7	6×10^{-4}	6×10^{-5}	7×10^{-4}	7×10^{-5}	
(43)	Hexabromo-cyclododecane (HBCDD)	See footnote 12 in Annex X to Directive 2000/60/EC	0,0016	0,0008	0,5	0,05	167
(44)	Heptachlor and heptachlor epoxide	76-44-8/1024-57-3	2×10^{-7}	1×10^{-8}	3×10^{-4}	3×10^{-5}	$6{,}7 \times 10^{-3}$
(45)	Terbutryn	886-50-0	0,065	0,0065	0,34	0,034	

(1) CAS: Chemical Abstracts Service.
(2) This parameter is the EQS expressed as an annual average value (AA-EQS). Unless otherwise specified, it applies to the total concentration of all isomers.
(3) Inland surface waters encompass rivers and lakes and related artificial or heavily modified water bodies.
(4) This parameter is the EQS expressed as a maximum allowable concentration (MAC-EQS). Where the MAC-EQS are marked as "not applicable", the AA-EQS values are considered protective against short-term pollution peaks in continuous discharges since they are significantly lower than the values derived on the basis of acute toxicity.
(5) For the group of priority substances covered by brominated diphenylethers (No 5), the EQS refers to the sum of the concentrations of congener numbers 28, 47, 99, 100, 153 and 154.
(6) For Cadmium and its compounds (No 6) the EQS values vary depending on the hardness of the water as specified in five class categories (Class 1: < 40 mg $CaCO_3$/l, Class 2: 40 to < 50 mg $CaCO_3$/l, Class 3: 50 to < 100 mg $CaCO_3$/l, Class 4: 100 to < 200 mg $CaCO_3$/l and Class 5: ≥ 200 mg $CaCO_3$/l).
(7) This substance is not a priority substance but one of the other pollutants for which the EQS are identical to those laid down in the legislation that applied prior to 13 January 2009.
(8) No indicative parameter is provided for this group of substances. The indicative parameter(s) must be defined through the analytical method.
(9) DDT total comprises the sum of the isomers 1,1,1-trichloro-2,2 bis (p-chlorophenyl) ethane (CAS number 50-29-3; EU number 200-024-3); 1,1,1-trichloro-2 (o-chlorophenyl)-2-(p-chlorophenyl) ethane (CAS number 789-02-6; EU Number 212-332-5); 1,1-dichloro-2,2 bis (p-chlorophenyl) ethylene (CAS number 72-55-9; EU Number 200-784-6); and 1,1-dichloro-2,2 bis (p-chloro-phenyl) ethane (CAS number 72-54-8; EU Number 200-783-0).
(10) There is insufficient information available to set a MAC-EQS for these substances.
(11) For the group of priority substances of polyaromatic hydrocarbons (PAH) (No 28), the biota EQS and corresponding AA-EQS in water refer to the concentration of benzo(a)pyrene, on the toxicity of which they are based. Benzo(a)pyrene can be considered as a marker for the other PAHs, hence only benzo(a)pyrene needs to be monitored for comparison with the biota EQS or the corresponding AA-EQS in water.
(12) Unless otherwise indicated, the biota EQS relate to fish. An alternative biota taxon, or another matrix, may be monitored instead, as long as the EQS applied provides an equivalent level of protection. For substances numbered 15 (Fluoranthene) and 28 (PAHs), the biota EQS refers to crustaceans and molluscs. For the purpose of assessing chemical status, monitoring of Fluoranthene and PAHs in fish is not appropriate. For substance number 37 (Dioxins and dioxin-like compounds), the biota EQS relates to fish, crustaceans and molluscs, in line with section 5.3 of the Annex to Commission Regulation (EU) No 1259/2011 of 2 December 2011 amending Regulation (EC) No 1881/2006 as regards maximum levels for dioxins, dioxin-like PCBs and non-dioxin-like PCBs in foodstuffs (OJ L 320, 3.12.2011, p. 18).
(13) These EQS refer to bioavailable concentrations of the substances.
(14) PCDD: polychlorinated dibenzo-p-dioxins; PCDF: polychlorinated dibenzofurans; PCB-DL: dioxin-like polychlorinated biphenyls; TEQ: toxic equivalents according to the World Health Organisation 2005 Toxic Equivalence Factors.'

Annex I of Commission Implementing Decision (EU) 2022/1037

Name of substance/group of substances	CAS number (1)	EU number (2)	Indicative analytical method (3) (4)	Maximum acceptable method detection or quantification limit (ng/l)
Sulfamethoxazole (5)	723-46-6	211-963-3	SPE-LC-MS-MS	100 (11)
Trimethoprim (5)	738-70-5	212-006-2	SPE-LC-MS-MS	100 (11)
Venlafaxine and O-desmethylvenlafaxine (6)	93413-69-5 93413-62-8	618-944-2 700-516-2	SPE-LC-MS-MS	6 (11)
Azole compounds (7)			SPE-LC-MS-MS	
Clotrimazole	23593-75-1	245-764-8		20 (11)
Fluconazole	86386-73-4	627-806-0		250 (11)
Imazalil	35554-44-0	252-615-0		800 (11)
Ipconazole	125225-28-7	603-038-1		44 (11)
Metconazole	125116-23-6	603-031-3		29 (11)
Miconazole	22916-47-8	245-324-5		200 (11)
Penconazole	66246-88-6	266-275-6		1 700 (11)
Prochloraz	67747-09-5	266-994-5		161 (11)
Tebuconazole	107534-96-3	403-640-2		240 (11)
Tetraconazole	112281-77-3	407-760-6		1 900 (11)
Dimoxystrobin Azoxystrobin (8)	149961-52-4 131860-33-8	604-712-8 603-524-3	SPE-LC-MS-MS	32 (11) 200 (12)
Famoxadone	131807-57-3	603-520-1	SPE-LC-MS-MS	8,5 (11)
Diflufenican	83164-33-4	617-446-2	SPE-LC-MS-MS	10 (12)
Fipronil	120068-37-3	424-610-5	SPE-HPLC-MS-MS	0,77 (12)
Clindamycin	18323-44-9	242-209-1	SPE-LC-MS-MS	44 (12)
Ofloxacin	82419-36-1	680-263-1	SPE-UPLC-MS-MS	26 (12)
Metformin and Guanylurea (9)	657-24-9 141-83-3	211-517-8 205-504-6	SPE-LC-MS-MS	156 000 (12) 100 000 (12)
Sunscreen agents (10)				
Butyl methoxydibenzoyl-methane	70356-09-1	274-581-6	SPE-LC-MS-MS/ESI	3 000 (12)
Octocrylene	6197-30-4	228-250-8		266 (12)
Benzophenone-3	131-57-7	205-031-5		670 (12)

(1) Chemical Abstracts Service
(2) European Union number – not available for all substances
(3) To ensure comparability of results from different Member States, all substances shall be monitored in whole water samples.
(4)
Extraction methods:

SPE – solid-phase extraction
Analytical methods:

HPLC-MS-MS – High-performance liquid chromatography (tandem) triple quadrupole mass spectrometry
LC-MS-MS – Liquid chromatography (tandem) triple quadrupole mass spectrometry
LC-MS-MS/ESI – Liquid chromatography (tandem) triple quadrupole mass spectrometry with positive electrospray ionisation
UPLC-MS-MS – Ultra-performance liquid chromatography (tandem) triple quadrupole mass spectrometry
(5) Sulfamethoxazole and trimethoprim, although not grouped, shall be analysed together in the same samples but reported as individual concentrations.
(6) Venlafaxine and O-desmethylvenlafaxine shall be analysed together in the same samples but reported as individual concentrations.
(7) The azole compounds shall be analysed together in the same samples but reported as individual concentrations.
(8) Dimoxystrobin and azoxystrobin shall be analysed together in the same samples but reported as individual concentrations.
(9) Metformin and guanylurea shall be analysed together in the same samples but reported as individual concentrations.
(10) The sunscreen agents shall be analysed together in the same samples but reported as individual concentrations.
(11) Maximum acceptable detection limit
(12) Maximum acceptable quantification limit

APPENDIX B: PESTICIDE ADI/ARFDS ESTABLISHED BY JMPR

Further to the health-based exposure guidelines documented in Table 17, the Joint FAO/WHO Meeting on Pesticide Residues (JMPR) have established ADIs and some ARfDs an extensive list of pesticides. These assessments can be found in the database on the JMPR website[157], and have been summarised in this table for ease of reference.

Table B.1. Pertinent guidelines and recommended exposure limits to selected pesticides.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
1,2-dichloropropane	300 (acute) 70 (int)				40	
1,2,4-triazole			0.2	0.3		
2,4-D	200 (int) 200 (chronic)	10	0.01		30	70
2,4,5-T		10	0.03 (<0.01 mg TCDD/kg)		9	
2-phenylphenol and its sodium salts			0.4			
Abamectin			0.001	0.003		
Acephate			0.03	0.1		
Acetamiprid			0.07	0.1		
Acetochlor		20	0.01	1		
Acibenzolar-S-methyl			0.08	0.5		
Acrylonitrile	100 (acute) 40 (chronic)					
Alachlor		10			20	2
Aldicarb		1	0.003	0.003	10	
Aldrin	2 (acute) 0.04 (chronic)	0.03	0.0001^ (P)		0.03[a]	
Aminocyclopyreachlor			3			
Aminopyralid			0.9			
Amitraz		2.5	0.01	0.01		
Amitrole			0.002			
AMPA			0.3[b]			
Anilazine			0.1			
Atrazine	10 (acute) 3 (int)	35	0.02[c]	0.1 [c]	100[d]	3
Azinphos-methyl			0.03	0.1		
Azocyclotin			0.003[e] (W)	0.2		
Azoxystrobin			0.2			
Benalaxyl			0.07 (W)	0.1		

[157] https://apps.who.int/pesticide-residues-jmpr-database/

Table B.1 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Bendiocarb			0.004			
Benomyl		50	0.1			
Bentazon		30	0.09	0.5	500*	
Benzovindiflupyr			0.05	0.1		
Bicyclopyrone			0.003	0.01		
Bifenazate			0.01			
Bifenthrin			0.01	0.01		
Bitertanol			0.01			
Bixafen			0.02	0.2		
Boscalid			0.04			
Bromomethane		1.4	1.0[f]			
Bromophos			0.04			
Bromophos-ethyl			0.003			
Bromopropylate			0.03			
Buprofezin			0.009	0.5		
Cadusafos			0.0005	0.001		
Captan		130	0.1 (W)	0.3		
Carbaryl		100	0.008	0.2	50*	
Carbendazim			0.03 (W)	0.1		
Carbofuran		5	0.001	0.001	7	40
Carbophenothion			0.0005			
Carbosulfan		10	0.01	0.02		
Chinomethionat			0.006			
Chlorantraniliprole			2			
Chlorbenside			0.01			
Chlordane	1 (acute) 0.6 (chronic)	0.5	0.0005 (P)		0.2	2
Chlorfenapyr			0.03	0.03		
Chlorfenson			0.01			
Chlorfenvinphos	2 (acute) 0.7 (chronic)		0.0005			
Chlormequat chloride			0.05	0.05		
Chlorobenzilate		20	0.02			
Chlorothalonil		15	0.02	0.6		
Chlorothalonil metabolite SDS-3701			0.008	0.03		
Chlorotoluron					30	
Chlorpropham		20~	0.05	0.5		
Chlopyrifos	3 (acute) 1 (chronic)		0.01	0.1	30	
Chlorpyrifos-methyl			0.01	0.1		
Clethodim			0.01			
Clofentezine			0.02			
Clothianidin			0.1	0.6		
Crufomate			0.1			
Cyanazine					0.6	
Cyantraniliprole			0.03			
Cyazofamid			0.2	0.2		
Cyclaniliprole			0.04			
Cycloxydim			0.07 (W)	2		
Cyflumetofen			0.1			
Cyfluthrin			0.04[g]	0.04[g]		
Cyhalothrin	10 (acute) 10 (int)	5~	0.02	0.02		

Table B.1 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Cyhexatin			0.003	0.02		
Cypermethrin	20 (acute)	10~	0.02	0.04		
Cyproconazole			0.02	0.06		
Cyprodinil			0.03			
Cyromazine		7.5~	0.06	0.1		
Daminozide			0.5			
DDT	0.5 (acute) 0.5 (chronic)	0.5	0.01 (P)		1.0[h]	
Deltamethrin			0.01	0.05		
Demeton-S-methyl		0.005	0.0003			
Diazinon	6 (acute) 0.7 (chronic)		0.003	0.03		
Dicamba		20	0.3	0.5		
Dichlobenil			0.01 (W)	0.5		
Dichloran			0.01			
Dichlorprop					100	
Dichlorvos	4 (acute) 0.5 (chronic)	0.5	0.004	0.1	20*	
Dicofol			0.002	0.2	10*	
Dieldrin	0.1 (int) 0.05 (chronic)	0.05			0.03[a]	
Difenoconazole			0.01	0.3		
Diflubenzuron		20	0.02			
Dimenthenamid-P			0.07	0.5		
Dimethipin		20~	0.02	0.2		
Dimethoate		0.2~	0.002	0.02	6	
Dimethomorph			0.2	0.6		
Dinocap			0.008	0.03		
Dinotefuran			0.2	1		
Dioxathion			0.0015			
Diphenyl			0.125			
Diphenylamine			0.08			
Diquat		2.2	0.006	0.8	30*	20
Disulfoton	0.3 (acute) 0.06 (chronic)	0.05	0.0003	0.003		
Dithianon			0.01	0.1		
Diuron		2				
Dodine		4~	0.1	0.2		
Edifenphos			0.003			
Emamectin benzoate			0.0005	0.02		
Endosulfan	7 (acute) 5 (chronic)	6	0.006	0.02	20*	
Endrin	0.6 (acute) 0.3 (chronic)	0.3	0.0002 (P)		0.6	2
Esfenvalerate			0.02	0.02		
Ethephon			0.05	0.05		
Ethiofencarb			0.1			
Ethion	2 (acute) 0.4 (chronic)	0.5	0.002			
Ethiprole			0.005	0.005		
Ethoprophos			0.0004	0.05		
Ethoxyquin			0.005[j]	0.5[j]		
Ethylenethiourea			0.004			
Etofenprox			0.03	1.0		

Table B.1 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Etoxazole			0.05			
Etrimfos			0.003			
Famoxadone			0.006	0.6		
Fenamidone			0.03	1.0		
Fenamiphos		0.25	0.0008	0.003		
Fenamirol			0.01			
Fenazaquin			0.05[j]	0.1		
Fenbuconazole			0.03	0.2		
Fenbutatin oxide			0.03			
Fenchlorphos			0.01			
Fenhexamid			0.2			
Fenitrothion			0.006	0.04	8*	
Fenoprop					9	
Fenpicoxamid			0.05			
Fenpropathrin			0.03	0.03		
Fenpopimorph			0.004	0.01 (W) 0.4		
Fenpyrazamine			0.3	0.8		
Fenpyroximate			0.01	0.01		
Fensulfothion			0.0003			
Fenthion			0.007	0.01		
Fentin acetate			0.0005[k]			
Fentin chloride			0.0005 [k]			
Fentin hydroxide			0.0005 [k]			
Fenvalerate			0.02	0.2		
Ferbam			0.003[l]			
Fipronil			0.0002[m]	0.003 [m]		
Flonicamid			0.07			
Fluaziop-p-butyl			0.004	0.4		
Flubendiamide			0.02	0.2		
Flucythrinate			0.02			
Fludioxonil			0.4			
Fluensulfone			0.01	0.3		
Flufenoxuron			0.04			
Flumethrin			0.004			
Flumioxazin			0.02	0.03		
Fluopicolide			0.08 (W)	0.6		
Fluopicolide metabolite 2,6-dichlorobenzamide			0.02	0.6		
Fluopyram			0.01	0.5		
Flupyradifurone			0.08	0.2		
Flusilazole			0.007	0.02		
Flutolanil		60~	0.09			
Flutriafol			0.01	0.05		
Fluxapyroxad			0.02	0.3		
Folpet		100~	0.1 (W)	0.2		
Fosetyl-aluminium		3,000	10			
Glufosinate ammonium		0.4~	0.01 [n]	0.01[n]		
Glyphosate	1,000 (acute) 1,000 (chronic)	100	1.0[a]		900*[#]	700
Haloxyfop		0.05	0.0007	0.08		
Heptachlor	0.6 (acute) 0.1 (chronic)	0.5	0.0001 (P)		0.03*	0.4

Table B.1 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Hexachlorobenzene	8 (acute) 0.07 (chronic)	0.8			1*	1
Hexaconazole			0.005			
Hexachlorocyclo-hexane α	2 (int) 0.9 (chronic)					
Hexachlorocyclo-hexane β	80 (acute) 0.6 (int)					
Hexachlorocyclo-hexane γ (lindane)	3 (acute) 0.008 (chronic)	0.3	0.005	0.06		
Hexythiazox			0.03			
Hydrogen cyanide		0.6	0.05			
Hydroxy-atrazine			0.04			
Imazalil		13~	0.03[o]	0.05 [o]		
Imazamox			3	3		
Imazapic			0.7			
Imazapyr			3			
Imazethapyr			0.6			
Imidacloprid			0.06	0.4		
Indoxacarb			0.01	0.1		
Iprodione		40	0.06			
Isofenphos			0.001			
Isofetamid			0.05	3		
Isoprothiolane			0.1[p]			
Isoproturon					9	
Isopyrazam			0.06	0.3		
Isoxaflutole			0.02			
Kresoxim-methyl			0.3			
Lufenuron			0.02			
Malathion	20 (acute) 20 (chronic)	20	0.3	2	900*	
Maleic hydrazide			0.3			
Mancozeb			0.03[p]			
Mandestrobin			0.2 (W)	3		
Mandipropamid			0.2			
Maneb		5	0.03			
MCPA		0.5	0.1	0.6	700*	
Mecarbam			0.002			
Mecoprop					10	
Meptydinocap			0.02			
Mesotrione			0.5			
Metaflumizone			0.1			
Metalaxyl and metalaxyl-m		60	0.08			
Methacrifos			0.006			
Methamidophos		0.05	0.004	0.01		
Methidathion		1~	0.001	0.01		
Methiocarb			0.02	0.02		
Methomyl		25	0.02	0.02		
Methoprene and s-methoprene			0.05			
Methoxychlor	5 (int)	5	0.1		20	40
Methoxyfenozide			0.1	0.9		
Methyl parathion	7 (int) 3 (chronic)	0.25			9*	

Table B.1 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Metolachlor		150			10	
Metiram			0.03 [p]			
Metrafenone			0.3			
Mevinphos			0.0008			
Mirex	0.3 (chronic)	0.2				
Molinate		2			6	
Monocrotophos			0.0006	0.002		
Myclobutanil			0.03	0.3 (W)		
N-acetyl-glufosinate			0.02[n]			
Natamycin			0			
N,N-diethyl-meta-toluamide (DEET)	1 (int)					
Norflurazon		40	0.005	0.3		
Novaluron			0.01			
Omethoate			withdrawn			
Organomercury compounds			No ADI			
Oxamyl		25	0.009[q]	0.009 [q]		200
Oxathiapiprolin			4			
Oxydemeton-methyl			0.0003[r]	0.002		
Paclobutrazol		13	0.1			
Paraquat		4.5	0.005	0.006		
Parathion	9 (int)		0.004	0.01	10*	
Parathion-methyl			0.003	0.03		
Penconazole			0.03	0.8		
Pendimethalin		40~	0.1	1.0	20	
Pentachlorophenol	5 (acute) 5 (chronic)	5			9	1
Penthyopyrad			0.1	1.0		
Permethrin	300 (acute) 200 (chronic)	50	0.05	1.5	300*	
Phenothrin			0.07			
Phenthoate			0.003			
Phorate			0.0007	0.003		
Phosalone			0.02	0.3		
Phosmet		20	0.01	0.2		
Phosphamidon			0.0005			
Phosphonic acid			1.0			
Phoxim			0.001			
Picoxystrobin			0.09	0.09		
Pinoxaden			0.1	0.3		
Piperonyl butoxide			0.2			
Pirimicarb			0.02	0.1		
Pirimiphos-methyl		10~	0.03	0.2		
Prochloraz		9	0.01	0.1		
Procymidone			0.1	0.1		
Profenofos			0.03	1.0		
Propamocarb			0.4	2.0		
Propargite		20~	0.01			
Propanil		5				
Propazine		20				
Propham		20	No ADI			
Propiconazole		13~	0.07	0.3		
Propineb			0.007	0.01 (P)		

Table B.1 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Propoxur			0.02			
Propylene oxide		30	0.04	0.04		
Propylene chlorohydrin			0.3	0.3		
Propylene bromohydrin			0.03	0.03		
Propylenethiourea			0.0003	0.003		
Prothioconazole			0.05 (W)	0.8		
Prothioconazole-desethio			0.01	0.01 (W) 1.0		
Pydiflumetofen			0.1	0.3		
Pymetrozine			0.03	0.1		
Pyraclostrobin			0.03	0.7		
Pyrazophos			0.004			
Pyrethrins			0.04	0.2		
Pyrimethanil			0.2			
Pyriproxifen			0.1			
Quinclorac			0.4	2.0		
Quinoxyfen			0.2			
Quintozene			0.01			
Resmethrin		30	0.03[s]			
Saflufenacil			0.05			
Sec-butylamine			withdrawn			
Sedaxane			0.1	0.3		
Simazine		5			2	4
Spinetoram			0.05			
Spinozad			0.02			
Spirodiclofen			0.01			
Spiromesifen			0.03			
Spirotetramate			0.05	1.0		
Sulfoxaflor			0.05	0.3		
Sulfuryl fluoride			0.01	0.3		
Tebuconazole			0.03	0.3		
Tebufenozide			0.02	0.9		
Teflubenzuron			0.005			
Terbufos			0.0006	0.002		
Terbuthylazine					7	
Terbutryn		1				
Thiabendazole			0.1	1.0		
Thiacloprid			0.01	0.03		
Thiamethoxam			0.08	1.0		
Thiodicarb			0.03	0.04		
Thiometon			0.003			
Thiophanate-methyl		80~	0.09	1.0		
Thiram		5~	0.01			
Tioxazafen			0.05	0.5		
Tolclofos-methyl			0.07			
Tolfenpyrad			0.006	0.01		
Tolylfluanid			0.08	0.5		
Toxaphene	5 (acute) 2 (chronic)					
Triadimefon			0.03	0.08		
Triadimenol			0.03	0.08		

Table B.1 continued.

Compound	Minimum Risk Level ATSDR (µg/kg bw/day)	Reference Dose, US EPA (µg/kg bw/day)	Acceptable Daily Intake, JMPR (mg/kg bw/day)	Acute Reference Dose, JMPR (mg/kg bw)	Drinking-water Guideline, WHO (µg/L)	Drinking-water Contaminant Limit US EPA (µg/L)
Triazole acetic acid			1[t]			
Triazole alanine			1[t]			
Triazophos			0.001	0.001		
Trichlorfon			0.002			
Trichloronate						
Trifloxystrobin			0.04			
Triflumezopyrim			0.2	1.0		
Triflumizole			0.04	0.3		
Trifluralin		7.5			20	
Triforine			0.03	0.3		
Trinexapac			0.3			
Vamidothion			0.008			
Vinclozolin		2.5~	0.01			
Zineb		50	0.03[p]			
Ziram			0.003[l]			
Zoxamide			0.5			

* Formal guideline value not established, however a 'health-based value' has been determined to provide guidance when there is a reason for local concern.

~ The EPA announced in 2004 that chemicals used as pesticides would not be reassessed by the IRIS Program. This entry is an archived value whose presence in the IRIS database was preserved at the request of the EPA program and regional offices; values were archived in 2016.

P – provisional tolerable daily intake. W – established for women of childbearing age; unnecessary for the general population unless a second value is specified.

a. Total for combined aldrin and dieldrin.
b. Sum of glyphosate and AMPA.
c. Group ADI and ARfD for atrazine, deethyl-atrazine, deisopropyl-atrazine and diaminochlorotriazine.
d . Group limit for atrazine and its chloro-s-triazine metabolites.
e. Group ADI and ARfD with cyhexatin.
f. As bromide ion.
g. Group ADI for cyfluthrin and beta-cyfluthrin.
h. Combined for DDT and its metabolites.
i. ADI and ARfD for ethoxyquin and three of its metabolites: methyl-ethoxyquin, dehydromethylethoxyquin and dihydroethoxyquin.
j. Group ADI for fenazaquin, TBPE and 4-OH.
k. Alone or in combination with other fentin compounds.
l. Group ADI for ferbam and ziram.
m. Group ADI and ARfD for fipronil and fipronil-desulfinyl.
n. ADI and ARfD apply to glufosinate-ammonium, n-aceteyl-gluphosinate and 3-(hydroxymethylphosphinoyl) propanoid acid, alone or in combination.
o. ADI and ARfD also apply to metabolites R061000 and R014821
p. Group ADI with maneb, metiram and zineb
q. ADI and ARfD apply to metabolites IN-A2213, IN-QKT34, IND2708 and IN-N009.
r. Group ADI established for demeton-s-methyl and related compounds, including oxydemeon-methyl
s. Estimated as bioresmethrin
t. Group ADI for triazole alanine and triazole acetic acid

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