HSE

AGENCY TECHNICAL REPORT

Substance Name: Formaldehyde in indoor air environments in Great Britian

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CAS Number: 50-00-0

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Summary

Formaldehyde is a reactive substance that exists as a gas at ambient temperature and standard atmospheric pressure. It is classified in the Great Britain (GB) Mandatory Classification and Labelling (MCL) list as a carcinogen category 1B, mutagen category 2, acute toxicant category 2 by the inhalation route and category 4 by the oral route, skin corrosive category 1B and skin sensitiser category 1A. It is irritant / corrosive to the eyes and respiratory tract. At lower concentrations, exposure to formaldehyde has also been linked with sensory irritation of the eye and upper respiratory tract and asthma-like respiratory symptoms.

Formaldehyde is one of several volatile organic compounds (VOC) which can be emitted from various sources into indoor air, including in people's homes. Indoor air pollutants (including VOCs and formaldehyde) are thought to cause significant adverse health effects. Some people have been reported to spend about 90% of their time indoors, much of it at home (around 66%). This is particularly the case for vulnerable populations, such as young children and elderly people. Consequently, improving indoor air quality has been highlighted as a priority for UK Government.

This report has been prepared by HSE, in conjunction with the UK Health Security Agency (UKHSA), the government agency responsible for public health protection, to collate information on sources that emit formaldehyde into indoor air environments in GB and understand the levels of formaldehyde that have been measured. Further, an outline of the evidence for adverse effects occurring in the general population, including children, at these levels is provided.

Risks to workers exposed in occupational settings were out of scope, as were risks from exposure to formaldehyde outdoors and risks to the environment.

Sources of formaldehyde in indoor air

There is a complex inter-relationship between factors that will contribute formaldehyde to indoor air (sources) and those that will remove formaldehyde from indoor air (sinks). Other factors that can influence formaldehyde levels include indoor air chemistry and environmental parameters such as temperature and humidity. Depending on the combination of factors, these could increase or decrease the level of formaldehyde that is emitted from various sources. For the purposes of this report, sources have been classified into continuous or intermittent sources.

Continuous sources include building materials such as engineered wood boards and articles such as furnishings, carpets, wall coverings and textiles. Often these sources have a large surface area, but emissions will diminish over time as articles age.

Emissions from articles occur in two phases. Initially, emissions appear to be mainly

driven by volatility and decline according to a first order exponential process, whereas longer term emissions (after around 3 months) may be driven by chemical reactions (hydrolysis) between moisture in the air and resins and naturally occurring components such as lignin in wood. Environmental parameters such as temperature and humidity are a key determinant of the scale of longer-term emissions from articles. Given that chamber studies are typically conducted under a standardised set of environmental conditions, it is not possible to extrapolate quantitative emissions data from chamber studies to emissions that might arise in domestic settings. However, data from chamber tests can be useful to rank articles according to their potential emissions. It is also relevant to note that because chamber tests are typically carried out on newly produced articles, the emissions data measured in these tests represent a worst-case situation and will not necessarily reflect the scale of emissions that will arise when that article is in situ in someone's home.

For continuous sources, historically, engineered wood boards were a major source of emissions. However, the introduction of the E1 emission standard has resulted in voluntary actions by board producers, including most EU-based and all GB-based producers, to supply lower emitting E1 boards which generate air concentrations of ≤ 124 μg/m^3 in EN 717-1 chamber tests when new. Boards that do not meet the E1 standard could emit considerably higher concentrations. Information the Wood Panel Industries Federation (WPIF) shared with HSE, indicates that 98 – 99% of the particleboard and oriented strand board (OSB) and 83% of the medium density fibreboard (MDF) supplied to the GB market originates in GB or the EU and is therefore likely to meet the E1 standard. These percentages do not include the supply of articles made using engineered wood boards such as furniture. HSE does not have data on the market share for different countries of origin for these articles, thus there is uncertainty about the proportions of high and lower emitting articles that are supplied to the GB market. Other continuous sources such as wallcoverings, mineral wool insulation, foams, paints and textiles appear to emit lower quantities of formaldehyde compared with boards.

Intermittent sources are typically point sources that may emit for varying durations ranging from a few minutes to several hours. These include combustion sources (e.g., open fires, wood burners, wood stoves, ethanol fireplaces, high temperature oven cleaning, smoking and vaping, incense burners, scented candles) and use of fragranced personal and household care products.

For intermittent sources, combustion processes generate some of the highest formaldehyde concentrations. A study in 2019 estimated the concentration in air for a standard reference room for a range of continuous and intermittent sources and found that during operation, emissions from intermittent combustion sources can be an order of magnitude higher (or more in some cases) than emissions from continuous sources and household and personal care products. The impact that intermittent sources have on the overall room concentration of formaldehyde will be dependent on the frequency and duration of operation of each source. These are heavily influenced by the activities

of individual householders. This is another reason why chamber studies are unlikely to provide an accurate indication of the levels of formaldehyde that will arise from specific activities when carried out within the home.

Sinks are materials that can adsorb formaldehyde, temporarily reducing indoor air concentrations, but are later able to re-emit it back into the environment. The extent to which formaldehyde adsorbs and desorbs can be complex and is dependent on numerous factors including the nature of the material and the conditions (e.g., the temperature and humidity). This includes deposition of dusts to which formaldehyde has adsorbed. Dust can also act as a source if this is re-entrained into the air and formaldehyde that has adsorbed onto the surface of dust particles can be released (desorb) back into the air.

Levels of formaldehyde measured indoors in domestic settings

For context, ambient outdoor concentrations of formaldehyde in European air range from around 1.2 – 27 µg/m^3 with a geometric mean (GM) of 4.3 µg/m^3. The higher concentrations result mainly from occasional photochemical reactions of atmospheric hydrocarbons.

A literature review carried out in 2023 identified eleven studies published over the last 25 years in which measurements were made of the levels of formaldehyde in homes in England. Studies of homes in Scotland and Wales were not found, but there is no reason to consider that levels of formaldehyde in homes in these regions will be significantly different from homes in England. Average formaldehyde levels in these studies ranged between 11 – 49 µg/m^3 and maxima ranged from 32 – 187 µg/m^3. Pooling datasets to obtain a weighted distribution resulted in a GM of 22.8 µg/m^3 (geometric standard deviation: 2.0 µg/m^3) and 5th and 95th percentiles at 6.5 µg/m^3 and 58.7 µg/m^3. These levels are similar to GMs from pooled studies where indoor monitoring was conducted pre-1998, suggesting little change over the past 25 years. These data are also consistent with average indoor air concentrations measured in other high-income countries.

Large scale studies in England and Germany have found that homes built after 1982 have around three times the level of indoor formaldehyde compared with homes built before 1919. This is thought to be the result of changes in building practices (greater use of engineered wood boards and requirements to increase the air tightness of buildings), introduced between the 1970s and 1990s. Where the air tightness of homes is increased to improve energy efficiency, ventilation will contribute to good air quality.

These studies from GB and Germany have identified several additional factors associated with increased indoor formaldehyde concentrations, including:

- rural location compared with suburban or urban location;
- detached homes compared with semi-detached, terrace, or flat;
- integral garage compared with detached garage;

- new particleboard furniture and/or flooring;
- frequent use of sanitary cleaners/disinfectants.

Health effects of exposure to formaldehyde in domestic settings

Formaldehyde is a known genotoxic carcinogen. Data from experimental animals confirm a link between long-term inhalation exposure and site-of-contact nasal tumours. The underlying mode of action is thought to be cell proliferation caused by repeated cycles of cell damage and repair arising from chronic irritation. This in turn leads to hyperplasia or metaplasia and ultimately the development of tumours. As such, this is a local, threshold effect. Irritation is a concentration-dependent effect. Exposure to concentrations of formaldehyde that are below the irritant threshold would not cause the initial cell damage that is the precursor event to tumour development.

Therefore, this report focuses on potential health effects at inhalation exposure concentrations that are relevant to general population (non-occupational) settings.

Most inhaled formaldehyde gas is retained in the upper airways; very little penetrates to the deep lung or lower airways. At concentrations that are relevant to indoor, non-occupational settings, inhaled formaldehyde is not expected to be systemically available to a great extent. As such, primary health effects likely to arise from such exposures amongst the general population are local effects to the upper airway tissues.

A sensitive and consistent response on exposure to formaldehyde in air at concentrations relevant to this report is sensory irritation, characterised as unpleasant sensations or discomfort in the eyes and nasal passages. Sensory irritation occurs when airborne irritants such as formaldehyde stimulate nerve endings located in the respiratory epithelium. It is different from histopathologically-detectable irritation (i.e., inflammation) and does not result in tissue damage. It is also reversible, and so quickly resolves when exposure ceases. Sensory irritation is a concentration-dependent effect: brief exposures to a set concentration can elicit the same intensity of responses as longer exposures to the same concentration.

There are varying expert views on the adversity of sensory-irritation effects: some consider that mild sensory irritation might not be an adverse effect, whilst others consider that irritation of mucous membranes could adversely impact workers or the general population if they were hampered in the safe performance of their occupation or driving, for example. Odour perception can increase the reporting of subjective effects such as eye/nasal discomfort, olfactory symptoms and annoyance. Objective parameters, such as conjunctival redness, eye blinking frequency (EBF), and nasal resistance or flow might be less affected by odour perception and were included in the design of some controlled chamber studies in volunteers, as were odour-masking chemicals.

Some observational epidemiology studies have reported an association between indoor formaldehyde exposure and worsening or increased prevalence of respiratory

conditions in children, existing asthmatics and those with impaired lung function. These conditions have included reduced pulmonary function and exacerbation of asthma and other respiratory allergic conditions. However, there are differing views on the susceptibility of children to effects of formaldehyde on asthma. No impact on lung function was observed in controlled exposure studies amongst human volunteers, including those in which subjects included individuals who were asthmatic or had dermal hypersensitivity to formaldehyde. Other observational epidemiology studies also did not find respiratory effects or changes in lung function linked to formaldehyde exposure.

Overall, there is some evidence to indicate that formaldehyde might have an adverse effect on asthma. Currently there is a lack of clear mechanistic understanding of whether formaldehyde induces asthma or exacerbates existing conditions and whether it acts as an allergen itself, or whether it acts as an irritant to induce asthma. The available evidence does not support clear dose-response or clear cause-effect relationships.

Abbreviations

ABS	Acrylonitrile-butadiene-styrene
ACH	Fixed air exchange rate
ADH3	Alcohol Dehydrogenase 3
AF	Assessment factor
AHHS	American Healthy Homes Survey
AHR	Airway hyper-responsiveness
AIR	German Umweltbundesamt's (UBA) Committee on Indoor Air Guide Values
AIRMEX	Indoor Air Monitoring and Exposure Assessment
AQEG	Defra Air Quality Expert Group
ASBP	The Alliance for Sustainable Building Products
ATSDR	US Agency for Toxic Substances and Disease Registry
BDF	German Association of Prefabricated Houses
BDO	1,4-Butanediol
BMC	Benchmark concentration
BMR	Benchmark response
CAS	Chemical Abstract Service
CBPB	Cement bonded particleboard
CMR	Carcinogenicity, Mutagenicity and Reproductive Toxicity
COC	Committee on Carcinogenicity of Chemical in Food, Consumer Products and the Environment
COM	Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment
CPHEA	US EPA's Centre for Public Health and Environmental Assessment
CPR	Construction Products Regulation
CSA	Chemical Safety Assessment
CWUKR	Comply with UK REACH
DALYs	Disability Adjusted Life Years
DHH	US Department of Healthy Housing
DLUHC	Department for Levelling up and Housing
DNEL	Derived no effect level
DPX	DNA-protein cross-links
DU	Downstream Users
DUINS	Downstream User Import Notifications
EBF	Eye blinking frequency
ECHA	European Chemicals Agency
EPA	US Environmental Protection Agency
EPHECT	Emissions, exposure patterns and health effect of consumer products in the EU
ESP	Electrostatic precipitators

EU	European Union
FEV_1	Forced Expiratory Volume in 1 second
FIFRA	US Federal Insecticide Fungicide and Rodenticide Act
FLEC	Field and Laboratory Emissions Cell
FVC	Forced Expiratory Vital Capacity
GB	Great Britian (England, Scotland and Wales)
HBGV	Health-Based Guidance Values
HIPS	High impact polystyrene
HSRB	Human Studies Review Board
IAQ	Indoor air quality
IAQG	Indoor air quality guidance
IARC	Internation Agency for Research on Cancer
IgE	Immunoglobulin E
IIA	Irritant-induced asthma
IQR	Interquartile range
IRIS	Integrated Risk Information System
JRC	Joint Research Centre
LCI	Lowest Concentration of Interest
LOAEC	Lowest observed adverse effect concentration
LOAEL	Lowest observed adverse effect limit
LOEL	Lowest observed effect level
LRT	Lower respiratory tract
MCL	Mandatory Classification and Labelling
MDF	Medium density fibreboard
MDI	Methylene diphenyl diisocyanate
ME	Microenvironments
MF	Melamine Formaldehyde
MHCLG	Ministry of Housing, Communities and Local Government
MRL	Minimal risk level
MUF	Melamine Urea Formaldehyde
MVHR	Heat recovery ventilation systems
NASEM	National Academies of Science, Engineering and Medicine
NHBC	National House Building Council
NICE	National Institute for Health and Care Excellence
NO	Nitric oxide
NOAEC	No observed adverse effect concentration
NOAEL	No observed adverse effect limit
OCSPP	Office of Chemical Safety and Pollution Prevention
OEHHA	California Office of Environmental Health Hazard Assessment
OEL	Occupation Exposure Limit
OPP	Office of Pesticide Programme
OPPT	Office of Pollution Prevention
ORD	US EPA Office of Research and Development
OSB	Oriented strand board

osRfC	organ-or system-specific reference concentration
PAF	Population Attributable Fraction
PARIS	Pollution and Asthma Risk Infant Study
PBT	Persistent, Bioaccumulative and Toxic
PEFR	Peak Expiratory Flow Rate
PEG	Polyethylene glycol
PF	Phenol formaldehyde
PHE	Public Health England
PLA	Polyactic acid
PMDI	Polymeric MDI
PoD	Point(s) of Departure
POM	Polyoxymethylene
PPB	Parts per billion
PPM	Parts per million
PRF	Phenol Resorcinol Formaldehyde
PU	Polyurethane
PVA	Polyvinyl alcohol
RAC	ECHA's Risk Assessment Committee
RADS	Reactive airway dysfunction syndrome
RCP	Royal College of Physicians
RCPCH	Royal College of Paediatrics and Child Health
REACH	Registration, evaluation, authorisation and restriction of chemicals
REL	Reference Exposure Level
RF	Resorcinol Formaldehyde
RfD	Reference dose
Rfc	Reference concentration
RH	Relative Humidity
SACC	TSCA Science Advisory Committee on Chemicals
SCOEL	Scientific Committee on Occupational Exposure Limits
SDS	Safety Data Sheet
THF	Tetrahydrofuran
TRV	Toxicological reference value
TSCA	Toxic Substances Control Act
TVOC	Total Volatile Organic Compounds
TWA	Time weighted average
UBA	Umweltbundesamt – German Environment Protection Agency
UF	Urea formaldehyde
UFFI	Urea formaldehyde foam insulation
UK	United Kingdom
URT	Upper respiratory tract
UKHSA	UK Health Security Agency
VOC	Volatile organic compound
vVOC	Very Volatile organic compound
WHO	World Health Organisation

WPIF Wood Panels Industry Federation

Definitions

Normal distribution: A normal distribution is a probability distribution of outcomes that are symmetrical or form a bell curve. In a normal distribution, 68% of the results fall within one standard deviation, and 95% fall within two standard deviations.

Log normal distribution: A random variable is said to have a log-normal distribution if its natural logarithm has a normal distribution. In other words, the exponential of a normal random variable has a log-normal distribution.

Uniform distribution: Type of probability distribution in which all outcomes are equally likely.

Disability Adjusted Life Year lost (DALY): Is a measure of the burden that the prevalence of a disease or health condition places on society as it captures both the impacts on morbidity (Years of life lived with disability) as well as impacts on mortality (Years of life lost premature). The measure puts everything on the scale of time so that it is easy to compare health burdens across different diseases and risk factors.

Population Attributable Fraction (PAF): Is an epidemiological measure which can be defined as the proportion of the new cases of disease or mortalities within a population and period of time that are attributable to the exposure distribution within the population.

Passivhaus is a standard for energy efficient homes. This was developed in Germany in order to be a leading standard for energy efficient design and construction for cold-climate houses. The Passivhaus standard is based on five fundamental concepts: super-insulation, thermal bridge-free construction, an airtight building envelope, use of high-performance doors and windows and heat recovery ventilation systems (MVHR) (Moreno-Rangel *et al.*, 2020). Passivhaus homes need less than 30% of the energy for space heating than an equivalent house built to the current Building Regulations standards. This standard is used in Germany, Austria and Ireland. The Passivhaus standard is typically ≤ 0.6 air changes per hour (ACH) @ 50 Pa, when the building is pressurised. (Passivhaus Trust, 2020)

Air permeability is the measure of airtightness of the building fabric. It is defined as the air leakage rate per hour per m^2 of envelope area at the test reference pressure differential of 50 Pa or 4 Pa. In 2021, England and Wales both introduced new building regulations that tightened the limiting air permeability for new buildings from 10 $m^3/hr.m^2$ to 8 $m^3/hr.m^2$ (Approved Document L1), however, the practice is to build new homes even more airtight (5 $m^3/hr.m^2$).

Formaldehyde unit conversions (from BS EN 717-1: 2004):

1 ppm = 1.24 mg/m³ (at 23 °C and 1 013 hPa).

1 mg/m³ = 0.81 ppm.

Unless stated otherwise, these factors will be used throughout this document.

1 Introduction

Formaldehyde is a reactive substance that exists as a gas at ambient temperature and standard atmospheric pressure. It is classified in the Great Britain (GB) Mandatory Classification and Labelling (MCL) list as a carcinogen category 1B, mutagen category 2, acute toxicant category 2 by the inhalation route and category 4 by the oral route, skin corrosive category 1B and skin sensitiser category 1A. Exposure to formaldehyde has also been linked with asthma-like respiratory symptoms.

Formaldehyde is predominantly used as a chemical intermediate to produce a range of formaldehyde-based resins and other chemicals. Formaldehyde-based resins are used extensively as adhesives and binders in the production of wood-based panels and other wood-based products like furniture and flooring. Articles made with formaldehyde-based resins are widely found in homes and workplaces and can release (off-gas) formaldehyde during their service life. Formaldehyde can also be introduced into indoor air from other sources such as burning candles, incense, wood burning, ethanol fires and use of fragranced household and personal care products.

Some people have been reported to spend about 90% of their time indoors, much of it at home (around 66%). This is particularly the case for vulnerable populations, such as young children and elderly people. Indoor air pollutants (including VOCs and formaldehyde) are thought to cause significant adverse health effects (NICE, 2020). Consequently, improving indoor air quality has been highlighted as a priority for UK Government.

This report has been prepared by HSE, in conjunction with the UK Health Security Agency (UKHSA), the government agency responsible for public health protection, to collate information on sources that emit formaldehyde into indoor air environments in GB and understand the levels of formaldehyde that have been measured. Further, an outline of the evidence for adverse effects occurring in the general population, including children, at these levels is provided.

Risks to workers exposed in occupational settings are out of scope as are risks from exposure to formaldehyde outdoors and risks to the environment. The information used for this report mainly comes from:

- Research carried out by the UKHSA
- Documents published by the European Chemicals Agency (ECHA) to support the introduction of (Commission Regulation (EU) 2023/1464) which sets limits on emissions from various formaldehyde emitting articles.
- An indoor air exposure assessment as part of a risk evaluation on formaldehyde by the United States Environmental Protection Agency (US EPA) in December 2024

- Reviews of formaldehyde inhalation toxicity by expert groups
- Information gathered during a call for evidence held between April and June 2023.

2 Substance(s) covered in this report

This section provides information about the REACH registration status of formaldehyde and of manufactured substances and polymers that have been identified as having the potential to release formaldehyde depending how they (or articles comprising them) are used. In addition to manufactured substances and polymers, other sources of formaldehyde are present in indoor settings. Data on the levels of formaldehyde emissions from various sources is discussed in Section 3.2.

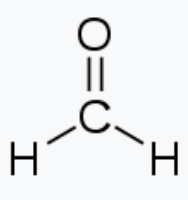
2.1 Formaldehyde

2.1.1 Substance identity

Table 1: Substance identity information for formaldehyde

EC Number:	200-001-8
CAS Registry Number:	50-00-0
IUPAC name (public):	Formaldehyde
Index number in the GB MCL list:	605-001-00-5
Molecular formula:	CH_2O
Molecular weight or molecular weight range:	30.026 g/mol
Synonyms:	Methyl aldehyde Formalin Methanal
Type of substance:	Mono-constituent

Structural formula:



Registration Dossier - ECHA (europa.eu)

2.1.2 Tonnage and registration status (UK REACH)

UK REACH applies to chemical substances that are manufactured in or imported into GB. This regulation applies to all individual chemical substances on their own, in mixtures or in articles. Manufacturers and importers of substances are required to understand the hazards of the substances they are supplying to the GB market.

Following the UK's exit from the EU, transitional provisions known as 'grandfathering' allowed GB based companies with existing EU REACH registrations to carry them over into the new UK REACH system. These transitional arrangements suspended the need to update registrations with GB specific use and exposure information until the relevant deadline for submission (depending on tonnage and hazard). At the time of searching the UK REACH registration database in August 2024, approximately 30 registrations had been submitted for formaldehyde under the transitional registration provisions, with an aggregated tonnage of 100,000 – 1,000,000 tonnes per annum.

2.1.3 Tonnage and registration status (EU REACH)

Formaldehyde is registered under EU REACH. It is manufactured and/or imported to the EU at ≥ 1,000,000 tonnes per annum. As of September 2024, ECHA held 271 active EU REACH registrations.

2.1.4 Manufacture and supply of formaldehyde

Formaldehyde is manufactured via the catalytic, vapour-phase oxidation of methanol (IARC, 2012). In 2015, 5 sites in the UK manufactured formaldehyde; at these sites formaldehyde manufacturing is integrated with the manufacturing of formaldehyde-based resins and/or other chemicals (ECHA, 2020a). According to information published by Formacare (this is the formaldehyde sector group of the European Chemical Industry Council, Cefic), the UK is the fifth largest producer of formaldehyde in the geographic region of Europe after Germany, Italy, Spain and the Netherlands (Formacare, 2023a).

In 2015, the total annual formaldehyde production in Europe (28 Member States (including the UK) plus Switzerland and Norway, 30 countries in total) was of the order of 3.2 million tonnes. Since formaldehyde is usually supplied as an aqueous solution, this equates to 8.6 million tonnes (37% aqueous solution) (ECHA 2020a). Between 2015 and 2017, around 20,000 – 30,000 tonnes per year of formaldehyde were imported into the EU (Eurostat 2018b, cited in ECHA 2020a). This indicates the majority of formaldehyde used in the EU is manufactured within the bloc. HSE does not have manufacture and import data specifically for the UK or GB.

Of the majority of formaldehyde manufactured or imported into the EU, 98% is used as a chemical intermediate in the production of formaldehyde-based resins, thermoplastics and other chemicals, which have applications in products such as wood panels and particleboard for construction and furniture making (ECHA, 2020a). There is no reason to consider that this percentage will be different for GB. The Alliance for Sustainable

Building Products (ASBP) states that formaldehyde is used as a raw material in as many as 85 industries and is used to make hundreds of different types of products (ASBP, 2017).

2.2 Manufactured substances identified as formaldehyde releasers

Substances can act as formaldehyde releasers because they have been manufactured using formaldehyde as a starting material. During their life-cycle, formaldehyde can be released either because the formaldehyde is present as a residue or because of degradation or other chemical reactions. Levels of formaldehyde residues depend on the manufacturing process and any post manufacturing treatments that are applied. Other factors will determine levels of formaldehyde released due to chemical processes such as degradation (see Section 3.1 for further details). Substances containing methylol groups (-CH_2-OH) could potentially be a source of formaldehyde depending on their chemical structure and factors such as pH (ECHA, 2017).

ECHA (2020b, Section B, Table B.2) lists around 40 manufactured substances which it identifies as potential formaldehyde releasers. This should not be regarded as an exhaustive list (ECHA, 2017). At the time of searching the UK REACH registration database in August 2024, 5 of these substances had been registered under UK REACH:

- **Methylene diphenyl diisocyanate (MDI) (CAS RN: 101-68-8)** – manufactured by reacting aniline with formaldehyde to produce 4,4' methylenedianiline and other diamine precursors which are then treated with phosgene to form a mixture of isocyanates. Most MDI goes into the production of polyurethane (PU) foams which are used in a number of applications including as an insulation material in construction and automobiles. ECHA's registration information (accessed August 2024) indicates that MDI may also be used in the manufacture of composite materials with wood or other fibrous materials, in certain coatings, adhesives and sealants and as a cleaning agent. Polyurethane foams, coatings and adhesives containing MDI are supplied for use by consumers.

- **1,4-Butanediol (BDO) (CAS RN: 110-63-4)** – manufactured by reacting acetylene with formaldehyde followed by a hydrogenation step. BDO is an intermediate in the production of tetrahydrofuran (THF) and polybutylene resins. THF resins are used to produce spandex fibres (for example Lycra) as well as elastomeric products such as buttons and rollers. Polybutylene resins have a variety of applications including the production of car bumpers and connectors and insulators in electrical components. In registration information (accessed August 2024), ECHA also lists use as a solvent/reactive processing aid in the manufacture of coatings and inks, use in adhesives and sealants and use in binders and release agents.

- **Pentaerythritol (Penta) (CAS RN: 115-77-5)** – manufactured by condensing acetaldehyde with formaldehyde in aqueous solution in the presence of an alkaline agent. The largest use of Penta in the EU is the production of alkyd resins. Alkyd resins are found in architectural coatings like paints and product finishes for automobiles. Penta is also used to make neopolyol esters which are used in engine lubricants for aeroplane turbines and automobile engines. ECHA's registration information (accessed August 2024) identifies use in the manufacture of intumescent polymers/paints (these expand on exposure to heat during a fire situation to create a heat resistant barrier thereby slowing the spread of fire) and PVC articles.

- **Methenamine (hexamine) (CAS RN: 100-97-0)** – manufactured by reacting formaldehyde with ammonia. It is primarily used to make epoxy resins. Hexamine is also used as an accelerator to create vulcanised rubber for tyres. ECHA's registration information (accessed August 2024) identifies professional and consumer use as a processing aid and as a functional fluid and use in cosmetics/personal care products. Although hexamine has been used as a solid fuel source for certain types of camping stoves, this use (and potentially other consumer uses that may be listed in ECHA's registration information) is likely to be impacted by restrictions that were introduced via the Control of Explosive Precursors and Poisons (Amendment) Regulations 2023 that came into effect from 1st October 2023 prohibiting the supply of this substance to the general public without a licence.

- **2,2',2''-(hexahydro-1,3,5-triazine-1,3,5-triyl)triethanol (CAS RN: 4719-04-4)** – manufactured by reacting formaldehyde with ethanolamine. ECHA's registration information (accessed August 2024) indicates that this substance is primarily used in oil and gas exploration and at refineries. It acts as a hydrogen sulfide scavenger to limit worker exposure to this hazardous gas. It is also approved for use as a biocide in GB and the EU (disinfectant and algicide) with applications for several product types (HSE, 2024).

There is evidence that a further 7 substances that can act as formaldehyde releasers are imported into GB. GB-based companies who imported substances from EU-based suppliers before the UK's exit from the EU (1st January 2021) had no EU REACH registration obligations as they were classed as Downstream Users (DUs). As they are now importers of chemicals from outside of GB, they may have registration obligations under UK REACH. However, a transitional measure allows former DUs to suspend the registration until the relevant deadline for submission (depending on tonnage and hazard). Where the identity of these imported substances was known, they could be included in a Downstream User Import Notification (DUIN) submitted to HSE. An initial search of data submitted in DUIN submissions in April 2024 identified submissions for the following substances, suggesting that these are imported from the EU (further information about the regulatory status of each of the formaldehyde releasing substances identified by ECHA is provided in Annex 2):

- **7a-ethyldihydro-1H,3H,5H-oxazolo[3,4-c]oxazole (CAS RN: 7747-35-5)** – This bicyclic compound is often manufactured through reactions involving amino alcohols and carboxylic acid derivatives, processes that can indirectly involve formaldehyde as a precursor or reagent. It is used as a chemical intermediate in the synthesis of pharmaceuticals and is listed by ECHA as a biocidal active substance.

- **1-[1,3-bis(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]-1,3-bis(hydroxymethyl)urea (Diazolidinyl Urea; CAS RN: 78491-02-8)** – manufactured by reacting allantoin and formaldehyde in the presence of sodium hydroxide solution and heat. This is used as an antimicrobial preservative in personal care products and household detergents.

- **N,N''-methylenebis[N'-[3-(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]urea] (Imidazolidinyl urea; CAS RN: 39236-46-9)** – chemically related to diazolidinyl urea. Imidazolidinyl urea can be used as a preservative in personal care products. Sigma Aldrich also lists use as a hydrogen-bonding reinforced factor to prepare polymeric supramolecular hydrogels by using polyethylene glycol (PEG) and diisocyanates also multi-functional polyacrylamide hydrogels.

- **1,3-bis(hydroxymethyl)-5,5-dimethylimidazolidine-2,4-dione (DMDM hydantoin; CAS RN: 6440-58-0)** – manufactured by reacting an excess of formaldehyde with dimethylhydantoin. This substance is used in the synthesis of pharmaceuticals and agrochemicals. It is also used as a preservative in personal care products.

- **Dimethoxymethane (CAS RN: 109-87-5) -** Dimethoxymethane, also known as methylal, can be manufactured by oxidation of methanol or by the reaction of formaldehyde with methanol. It is used as a solvent and chemical intermediate.

- **1,3-Bis(hydroxymethyl) urea (CAS RN: 140-95-4)** – manufactured by reacting an excess of formaldehyde with urea. 1,3-Bis(hydroxymethyl)urea is used as a formaldehyde-releasing preservative in cosmetics. The US National Library of Medicine indicates that it is also used in the production of urea-formaldehyde resins and as a textile finishing agent.

- **2-(Hydroxymethylamino)ethanol (CAS RN: 34375-28-5)** – manufactured by reacting ethylenediamine with ethylene oxide, 2-(Hydroxymethylamino)ethanol is used as an intermediate in the production of other chemicals.

DUINs could be submitted for substances imported from the EU-27 into GB at any point within the two years prior to EU exit. They represent an approximation of substances on the GB market in the period before EU exit. However, as DUIN submission was a simple process and free of charge, companies may have under or over-reported substances (potentially erring on the side of caution to be compliant). Consequently, the DUIN data needs to be treated with caution. Many former DUs will not (currently)

have full information on the identity of the substances they import from the EU. This is because most substances are placed on the market as mixtures and the full composition of those mixtures is not always given on a Safety Data Sheet (SDS) or a SDS may not be required. In addition, substances could be imported by multiple importers in lower volumes (i.e. below 1 tonne/year/importer); in these cases, the substances did not need to be included in a DUIN submission as there would be no registration duty under UK REACH for each importer.

UK and EU registration data cannot provide a complete picture of the respective markets for these substances as they do not include substances manufactured or imported by individual companies below one tonne per year. It may be the case that these and other formaldehyde releasing substances are present in small quantities in semi-finished or finished imported goods (articles), but there is no requirement to register these imports unless the articles are designed to intentionally release the substance during service life and the aggregate import equals or exceeds one tonne per year. Importers/ suppliers may in any case be unaware of their presence, due to commercial confidentiality. In addition, there is no registration requirement for polymers under UK or EU REACH, however polymers may be a major source of formaldehyde released into indoor air.

Commercially important polymers/resins that are manufactured using formaldehyde and which could release formaldehyde during the service life of articles include:

- **Phenol formaldehyde (PF) resins** – manufactured by reacting phenol with formaldehyde directly to produce a thermosetting network polymer or (where the ratio of formaldehyde to phenol is < 1) to produce a prepolymer referred to as a novolac. PF resins (including Bakelite) have high moisture and chemical resistance as well as high heat resistance. End-use applications include fiberglass insulation, decorative and industrial laminates and under-the-hood components (e.g. friction materials) in automobiles. ECHA (2020a) states that around 60% of PF resins are put to these uses.

- **Urea formaldehyde (UF) resins** – amino thermosetting resins produced by combining urea and formaldehyde heated with a mild acid catalyst like ammonia. The largest application for formaldehyde is the production of UF resins. These are thermosetting adhesives which are resistant to abrasion. Most UF resins in the EU (around 95% according to ECHA, 2020a) are used to make building materials such as particleboard and plywood. UF polymers are also used in the manufacture of urea-based controlled-release fertilisers which are used in agriculture, professional horticulture and landscaping.

- **Melamine Urea Formaldehyde (MUF) and Melamine Formaldehyde (MF) Resins** - These are poly-condensation products of the reaction of formaldehyde with urea and melamine. MUF and MF resins have similar processing applications and applications to urea-formaldehyde resins, MF and MUF resins

are less susceptible than UF resins to release of formaldehyde (Conner, 2001). Formaldehyde free melamine-urea resins are also available.

- **Resorcinol formaldehyde (RF) and phenol resorcinol formaldehyde (PRF) resins –** produced by replacing phenol with resorcinol onto the active ethynol groups. These resins are used for exterior-grade timber structures because of their strong, water and weather resistant bonds (Conner, 2001).

- **Polymeric forms of formaldehyde** – polyoxymethylene (POM; CAS RN: 66455-31-0). POM is an engineering thermoplastic, also referred to as polyformaldehyde. Polyoxymethylene homopolymer is manufactured by the reaction of aqueous formaldehyde with alcohol followed by polymerisation. To produce polyoxymethylene copolymer, formaldehyde is converted to trioxane before polymerisation with a co-monomer such as dioxolane (which is formed by reacting ethylene glycol with aqueous formaldehyde). Polyoxymethylenes (POM) are used to make precision parts that require high stiffness, low friction, and can be moulded into highly precise, complex shapes. As a result, they have replaced metals and other plastics in many applications, combining high resistance properties and lighter weights. In the EU, POM are primarily used in a wide range of industrial and automotive applications (ECHA, 2020a).

HSE does not have information about the extent to which these resins are manufactured in GB or imported in an uncured state. Finished articles made using these resins will also be imported.

There may be other substances which have not been manufactured using formaldehyde as a raw material, but which can release formaldehyde as a result of chemical processes. One example may be artificially synthesised compounds that also occur in nature such as the terpene limonene. The role of indoor air chemistry on indoor air pollution is discussed in Section 3.1.2.

3 Quantitative data on emissions and exposure

3.1 Factors affecting levels of formaldehyde in indoor settings

There is a complex inter-relationship between factors that will contribute formaldehyde to indoor air (sources) and those factors that will remove formaldehyde from indoor air (sinks). Other factors that can influence formaldehyde levels include indoor air chemistry and environmental parameters such as temperature and humidity.

3.1.1 Sources and sinks

Sources that release formaldehyde into indoor air may be categorised in several ways. For the purposes of this report, sources have been classified into continuous or intermittent sources.

3.1.1.1 Continuous sources

Continuous sources are those such as building materials and furniture. These are always or nearly always present and provide a permanent but declining release of formaldehyde over time. Based on its modelling work and supported by data from He *et al.* (2019), the US EPA has proposed that emissions from articles follow a biphasic emission profile, i.e., rapid emission of formaldehyde vapour when the product is new followed by a much slower emission of formaldehyde which may be generated from reactions arising from indoor air chemistry (see Section 3.1.2 for further information about the influence of indoor air chemistry and environmental parameters on formaldehyde emissions) (US EPA, 2024a). Continuous sources can have a large surface area such as carpets, floors and walls and can therefore provide a major contribution to the level of formaldehyde in indoor air, but typically this contribution will diminish over time (sometimes referred to as the ageing effect). Emissions could be in the form of gaseous formaldehyde but could also be as dusts generated from wear and tear during the service life of the article to which formaldehyde has adsorbed. Where coatings are applied (e.g., primer, paint, wallpaper) this can substantially reduce the levels of formaldehyde that will be emitted (see Section 3.2.2.2 for further information). This may not be the case if the coating is also a formaldehyde releaser (see Section 3.2.2.3, 'Other continuous sources' for further information).

3.1.1.2 Intermittent or transient sources

Intermittent or transient sources are typically point sources that emit for some but not all of the time, e.g., combustion (open fires, wood burners, wood stoves, ethanol fireplaces, high temperature oven cleaning, smoking and vaping, incense burners, scented candles) or use of fragranced personal and household care products. These

may release formaldehyde in higher concentrations than continuous sources but for shorter periods of time, i.e. only whilst the activity takes place. The extent to which these contribute to total indoor formaldehyde will depend on the frequency with which the activity is carried out and the scale of use. As before, emissions could be in the form of gaseous or particulate-adsorbed formaldehyde. Where activities generate particles, e.g. combustion, it may be the case that a greater proportion of formaldehyde is present as particulate-adsorbed formaldehyde compared with activities that do not generate particles to the same extent. HSE has not found any experimental data that quantifies the relative amount of gaseous versus particulate-adsorbed formaldehyde from any source and it is not clear whether the form in which formaldehyde is present in indoor air has relevance for the risk to health that may be created (this may be the case if deposition patterns in the respiratory tract change between gaseous and particulate-adsorbed formaldehyde).

3.1.1.3 Sinks

Adsorption and desorption of formaldehyde are processes related to the capture and release of formaldehyde in various materials. Adsorption is profoundly affected by both the pore structure and the surface chemistry of an adsorbent and acts as a sink together with the deposition of dusts to which formaldehyde has adsorbed onto surfaces indoors. However, desorption of formaldehyde from the material depends on indoor temperature (as explained in indoor chemistry below) and re-entrainment of dusts to which formaldehyde has adsorbed (this could be any indoor dust not just dusts originating from specific articles) can act again as a source. Formaldehyde can be removed from indoor air by ventilation (including exfiltration through the building fabric and opening windows); however, the hierarchy of control in terms of indoor air pollution requires interventions that remove the source to be considered as far as possible before the use of ventilation to maintain good indoor air quality.

The year when a building is constructed influences the airtightness of the building fabric and its ventilation characteristics, according to the corresponding building regulations at that time. Raw *et al*. (2004) found that homes built between 1991 – 1998 had higher indoor concentrations of formaldehyde (geometric mean (GM) ~ 40 µg/m^3) than older homes built before 1919 (GM ~16 – 17 µg/m^3), which were much leakier, with cracks and gaps in the building envelope, and consequently not energy efficient (this is discussed further in Section 3.3.2.2).

3.1.2 Indoor chemistry and environmental parameters

Other factors that influence formaldehyde levels include indoor air chemistry (this relates to chemical reactions that occur as a result of the composition of indoor air) and environmental parameters such as temperature and humidity. Depending on the combination of factors, these could increase or decrease the level of formaldehyde that is emitted from various sources.

3.1.2.1 Indoor air chemistry

3.1.2.2.1 Homogenous chemistry

Homogenous chemistry occurs when reactants are components in the same phase and exist in the liquid, solid, or gaseous phase. Hydrolysis is an important reaction that takes place in building materials and describes the decomposition of a chemical substance by reaction with water. It has been reported that at a relative humidity of 65%, aqueous surface films are common, and water is a substantial fraction (~30%) of airborne particles (Ho *et al.*, 1974 cited in Weschler and Carslaw, 2018). Hydrolytic decomposition of urea-formaldehyde resins, used in the manufacture of engineered wood boards, is of particular importance for the indoor environment. This reaction is known to be one of the main sources of indoor formaldehyde (Salthammer and Morrison, 2022).

3.1.2.2.2 Heterogeneous chemistry

Heterogeneous chemistry occurs when reactants are components of two or more phases. One example is ozone (a component of the gas phase), which deposits and reacts at the interface or within a condensed phase, such as a surface layer of grime. Ozone reactions with condensed phase unsaturated organics generate a wide range of products, from nearly volatile to very volatile, such as formaldehyde. It has been observed that at higher temperatures, reaction products volatilise more rapidly from surfaces.

Of particular importance to indoor formaldehyde levels are the reactions of ozone with monoterpene species from consumer products (Weschler and Carslaw, 2018). These reactions have been extensively studied (AQEG, 2022):

- monoterpenes, such as limonene and alpha-pinene, are ubiquitous indoors because they are used in large quantities in personal care and cleaning products, to add fragrance (Nazaroff and Weschler, 2004);

- ozone is also ubiquitous indoors; indoor ozone mainly originates from outdoors and enters the indoor environment by infiltration through the building envelope as well as by natural, or mechanical ventilation. The review on indoor ozone concentrations and influencing factors by Nazaroff and Weschler (2022) reported that measurements in approximately 2000 indoor environments (residences, schools, and offices) show a tendency for average indoor ozone concentrations of 4 – 6 ppb and an indoor-to-outdoor concentration ratio of about 1:4 (0.25). Ozone concentrations indoors are much lower than outdoors, as it rapidly deposits on indoor surfaces or human occupants and also reacts with nitric oxide (NO). However, indoor emission sources of ozone do exist; indoor ozone is generated from laser printers and photocopiers, from electrostatic precipitators (ESPs) designed for particle control as well as from portable air ionizers marketed for indoor air cleaning.

- ozone-monoterpene reactions are fast enough to compete with typical air exchange rates indoors (Weschler and Carslaw, 2018) and can form both formaldehyde and secondary aerosols as oxidation by-products (Abbatt and Wang, 2020).

3.1.2.2 Environmental parameters

In a literature review, Salthammer and Morrison (2022) investigated the impact of temperature on indoor air pollutant concentrations, including formaldehyde. Because of its reactivity, chemical reactions which can be modified by ambient temperature and humidity have a greater effect on formaldehyde emissions from engineered wood boards than diffusion. Diffusion is more relevant for emissions of non-reactive substances. Parthasarathy *et al*. (2010) carried out chamber experiments to gauge the effect of temperature and humidity on formaldehyde emissions. Tests conducted under various combinations of temperature (15, 25 and 35°C) and relative humidity (50% and 85%) on samples from 4 articles made using engineered wood boards revealed emissions under high temperature and high humidity conditions were at least 10 times greater than emissions under low temperature and low humidity conditions.

Temperature not only affects emissions of formaldehyde from building products but can affect desorption rates from surfaces in the home (US EPA, 2024b). As noted previously, formaldehyde adsorption to surfaces can be considered as “indoor sinks” (Plaisance *et al*., 2013). Increased temperature and energy control in newer homes reduces the ventilation rates and raises temperatures. At higher temperatures, formaldehyde may re-emit from surfaces it is adsorbed to, indicating adsorption to surfaces in the home is not always a route of dissipation for formaldehyde and may result in persistence of the material in indoor air (US EPA, 2024b, Plaisance *et al*., 2013).

3.2 Quantitative emissions data

The majority of the emissions data currently available relates to emissions from continuous sources, in particular engineered wood boards and products made with those boards. Less information is available about other continuous sources and intermittent sources. As continuous sources age and formaldehyde emissions from them taper off, intermittent sources will provide a larger share of indoor formaldehyde compared with the situation in a newly built or newly furnished dwelling. Whether the contribution from intermittent sources exceeds that from continuous sources will depend on the age of continuously emitting sources and the frequency and scale of activities that give rise to intermittent sources. Both of these factors are influenced by the activities of individual householders. For this reason, to inform regulatory decision making, ECHA and the US EPA used modelling approaches to estimate the indoor air concentration of formaldehyde that could arise with various combinations of continuous and intermittent sources.

The following text summarises the methods used to measure emissions from articles

and intermittent sources, quantitative emissions data identified by the HSE and the modelling approaches that have been applied to examine how indoor air concentrations may vary with different combinations of sources.

3.2.1 Emissions testing and interpreting emissions data

Emissions of formaldehyde from articles and other sources can be tested for and the results expressed in different ways. A description of various testing approaches which may be used is available in Salthammer *et al*. (2010). To ensure tests are conducted under defined conditions, various standards organisations have specified methods, and these are often harmonised across multiple standards setting bodies. British Standard BS EN 717 (which replicates EN 717 for GB) defines methods for determining the formaldehyde emissions of individual engineered wood boards and panels under standard conditions (i.e. temperature (23 ± 0.5°C), relative humidity (45 ± 3%), air velocity (0.1 – 0.3 m/s at the surface of the test piece) and air exchange rate (1 ± 0.05/h)). Methods incorporated into this standard include BS EN 717-1 (the chamber method), BS EN 717-2 (the gas analysis method) and BS EN 717-3 (the flask method). These methods can be used for other product types in addition to wood boards. The amount of formaldehyde that is released can be expressed both in terms of concentrations in the chamber under the specific conditions (ppm or µg/m^3) and in terms of emission rates (µg/m^{2}h).

Other standards that may be referred to include BS EN 16516:2017+A1:2020 for construction products, BS EN ISO 12460 for uncoated and coated engineered wood boards and other materials and BS EN ISO 16000-9, a chamber method for the determination of emissions of volatile organic compounds from samples of building products.

The test conditions in BS EN 717 and other standards will not replicate the variations in environmental conditions that will exist across all indoor settings. For this reason, emissions data only give an indication about which sources could provide a greater or smaller share of indoor formaldehyde emissions. Also, emissions tests are typically calculated on new products and, therefore, for continuous sources, emissions test results are likely to represent a worst-case for that source and will not reflect the decline in emissions that occurs as articles age.

3.2.2 Measured data for continuous sources

Any article that has been made using polymers that may contain formaldehyde residues or can emit formaldehyde as a result of indoor air chemistry can act as a continuous emitter of formaldehyde.

ECHA (2020b), provides emissions data from chamber tests and the methods used to generate that data for various continuous sources. This has been summarised into Table 2 (emission rates; µg/(m^2/h) or µg/h) and Table 3 (air concentration; µg/m^3). It is not possible to make direct comparisons between emission rates and air concentrations.

Table 2: Measured emission rates for various articles reported in ECHA (2020b)

Product	Min	Average (GM, AM)	Max	Used method	Reference
Solid wood					
Solid wood (six different wood species)	0.014 mg/(m²h)		0.084 mg/(m²h)	EN 717-2	Böhm *et al.* (2012)
Wood-based products (plywood, particleboard, OSB, MDF, laminate flooring)					
Plywood, 22 mm	0.35 mg/(m²h)		2.65 mg/(m²h)	EN 717-2	Böhm *et al.* (2012)
Plywood, 8 mm	0.13 mg/(m²h)		1.66 mg/(m²h)	EN 717-2	Böhm *et al.* (2012)
Plywood, UF	0.3 mg/(m²h)		2.5 mg/(m²h)	EN 717-2	KEMI (2015)
Plywood, MUF	0.2 mg/(m²h)		2.0 mg/(m²h)	EN 717-2	KEMI (2015)
Plywood, PF	0.1 mg/(m²h)		0.4 mg/(m²h)	EN 717-2	KEMI (2015)
Plywood, uncovered, interior use (15 and 19 mm)	0.26 mg/(m²h)		0.36 mg/(m²h)	EN 717-2	Salem *et al.* (2012)
Plywood, uncovered, construction use (15 and 19 mm)	0.15 mg/(m²h)		0.18 mg/(m²h)	EN 717-2	Salem *et al.* (2012)
Particleboard, UF and unknown	2.4 mg/(m²h)		4.7 mg/(m²h)	EN 717-2	KEMI (2015)
Particleboard, uncoated (8, 10, 15, and 22 mm)	0.4 mg/(m²h)		0.84 mg/(m²h)	EN 717-2	Salem *et al.* (2012)
Particleboard, veneered (8, 10, 15, and 22 mm)	0.7 mg/(m²h)		2.52 mg/(m²h)	EN 717-2	Salem *et al.* (2012)
Particleboard, laminated, (8, 10, 15, and 22 mm)	0.22 mg/(m²h)		0.65 mg/(m²h)	EN 717-2	Salem *et al.* (2012)
Particleboard, UF		57.6 µg/m³ / 58.5 µg/(m²h)		ISO 16000-9	Yrieix *et al.* (2010)
Particleboard, uncoated		0.041 mg/(m²h)		EN 717-1	Yu and Kim (2012)
Particleboard, coated with MF paper		0.04 mg/(m²h)		EN 717-1	Yu and Kim (2012)

Product	Min	Average (GM, AM)	Max	Used method	Reference
OSB, UF		1.0 mg/(m²h)		EN 717-2	KEMI (2015)
MDF, UF and unknown	3.1 mg/(m²h)		3.6 mg/(m²h)	EN 717-2	KEMI (2015)
MDF, uncoated (3-22 mm)	0.23 mg/(m²h)		0.73 mg/(m²h)	EN 717-2	Salem *et al.* (2012)
MDF, laminated (2.5 and 3 mm)	0.2 mg/(m²h)		0.20 mg/(m²h)	EN 717-2	Salem *et al.* (2012)
MDF, uncoated		0.096 mg/(m²h)		EN 717-1	Yu and Kim (2012)
MDF, coated with PVC laminates		0.007 mg/(m²h)		EN 717-1	Yu and Kim (2012)
Laminate	< 3 µg/(m²h)		28 µg/(m²h)	Chamber test, unpublished data	Salthammer and Gunschera (2017)
Laminate (bonded laminate with particleboard)		0.410 mg/(m²h) (initial); 0.04 mg/(m²h) (14 days)		Chamber test	Wiglusz *et al*. (2002)
Laminate (thermofused saturated papers with HDF)		Initial emissions 14 times lower than in previous row		Chamber test	Wiglusz *et al.* (2002)
Furniture					
Shelf, MUF		1.1 mg/(m²h)		EN 717-2	KEMI (2015)
Wallcoverings					
Wallcoverings (N = 144)	107 samples < 1 µg/(m²h)		3 samples 31-60 µg/(m²h)	Chamber test, unpublished data	Salthammer and Gunschera (2017)

Product	Min	Average (GM, AM)	Max	Used method	Reference
Wallcoverings (N = 97)	89 samples < 1 µg/(m²h)		1 sample 11-30 µg/(m²h)	Chamber test, unpublished data	Salthammer and Gunschera (2017)
Paints					
Paints and lacquers	1 µg/(m²h) (10 days); 1 µg/(m²h) (28 days)		8 µg/(m²h) (10 days); 5 µg/(m²h) (28 days)	Chamber test	Horn *et al.* (2007) as reported in Salthammer and Gunschera (2017)
Paints	8.1 µg/(m²h)		9.8 µg/(m²h)		Salthammer *et al.* (2010)
Mineral wool, insulating materials					
Mineral wool board	22 µg/(m²h)	70-76 µg/(m²h)	225 µg/(m²h)	Chamber test	Wiglusz *et al.* (2000)
Textiles					
Carpet	3 µg/(m²h); 13 µg/(m²h)		16 µg/(m²h); 29 µg/(m²h)	Without O_3; with O_3	Abbass *et al.* (2017)
Carpet	3.5 µg/(m²h)		17.5 µg/(m²h)	Chamber test	Katsoyiannis *et al.* (2008)

Table 3: Measured air concentrations under standard test conditions for various articles reported in ECHA (2020b)

Product	Min	Average (GM, AM)	Max	Used method	Reference
Solid wood					
Solid wood (six different wood species)	0.004 mg/m³		0.008 mg/m³	EN 717-1	Böhm *et al.* (2012)
Solid wood flooring (spruce, 10 and 15.5 mm)	0.035 mg/m³		0.125 mg/m³	EN 717-1	Salem *et al.* (2012)
Solid wood flooring (oak, 10 and 15 mm)	0.021 mg/m³		0.041 mg/m³	EN 717-1	Salem *et al.* (2012)
Solid wood flooring (bamboo, 12 and 15 mm)	0.01 mg/m³		0.082 mg/m³	EN 717-1	Salem *et al.* (2012)
Wood-based products (plywood, particleboard, OSB, MDF, laminate flooring)					
Blockboard, uncoated	0.015 mg/m³		0.023 mg/m³	EN 717-1	Böhm *et al.* (2012)
Particleboard (chipboard)	0.042 mg/m³		0.098 mg/m³	EN 717-1	Kolarik *et al.* (2012)
OSB	< 0.01 mg/m³		0.042 mg/m³	EN 717-1	Kolarik *et al.* (2012)
MDF, UF and unknown	0.10 mg/m³		0.13 mg/m³	EN 717-1	KEMI (2015)
MDF	< 0.01 mg/m³		0.101 mg/m³	EN 717-1	Kolarik *et al.* (2012)
Flooring laminate	0.006 mg/m³		0.018 mg/m³	EN 717-1	Böhm *et al.* (2012)
HDF laminate (7, 8, 11, and 12 mm)	0.042 mg/m³		0.123 mg/m³	EN 717-1	Salem *et al.* (2012)
PVC/HPL laminate (5 and 13.5 mm)	0.025 mg/m³		0.041 mg/m³	EN 717-1	Salem *et al.* (2012)
PVC-laminate with UV-durable layer	0.003 mg/m³		0.008 mg/m³	EN 717-1	Salem *et al.* (2012)
Furniture					

Product	Min	Average (GM, AM)	Max	Used method	Reference
Stool, chair	0.10 mg/m³		0.18 mg/m³	EN 717-1	Andersen *et al.* (2016)
Kitchen front door	< 0.01 mg/m³		0.15 mg/m³	EN 717-1	Andersen *et al.* (2016)
Bookcase, chest of drawers	0.01 mg/m³		0.03 mg/m³	EN 717-1	Andersen *et al.* (2016)
Table, cabinet, armchair	< 0.01 mg/m³		0.02 mg/m³	EN 717-1	Andersen *et al.* (2016)
Bookcase and drawer (chipboard)	0.027 mg/m³		0.046 mg/m³	EN 717-1	Kolarik *et al.* (2012)
Paints					
Paints	< 0.01 mg/m³		0.010 mg/m³	EN 717-1	Kolarik *et al.* (2012)
Photocatalytic paints (lights on)			Peak value of 76 µg/m³	Chamber test	Salthammer and Fuhrmann (2007)
Mineral wool, insulating materials					
Insulating materials	< 0.01 mg/m³		0.011 mg/m³	EN 717-1	Kolarik *et al.* (2012)
Textiles					
Carpet	< 0.01 mg/m³		< 0.01 mg/m³	EN 717-1	Kolarik *et al.* (2012)
Curtain	1.0 µg/m³	2.5 µg/m³	5.3 µg/m³	EN 717-1	Aldag *et al.* (2017)
Curtain		< 0.01 mg/m³		EN 717-1	Kolarik *et al.* (2012)
Roller blind		0.047 mg/m³		EN 717-1	Kolarik *et al.* (2012)

3.2.2.1 Solid wood

Formaldehyde is a decomposition product of lignin and is therefore released in small quantities from solid wood products. ASBP (2017) reports emission rates ranging between 14 – 84 µg/(m^2h) for various wood species including oak, poplar, pine, birch, spruce and beech in order of increasing emission rate. ECHA (2020b) reports that air concentrations for these wood species range from 0.4 – 0.8 µg/m^3 when tested according to EN 717-1. Higher air concentrations ranging from 10 – 125 µg/m^3 were reported for solid wood flooring when tested according to EN 717-1.

3.2.2.2 Engineered wood boards

Engineered wood boards are typically made by binding together wood fibres (occasionally other sources of fibres are used) with a binder, typically a synthetic resin. Different types of engineered wood boards are made depending on the particle size of the wood material and how it is oriented within the panel. The type of resin that is used determines the levels of formaldehyde that may be emitted.

ECHA (2020a) provides the following about emissions relating to the resins used to produce engineered wood boards:

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- Urea formaldehyde (UF) resins are used in raw and covered wood-based materials, laminates, furniture, windows, and doors. UF resins are suitable only for indoor applications as wood-based materials containing UF resins are not water resistant. Moisture causes depolymerisation which releases formaldehyde. Average formaldehyde emission rates for UF-based wood products (bare) are 164 µg/(m^2h) (range 8.6- 1 580 µg/(m^2h)) (Salthammer et al., 2010).

- Phenol formaldehyde (PF) resins are water resistant and they are suitable for indoor as well as outdoor uses. The emission rates for PF-based wood products (bare) are in the range of 4.1-9.2 µg/(m^2h) (Salthammer et al., 2010).

- Melamine formaldehyde (MF) resins can be used in indoor and outdoor applications. They are water resistant and the formaldehyde emission rate is estimated to be around one-fifth of that related to UF resins (BAAQMD, 2012). Melamine urea formaldehyde (MUF) resins are also water resistant and their formaldehyde emissions are low compared to UF resins – in the area of 50% of the emissions related to UF resins (Salem et al., 2011).

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Appendix B of BS EN 13986 (the harmonised British and European standard for engineered wood boards used in construction) specifies two formaldehyde emission classes for engineered wood boards. Boards that release ≤124 µg/m^3 formaldehyde when tested according to EN 717-1 meet emission class E1. Boards which release >124 µg/m^3 meet emission class E2 (ASBP, 2017). There is no lower limit for emissions

for E1 boards and no upper limit for emissions for E2 boards. Panels to which no formaldehyde-containing materials were added during production or in post-production processing may be classed as E1 without testing. Such panels include unfaced cement bonded particleboards, certain unfaced wet-process fibreboards and unfaced, coated or overlaid boards glued with resins that do not emit formaldehyde or emit negligible amounts of formaldehyde (e.g. isocyanate-based or phenolic glues).

Since 2007, a voluntary agreement has been in place between European board producers to only supply E1 boards (ECHA, 2020a). This agreement includes all UK-based producers according to information provided to HSE by the Wood Panel Industries Federation (WPIF; trade body representing all UK-based wood panel producers). A minority (about 5%) of EU-based producers do not follow this voluntary agreement. ECHA (2020b) provides information on the share of boards meeting class E1 and class E2 produced in the EU (including the UK) and imported into the EU from various non-EU-28 countries. Table C3 of this ECHA document indicates that in 2017, no E2 panels were produced in UK. Across the EU as a whole 3% of EU-produced plywood, 4% of EU-produced particleboard and 2% of EU-produced MDF was classed as E2.

Some websites refer to boards of other classes e.g. E0.5 or E0. These classes are not recognised within British or European standard but may be used by suppliers for marketing purposes. Boards are also marketed as zero added formaldehyde. This is used for boards produced using formaldehyde-free resins.

Other, lower limits are also set in different countries, regions and by various organisations. To apply the voluntary Blue Angel label (accessed October 2024; this is an eco-labelling scheme developed by the German Federal Government) to a board would require emission levels of below 0.05 ppm (62 µg/m^3) when tested according to recognised methods including EN 717, while the eco-label Natureplus (an independent European eco label for construction products) would be 0.036 ppm (around 40 µg/m^3) which is in line with the standards set by members of the German Association of Prefabricated Houses (BDF) requiring members to uses boards with 0.03 ppm emissions (British Woodworking Federation, 2017).

Where wood panels are coated (e.g. primer, gypsum board, paint), this can significantly reduce emissions of formaldehyde. Salthammer (2019a) reported emission reductions ranging from 70 – 98% depending on the number and type of coating materials (primer alone or primer plus dispersion paint, plaster, fleece wallpaper or latex paint). Another study reported by Salthammer (2019a) measured air concentrations of 0.13 ppm (~ 160 µg/m^3) for uncoated particleboard dropping to 0.02 ppm (~25 µg/m^3) for particleboard covered by a diffusion barrier film, gypsum plasterboard and vinyl wallpaper. Coatings do not automatically reduce emissions. ECHA (2020b) reports data from a study in which veneered particleboard of various thicknesses gave rise to higher emissions (0.7 – 2.52 mg/(m^{2}h)) compared with the same thicknesses of uncoated particleboard (0.4 – 0.84 mg/(m^{2}h)) (see Table 2).

3.2.2.2.1 Main types of engineered wood boards supplied to the GB market

3.2.2.2.1.1 Particleboard

Particleboard (sometimes referred to as chipboard) is an engineered wood-based sheet material in which typically soft wood chips such as spruce, pine and fir but sometimes hardwood chips such as birch are bonded with a resin adhesive such as UF, MUF, PF or PMDI (Polymeric MDI). UF is the most commonly used resin but is only suitable where the panel will be used in dry conditions. Alternative resins (MUF, PF, PDMI) are used for environments such as bathrooms or kitchens where damp and moisture may be present. The typical constituents of particleboard are 83 – 88% by weight wood chips, 6 – 8% formaldehyde-based resin or 2 – 3% PMDI, 5 – 7% water and 1 – 2% paraffin wax solids.

Particleboard can be used for a wide range of furniture and construction applications including kitchen units, worktops, dining room and bedroom units where it typically has a veneered or laminated finish and load-bearing applications such as flooring (GreenSpec, accessed Sept 2024). Information the WPIF provided to HSE indicates that around 78% of particleboard supplied to the GB market is manufactured in GB. The majority (98%) of the remaining market share is supplied from the EU with only 2% being imported from the rest of the world. These percentages do not take account of particleboard supplied in furnishings produced outside GB.

3.2.2.2.1.2 Oriented strand board

Oriented strand board (OSB) is an engineered wood-based material in which longer ‘flakes’ of wood, e.g. softwood such as spruce and pine or hardwood such as aspen, are bound with resin adhesive, mainly PF, MDI, PMDI or MUF (which confer moisture resistance), often with strands oriented in a particular direction. The degree of orientation varies widely, even within a single panel. This can be used to tailor the technical properties of the board. The removal of fine particulates before resin application means that OSB contains lower amounts of resin, typically 2 – 3% by weight. OSB is primarily used for construction e.g. flooring, flat roof decking and wall sheathing but may be used for furniture where its presence is not visible. For the same loading conditions, a thinner board of OSB can be used compared with particleboard (GreenSpec, accessed Sept 2024). Information provided to HSE by the WPIF indicates that around 64% of OSB supplied to the GB market is manufactured in GB. The majority (99%) of the remaining market share is supplied from the EU with only 1% being imported from the rest of the world.

3.2.2.2.1.3 Medium density fibreboard

Medium density fibreboard (MDF) is an engineered wood-based sheet material which is made by bonding soft or hardwood fibres that have undergone a thermal softening process with resins, mainly UF. Other resins such as MUF, phenolic resins and PMDI may be used where moisture resistance is required. MDF typically contains 82% wood fibre, 10% resin, 7% water, > 1% paraffin wax and > 0.05% silicon. Flame retardants can be added if fire resistance is required (GreenSpec, accessed Sept 2024).

Information from the WPIF indicates that around 48% of MDF supplied to the GB market is manufactured in GB. The majority (83%) of the remaining market share is supplied from the EU with 17% being imported from the rest of the world (11% Asia, 6% South America).

MDF is used for a wide range of construction and furniture applications including decorative features such as skirtings, architraves, window boards and decorative facades as well as in flooring materials. It is also used extensively in furniture because its smooth, dense surface provides a good base for painting, veneering and laminating (GreenSpec, accessed Sept 2024).

3.2.2.2.1.4 Wet process fibreboard

Wet process fibreboards are typically produced without resin using a method similar to papermaking. As with MDF, these fibreboards are made using thermally softened wood fibres (and occasionally recycled paper fibre) but instead of resins, this process mainly relies on the natural adhesive properties and felting behaviour of fibres to achieve bonding. Occasionally synthetic resins may be used and additives including wax, bitumen emulsion, natural oil (not further specified) or flame retardants may be added depending on the intended end use.

Fibreboards are classified according to their density:

- Hardboards have a density > 900 kg/m^3
- Mediumboards have a density > 400 kg/m^3 to <900 kg/m^3 (split into high density mediumboards 560 kg/m^3 to <900 kg/m^3 and low density mediumboards 400 kg/m^3 to <560 kg/m^3)
- Softboards have a density > 230 kg/m^3 to <400 kg/m^3

Wet process fibreboards find use in a wide range of construction and furniture related applications. Hardboards are used as drawer bottoms and unit backs also door facing, caravan interiors and floor coverings. High density mediumboards may be used as wall and ceiling linings. Low density mediumboards may be used as pinboards or components of partitioning systems. Softboards may be used as pinboards, underlay materials and acoustic absorbents (GreenSpec, accessed Sept 2024). HSE does not have GB specific information on manufacture and import for wet process fibreboards.

3.2.2.2.1.5 Plywood

Plywood can be made from hard or soft wood species or a mixture. Veneer plywood is produced by binding thin sheets of wood with an adhesive resin such as UF, MUF or PF. Typically sheets of veneer are layered with the grain of each layer oriented at 90° to the adjacent veneer but this can vary to meet specific technical performance requirements. Core plywood uses strips of wood (about 25 mm wide for blockboard, oriented vertically for laminboard) and resin binders to produce panels suitable for interior uses such a joinery, door blanks and furniture. The type of resin used determines the applications for which plywood is suited.

Plywood performance is governed by BS EN 314-2 which sets out three bond performance classes:

- BS EN 314 Class 1 for dry interior uses
- BS EN 314 Class 2 for high humidity environments such as covered exterior uses
- BS EN 314 Class 3 for exterior uses out of ground contact

UF panels are suitable for interior use and normally achieve class 1. MUF panels are more resistant to moisture and can achieve class 2. Increasing the melamine content improves moisture resistance to achieve class 3 and marine plywood standards (BS 1088). PF panels also have better moisture resistance than UF panels and can achieve class 3. Other standards apply to durability and appearance of plywood (e.g., BS EN 636: 2012+A1: 2015).

The WPIF indicates that all plywood supplied to the GB market is imported mainly from non-EU countries such as Brazil and other South American countries, with around 15% originating in China.

Structural plywood is used for floor decking, wall sheathing, flat roofing, concrete formwork and external cladding. Other types of plywood have a range of uses and can be overlaid with decorative veneers, phenolic resin film coatings or other decorative finishes.

3.2.2.2.1.6 Cement bonded particleboard

Cement bonded particleboard (CBPB) panels are a mixture of wood particles and Portland cement with a ratio of 60% cement, 20% wood and 20% water. The panels are heavier than wood and resin panels but are more resistant to fire, have high stiffness, high durability and are good sound insulators. They tend to be used for specialised applications in construction e.g., internal wall construction in public places, lift shaft linings, cabling ducts, motorway acoustic fencing and cladding of prefabricated housing units. HSE does not have specific information on the production of CBPB in GB.

3.2.2.2.1.7 Flaxboard

Flaxboard is an engineered sheet material made using flax shives (derived from the stems of the flax plant and are a by-product of linen) and resins such as UF, MUF, PF or PMDI. As for wood-based panels the resin type affects moisture resistance. The typical composition is at least 70% flax shives (wood flakes, chips and sawdust may also be included in the biomass content) and resin. Flaxboards are not suitable for load-bearing applications but may be used for fire-resistant door cores and partitions because this type of board has better fire-resistance compared with other board types. Flaxboard can also be used to make furniture, shelves and worktops (GreenSpec, accessed Sept 2024). HSE does not have GB specific information on production and import of flaxboard.

3.2.2.2.2 Measured emissions from engineered wood boards

Formaldehyde emissions from engineered wood boards vary widely depending on the type of board, its thickness, the bonding resin that has been used and whether a coating has been applied. In the EU, over the last 30 years, formaldehyde emissions from engineered wood boards have fallen from a measured concentration of 3 ppm (3,720 µg/m^3) in 1978 to approximately 0.1 ppm (124 µg/m^3) in 2007, measured in environmental chambers, as defined by the relevant standards EN 717-1 and EN 717-2 (Marutzky, 2008; cited by ASBP and British Woodworking Federation, 2017).

Salthammer and Gunschera (2017, as reported in ECHA 2020b) summarised area specific emission rates measured according to EN 717-1 for various types of uncovered boards (sometime referred to as raw boards). When converted to emission rates (expressed as µg/(m^{2}h) assuming steady state conditions), the median emission rates for different board types were:

- 99.5 µg/(m^{2}h) for particleboard (48 samples),
- 87 µg/(m^{2}h) for MDF (31 samples),
- 62 µg/(m^{2}h) for OSB (39 samples) and
- 50 µg/(m^{2}h) for plywood (30 samples).

This highlights particleboard and MDF as higher emitting board types compared with OSB or plywood which is consistent with the higher percentages of bonding resins that are required for these board types compared with other types.

Other smaller scale studies carried out according to EN 717-2 reported by ECHA (2020b) include results from an enforcement project carried out by the Swedish Chemicals Agency (KEMI) in 2014. Eighteen boards from 9 suppliers were tested. Emission rates ranged from 40 to 4,700 µg/(m^{2}h) for particleboard, 200 to 3,600 µg/(m^{2}h) for MDF and laminated MDF, 1000 µg/(m^{2}h) for one sample of OSB and 150 to 360 µg/(m^{2}h) for plywood. These ranges demonstrate the high variability in emission rates between and within board types. Higher rates were reported for thicker boards and boards bonded with UF resin or boards where the type of bonding resin was not known. The highest emissions rates came from boards that did not meet the E1 emission standard. KEMI followed this initial testing with EN 717-1 chamber tests for 7 panels (two UF-bonded particleboards and one particleboard where the binding resin was not known, two UF-bonded MDF boards and one MDF board where the binding resin was not known and one UF-bonded plywood) which did not meet the E1 standard. The average chamber concentration across all tested boards was 110 µg/m^3, particleboards gave chamber concentrations of 70 – 200 µg/m^3, MDF samples gave chamber concentrations of 100 – 130 µg/m^3 and the plywood sample gave a chamber concentration of 40 µg/m^3.

It is not clear if the emissions data reported by ECHA (2020b) are representative for

boards currently supplied to the GB market. The chamber concentrations reported by KEMI likely reflect a worst-case situation because the tests were performed using the highest emitting sample from 5 test samples taken from each of the 7 panels that did not meet the E1 standard. Since 2007, a voluntary agreement has been in place between European (including the UK) board producers to only supply panels that meet the E1 standard. While these tests were performed after this voluntary agreement was in place, the data is now over 10 years old and it is not known if anyone still supplies panels like those tested by KEMI.

A review of the primary literature may help to resolve some uncertainties around the characteristics of the materials that have been tested. While it cannot be assumed that these data are representative for current products on the market, these data illustrate the scale of emissions that are possible from boards that do not meet the E1 emissions rating.

3.2.2.3 Other continuous sources

Much less information is available for other continuous sources compared with the data that is available for engineered wood boards. The emissions data reported by ECHA (2020b) suggest lower rates and lower chamber concentrations from other continuous sources compared with engineered wood boards.

3.2.2.3.1 Furniture

Furniture is a major market for engineered wood boards, particularly MDF and particleboard. Whereas the majority of engineered wood boards supplied to the GB market (with the exception of plywood) originate in GB or the EU, there is less clarity about the origin of the boards used to make imported furniture. In many furniture applications, boards are used with some form of applied facing, which can include wood veneer, laminate, melamine and many others. ECHA (2020b) states that in addition to boards, surface veneers and lacquers are a source of formaldehyde emissions. If formaldehyde has been used as a fumigant or preservative in fabrics and foams used to produce the furniture, this could also act as a source of formaldehyde.

The current EU legislation focus on reduction of emissions from furniture, as these may have high density in other environments, such as schools. Measurements in French schools (160 establishments) showed annual mean levels of formaldehyde up to 30 $\mu g/m^3$ in 89.4% of schools (Michelot *et al.*, 2013) with the main source being furniture and cleaning activities. Increased ventilation was proved to reduce formaldehyde levels.

3.2.2.3.2 Wallcoverings

Historically, wallcoverings were a source of formaldehyde because of the use of adhesives to assemble layers of paper. Modern wall coverings tend to use formaldehyde-free fleece as the backing material giving rise to much lower emissions (Salthammer, 2019b).

3.2.2.3.3 Paints

If polymers that have been used to produce paints and lacquers contain monomer residues and those monomers have methanol groups, this could become a source of formaldehyde emissions.

3.2.2.3.4 Mineral wool

ECHA (2020b) identifies mineral wool insulation as a possible source of formaldehyde emissions as a result of the use of UPF resins as binders which can be around 3% by mass of this product.

3.2.2.3.5 Foams

Foams made using formaldehyde-based resins such as UF, MF or PF may be used for insulation or as padding in the manufacture of furniture. These may be a minor source of formaldehyde. Information provided to the US EPA by the North American Insulation Manufacturers Association noted that insulation products in which formaldehyde is a component of the binder are often cured at high temperatures which destroys formaldehyde residues (US EPA, 2024a). ECHA (2020b) reports emissions from polyurethane foams (PUF) are typically below 10 µg/m^3. This type of foam is mainly used in upholstered furniture and mattresses.

3.2.2.3.6 Textiles

ECHA (2020b) notes various uses for formaldehyde in textile production. If residues remain in the finished textiles this could be released during the service life of textiles (e.g. curtains or carpets) and articles with textile coverings.

3.2.2.4 Quantitative data on rates of decline in emissions from continuous sources

The review by Salthammer (2019a) identified few studies dealing with the long-term emission behaviour of materials and products. Most studies measured formaldehyde emissions from newly produced materials. In one study, an empirical potential function was applied to extrapolate the formaldehyde emission rate of particleboard, fibreboard and plywood. Taking the 28-day emission value as a starting point, it was estimated that:

1) for plywood, emissions could reduce by 33% after 1 year and 42% after 2 years, and

2) for particleboard, emissions could reduce by 45% after 1 year and 66% after 2 years.

Salthammer (2019a) estimated that when emissions from engineered wood boards are averaged over the lifetime of the board (assumed to be 10 years) they are about 40% of the emissions when new.

The US EPA (2024a) reports that formaldehyde emissions from articles are expected to decline over time according to a first-order exponential process. Chamber studies suggest emissions half-lives range from 1.5 to 2 years. With an emission half-life of 1.5 years, it was estimated that after 10 years, formaldehyde emissions from engineered wood boards would be approximately 1 percent of the initial emission rate. This may change depending on factors such as the article surface area, use pattern for the article, also temperature and humidity.

The Government of Canada is running a new large-scale study continuously measuring indoor air quality (IAQ) and formaldehyde in newly built homes up to 1 year post occupation (Government of Canada, 2019). This study may provide data for material aging under real home occupancy. The results are expected to be published late summer/early autumn 2025.

3.2.3 Measured data for intermittent sources

Combustion activities and use of fragranced products, combined with indoor air chemistry, provide intermittent sources of formaldehyde. Unlike continuous sources, intermittent sources provide a pattern of short (minutes) and intermediate (hours) duration peaks. The quantity of formaldehyde that is emitted from intermittent sources depends on the frequency with which the activity is carried out and the scale of use. These are heavily influenced by the activities of individual householders.

3.2.3.1 Household and personal care products

The EPHECT (Emissions, Exposure Patterns and Health Effects of Consumer Products in the EU) project, funded by DG SANCO (2012-2015), generated emissions data for the most frequently purchased brands of fifteen consumer products including all-purpose cleaners, kitchen cleaners, floor cleaners, glass and window cleaners, bathroom cleaners, furniture and floor polish products, combustible air fresheners, spray air fresheners, electric air fresheners, passive air fresheners, coating products for leather and textiles, hair styling products, spray deodorants and perfumes. Formaldehyde emissions arose from many of the consumer products that were tested; i.e., all-purpose cleaners (A1), kitchen cleaners (A2), floor cleaners (A3), furniture polish (A6), floor polish (A7), combustible air fresheners (A8), electric air fresheners (A11), and perfumes (A15).

Modelling was used to estimate the formaldehyde concentrations that might arise in different home microenvironments (ME) (Dimitroulopoulou *et al.*, 2015a). To estimate a realistic formaldehyde exposure of the residents, it was assumed that several consumer products were used within each home ME during the day. The range of consumer products assumed to be used and the locations in which they were used was based on information collected during the household survey carried out within the framework of the EPHECT project (Dimitroulopoulou *et al.*, 2015b). This survey identified the most popular consumer products, as well as collected detailed information on product use in terms of frequency, quantities, location, dilution and ventilation conditions during the use. A ‘most representative worst-case scenario’ was followed:

the scenarios reflecting the realistic worst cases of product use, as reported in the household survey, under the most representative conditions of use in Europe, were employed for exposure and health risk assessment. The pattern of movement of a typical resident through a dwelling including time spent sleeping in the bedroom was used to estimate the length of time a householder would spend in each ME. This information was used to estimate the formaldehyde exposure level that a typical householder may attain over a 30-minute or 24-hour period. Average ventilation rate of 0.35 ACH (referred to as 'normal ventilation conditions') in dwellings in Western Europe, represented by France and UK (Dimitroulopoulou, 2012) was used in the model simulations. In addition, the ventilation rate of 0.1 ACH was used to represent low ventilation conditions resulting from future building regulations on energy saving (infiltration rates in airtight homes are around 0.15 ACH and around 0 ACH for highly airtight homes), or inappropriate installation, maintenance and/or operation of ventilation systems in dwellings. Table 4 shows the results for formaldehyde indoor air concentrations in domestic micro-environments and the potential exposure to formaldehyde in West Europe. The consumer products that were used are reported under the table.

Table 4: Modelled formaldehyde concentrations in the home and extrapolated potential 24-hour exposures for West Europe (from Dimitroulopoulou *et al.*, 2015a).

Microenvironment	Product used	24-h mean Conc (µg/m³) (0.35 ACH)	Max 30-min Conc µg/m³ (0.35 ACH)	24-h mean Conc µg/m³ (0.1 ACH)	Max 30-min Conc µg/m³ (0.1 ACH)
Bathroom	A1, A2, A3, A11,A15	17	78	43	89
Living Room	A1, A3, A6, A7, A8	4	17	8	25
Kitchen	A1, A2, A3, A7	7	51	18	54
Bedroom	A1, A3, A6, A7	5	34	13	36
Exposure of a resident, based on representative use patterns and home occupancy behaviour		3	27	8	38

(A1) all-purpose cleaners, (A2) kitchen cleaners, (A3) floor cleaners, (A6) furniture polish, (A7) floor polish, (A8) combustible air fresheners, (A11) electric air fresheners, (A15) perfumes.

These data indicate that depending on product use and ventilation rate, use of household products can result in 24-hr mean exposures of residents between 3 and 8 µg/m³.

Based on the results from this work, Salthammer (2019b) estimated that consumer products such as cleaning agents, air fresheners, polish and perfume may contribute between 1 µg/m³ to 5 µg/m³ of formaldehyde to indoor air per item.

3.2.3.2 Data for combustion and other sources reviewed in Salthammer (2019a; b)

Salthammer and Gunshcera (2017) collated data on emission rates for major indoor sources of formaldehyde including both continuous and intermittent sources. This work which is summarised in Table 5, has been published by Salthammer (2019a; b) and provided key information for the EU Annex 15 restriction dossier. Where sufficient data were available, Salthammer derived statistical distribution functions (log-normal, normal, uniform) for the various data sets they had collated, and Monte Carlo simulations were used to identify various statistical parameters such as the mean, median, standard deviation, 25th and 75th percentiles. Where insufficient data were available to run Monte Carlo simulations, Salthammer (2019b) provided an estimated

range of emissions. As before, some data are reported as emission rates (µg/(m^2/h) or µg/h) and some results are reported as air concentrations (µg/m^3). It is not possible to make direct comparisons between emission rates and air concentrations.

Table 5: Distribution functions (log-normal, normal, uniform) and statistical parameters for formaldehyde emissions related to different products and processes in the indoor environment (Salthammer, 2019b).**

Product	Function	Emission factor
burning candles * (both scented and unscented)	log-normal	GM=192.5 µg/h, σ_g=1.42 µg/h
burning incense sticks *	uniform	Min=3 µg/m³, Max=39 µg/m³
burning mosquito coils *	uniform	Min=0.54 mg/h, Max=7.52 mg/h
tobacco smoking	uniform	Min=20 µg/m³, Max >1,000 µg/m³
electronic cigarettes	uniform	Min=1 µg/m³, Max=135 µg/m³
decorative fireplaces (during the burning phase, with different type of fuel: ethanol and gel-type) *	uniform	Min=698 µg/h, Max=10,637 µg/h
wood-burning fireplaces *	uniform	Min=5 µg/m³, Max=48 µg/m³
Cooking and cooking related activities such as high temperature oven cleaning	normal	µ=700 µg/h, σ=100 µg/h
air cleaning devices *	uniform	Min=2 µg/m³, Max=25 µg/m³
textile	log-normal	GM=1.9 µg/(m^2 h), σg=1.38 µg/(m^2 h)
carpet	log-normal	GM=3.9 µg/(m^2 h), σg=1.65 µg/(m^2 h)
surface coatings	log-normal	GM=2.3 µg/(m^2 h), σg=1.56 µg/(m^2 h)
wallcoverings	log-normal	GM=0.5 µg/(m^2 h), σg=2.23 µg/(m^2 h)
solid wood	normal	µ=4 µg/(m^2 h), σ=1 µg/(m^2 h)
particleboard	log-normal - normal	GM=79 µg/(m^2 h), σg=1.37 µg/(m^2 h)
OSB (Oriented strand	log-normal -	GM=39 µg/(m^2 h), σg=1.96

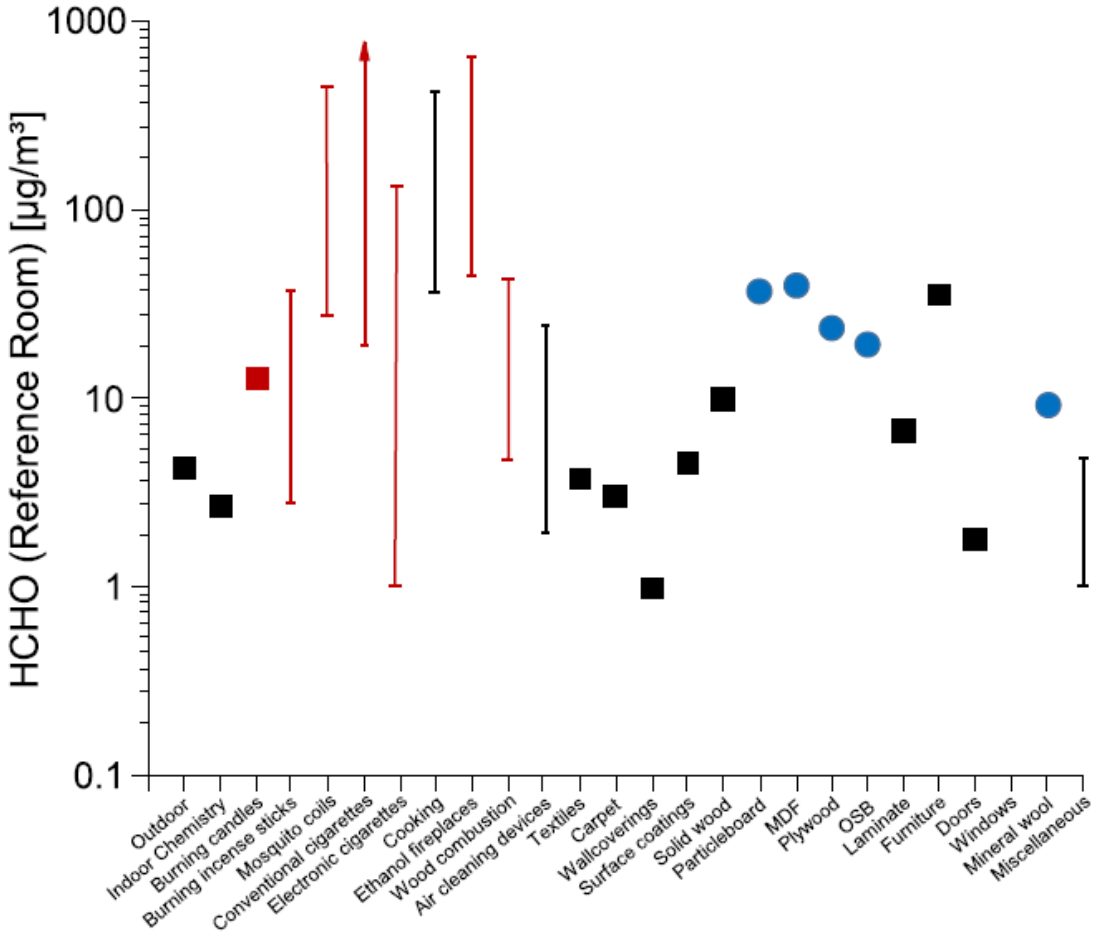
Product	Function	Emission factor
board)	normal	µg/(m^2 h)
MDF	n.a.	GM=80 µg/(m^2 h)
Plywood (uncovered)	n.a.	GM=48 µg/(m^2 h)
laminate	log-normal	GM=8.5 µg/(m^2 h), σg=1.8 µg/(m^2 h)
furniture	log-normal - normal	GM=17.8 µg/(m^2 h), σg=2.54 µg/(m^2 h)
doors	log-normal	GM=18.2 µg/(m^2 h), σg=2.7 µg/(m^2 h)
mineral wool	n.a.	GM=31.0 µg/(m^2 h)

∗Only one product (not several) was tested each time in the chamber; the distribution was then defined by testing different brands for the same product

**Distribution functions (see definitions for details) were developed by Salthammer (2019b) from the available data and combined by use of simulation methods. The shape of the distributions were informed by the available input data from Salthammer (2019a).

Although it is not possible to make direct comparisons between emission rates and air concentrations, Salthammer (2019b) converted the emission rates from various permanent and intermittent formaldehyde emitting product groups, activities, and processes, reported in Table 5, to concentrations in a European reference room, as described in EN 16516 (see Appendix 4) to obtain the graph shown in Figure 1. For unit specific emission rates, one single unit or item was assumed. Use of multiple items of one type (e.g. multiple scented candles or incense sticks) would increase emissions above the levels shown in Figure 1.

Figure 1: Comparison of Reference Room (30 m³) concentrations for a fixed air exchange rate (ACH) of 0.5 h^{-1} for different products and materials (geometric means and ranges) (from Salthammer, 2019b).



The blue dots represent concentrations calculated from engineered wood boards and mineral wool, whereas the red bars and the red square represent concentration ranges calculated for combustion processes. In this case cooking, represented by a black bar, includes high temperature oven cleaning as well as roasting fish or meat and stove top frying with various types of cooking oils.

It was concluded that among intermittent sources, combustion processes (e.g., conventional cigarettes, ethanol fireplaces, burning incense sticks and high temperature oven cleaning) generate some of the highest formaldehyde concentrations.

3.2.3.3 3D Printers

Small scale 3D printers are now widely available to the general public and may be used in various locations around homes including bedrooms. Chamber tests and studies in a test room (27 m^2, with air exchange approximately of 5 h^{-1}) with extrusion printers using

acrylonitrile-butadiene-styrene (ABS) and polylactic acid (PLA) (commonly used polymers for this type of 3D printing) found air formaldehyde concentrations of 2 to 3 $\mu g/m^3$ (Mendes *et al*., 2017).

The polymer type that is used will affect emissions. Davis *et al*. (2019) measured the following emissions of formaldehyde from several polymers that are used for 3D printing including ABS, PLA, nylon, high impact polystyrene (HIPS) and polyvinyl alcohol (PVA).

Table 6: Average formaldehyde emissions rates (ER) in µg/h for different polymers used in 3D printing

Polymer	ABS (N=12)	PLA (N= 9)	Nylon (N=2)	HIPS (N=1)	PVA
ER (µg/h)	24.7	7.0	6.1	30.2	21.6

Table 7: Minimum, average, and maximum emission yields of formaldehyde yields (µg/g) for different polymers used in 3D printing

Polymer	Minimum	Average	Maximum
ABS (n = 12)	0.6	2.2	6.2
PLA (n = 9)	0.2	0.4	1.0
Nylon (n = 2)	0.4	0.5	0.5
HIPS (n = 1)	-	2.4	-
PVA (n = 1)	-	1.9	-

These results indicate that use of 3D printers can contribute to indoor formaldehyde. Noting the ventilation rate of 5 ACH adopted by Mendes *et al*. (2017) is quite high compared with typical ventilation rates in domestic settings, the air concentrations that were measured in this study could underestimate real world emissions from 3D printers used in homes.

3.2.4 Modelling approaches

Given that there will be multiple sources emitting formaldehyde into homes, ECHA and the US EPA used modelling approaches to understand how air concentrations of formaldehyde might change with different combinations of sources.

3.2.4.1 Modelling approach used by ECHA

ECHA (2020a) constructed an exposure scenario intended to represent a newly built home in which engineered wood boards were used as the construction material and where various formaldehyde emitting articles were present. Three scenarios were devised to reflect rooms constructed with differing amounts of formaldehyde emitting materials but with the same level of formaldehyde emitting furnishings and textiles. Account was taken in the calculations of the barrier effects of paint on emissions from ceilings and walls, and of the sink effect of adsorption onto articles. The calculations did

not take account of the reduction in formaldehyde emissions as articles age. Combustion and other intermittent sources were also not taken into account. This approach is therefore tailored towards understanding the contribution to indoor formaldehyde that is made from articles, primarily engineered wood boards, used for construction and furnishings made with formaldehyde emitting materials.

Monte Carlo simulations were applied to generate a statistical distribution of formaldehyde in air concentrations under each scenario. This resulted in median formaldehyde concentrations of 56 – 88 µg/m^3, with a 95th percentile of 129 – 164 µg/m^3. When these are compared with the average concentrations reported for formaldehyde in homes (in the range of 20 – 40 µg/m^3; see Section 3.3), this shows the extent to which this modelling may be overestimating the contribution continuous sources make to indoor formaldehyde. Overestimation may arise because emissions values were derived for new articles and no account was taken of the decline in emissions as articles age. The ability of this type of modelling to generate realistic information about formaldehyde in air is very dependent on the parameters that are assumed for the room, the emissions that are assigned to different sources and the reductions that are applied to take account of the barrier effect of coatings, the ageing of articles and the removal of formaldehyde by sinks (e.g. deposition). If insufficient adjustments were made to take account of the barrier effect of coatings on boards used to construct homes and the ability of sinks to reduce levels of formaldehyde in indoor air this would also result in overestimation.

A further source of uncertainty is how representative the characteristics assumed for the European Reference Room to GB housing are. Salthammer (2019b) considered that while calculations using the Reference Room concept may be useful to compare emission behaviours of different products, a key weakness is that no account has been taken of the contribution from intermittent sources. Therefore, this approach does not reflect real living conditions.

3.2.4.2 Modelling approach used by the US EPA

For its risk evaluation, the US EPA used a two-tier approach to model formaldehyde in indoor air (US EPA 2024a). An initial assessment was performed using the US EPA's tier 1 Conceptual Exposure Model (CEM), version 3.2. Emissions data for various categories of article were used to estimate 1-year average daily indoor formaldehyde concentrations that could be generated from each article type. The estimated air concentrations generated by the CEM are dependent on the emission rate that is assumed for the article (the data used by the US EPA reflects emissions from new articles), and the expected surface area of the product in a room. Some account is taken of sinks such as air exchange between rooms but no account is taken of the ageing effect (the CEM assumes emissions are at a constant rate over time). It is therefore likely that the CEM will overestimate indoor air concentrations that will arise under realistic conditions of use. It is also the case that the CEM does not aggregate exposures from multiple sources. This modelling is therefore not suitable to determine indoor concentrations that could arise under real world conditions.

For this reason, the US EPA performed additional modelling calculations with the higher tier IECCU model (Indoor Environmental Concentrations in Buildings with Conditioned and Unconditioned Zones), version 1.1. This model allows emissions from several sources to be aggregated and an exponential rate of decay in emissions was assumed to take account of the ageing effect. As with ECHA's modelling and the CEM, this model does not take account of the barrier effect of coatings or the impacts of differing thicknesses of articles which may accelerate, delay and/or prolong emissions over time. Also, the IECCU model does not take account of environmental factors such as temperature and humidity which could also influence emissions.

The IECCU model predicted that emissions would tend towards zero after about 3 months which the US EPA concluded underestimated emissions. This was explained by the US EPA as suggestive that formaldehyde emissions from articles occurs in two phases with emissions during the initial phase mainly driven by volatility and longer-term emissions (after around 3 months) being driven by hydrolysis reactions between airborne moisture and resins and naturally occurring lignins. The IECCU model does not take account of emissions arising from chemical reactions with the article.

EPA compared these two models against the monitoring results of the American Healthy Homes Survey II (AHHS II). AHHS II is a national study which aimed to measure indoor formaldehyde and other indoor pollutant concentrations in US homes covering 689 homes from 78 cities across 37 states; measurements were taken between March 2018 and June 2019 (see Section 3.3.2.4 for further information). As with the modelling performed by ECHA, the models used by the US EPA did not take account of emissions from intermittent sources so do not represent real-world exposures. Whilst the Tier 1 model overestimates exposure by up to four-fold the Tier 2 model underestimated exposures when compared against the AHHS II survey.

In summary, both the ECHA and EPA indoor air modelling provide useful information on the source, scale, and relative importance of emissions from different sources, but do not represent exposures under real-world conditions. Such information can be provided through monitoring surveys, but full details of the conditions pertaining to the monitoring data must be known to compare studies.

3.2.5 Summary of emissions

Quantitative emissions data are available for various sources of continuous and intermittent emissions. This data will not provide accurate information about the levels of formaldehyde that will arise in homes where these sources are present because this data does not take account of environmental factors such as temperature and humidity which will affect emissions and will vary between homes. However, the data allows comparisons to be made about the relative contributions from various sources. It is clear from the available data that emissions between sources and even for different examples of a single type of source vary widely even without the additional variation due to household specific environmental factors.

For continuous sources, historically, engineered wood boards were a major source of emissions. However, the introduction of the E1 emission standard has resulted in voluntary actions by board producers, including most EU-based and all UK-based producers (ECHA, 2020b), to supply lower emitting E1 boards (to meet the E1 standard, boards must generate air concentrations of ≤ 124 µg/m^3 in EN 717-1 chamber tests when new). Boards that do not meet the E1 standard could emit considerably higher concentrations. Information supplied to HSE by the WPIF indicates that 98 – 99% of the particleboard and OSB and 83% of the MDF supplied to the GB market originates in GB or the EU and is therefore likely to meet the E1 standard. These percentages do not include the supply of articles made using engineered wood boards such as furniture. HSE does not have data on the market share for different countries of origin for these articles, thus there is uncertainty about the proportions of high and lower emitting articles that are supplied to the GB market. Other continuous sources such as wallcoverings, mineral wool insulation, foams, paints and textiles appear to emit lower quantities compared with boards.

As continuous sources age, emissions decline with an initial rapid decline in emissions followed by a long gradual tail off. Emissions data obtained from new articles therefore represent worst case emissions. Applying coatings to boards such as veneers or paint etc reduces emissions. The extent will depend on the type of coating and whether the coating is itself a formaldehyde emitter.

For intermittent sources, combustion processes (e.g., conventional cigarettes, ethanol fireplaces, burning incense sticks and high temperature oven cleaning) generate some of the highest formaldehyde concentrations. In order to compare sources, Salthammer (2019b) estimated the concentration in air for a standard reference room for a range of continuous and intermittent sources using geometric mean values of emission rates for articles of a defined area or single units of intermittent sources and assuming an air exchange rate of 0.5 h^{-1} (Figure 1). This shows that emissions from combustion sources during operation can be an order of magnitude higher than emissions from continuous sources. The impact that intermittent sources have on the overall room concentration of formaldehyde will be very dependent on the frequency and duration of operation of each intermittent source.

3.2.6 Uncertainties in emissions data

- There is a complex inter-relationship between factors that will contribute formaldehyde to indoor air (sources) and factors that will remove formaldehyde from indoor air (sinks). Other factors that can influence formaldehyde levels include indoor air chemistry, environmental parameters such as temperature and humidity, emission sources (e.g. type and loading of furniture, flooring, carpets, etc).

- In tests, there is considerable variability in emissions between sources and for different examples of a single type of source.

- Emissions from continuous sources decline as the article ages therefore emissions from new articles represent a worst case.
- Emissions from intermittent sources are very dependent on the frequency and duration of operation of each intermittent source.
- Emission values obtained from chamber studies will not necessarily reflect emissions under real-world conditions, which will be affected by variations in temperature and humidity compared with the standard test conditions.

3.3 Quantitative data on formaldehyde levels indoors

Monitoring data are available for homes in England to help understand the levels of formaldehyde that are present in indoor air in domestic settings in GB. Data are also available for Northern Ireland, Germany and certain other EU countries, Canada and the US; they have been summarised along with data on levels that arise outdoors to act as comparators to the GB data. A key challenge for the interpretation of this data is that, for practical reasons, recent monitoring in GB has been performed for a very small number of homes, creating uncertainty around how representative these measurements are for the current GB housing stock; the most recent larger scale study was published in 2004. It is important to recognise this uncertainty when analysing the available monitoring data.

Salthammer (2010) cautioned that care must be taken when comparing studies. Two strategies are commonly applied: (a) the collection of samples from many randomly selected homes to estimate the exposure of the general population and (b) the monitoring of a limited number of homes to measure the time vs concentration behaviour and to see the influence of aging. Results are generally log-normal, so the geometric mean, or mean and median and the 90th or 95th percentiles should be reported with the supporting information including the monitoring strategy and supporting information on the method and duration of measurement.

3.3.1 Exposure to formaldehyde outdoors

Emission sources of formaldehyde differ between outdoors and indoors, as has been detailed by Salthammer (2019b). In ambient air, formaldehyde can be produced by the biosphere, atmospheric reactions of volatile organic compounds, and the combustion of biomass (e.g. wood) and fuels from vehicles (Rodrigues *et al.*, 2012; Dugheri *et al.*, 2021). In urban environments, fuel combustion from traffic is a significant source of aldehydes in the air because of direct emissions and also the release of hydrocarbons that were transformed to aldehydes by photochemical oxidation.

Bruinen de Bruin *et al*. (2008) measured average outdoor formaldehyde concentrations between 0.3 ppb (~ 0.4 µg/m^3) and 4.0 ppb (~ 5 µg/m^3) in 11 EU cities. The average formaldehyde concentrations in European ambient air are lower in comparison to non-European areas (Salthammer, 2013). Salthammer (2019a, b) assumed for his

modelling work that the distribution of formaldehyde concentrations in outdoor air had a geometric mean of 4.31 µg/m³, and a range from 1 ppb (~ 1.2 µg/m^3) to 20 ppb (~27 µg/m^3) with the higher concentrations resulting mainly from photochemical reactions of atmospheric hydrocarbons.

Formaldehyde does not persist in outdoor air. Formaldehyde is mainly removed from the air by direct photolysis (with a half-life of 1 – 4 hours) and oxidation by photochemically produced hydroxyl and nitrate radicals (with longer half-lives measured in days) (US EPA, 2024b). Formaldehyde may also be removed by deposition after transfer into rain, fog or clouds where it is likely to convert to methylene glycol or formic acid. While formaldehyde is not persistent in the environment, environmental exposure may still occur where release is continuous.

The current report focusses on indoor air; therefore, the above data are given for information.

3.3.2 Exposure to formaldehyde indoors in domestic settings

3.3.2.1 Methods to determine formaldehyde in indoor air in domestic settings

Formaldehyde is found in two forms in the indoor environment. It is found as gas, because of off gassing from materials, and it may be adsorbed onto the surface of dust particulates. Air monitoring for vapours can be collected by diffusion or active pumped sampling through a sorbent tube. Whilst active sampling is more accurate than diffusion sampling, diffusion sampling is preferred for indoor air quality surveys as the sampler can be installed and left alone, whilst active sampling requires the pump to be monitored constantly. Active sampling is typically carried out for several hours. Diffusion sampling is typically carried out over days or weeks.

Neither diffusion sampling nor use of a sorbent tube will detect formaldehyde that is bound to particulates. Particles can be collected on a treated filter or through a liquid impinger. In these cases, substances in the liquid or treated filter react with the formaldehyde to form a stable complex, which can then be analysed in a laboratory. A key disadvantage of filter sampling is that as air passes through the filter, the liquid which needs to react with formaldehyde dries out as air passes through the filter. At this point, formaldehyde cannot react with the liquid and therefore this sampling method can underestimate levels of particulate-bound formaldehyde, particularly with longer sampling durations. Liquid impingers collect all particulate-bound formaldehyde and do not dry out in the same way that a treated filter can do.

Concentrations of formaldehyde in air can be expressed in various terms, such as parts per million (ppm), parts per billion (ppb), mg/m^3 or µg/m^3. Conversion from ppm, ppb to mg/m^3, µg/m^3 is temperature and pressure dependent. In UK and EU 20°C is used, whilst 23°C is used in test chamber tests and 25°C is used in the US. In all cases a pressure of 101.325 Kpa is used. Whilst the variance in pressure causes a slight difference in conversion, ±0.01, the pressure is usually not known. Furthermore, the humidity content of air (typically 30 – 70 % relative humidity (RH) in indoor air) will

affect formaldehyde concentrations (higher relative humidity is associated with higher formaldehyde concentrations). As such, undue precision should not be given to measurements or conversions.

If diffusion sampling or sorbent tubes are used, this will miss a possible additional contribution from particulate-adsorbed formaldehyde, but this may be an important factor for respiratory symptoms. Particulate-adsorbed formaldehyde will have a different deposition pattern in the respiratory tract compared with vapour-phase formaldehyde. Formaldehyde gas at low concentrations is highly water soluble so reacts with the mucus membranes of the eyes, nose and throat, whereas small particles (0.5 – 5 µm) are deposited in the lower airways and gas exchange region. Therefore, formaldehyde sampling methodology may impact the interpretation of epidemiological data.

3.3.2.2 Measured indoor air concentrations in GB homes

Crump and Gardiner (1989) demonstrated the potential of a wide range of building, furnishing and consumer products to release formaldehyde to indoor air, including particleboard, textiles, urea formaldehyde foam insulation (UFFI) and some furniture polishes. As well as occurring in the air, formaldehyde is found in household dust (Brown *et al*., 1988).

Berry *et al*. (1996) carried out the Indoor Environment Study over a 12-month period, including formaldehyde measurements over a 3-day period each month. This was carried out in collaboration with the ALSPAC study (174 homes located in Avon) and 6 more homes located in Hertfordshire. The aim was to identify the range and concentration of pollutants in UK homes and identify the factors such as household characteristics and occupant activities, which may have an impact on the levels of the pollutants. In the Avon homes (n = 174), formaldehyde levels measured in the main and second bedroom were the same (25 µg/m^3), levels were slightly higher than in living rooms and kitchens (23 µg/m^3). The lowest levels were measured in bathrooms (19 µg/m^3), whereas the outside concentration was only 2 µg/m^3. In the Hertfordshire homes (n = 6), there is the same trend for higher concentrations in the main and second bedrooms (18 µg/m^3) than in the living rooms (15 µg/m^3) and bathrooms (13 µg/m^3), but similar to the kitchen concentrations (18 µg/m^3).

Berry *et al*. (1996) carried out a statistical analysis, aiming to define the following relationships:

1. Between the annual mean formaldehyde concentration in the living room and main bedroom with the following factors: a) type of home location, b) type of dwellings, c) dwelling with or without garage; d) type of cooking fuel (electricity or natural gas; e) type of main heating fuel; f) age of dwelling; g) dwelling with or without an extractor fan; h) dwelling with smoking activity; i) dwellings where occupants consider if fresh air supply was adequate; j) type of main heating system;

2. Relationship between first formaldehyde reading and recent changes to

dwellings and occupant behaviour: a) painting activity in the past year; b) decorating activity in the past year, c) new curtains in the past year; d) new flooring in the past year; e) new chipboard furniture in the past year; f) period of opening windows during the day; g) period of opening windows during the night; h) presence of mould in the past year, new furniture of any type in the past year; time for heating in winter compared with winter formaldehyde concentrations

3. Relationship between formaldehyde reading in the 2nd month of monitoring with recent changes and activities recorded in the updated questionnaire: a) building work in the previous month; b) painting activity; c) decorating activity; d) new curtains; e) new furniture; f) new floor covering.

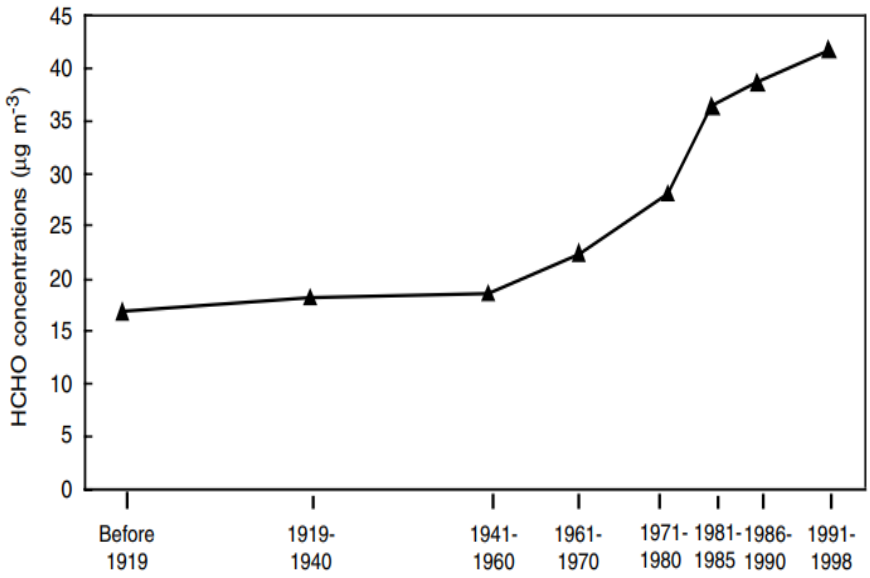
The most significant relationship was between the formaldehyde concentration in the main bedroom and living room with the age of the dwelling, which is an increasing trend: 52 μg/m^3 in bedrooms and 41 μg/m^3 in living rooms for newer homes built since 1982, vs 16 μg/m^3 in both bedrooms and living rooms for homes built pre-1919. The second factor was the area: in rural areas the formaldehyde levels were higher in bedrooms and living rooms (36 μg/m^3 and 32 μg/m^3, respectively) than in suburban areas (25 μg/m^3 and 23 μg/m^3, respectively) and higher than in towns (22 μg/m^3 and 20 μg/m^3, respectively). The third factor was the dwelling type with higher concentrations in detached dwellings in bedrooms and living rooms (36 μg/m^3 and 30 μg/m^3, respectively), compared to flats (20 μg/m^3 and 21 μg/m^3, respectively), terraces (21 μg/m^3 and 19 μg/m^3, respectively), and semi-detached (26 μg/m^3 and 23 μg/m^3, respectively). The fourth factor was that bedrooms and living rooms in homes with attached garages had higher formaldehyde concentrations (31 μg/m^3 and 26 μg/m^3, respectively) than in homes without (19 μg/m^3 in both rooms). Berry *et al*. (1996) reported that a detailed examination of the data for the above factors showed that these associations are the result of the distribution of dwellings of different age within these groups, and it is only the age of dwelling that is the key factor. The outdoor data also indicate that the location is not a significant factor compared to indoor sources that contribute to formaldehyde concentrations. Further investigation of the influence of new chipboard furniture in the past year shows that the main bedroom with new furniture had higher concentrations (27 μg/m^3) compared to rooms with no new furniture (21 μg/m^3). Also, new furniture in the previous month in the main bedroom and living room resulting in higher formaldehyde concentrations (29 μg/m^3 and 25 μg/m^3, respectively) compared to rooms with no furniture (21 μg/m^3 and 19 μg/m^3, respectively), whereas new floor covering during previous month resulted again in higher concentrations in bedrooms and living rooms (29 μg/m^3 and 24 μg/m^3, respectively) compared to rooms with no floor covering (21 μg/m^3 and 19 μg/m^3, respectively). The above results are not statistically significant because of the small number of homes with new furniture or new floor covering in the whole sample, but they provide an indication of the main formaldehyde sources in homes. Furthermore, all the other statistical tests examining the various relationships of formaldehyde concentrations with the factors reported above were not statistically significant.

Berry *et al*. (1996) concluded that particleboard (known also as chipboard) e.g. used for furniture and urea formaldehyde foam insulation in cavity walls are likely to be major sources of formaldehyde emissions into indoor air.

Raw *et al*. (2004) measured formaldehyde concentrations in 833 homes located in England. They found an increasing trend over time with statistically significant higher concentration of formaldehyde in homes built after 1982 (mean: bedroom 52 µg/m^3, living-room 41 µg/m^3) compared to homes built before 1982 (mean: bedroom 41 µg/m^3, living-room <29 µg/m^3). Raw *et al*. (2004) also showed that formaldehyde levels have more than doubled since the 1960s. This is probably associated with the replacement of natural timber with engineered wood board products and the increased air tightness of builds after 1973. The overall geometric mean in bedrooms for all homes was 22 µg/m^3 (see Table 8 and Figure 2).

Homes with particleboard floors and furniture had significantly higher mean formaldehyde levels than those without. Raw *et al*. (2004) reported that indoor concentrations were 50% higher in properties having particleboard floors (32 µg/m^3) than in properties without particleboard floors (20.3 µg/m^3). Raw *et al*. (2004) also showed that formaldehyde in homes where new particleboard furniture had been brought into the bedroom during the four weeks before the sampling period had higher levels of formaldehyde in the bedroom (36.7 µg/m^3) than other homes (22.2 µg/m^3).

Figure 2: Geometric mean concentrations of Formaldehyde over time. From Raw *et al*. (2004).



The year when a building is constructed influences the airtightness of the building fabric and its ventilation characteristics, according to the corresponding building regulations at that time. Raw *et al*. (2004) found that homes built between 1991 – 1998 had higher indoor concentrations of formaldehyde (GM ~ 40 µg/m^3) than older homes built before

1919 (GM ~16-17 µg/m^3), which were much leakier, with cracks and gaps in the building envelope, and consequently not energy efficient.

Table 8: Mean formaldehyde levels (µg/m^3) by UK building date (adapted from Coward *et al*., 2001)

When built	Mean (µg/m^3)	N
Before 1919	16.9	146
1919-1940	18.2	152
1941-1960	18.7	163
1961-1970	22.3	135
1971-1980	28.0	110
1981-1990	37.3	79
1991-1998	45.0	48

Table 8 shows that mean formaldehyde concentrations in UK buildings were fairly constant until 1960 with the use of traditional construction techniques and natural wood products. There was an increase in the 1960s with a greater use of engineered wood boards. Following the oil crisis in 1973, measures were put in place to increase the energy efficiency of buildings to reduce the need for oil. This resulted in buildings becoming “tighter” with greater releases of formaldehyde indoors. During the 1970s and 1980s there was also greater use of “flat pack” furniture. This is shown by the more than doubling of mean formaldehyde levels in the 1980s and 1990s.

Clark *et al*. (2023) systematically reviewed the literature to derive a distribution of annual average indoor formaldehyde concentrations representative of the English housing stock. They identified 11 studies published between 1996 and 2022, providing either raw data or summaries (e.g., means, medians, ranges) on indoor formaldehyde concentrations in English dwellings (1,700 homes, Table 9). Their search was also expanded to include studies conducted in Northern Ireland, Scotland, and Wales, although no studies were identified from Scotland and Wales. Monitoring durations in each home were between 24 hours and 7 days.

Table 9: Characteristics of formaldehyde monitoring studies in English homes from 1996 to 2022 (from Clark *et al.*, 2023).

Study	Location	Number of residences monitored	Time period of monitoring (duration in each home)	Type of dwellings	Summary of measured formaldehyde concentrations in µg/m^3
Brown *et al.*, 1996 (in Berry *et al.*, 1996)	Avon	174	1991-1993 Monthly measurements throughout year (3-days)	Existing homes, constructed prior to 1993	*Bedroom* – AM: 25, SD: 17, Min: 6, Max: 130 *Living room* – AM: 27, SD: 13, Min: 4, Max: 74
Möhle *et al.*, 2003	Hertfordshire, London, Berkshire, Bedfordshire, Oxfordshire	10	2001 (3-days)	Existing homes built before 2000	*Bedroom* – AM: 37.5, Min: 17, Max: 70 *Living room* – AM: 28.1, Min: 14, Max: 54
Venn *et al.*, 2003*	Nottingham	416	1998-1999 (3-days)	Existing homes built before 1998	*Cases (Bedroom)* – Proportion: 0-16 (25.8%); 16-22 (24.2%); 22-32 (26.8%); >32 (23.2%) *Controls (Bedroom)* – Proportion: 0-16 (27.1%); 16-22 (22.4%); 22-32 (26.6%); >32 (23.8%)
Raw *et al.*, 2004	England (dwellings across the country)	833	1997-1999 (3-days)	Existing homes (built before 1919 – 1998)	*Bedroom* – Median: 24, GM: 22, Min: 1, Max: 171
Crump *et al.*, 2005	South of England	37	2002 heating and non-heating seasons (3-days)	New homes built since 1995 and prior to 2002	*Heating season (Living room)* – GM: 24, Min: 10, Max: 75 *Non-heating season (Living room)* – GM: 26, Min: 1, Max: 61
Gee *et al.*, 2005	Manchester area	200	--- (5-days)	Existing homes	*Bedroom* – Median: 49* *Living room* – Median: 37* ** Reported in paper as ppm (Bedroom: 0.04 ppm; Living room: 0.03 ppm). Converted to µg/m^3*

Study	Location	Number of residences monitored	Time period of monitoring (duration in each home)	Type of dwellings	Summary of measured formaldehyde concentrations in µg/m^3
McGill *et al*., 2015	England	7	2012-2013 non-heating season (24-hr)	New zero carbon social rented properties (Passivhaus), built since 2012	*Living room* – Median: 37.5*, Min: 12.5, Max: 187.4 **Reported in the paper as ppm. Converted to µg/m^3*
Wang *et al*., 2017	York	3	2015 (3-days)	Existing homes	*Living room* – AM: 48.7
MHCLG, 2019	Leeds, Manchester, Bolton, London, Didcot, Bristol	10	2015-2016 (7-days)	New homes, constructed prior to the end of 2014	*Bedroom* – AM: 40, SD: 17, Min: 19, Max: 72 *Living room* – AM: 34, SD: 16, Min: 15, Max: 65 *Outside* – AM: 2, Median: 2
Burman and Stamp, 2019	East London	5	2018 heating and non-heating seasons (7-days)	New, low energy apartment blocks completed in 2014 and 2015	*Bedroom, living room, kitchen* - AM: 16.8, SD: 16.3, Min: 1.15, Max: 31.9 (values combined for the heating and non-heating seasons).
Stamp *et al*., 2022	London	5	2018 Heating and non-heating seasons (7-days)	New low energy apartments (built 2015)	*Heating season (Bedroom, living room, kitchen)* – AM: 22.8 *Non-heating season (Bedroom, living room, kitchen)* – AM: 10.8 *Outdoor* - <3

AM: Arithmetic mean, GM: Geometric mean, Min: Minimum, Max: Maximum, SD: Standard deviation, ---: Unknown

*Venn *et al*., 2003 was an epidemiological study of 193 children with persistent wheezing illness (cases) and 223 children without (controls).

Table 10: Reproduction of table S3 from Clark *et al.* (2023) providing summary statistics of the pooled dataset of indoor formaldehyde concentrations in English residences. These summaries show the full pooled data distribution prior to the data on the extremes being trimmed off for the burden of disease analysis (see Methods in Clark *et al.*)

	N studies	GM (GSD)	Median	Min	Max	References
England Full pool of studies published between 2003 and 2022	10	22.5 (2.2)	24.6	0.1	187.4	(Burman and Stamp 2019; Crump *et al.* 2005, Gee *et al*. 2005; McGill *et al*. 2015a; MHCLG 2019; Möhle *et al*. 2003; Raw *et al*. 2004; Stamp *et al*. 2022; Venn *et al*. 2003; Wang *et al*. 2017)
New homes built since 2010 Mixture of energy efficient, Passivhaus, social housing and naturally and mechanically ventilated homes	4	23.8 (2.1)	24.3	6.2	187.4	(Burman and Stamp 2019; McGill *et al*. 2015a; MHCLG 2019; Stamp *et al*. 2022)
Housing stock built prior to 1988	2	22.3	22.5	1.0	171.0	(Berry *et al*. 1996; Raw *et al.* 2004)

GM: Geometric mean; GSD: Geometric standard deviation

Tables 9 and 10 show that over the last 25 years in many GB homes under normal living conditions, formaldehyde concentrations were typically between 11 – 49 µg/m^3 (arithmetic/geometric mean or median, see definitions for details) and the maxima range from 32 – 187 µg/m^3. Clark *et al*. (2023) further estimated a weighted distribution by pooling data from these studies, giving a geometric mean of 22.8 µg/m^3 (geometric standard deviation: 2.0 µg/m^3) and 5th and 95th percentiles at 6.5 µg/m^3 and 58.7 µg/m^3, assuming a log-normal distribution (see paper for detailed methods). Clark *et al*. (2023) also estimated a historical distribution using only studies where housing stock was built pre-1998 (Brown *et al*., 1996, Raw *et al*., 2004), and found that the pooled geometric mean was 21.7 µg/m^3, however, there was a slightly higher percentage of households at the upper end of the distribution (>60 µg/m^3) pre-1998 (5%) compared with the 'current' estimated distribution (3%) (note that the 'current' distribution is made up of data from studies where indoor monitoring was conducted between 1998 – 2022). We may infer from these results that there has been minimal change in indoor formaldehyde concentrations on average across GB homes, over the last 25 years.

Concerns that steps taken to improve energy efficiency of new builds may be impacting indoor air quality, led the National House Building Council (NHBC) to undertake an indoor air quality monitoring programme in collaboration with Scottish and Southern Energy in 10 new build zero-carbon rental homes (NHBC, 2013).

Concentrations of formaldehyde in air were determined at 4 time points by pumped cartridge sampling and high performance liquid chromatography analysis according to the international standard method BS ISO 16000-3. The first measurements were taken shortly before construction was completed. Occupants started to move into these properties about two weeks after the September 2010 samples were taken. The characteristics and results for each home expressed as the mean of 3 sampling locations (kitchen, living room, master bedroom) are presented in Table 11. Measurements were also made of total volatile organic compounds (TVOC; results not presented here but are reported in NHBC (2013)).

Table 11: Summary of mean formaldehyde results (adapted from NHBC, 2013)

Home no.	Mean formaldehyde concentrations			
	September 2010[(a)] (µg/m^3)	**February 2011** (µg/m^3)	**July 2011** (µg/m^3)	**March 2012** (µg/m^3)
1 (timber frame, 3-bed detached)	19	38	52	30
2 (timber frame, 2-bed end of terrace)	52	23	27	22
3 (timber frame, 2-bed mid terrace)	40	28	34	26
4 (timber frame, 2-bed end of terrace)	37	51	55	25
5 (masonry, 1-bed first floor flat)	91	31	30	22

Home no.	Mean formaldehyde concentrations			
	September 2010[(a)] ($\mu g/m^3$)	**February 2011** ($\mu g/m^3$)	**July 2011** ($\mu g/m^3$)	**March 2012** ($\mu g/m^3$)
6 (masonry, 1-bed first floor flat)	24	(b)	48	23
7 (masonry, 3-bed end of terrace)	48	36	76	32
8 (masonry, 3-bed mid terrace)	37	33	27	23
9 (masonry, 3-bed end of terrace, intermittently occupied show home)	55	33	37	17
10 (masonry, 3 bed detached)	71	25	61	21

(a) Sampling coincided with painting, sealing and other post-second fix finishing
(b) No measurement

Elevated levels of VOCs and formaldehyde were present immediately after construction and persisted for over six months post construction. Different VOCs were found as the occupancy phase progressed, but overall TVOC levels followed a downward trend with time. As the occupancy phase proceeded, the main VOCs found in the air were considered by the study authors to be those associated with occupant activities and use of consumer products. The March 2012 results for formaldehyde are consistent with the geometric mean concentration reported by Clark *et al*. (2023). A key focus for this work was to understand the effectiveness of the mechanical ventilation and heat recovery (MHVR) systems that were installed in these new builds. During the study period, various interventions were made to ensure optimal function of the MHVR systems in these homes. It cannot be excluded that had these interventions not taken place, or where the MHVR system is incorrectly designed, installed, operated and maintained, higher levels of contaminants including formaldehyde may have arisen.

Within this work a small-scale study was performed to assess air quality during cooking (boiling potatoes, frying breakfast or frying steak using one ring on either an induction electric hob or gas hob). Formaldehyde levels were measured by pumped cartridge sampling and high performance liquid chromatography. Sampling points were situated near to the cooker (at close to breathing height when standing to cook) and in the lounge area of the open-plan ground floor (at sitting height). Contaminant levels measured at both locations were similar therefore only the results for the kitchen were reported. These are presented in Table 12.

Table 12: Formaldehyde levels measuring during cooking activities (home 9)

Cooking fuel	Cooking type	Ventilation mode	30-minute mean formaldehyde concentration ($\mu g/m^3$)
Electricity	Boiling potatoes	Normal	60
Electricity	Frying breakfast	Normal	50
Electricity	Frying steak	Normal	40
Gas	Boiling potatoes	Normal	39
Gas	Frying breakfast	Normal	43
Gas	Frying steak	Normal	40
Gas	Boiling potatoes	Off	45
Gas	Frying breakfast	Off	51
Gas	Frying steak	Off	35

The study authors suggested that longer cooking times or use of more than one ring could result in higher formaldehyde emissions, and if the kitchen was inadequately ventilated this could also result in higher air concentrations.

A report by Ministry of Housing, Communities and Local Government (MHCLG, 2019) reported 7-day average concentrations of formaldehyde in 10 new homes constructed prior to the end of 2014 in accordance with the energy efficiency standards established by Part L 2010 or Part L 2013 of the Building Regulations. The study aimed to evaluate if the ventilation standards established in Part F 2010 of the Building Regulations provide satisfactory indoor air quality in new homes. Measurements were taken when homes had been occupied for around one year. The study found that mean concentrations measured between December 2015 and February 2016 were 34 $\mu g/m^3$ (living rooms) and 40 $\mu g/m^3$ (bedrooms), with maximum measured levels at 65 $\mu g/m^3$ (living room) and 72 $\mu g/m^3$ (bedroom). The whole house ventilation rates in these homes had a mean of 0.33 air changes per hour (ACH; range: 0.19 – 0.63 ACH), with the higher ventilation rates observed in houses with open windows for approximately one hour each day to 'air' the house or during periods when smoking indoors. Stamp *et al*. (2022) also found that indoor formaldehyde concentrations were higher in the heating seasons because of reduced ventilation in the London apartments covered by this study.

Research from Northern Ireland found that mean levels monitored in real-time over 24hr periods in kitchens and open plan living rooms in three newly built Passivhaus homes (1 year post construction), which represent energy efficient homes with super-insulation materials, airtight building envelope, and heat recovery ventilation systems (MVHR) reached as high as 1,400 µg/m^3 (1.13 ppm) in one home in the winter (heating) months. In another home, mean concentrations in the winter were measured at ~350 µg/m^3 (0.29 ppm) and between ~0-270 µg/m^3 (0-0.22 ppm) in the three homes in the summer months (McGill *et al*., 2017).

Airborne concentrations of formaldehyde have also been studied in other non-industrial microenvironments in GB. In 19 nursery schools in London, three-month mean formaldehyde concentrations ranged from 4 µg/m^3 to 25 µg/m^3 (Mayor of London, 2020).

The above average formaldehyde concentrations in homes reported in Table 9 consistently exceed the long-term (annual average) PHE health-based guideline value of 10 µg/m^3, which has been included in the recently revised Building Regulations (DLUHC, 2021, Approved Document F) and also cited in the National Institute for Health and Care Excellence (NICE) Guidelines for indoor air quality (NICE, 2020).

3.3.2.3 Indoor air concentrations in European homes

3.3.2.3.1.Results from German studies

3.3.2.3.1.1 Umwelt-Survey Band IIIc (Krause *et al*., 1991)

The German Institute for Water, Soil and Air Hygiene carried out a research project "Environment and Health - Measurements and Analysis of Environmental Exposure Factors in the Federal Republic of Germany 1985/86" (1st Environment Survey) in 329 homes.

The estimated formaldehyde median concentration in indoor air was 55.0 µg/m^3 (sample size: n = 329). The GM is estimated at 49.40 µg/m^3, the standard deviation at 1.87. The estimated arithmetic mean is 58.55 µg/m^3, the standard deviation is 32.73 µg/m^3. The maximum value was measured 309.0 µg/m^3. 17.0% of the values were below the detection limit of 30 µg/m^3.

The houses built "until 1919" had the lowest formaldehyde concentrations (38.59 µg/m^3) compared to houses built recently (1974 - 1986), with the highest concentration of 55.17 µg/m^3.

Among the different types of heating, the highest estimates are for households with underfloor heating (GM: 66.75 µg/m^3). Households with central heating had a GM of 47.70 µg/m^3, whereas the lowest was for households with oil/wood/gas stoves (45.79 µg/m^3).

If the furniture was purchased "after 1980" formaldehyde concentrations were higher (GM: 55.15 µg/m^3) than households with furniture before 1980 (GM: 47.80 µg/m^3).

Households without “furniture made of pressed chipboard” resulted in the lowest concentrations (35.34 µg/m^3), compared to households with some furniture made of pressed chipboard, which had the highest concentrations (56.88 µg/m^3).

Finally, lower concentrations were measured in households which did not use sanitary cleaners/disinfectants (43.29 µg/m^3), compared to those which use them frequently (52.93 ug/m^3).

3.3.2.3.1.2 Kinder-Umwelt-Survey (KUS) 2003/06 (Schulz *et al*., 2010)

The German Environmental Survey (GerES) aimed to determine the exposure of Germany’s general population to pollutants. The survey was carried out by the Federal Environment Agency from the mid-1980s. It used a randomly selected sub-sample from KiGGS (the German Health Interview and Examination Survey for Children and Adolescents) composed of 1,790 children 3 to 14 years of age from 150 study locations and produced extensive data on exposure to chemical pollutants, biological contaminants (such as moulds and house dust mites) and noise.

Sampling was carried out in the room in which the child (3 to 14 years of age) normally spent most time over a 24-hour day. In about 95% of the cases, this was the child’s own room, in 4% of the cases, the parents’ bedroom and in 1% of the cases, another room in the dwelling. Passive sampling was conducted over one week. The results showed that the geometric mean of formaldehyde concentrations in households with children aged 3 to 14 in Germany (N=586) was 23.3 µg/m^3, with an arithmetic mean of 25.7 µg/m^3 and max of 68.9 µg/m^3.

The report presents values of VOC levels for different conditions, covering, among others, potential factors influencing specific substances such as renovation work, presence of chipboard furniture and use of household chemicals/products.

High formaldehyde concentrations were measured when renovation in the sampling room had been performed in the last 12 months compared to houses with no renovation (GM: 26.3 µg/m^3 and 23.3 µg/m^3, respectively), as well as when linoleum or cork floor existed in the sampling room compared to rooms without such flooring (GM: 25.1 µg/m^3 and 23.1 µg/m^3, respectively). The highest concentrations were measured in rooms with furniture made of chipboard. The average formaldehyde concentration in rooms with a lot of furniture made of chipboard were significantly higher than in rooms with none or few such furniture (27.1 µg/m^3 and 19.5 µg/m^3, respectively).

The sampling room equipped with furniture made of solid wood and/or wood wall, ceiling and/or floor resulted in significantly lower average concentrations of formaldehyde (and a number of other aldehydes) in the indoor air compared to the rooms, which did not have such furnishings with (solid) wood (GM: 21.4 µg/m^3 and 24.4 µg/m^3, respectively).

Because formaldehyde emissions had been increasing in Germany since around the mid-1980s from articles made of chipboard, the Chemicals Ban Ordinance, determined

that coated and uncoated wood materials (chipboard, carpentry, Veneer and fibreboard) may not be placed on the market, if it resulted in concentration of formaldehyde in the air of a test room higher than 0.1 mL/m^3 (ppm) under test conditions ("emission class E1").

This report concluded that although legal regulations (Hazardous Substances Ordinance/ Prohibition of Chemicals Ordinance) have led to a significant reduction in formaldehyde emissions from wood-based materials in recent decades, wood-based materials produced with urea/formaldehyde glues are still a significant source of emissions of formaldehyde into indoor air. This is due to the fact that their formaldehyde emissions barely decrease over time and they are still frequently used in many places in both house building and interior construction work (UBA, 2020).

3.3.2.3.1.3 German Environmental Survey 2014–2017 (GerES V) (Birmili *et al*., 2021)

Indoor air concentrations of formaldehyde and other aldehydes (C2-C11) were measured in residences of 639 participants in the German Environmental Survey for Children and Adolescents between 2014–2017 (GerES V). Sampling was conducted using passive samplers over periods of approximately seven days for each participant. A comparison of GerES IV (2003–2006; 579 samples) and GerES V (2014–2017; 533 samples) shows that both studies used the same experimental techniques, but GerES IV was based on the sub-set of 3- to 14-year-old children.

The results show that the most abundant compound was formaldehyde and that the geometric mean of formaldehyde concentrations was decreased by 3% in 2014–2017 (22.2 µg/m^3), compared to 2003-2006 study (23.3 µg/m^3). This was a non-statistical difference.

The study considered that the lack of large change in formaldehyde concentrations between GerES V and GerES IV survey is associated with formaldehyde emissions from furniture with wood-based materials. Although the E1 emission class implies low, albeit non-zero formaldehyde emissions, even products advertised as "free of formaldehyde" may still emit formaldehyde (Birmili *et al*., 2021). Measurements in a test cabin built from "formaldehyde-free" materials (solid beech frame; gypsum board floor, walls and ceiling; inner surfaces treated with primer and covered with low-emitting wallpaper) yielded, at an air exchange rate inside the cabin of 0.3 h^{-1}, a room concentration of formaldehyde of approximately 22 µg/m^3 (Salthammer and Mentese, 2008).

Excluding wood-based materials as a source and taking other sources and sinks into account, Birmili *et al*. (2021) estimated that indoor formaldehyde concentrations are expected to be in the order of 10–15 µg/m^3. The inclusion of wood-based materials would roughly double these values. Their results showed that rooms containing pieces of furniture with particleboard tended to have higher formaldehyde concentrations than rooms without such furniture (22.6 µg/m^3 vs 16.3 µg/m^3). However, this difference is not statistically significant, which might be due to the relatively small subgroup of 43

participants (out of 639) with no self-reported furniture made from particleboard.

It is difficult to connect these results with previous measurements in test chambers of formaldehyde emissions from furniture. At present, there are no reliable data on the release of formaldehyde and other very volatile and volatile organic compounds (VVOCs/ VOCs) from furniture over a period of several years. It is known, at least, that the formaldehyde emission rate declines nonlinearly within the first weeks and months. Birmili *et al*. (2021) reported that over a period of 10 years or more, which is the lifetime of wood-based materials in housing, the average formaldehyde emission rate from furniture corresponds to 40% of their respective emissions when new (estimated by Salthammer, 2019a, b).

3.3.2.3.2 Other European studies

The above geometric mean concentration of 22.8 µg/m^3 which is currently measured in the GB homes, is in line with the mean background level of 22 µg/m^3 that was reported 15-20 years ago for dwellings across Europe in the EU AIRMEX study (Geiss *et al*., 2011) and by the INDEX project (Kotzias *et al*., 2005). Dimitroulopoulou *et al*. (2015a) grouped together the background concentration levels for dwellings across Europe for four European geographical regions which had been examined in the EPHECT project and reported in the AIRMEX study (Geiss *et al*., 2011). The formaldehyde concentrations were in the range of 11-45 µg/m^3 in dwellings of North Europe, 4-57 µg/m^3 in West Europe, 11-41 µg/m^3 in East Europe and 7-52 µg/m^3 in South Europe, resulting in a mean background concentration of 22 µg/m^3 across Europe.

Sarigiannis *et al*. (2011) also carried out a review of Volatile Organic Compound (VOC) concentrations in European indoor environments (period: 1990-2008) and their impact on health, focussing on nine VOCs (benzene, toluene, xylenes, styrene, formaldehyde acetaldehyde, limonene, naphthalene, a-pinene), identified by the European Commission's INDEX strategy report as the priority pollutants requiring regulation in relation to the indoor environment (Kotzias *et al*., 2005) and reported similar levels.

Furthermore, based on a literature review and Monte-Carlo simulations, Salthammer (2019b) estimated that formaldehyde concentrations in European homes under normal living conditions were likely to be within the range of 0-120 µg/m^3, with a geometric mean of 23.1 µg/m^3, and interquartile range (IQR) of 15.7 µg/m^3 and 34.0 µg/m^3. In this analysis, the UK data from 876 homes published by Raw *et al*. (2004) were included.

3.3.2.4 Indoor air concentrations in US and Canadian homes

The US Department of Healthy Housing (DHH) and US EPA carried out a second joint survey of American housing (AHHS II) in 2018/2019. A formaldehyde sample was collected in 689 of the 703 housing units in which the survey was conducted (98%). The main reason a sample was not collected in 14 units was failure of the sampling pump. The limit of detection based on the sampling time varied from a minimum of 0.06 ppb (0.07 µg/m^3) to a maximum of 0.33 ppb (0.4 µg/m^3), with an average of 0.15 ppb (0.18 µg/m^3). Only one sample of the 689 was below the detection limit. All but one of

the 689 homes tested were below 0.1 ppm (0.123 µg/m^3). The maximum formaldehyde level was 100.9 ppb or 0.101 ppm (0.123 µg/m^3).

The AHHS II (US EPA, 2024a) reported monitoring data suggests that concentrations of formaldehyde may range from 0.27 to 124.2 µg/m^3 (19.77 µg/m^3: 50 percentile for all homes, with 90 percent of homes having concentrations below 41.8 µg/m^3 (QuanTech, 2021). Those data include formaldehyde-containing materials and sources such as tobacco smoke or the use of fireplaces, gas-burning appliances, candles, and air purifiers (QuanTech, 2021), which do not contain formaldehyde but rather lead to the formation of formaldehyde during use.

The US EPA states that the AHHS II air sampling was not performed throughout the entire home, and across multiple seasons. It should also be reiterated that formaldehyde emission rates decrease over time. Generally, it is expected that after the installation of formaldehyde-bearing materials in a home, there is an initial rise of formaldehyde concentration, followed by a levelling-off period that may be as brief as 30 days or less, which is followed by a longer gradual decline of formaldehyde concentration over time (US EPA, 2024a). Table 13 summarises the formaldehyde monitoring data from AHHS II.

Table 13: Range and weighted quantiles of AHHS II residential indoor air formaldehyde concentrations (µg/m^3)

Minimum	10th Percentile	Median	90th Percentile	Maximum
0.27	7.54	19.8	41.8	124.2

Results from studies carried out in Canada since the early 1990s consistently indicate that formaldehyde concentrations in Canadian homes range between 2.5 and 88 µg/m^3 with an average between 30 and 40 µg/m^3 (Health Canada, 2005).

3.3.3 Summary of indoor air surveys of formaldehyde

Ambient outdoor concentrations of formaldehyde in European air range from around 1.2 – 27 µg/m^3 with a geometric mean of 4.3 µg/m^3. The higher concentrations result mainly from photochemical reactions of atmospheric hydrocarbons.

Based on a systematic literature review, Clark *et al*. (2023) calculated that the average formaldehyde concentrations in homes in England over the last 25 years ranged between 11 – 49 µg/m^3, maxima ranged from 32 – 187 µg/m^3. Pooling data to obtain a weighted distribution resulted in a geometric mean of 22.8 µg/m^3 (geometric standard deviation: 2.0 µg/m^3) and 5th and 95th % percentiles at 6.5 µg/m^3 and 58.7 µg/m^3, assuming a log-normal distribution. These levels are similar to GMs from pooled studies where indoor monitoring was conducted pre-1998, suggesting little change over the past 25 years. Although Clark did not have data for homes in Scotland and Wales, there is no reason to consider that levels in homes in these regions will be significantly

different to homes in England.

The currently measured formaldehyde concentrations in English homes are in line with an average of 22 µg/m^3 calculated from data gathered 15 – 20 years ago in the EU AIRMEX and INDEX studies and the GM concentration of 22.2 µg/m^3 reported by Birmili *et al*. (2021) from samples collected in Germany between 2014 – 17. Results from studies carried out in Canada since the early 1990s consistently indicate that formaldehyde concentrations in Canadian homes range between 2.5 and 88 µg/m^3 with an average between 30 and 40 µg/m^3 (Health Canada, 2005). Data collected between 2018/19 in the USA indicate that concentrations of formaldehyde may range from 0.27 to 124.2 µg/m^3 (median 19.77 µg/m^3), with 90 percent of homes having concentrations below 41.8 µg/m^3 (US EPA, 2024a). Based on these data, the HSE concludes that formaldehyde levels in English (and by extrapolation GB) homes are equivalent to levels found in other high-income countries around the world.

It is noteworthy that in a study of 10 new English homes built before 2014, arithmetic mean formaldehyde concentrations of 34 µg/m^3 (living rooms) and 40 µg/m^3 (bedrooms) were found (MHCLG, 2019). Also, a study conducted in Northern Ireland revealed levels up to 1,400 µg/m^3 in three houses constructed to meet Passivhaus standards. This suggests that steps that are taken to improve air tightness and hence the energy efficiency of homes may result in poorer indoor air quality.

Data sets for some indoor air monitoring studies were large enough to investigate the impact of different types of sources on indoor air concentrations. For homes in England, Berry *et al*. (1996) and Raw *et al*. (2004) found that the age of home had a significant impact on indoor formaldehyde levels, with homes built after 1982 having around three times the level of indoor formaldehyde compared with homes built before 1919. This is likely to be the result of changes in building practices (greater use of engineered wood boards and requirements to increase the air tightness of buildings), introduced between the 1970s and 1990s. Similar findings were obtained in German studies.

These studies from GB and Germany have identified several additional factors associated with increased indoor formaldehyde concentrations, including:

- rural location compared with suburban or urban location;
- detached homes compared with semi-detached, terrace, or flat;
- integral garage compared with detached garage;
- new particleboard furniture and/or flooring;
- frequent use of sanitary cleaners/disinfectants.

The difference for each factor was around 10 – 20 µg/m^3.

Based on data published by Salthammer (2919a), Birmili *et al*. (2021) concluded that:

“Ignoring wood-based materials as a source and taking sinks into account, indoor formaldehyde concentrations in order of 10 – 15 µg/m^3 are expected. We estimate that

the inclusion of wood-based materials would roughly double these values."

Birmili *et al*. (2021) also notes that:

"Measurements in a test cabin built from "formaldehyde-free" materials yielded, at an air exchange rate of 0.3 h^{-1}, room concentrations of formaldehyde between 15 and 20 $\mu g.m^3$."

These levels are within the range of averages recorded in studies of GB homes.

3.3.4 Uncertainties in indoor air monitoring data

- Formaldehyde in air measurements are only available for a very small fraction of GB homes, and the larger data sets were obtained pre-2000, creating uncertainty around how representative these measurements are for the current GB housing stock. More recent data are available from studies in Germany and other European countries, also the US. These data are consistent with the available GB data which reduces the uncertainty associated with the use of data from older studies.

- Newer studies in the UK are small and report arithmetical means which are heavily influenced by high results, sample size and statistical distribution of monitoring results.

- It is not clear how representative the monitoring data are for situations such as very small and/or highly furnished rooms, e.g. small bedrooms with built in wardrobes or highly energy efficient homes or cases where much of the furniture is new and has been made using high formaldehyde-emitting materials.

- Based on data published by Salthammer (2019a), Birmili *et al*. (2021) performed calculations to understand the relative contribution to indoor air from continuous and intermittent sources which suggest on average there is a roughly equal, 50:50, split between continuous and intermittent sources.

- The Agency does not know how widely this ratio holds across GB homes. There will be homes that deviate from this ratio based on the age of the house, its ventilation characteristics, the age and type of the furnishings in the house and the activities of residents around frequency and duration of use for various intermittent sources.

4 Hazards (including classification)

4.1 Classification

Table 14: Mandatory classification in the GB MCL list

Index No	International Chemical Identification	EC No	CAS RN	Classification		Spec. Conc. Limits, M-factors	Notes
				Hazard Class and Category Code(s)	Hazard statement code(s)		
605-001-00-5	Formaldehyde	200-001-8	50-00 - 0	Carc. 1B Muta. 2 Acute Tox. 4 Acute Tox. 2 Skin Corr. 1B Skin Sens. 1A	H350 H341 H302 H330 H314 H317	Oral: ATE = 500 mg/kg bw Inhalation ATE = 100 ppmV (gases) Skin Corr. 1B; H314: C ≥ 25 % Skin Irrit. 2; H315: 5 % ≤ C < 25 % Eye Irrit. 2; H319: 5 % ≤ C < 25 % STOT SE 3; H335: C ≥ 5 % < 25% EUH071; C ≥ 25%	B, D, F

Note B: Relates to substances that are placed on the market in aqueous solutions. The required classification and labelling may be different at different concentrations. This note specifies that the supplier of an aqueous solution of such a substance must state the percentage concentration of the solution on the label.

Note D: Substances which are susceptible to spontaneous polymerisation or decomposition are generally placed on the market in stabilised form. However, if placed on the market in a non-stabilised form, the supplier must state on the label the name of the substance, followed by the words ‘non-stabilised’.

Note F: This substance may contain a stabiliser. If the stabiliser changes the hazardous

properties of the substance, as indicated by the classification in Part 3, classification and labelling should be provided in accordance with the rules for classification and labelling of hazardous mixtures.

Details on any mandatory classification and labelling that may have been adopted for the formaldehyde releasers identified in Table B.2 of the Annex to ECHA's restriction dossier (ECHA, 2020a) is available in Annex 2.

4.2 Relevant key hazards

The hazards of formaldehyde have been well characterised, with the chemical having a mandatory classification under GB CLP (Section 4.1). Since this technical report aims to summarise the risk to the general population from inhalation exposure to formaldehyde in indoor settings, the focus of this section is on relevant human-health hazards from its inhalation. This report has not concluded on thresholds of effect. Instead, this section presents the reviews undertaken by other authoritative bodies that have considered the hazards of formaldehyde inhalation exposure and the approaches they have taken to determine health-based guidance values (HBGVs).

For transparency in reporting and clear interpretation of toxicological data, inhaled formaldehyde concentrations have been recorded as provided in original study reports. For consistency and comparison of inhaled concentrations of formaldehyde, where units have not been provided in study reports, units have been converted such that 1 ppm = 1.23 mg/m^3 = 1,230 µg/m^3 at 25 °C (ATSDR, 1999; WHO, 2010; SCOEL, 2016). It is acknowledged that different authoritative bodies have used different conversion factors, and rounded values differently, which could lead to slight differences in µg/m^3 values.

4.2.1 Toxicokinetics

Following inhalation of exogenous formaldehyde, tissues of the upper respiratory tract (URT) are the first sites of contact. Based on dosimetry modelling in humans, 90% of inhaled formaldehyde gas is retained in the upper airways, predominantly in the nasal mucosa. Very little exogenous formaldehyde gas penetrates to the lower airways (WHO, 2010; Kimbell *et al*., 2001; US EPA, IRIS, 2024). Most of the inhaled formaldehyde is expected to be hydrated by water contained within the lining of the URT to produce methanediol, leaving only a small fraction unchanged. Once absorbed into respiratory tissue, any unchanged formaldehyde is rapidly metabolised to formate, which may be further oxidised to form carbon dioxide or used in the one-carbon biosynthetic pathways leading to protein and nucleic acid synthesis.

Whilst there is evidence that exogenous formaldehyde gas does not penetrate to the deep lung in significant quantities, formaldehyde gas molecules can adsorb to the surface of particulates of respirable size. It has been suggested that these particulates might then serve as carriers, able to penetrate to the peripheral airways and deliver formaldehyde deeper into the lung, rather than remaining in the URT. At sites of contact

in the lower airways, formaldehyde is likely to act as an irritant, potentially resulting in local inflammatory responses (US EPA, 2005; Dhar, 2020).

Formaldehyde is produced endogenously as a result of normal cellular metabolism, with blood concentrations of endogenous formaldehyde in human subjects reported to be in the range of 2-3 mg/L (Heck *et al*., 1985). This endogenous formaldehyde can impact the uptake of exogenous formaldehyde: at low concentrations of formaldehyde in the air, the levels of endogenous formaldehyde present in the nasal mucosa reduce the concentration gradient across the tissue such that the uptake of exogenous formaldehyde is significantly reduced (US EPA, IRIS, 2024; SCOEL, 2016). The retention of the majority of inhaled formaldehyde gas in the URT is consistent with experimental data in both human volunteers and experimental animals, where exposure to exogenous formaldehyde at concentrations ranging from 1.9 ppm to 14.4 ppm [2,340 µg/m^3 – 17,710 µg/m^3 respectively] does not significantly alter the blood formaldehyde concentration (ATSDR, 1999; US EPA, IRIS, 2024; Casanova *et al*., 1988; Heck *et al*., 1985; Kleinnijenhuis *et al*., 2013). Following from the points above, the lack of increased blood formaldehyde concentrations is likely due to rapid metabolism or reaction with cellular macromolecules (DNA, RNA, proteins) at first sites of contact in the URT (ATSDR, 1999).

Considering the above, formaldehyde is not expected to be systemically available following inhalation exposures at levels occurring in GB homes (Section 3.3.2.2). Therefore, the primary health effects relevant to this assessment are local effects to the upper airway tissues (US EPA, IRIS, 2024; ECHA, 2020c).

4.2.2 Inhalation effects of formaldehyde

As noted previously, the primary effects following formaldehyde inhalation are expected to be local in the respiratory tract. The local effects potentially resulting from formaldehyde inhalation exposure can be categorised as sensory irritation, exacerbation of asthma and other respiratory conditions, and tumorigenesis through site-of-contact tumour formation.

Of the available human studies (epidemiological, occupational, or controlled exposures) investigating the effects of formaldehyde inhalation, some include subjective reporting of symptoms. Odour perception can increase the reporting of subjective effects such as eye/nasal discomfort, olfactory symptoms and annoyance. Objective parameters, such as conjunctival redness, eye blinking frequency (EBF), and nasal resistance or flow might be less affected by odour perception (WHO, 2010; SCOEL, 2016). Multiple odour thresholds for formaldehyde have been reported, ranging from 50 – 500 µg/m^3 (WHO, 2010). The available investigations into odour thresholds suggest that odour perception for individuals depends on factors such as air purity, smoking status and previous exposure to formaldehyde. One study that used a bi-sensory method to determine thresholds for odour and sensory irritation found that odour thresholds for individuals ranged from 0.02 – 0.5 ppm [25 – 615 µg/m^3]. In the same study, 50% of 31 participants were able to detect formaldehyde at concentrations of 0.1 ppm [123 µg/m^3],

thereafter referred to as the P50 concentration (Berglund *et al.*, 2012, cited in SCOEL, 2016).

4.2.2.1 Sensory irritation

Airborne irritants like formaldehyde can stimulate trigeminal nerve endings located in the respiratory epithelium, which is perceived as unpleasant sensations in the eyes and upper airways (WHO, 2010; ECHA, 2020c; US EPA, IRIS, 2024). The eyes are reportedly more sensitive to volatile chemical irritants than the upper airways (Doty *et al*, 2004; WHO, 2010). Sensory irritation is different from histopathologically-detectable irritation (i.e., inflammation). It is also reversible, and so quickly resolves when exposure is stopped. The severity of sensory irritation is not a function of concentration and time, but is instead related to exposure concentrations: brief exposures can elicit the same intensity of responses as longer exposures.

Across the available controlled human exposure studies, sensory irritation reactions, affecting the eyes, nose, and upper airways, have been reported for formaldehyde inhalation exposures ranging from approximately 370 µg/m^3 to 3,690 µg/m^3 (ATSDR, 1999; Health Canada, 2005; WHO, 2010; SCOEL, 2016; ECHA, 2020c; US EPA, IRIS, 2024). Many of these studies include subjective reporting of irritation symptoms, alongside or in place of objective parameters. As noted previously, when subjective parameters are employed, confounding factors, such as personality traits or odour perception, can result in an over-estimation of the sensitivity of sensory irritation responses when compared with objective parameters (WHO, 2010; SCOEL, 2016). It is also noted that sensory habituation may occur, where there is a reduced response to the same exposures of formaldehyde concentrations. The US EPA noted that it is not clear whether desensitisation occurs over time, or the concentrations or timeframes over which this might occur (US EPA, IRIS, 2024).

4.2.2.2 Asthma and respiratory conditions

Asthma is a clinical diagnosis encompassing varying symptoms and physiological mechanisms, though it is primarily characterised by a restriction of airflow resulting from inflammation narrowing the airways.

Allergic asthma is thought to be the most common form of asthma and often has an early onset of symptoms. With immunological asthma, an individual becomes sensitised to an allergen, e.g., dust mites, grass, pollens, fungal spores, or animal dander. This induction phase is then followed by the elicitation phase, where repeated exposure to the allergen results in the presentation of symptoms. The immune system sensitised to a specific allergen produces immunoglobulin E (IgE) antibodies, which activate IgE-dependent mast cells that recruit eosinophils to the lungs upon repeated exposures. This starts a cascade of inflammatory responses, ultimately leading to airway hyper-responsiveness (AHR), mucus over-production, and airway remodelling (Boonpiyathad *et al*., 2019).

In comparison, non-allergic asthma is an irregular innate immune response and does

not involve IgE production, therefore has no period of latency and can occur following a single high exposure to non-specific stimuli. These stimuli might include viruses, cigarette smoke and environmental pollutants (Boonpiyathad *et al*., 2019). A form of occupational asthma, irritant-induced asthma (previously known as reactive airway dysfunction syndrome (RADS)) is considered to have a non-allergic mechanism. It is a term used to describe the asthma-like symptoms, non-specific bronchial hyper-responsiveness and airway inflammation, caused by irritant mechanisms. Inhalation of high concentrations of formaldehyde has been associated with cases of irritant-induced asthma in the workplace (Vandenplas *et al*., 2014; Fishwick *et al*., 2023). Clinical division into allergic and non-allergic asthma phenotypes is thought to be an over-simplification, the reality being a more complex continuum of underlying pathophysiological mechanisms. As such, asthma endotypes have been defined to reflect the varying mechanisms (Hammad and Lambrecht, 2021).

As noted above, the local effects reportedly associated with formaldehyde inhalation exposures include exacerbation of asthma and other respiratory conditions. Although a known skin sensitiser, there is limited evidence to support formaldehyde acting as an allergen in an immunologically-mediated mechanism of asthma (ATSDR, 1999; WHO, 2010). Whilst there have been reports of anaphylaxis amongst dermally-sensitised individuals following dermal and inhalation exposures to formaldehyde, which indicate IgE-mediated reactions are possible, the frequency of occurrence is very rare (Maurice *et al*., 1986). Rather, formaldehyde may act to augment immune responses to other allergens, potentially affecting the prevalence and severity of existing asthma cases (WHO, 2010; US EPA, IRIS, 2024). As such, there are potentially several vulnerable populations with respect to effects on asthma and other respiratory conditions, as discussed below.

<u>Asthmatics and other existing respiratory conditions</u>:

The US Agency for Toxic Substances and Disease Registry (ATSDR, 1999) noted that the evidence for an immunologically-mediated sensitisation of the respiratory tract by formaldehyde was weak. There was no response to challenge with formaldehyde in several controlled human exposure studies, despite subjects having previous exposure and complaints of asthma-like symptoms. Similarly, amongst the available occupational studies there were very few case reports of workers with significantly altered pulmonary function in response to challenge with formaldehyde that would be consistent with immunologically-mediated mechanisms. There was also a lack of consistent evidence for increased serum levels of formaldehyde-specific IgE antibodies in exposed subjects complaining of asthma-like symptoms (ATSDR, 1999). Other studies, including epidemiology studies, presented evidence for formaldehyde-induced changes in pulmonary function in workers or those exposed in residential settings, although the changes were small/subtle (Krzyzanowski *et al*., 1990; ATSDR, 1999).

The World Health Organisation (WHO, 2010) noted that, although formaldehyde was not considered a respiratory sensitiser, two hypotheses were suggested to explain

formaldehyde-induced respiratory symptoms: inflammation in the lungs; and formaldehyde acting as an adjuvant for allergens (WHO, 2010). The US EPA has also suggested that formaldehyde exposures in general population studies might enhance the immune hypersensitivity response to allergens (US EPA, IRIS, 2024). Given this information, it is possible that existing asthmatics, or those with respiratory conditions impairing lung function, might potentially be more susceptible to effects from formaldehyde inhalation.

Children:

The lungs are under-developed in neonates and are not reported to be fully functional until 6 – 8 years of age, with children's airways remaining narrower than those of adults (Burri, 1984, cited in Bateson and Schwartz, 2008; OEHHA, 2008). The authors of several expert reviews of inhalation toxicity have postulated that children might be more susceptible than adults to effects on the respiratory system resulting from the inhalation of formaldehyde and other chemical agents. Studies on other chemicals have also suggested that human sensitivity to sensory irritation might be dependent on age, again highlighting differences in the physiology of children and adults (Shusterman *et al.,* 2006; Hummel *et al.,* 2003).

There is some evidence to support the hypothesis that children might be more susceptible to effects of formaldehyde inhalation on the respiratory system than adults. The Royal College of Paediatrics and Child Health (RCPCH) and the Royal College of Physicians (RCP) reported that respiratory problems among children were exacerbated by exposure to formaldehyde in homes (RCPCH / RCP, 2020). This conclusion was based on:

i) The Pollution and Asthma Risk Infant Study (PARIS), in which indoor renovation was reported to be a risk factor for respiratory symptoms or allergies (Herr *et al*., 2012). Evidence from that cohort suggested that the risk might be attributable to formaldehyde (Roda *et al*., 2011; 2013).

ii) A survey of over 6,500 schoolchildren in France, in which rhino-conjunctivitis and asthma were significantly associated with high levels of formaldehyde or acrolein in classrooms (Annesi-Maesano *et al*., 2012; see Annex 5 for more information on this study).

The ATSDR addendum to the toxicological profile on formaldehyde (2010) reviewed available epidemiology studies examining the relationship between residential formaldehyde exposure and asthma and/or allergies. Although inconsistent findings were reported, the results of these studies indicated that low residential indoor air levels of formaldehyde might predispose young children to asthma or allergies. However, the ATSDR noted the dose-response relationship had not been clearly established and further research was necessary (ATSDR, 1999; ATSDR, 2010).

Similarly, the US EPA's Integrated Risk Information System (IRIS) reviewed the

available hazard data relevant to inhalation exposure to formaldehyde. This assessment reported epidemiological studies in which children had an increased sensitivity to formaldehyde exposure-induced respiratory effects under 'sufficient exposure conditions'. It was also noted that children younger than five years of age might experience symptoms consistent with lower respiratory infections in association with residential formaldehyde levels lower than those at which older individuals experienced these symptoms (Roda *et al*., 2011; Rumchev *et al*., 2002). Overall, the review concluded that formaldehyde was likely to cause an increased risk of prevalence of asthma and allergic conditions (e.g., rhinitis and rhinoconjunctivitis), and decreased control of asthma symptoms (US EPA, IRIS, 2024). These findings were primarily supported by studies in occupational settings (> 100 µg/m^3) and epidemiology studies with formaldehyde concentration ranging between 50 – <100 µg/m^3 (US EPA, IRIS, 2024).

Conversely, the WHO stated that, in relation to non-cancer respiratory effects, the available data indicated that children were not more susceptible than adults, taking into account information on oronasal breathing in children and respiration rates. This conclusion agreed with predicted formaldehyde absorption rates per unit surface area of the nasal cavity, which were reported to be equal in both children and adults.

The WHO (2010) acknowledged the association between low formaldehyde exposure and asthma or sensitisation, but noted the epidemiology data supporting this association often included complex co-exposure scenarios, making it difficult to establish dose-response relationships (WHO, 2010). The WHO further noted the lack of consistent findings in controlled human exposure studies and epidemiology studies (<1000 µg/m^3), specifically with respect to cause-effect and dose-response relationships between formaldehyde and measurable lung effects.

ECHA (2020c) also did not consider there was sufficient evidence to conclude that children are more susceptible and cited the 2016 German Umweltbundesamt's (UBA's) Committee on Indoor Air Guide Values (AIR). This states: "there is no clear association between formaldehyde exposure in the indoor environment and asthma in children. Mainly, the epidemiological studies suffer from small sample sizes, implausible formaldehyde concentrations, and the fact that other substances or factors initiating asthma and asthma-like complaints were not adequately considered. Results derived from controlled human exposure studies as well as animal experiments support this opinion."

Overall, children might have an increased susceptibility to some effects resulting from formaldehyde inhalation exposures. Specifically, in some studies reduced pulmonary function, increased prevalence of asthma and reduced asthma control have been reported in children following formaldehyde exposures in indoor air (ATSDR, 1999, 2010; OEHHA, 2008; US EPA, IRIS, 2024; Lam *et al.,* 2021).

4.2.2.3 Carcinogenicity

Formaldehyde is a known genotoxic carcinogen in experimental animals (rodents), causing site-of-contact nasal tumours following chronic inhalation exposure (ECHA, 2020c; SCOEL, 2016; WHO, 2010; US EPA, IRIS, 2024). The critical cancer type in rats is nasal squamous cell carcinoma.

Site-of-contact tumour induction by formaldehyde is driven by sustained cytotoxicity and chronic cell proliferation in combination with DNA alterations by endogenous and exogenous formaldehyde. The dose-response relationship for all the parameters investigated (damage to the nasal epithelium, cell proliferation, tumour incidence, the formation of DNA-protein cross-links (DPX) and DNA adducts) is very flat for low level exposures and becomes steeper at higher levels of exposure, ≥ 2 ppm [2,460 µg/m^3] in experimental animals (SCOEL, 2016). No-effect concentrations have been demonstrated for all effects except DPX and DNA-adduct formation. At the lowest concentrations investigated, adducts caused by endogenous formaldehyde far outweighed those caused by exogenous formaldehyde. Proliferation of respiratory epithelial cells is also not increased at low formaldehyde concentrations ≤ 2 ppm [≤2,460 µg/m^3]. Furthermore, even though DPX are formed at low concentrations, these are rapidly repaired and might be a source of DNA adducts (SCOEL, 2016). The observed experimental genotoxicity of formaldehyde therefore does not appear to play a key role in its carcinogenic potential at concentrations that do not lead to increased cell proliferation. Therefore, a threshold can be established for concentrations that don't lead to sustained cell proliferation and histopathological alterations (ECHA, 2020c; WHO, 2010; SCOEL, 2016).

The formaldehyde concentration might be more important for cytotoxicity than the total formaldehyde dose. There are also likely to be species differences in responses. SCOEL (2016) and WHO (2010) considered that rats are more sensitive to DPX formation than monkeys, and that monkeys are probably more sensitive than humans; they also reported that the number of DNA adducts is higher in rats than in monkeys at comparable exposure concentrations. Given its different respiratory physiology, the rat might be a poor and over-sensitive model, whilst the monkey exhibits similarities to humans (DeSesso, 1993, cited in SCOEL, 2016).

Inhalation exposure to formaldehyde in occupational settings can induce nasopharyngeal cancer in humans (WHO, 2010; IARC, 2006; ECHA, 2012). The available evidence indicates that inhaled formaldehyde causes DPX formation in nasopharyngeal tissue. The WHO (2010) stated that the strongly non-linear relationship between formaldehyde exposure and the development of squamous cell carcinoma in rats was largely corroborated by epidemiological studies. Therefore, avoidance of sensory irritation of the eye and URT would imply a safety margin to also avoid cytotoxic irritation-induced local cell proliferation as a first step to tumour induction in humans.

The International Agency for Research on Cancer (IARC) concluded there was strong

but not sufficient evidence for a causal association between leukaemia and occupational exposures to formaldehyde (IARC, 2006). IARC noted that increased risk for leukaemia was consistently observed in studies of professional workers and in two of the three evaluated studies of industrial workers. However, IARC recognised limitations in the findings from the cohorts of some studies on professional and industrial workers, and noted that they conflicted with the negative findings from another cohort of industrial workers. Long-term inhalation carcinogenicity studies in experimental animals have presented no evidence of lymphohematopoietic malignancies at concentrations at which nasal cancers developed (WHO, 2010).

Overall, the association of formaldehyde and the development of nasopharyngeal malignancies or leukaemia in humans is likely to be limited to high inhalation exposures (higher than exposures typically experienced in UK indoor environments).

ECHA's Risk Assessment Committee (RAC) relied upon findings in animal studies (primarily nasal tumours in rats) in supporting a classification under CLP of formaldehyde as a presumed human carcinogen (Category 1B); it considered that the evidence in humans was insufficient to warrant classification for carcinogenicity Category 1A (ECHA, 2012).

4.2.3 Summary of key studies addressing the inhalation toxicity of formaldehyde

The available data investigating the health effects of formaldehyde inhalation largely comprise epidemiology studies, occupational studies, controlled human exposure studies and animal studies. Key studies identified by expert groups and relied upon in the derivation of health-based guidance values (HBGVs), have been summarised below (Table 15). For Points of Departure (PoDs) derived by expert groups to set HBGVs please see (Table 16) Section 4.2.5.

Additional studies and further details for each study referenced, including recognised strengths and limitations, have been included in Annex 5, Section A.5.1.

Table 15: Key studies investigating inhalation effects of formaldehyde.

Reference	Type of study	Population / species	Critical effects	Expert Group Identifying as key / supportive study
Epidemiology studies (general population)				
Hanrahan *et al*., 1984	Cross-sectional	Wisconsin, US, teenagers and adults living in mobile homes (smokers included).	Ocular discomfort – burning eyes and irritation.	US EPA, IRIS, 2024. Derivation of point of departure (PoD) for sensory irritation.
Krzyzanowski *et al*., 1990	Cross-sectional	US adults & children (6 – 15 years).	Decrease in peak expiratory flow rate (PEFR).	US EPA, IRIS, 2024. Derivation of PoD for pulmonary function.
Annesi-Maesano *et al.*, 2012	Cross-sectional	Children in France (9 – 10 years).	Prevalence of rhinoconjunctivitis and asthma.	US EPA, IRIS, 2024. Derivation of PoDs for rhinoconjunctivitis and asthma prevalence.
Matsunaga *et al*., 2008	Cross-sectional	Pregnant adult women in Japan.	Study authors found no clear association between formaldehyde concentration and the prevalence of asthma nor allergic rhinitis. Increased prevalence of atopic eczema in the highest dose group.	US EPA, IRIS, 2024. Derivation of PoD for the prevalence of atopic eczema.

Reference	Type of study	Population / species	Critical effects	Expert Group Identifying as key / supportive study
Rumchev *et al.*, 2002	Case-control	Children aged between 6 months and 3 years in Perth, Western Australia.	Increased risk of asthma.	Health Canada, 2005. Basis for setting a long-term (8 hour) guidance value.
Venn *et al.*, 2003	Nested case-control	Children (9 – 11 years) in Nottingham, UK.	Study authors conclude formaldehyde and damp were associated with more frequent nocturnal symptoms of asthma.	US EPA, IRIS, 2024. Derivation of PoD for degree of asthma control.
Epidemiology studies (occupational)				
Holmström *et al.,* 1989	Cross-sectional	Workers in a chemical plant, furniture factory and control group.	Histological changes in the nasal mucosa.	ATSDR, 1999. Derivation of PoD for histopathological changes in the nasal epithelial tissue.
Wilhelmsson and Holmström, 1992	Cross-sectional	Workers in a chemical plant, office workers as controls.	Nasal obstruction and discomfort, lower airway discomfort, eye irritation.	OEHHA, 2008. Derivation of PoD for sensory irritation.
Controlled human exposure studies				
Andersen and Mølhave,	Controlled	Healthy adults in Denmark.	Irritative symptoms, including eye blinking frequency, subjectively	US EPA, IRIS, 2024.

Reference	Type of study	Population / species	Critical effects	Expert Group Identifying as key / supportive study
1983	exposure		reported discomfort and dryness in the throat and nose. Also included objective measurements.	Derivation of PoD for sensory irritation.
Pazdrak *et al.,* 1993	Controlled exposure	Occupationally-exposed patients with skin hypersensitivity to formaldehyde and unexposed controls.	Sensory irritation, mucosal congestion, transient burning of the eyes and nasal passages, elevated eosinophil counts. Subjective and objective measurements.	ATSDR, 1999. Derivation of PoD for sensory irritation effects.
Kulle *et al.*, 1987 Kulle, 1993 (re-examination of original dataset)	Controlled exposure	Healthy, non-smoking adults (male and female).	Sensory eye irritation. No significant effects on pulmonary function. Objective and subjective measurements.	Health Canada 2005; OEHHA, 2008; US EPA IRIS 2024. Derivation of PoD for eye irritation symptoms.
Lang *et al.*, 2008	Controlled exposure	Healthy adult males and females.	Subjective and objective reports of sensory irritation (eyes and URT).	WHO, 2010; SCOEL, 2016; UBA, 2016; ANSES, 2018; ECHA 2020c. Derivation of PoDs for sensory irritation.
Mueller *et al.*, 2013	Controlled exposure	Adult males, hypo- and hyper-sensitive (based on CO_2 sensitivity measurements in nasal	Subjective and objective reports of sensory irritation – eye and nasal discomfort.	US EPA, 2024. Derivation of PoD for sensory

Reference	Type of study	Population / species	Critical effects	Expert Group Identifying as key / supportive study
		mucosa).		irritation.
Experimental studies in animals				
Rusch *et al*., 1983	Inhalation for 26 weeks	Cynomolgus monkeys, Fischer F344 rats, Syrian golden hamsters.	Nasopharyngeal irritation and lesions in nasal epithelium (squamous metaplasia and hyperplasia).	ATSDR, 1999; WHO, 2010; ECHA, 2020c. Derivation of PoD for metaplasia and hyperplasia in nasal epithelium.
Appelman *et al*., 1988	Inhalation for 13 or 52 weeks	SPF Wistar rats (males).	Basal cell hyperplasia and squamous metaplasia of the nasal epithelium.	WHO, 2010. Derivation of PoD for increased cell proliferation in the nasal mucosa.
Woutersen *et al*., 1989	Inhalation for 3 or 28 months; sacrifice at 28 months	Wistar rats (males).	Increased incidence of squamous metaplasia of the respiratory epithelium.	WHO, 2010. Derivation of PoD for increased cell proliferation in the nasal mucosa.

4.2.4 Review of publications on formaldehyde inhalation toxicity by expert groups

The available authoritative reviews have assessed the extent to which formaldehyde inhalation is associated with sensory irritation, tumorigenesis and the development, or exacerbation, of respiratory conditions such as rhinitis, rhinoconjunctivitis, and asthma. The conclusions of prominent expert groups on the health effects of formaldehyde from inhalation exposure, and the health-based guidance values they proposed, are summarised below.

4.2.4.1 Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Formaldehyde, 1999

The ATSDR reviewed the available data set in 1999 and published an addendum in 2010 (see below). The data comprised controlled exposure studies in humans, epidemiology studies (occupational and non-occupational) and animal studies. The ATSDR estimated levels that posed minimal risk to humans (minimal risk levels or MRLs) to inform health professionals and the general population. An MRL is 'an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Exposure to a level above the MRL does not mean that adverse health effects will occur. MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely' (ATSDR, 1999).

ATSDR also noted the limited data-set available to conclude on the susceptibility of children to formaldehyde inhalation compared with adults:

*'*Two studies were available providing suggestive evidence that children may be more sensitive than adults to the irritant properties of airborne formaldehyde (Krzyzanowski *et al*., 1990; Wantke *et al*., 1996). Additional research is necessary to confirm or discard the hypothesis that children may be more susceptible than adults to the irritant effects of formaldehyde and to understand the mechanistic basis of this possible difference*.'*

Overall, the ATSDR concluded that formaldehyde was irritating to the upper respiratory tract and eyes following inhalation exposures. ATSDR noted that the evidence that formaldehyde sensitised the respiratory tract was lacking.

4.2.4.1.1 Acute value

Controlled exposure studies in humans indicated eye, nose, and throat irritation at 0.4 – 3 ppm [490 – 3,690 µg/m^3]. ATSDR identified the critical LOAEC from which to derive an acute (14 days of exposure or less) minimal risk level (MRL) as 0.4 ppm [490 µg/m^3]. At this concentration, there was increased itching, sneezing, mucosal congestion and transient burning of the eyes and nasal passages, elevated eosinophil counts and transient increase in nasal lavage fluid (i.e., objective measures) in a

controlled human exposure study in which volunteers were exposed for 2 hours (Pazdrak *et al*., 1993). ATSDR applied an assessment factor (AF) of 9 (3 for the point of departure (PoD) being a LOAEC and 3 for human variability) to derive an acute MRL of 0.04 ppm [rounded to **50 µg/m^3**]. The justification for the assessment factor of 3 for use of a minimal LOAEL was that the symptoms of irritation were mild and reversible, and the clinical significance of changes in nasal lavage fluid was uncertain. An AF of 3 for human variability was justified on the grounds of the inclusion of a potentially sensitive group of subjects, i.e., those that displayed dermal sensitivity to formaldehyde. Given the inclusion of dermally sensitive individuals and the weight of evidence that some people wouldn't experience eye or URT irritation even at 1 ppm [1,230 µg/m^3] formaldehyde, ATSDR considered the derived value to be conservative.

4.2.4.1.2 Intermediate value

ATSDR did not identify studies of humans exposed for intermediate durations but noted the numerous animal studies. The study by Rusch *et al*. (1983) examined several species and identified the lowest effect level among the available sets of data. Given this observation, the absence of human intermediate-duration data, and the putatively greater relevance of monkeys, compared with rodents, to humans, ATSDR chose the Cynomolgus monkey NOAEC of 0.98 ppm [1,210 µg/m^3] and LOAEL of 2.95 ppm [3,630 µg/m^3] as the basis of the intermediate-duration (15 – 364 days of exposure) MRL. In this study, there were clinical signs of nasopharyngeal irritation (hoarseness, nasal congestion/discharge) and lesions in nasal epithelium (squamous metaplasia and hyperplasia) from 2.95 ppm [3,630 µg/m^3] formaldehyde for 26 weeks.

ATSDR applied an AF of 3 for extrapolation from animals to humans rather than the default of 10 because similar nasal effects were reported at similar concentrations in different species and different studies; hence, pharmacodynamic differences in species susceptibility were not expected. ATSDR then applied an assessment factor of 10 for human variability. Application of the overall assessment factor of 30 to the NOAEC gave an intermediate MRL of 0.033 ppm [**40 µg/m^3**].

4.2.4.1.3 Long-term value

ATSDR set a chronic MRL (365 days or more of exposure) from an occupational study in chemical-plant workers (producing formaldehyde and formaldehyde resin), furniture-factory workers (working with particle-board and glue components) and office workers ('referents' from the same village as the furniture factories) (Holmström *et al*., 1989). ATSDR selected this study over cross-sectional studies primarily because the statistically significant effects were found in a group exposed to formaldehyde in the absence of potentially confounding exposures to wood dust (the furniture-factory workers in this study were co-exposed to wood dust).

In Holmström *et al*. (1989), nasal mucosa specimens for histological analysis were taken from the medial or inferior aspects of the middle turbinate. Histological changes included loss of cilia, goblet cell hyperplasia, and cuboidal and squamous cell

metaplasia replacing columnar epithelium from nasal tissue specimens. The mean histological score for the chemical workers (2.16) was reported to be significantly different from the control score (1.46), although the ranges of scores was the same for both groups (0 – 4 on a scale from 0 to 8). Furthermore, the estimated formaldehyde exposures of the office workers (0.07 – 0.13 ppm [86 – 160 µg/m^3] in late summer, year-round median 0.07 ppm [86 µg/m^3]) over-lapped with that of the chemical workers (0.04 – 0.4 ppm [49 – 492 µg/m^3], median 0.24 ppm [295 µg/m^3]). The WHO (2010) noted the absence of an exposure-dependent effect in histopathology in this study.

From Holmström *et al*. (1989), ATSDR noted clinical symptoms of mild irritation of the eyes and upper respiratory tract and mild damage to the nasal epithelium in chemical-plant workers exposed for 10.4 years (range 1 – 36 years) to an average time-weighted average (TWA) concentration of 0.24 ppm [295 µg/m^3]. ATSDR considered this LOAEC of 0.24 ppm [295 µg/m^3] to be a minimal LOAEC.

ATSDR then applied an AF of 3 for the use of a LOAEL; the histological effects in the exposed groups were mild and sub-clinical, suitable for 0.24 ppm [295 µg/m^3] to be designated as a minimal LOAEC. An AF of 10 was applied for human variability; the full factor of 10 was applied because the observed mild effects were observed in workers that were in apparently good health, hence a 'health-worker' effect might have resulted in sensitive individuals avoiding employment in the studied workplaces. The chronic MRL was 0.008 ppm [**10 µg/m^3**].

ATSDR did not adjust the exposure duration to a continuous exposure basis because the evidence indicated that the concentration was more important than the product of concentration and duration of exposure in determining the severity of formaldehyde-induced epithelial damage in the URT. It considered the chronic MRL to be sufficiently protective of the development of nasal tumours, given it was below the intermediate-duration inhalation MRL derived from studies in animals for the development of clinical signs of nasopharyngeal irritation and lesions in the nasal epithelium.

4.2.4.1.4 Addendum to the ATSDR toxicological profile

The ATSDR published an addendum to its toxicological profile for formaldehyde in 2010 (ATSDR, 2010). This took into account the scientific data that had been published in the open peer-reviewed literature since the release of the original profile.

In reviewing the available controlled human exposure studies, ATSDR referred to several (including Lang *et al*., 2008; Kulle *et al.,* 1987; Kulle, 1993) that did not show an effect of formaldehyde inhalation on lung function. This included studies in which subjects were described as asthmatic (Witek *et al*., 1987; Ezratty *et al*., 2007; Krakowiak *et al*., 1998).

In terms of the earlier repeated-exposure human studies in occupational or residential settings, ATSDR noted that they provided 'limited evidence that pulmonary functions may be adversely affected by repeated exposure to formaldehyde' (citations included

Krzyzanowski *et al*., 1990). ATSDR reviewed the newly available studies and also considered children's susceptibility. Regarding the latter, ATSDR concluded the findings were inconsistent, citing the studies of Krzyzanowski *et al*. (1990), Rumchev *et al*. (2002), Venn *et al*. (2003), Garrett *et al*. (1999; see Annex 5), Delfino *et al.* (2003; see Annex 5) and Tavernier *et al*. (2006; see Annex 5). The overall conclusion was that *'*The results of these studies indicated that low residential indoor air levels of formaldehyde may predispose young children to asthma or allergies. However, the dose-response relationship has not been clearly established and further research is necessary*.'*

4.2.4.2 Health Canada Residential Indoor Air Quality Guideline, 2005

Health Canada reviewed epidemiological studies, controlled human exposure studies and animal studies to derive a short-term and a long-term indoor air quality guideline (IAQG) value.

4.2.4.2.1 Short-term value

Health Canada concluded from the human clinical studies and animal experiments that the primary effects of acute inhalation exposure to formaldehyde were irritation of the mucosa of the URT and the eyes. In relation to histopathological changes in the nasal cavity in animal studies, which occurred at concentrations of 3,700 µg/m^3 and above, Health Canada noted that these were a function of the formaldehyde concentration in inhaled air rather than of the cumulative dose.

Health Canada concluded that the controlled exposure studies showed largely consistent patterns, in that exposures between 2,460 and 3,690 µg/m^3 caused eye, nose and throat irritation, and that exposure to 3,690 µg/m^3 caused transient lung function changes in healthy subjects, but not asthmatics (Green *et al*., 1987).

As the study by Kulle *et al*. (1987) was the only study available at that time that enabled the assessment of an exposure-response relationship, Health Canada used it to set the short-term IAQG value. It is noted that the original study conducted in 1987 was re-examined with additional statistical methods in 1993 (Kulle, 1993), see Annex 5; Section A.5.1. The most sensitive effect reported was self-perceived sensory eye irritation, with a NOAEC of 615 µg/m^3 and a LOAEC of 1,230 µg/m^3. The recommended short-term (1-hour averaged) guideline value was **123 µg/m^3**, which was one-tenth of the lowest concentration at which eye irritation was reported by Kulle *et al*. (1987) and Kulle (1993) (Health Canada, 2005).

4.2.4.2.2 Long-term value

Health Canada concluded that epidemiological studies on the effects of long-term exposure to formaldehyde reported respiratory and allergic effects at concentrations below the short-term guideline value. Health Canada considered that an association between low-level exposure to formaldehyde and the development of allergic sensitisation and/or asthma was biologically plausible as it was consistent with

observations in animals: formaldehyde enhanced allergic sensitisation to ovalbumin in mice and guinea pigs.

The long-term guideline value was based on the Rumchev *et al*. (2002) study, a case-control study on childhood asthma that reported an association between formaldehyde exposure and hospitalisation for asthma. Health Canada considered it to be the most suitable study because of the observed concentration-response relationship and the study design (including the control of confounding variables). One limitation noted was the retrospective design: formaldehyde levels in air were measured after onset of symptoms in cases and after the assessment of health status of cases and controls. See the WHO and UBA sections below for further critique of this study. No effects were found in children exposed to 10 to 29 µg/m^3 and 30 to 49 µg/m^3 formaldehyde, a non-significant increase of risk was observed at 50 to 59 µg/m^3 and a significantly increased risk was observed at 60 µg/m^3. Health Canada concluded that long-term exposure to formaldehyde levels below 50 µg/m^3 appeared not to be associated with adverse effects.

Therefore, Health Canada's long-term (8-hour averaged) guideline value was **50 µg/m^3**.

4.2.4.3 The Office of Environmental Health Hazard Assessment (OEHHA) Reference Exposure Levels, 2008

The California Office of Environmental Health Hazard Assessment (OEHHA) is the responsible department within the California Environmental Protection Agency for evaluating health risks related to chemical contaminants. The OEHHA sets health-based exposure levels (reference exposure levels, or RELs) for health conditions other than cancer (OEHHA, 2008). RELs are set for short-term (acute) exposures of one hour or less, 8-hour exposures and long-term (chronic) exposures of between one year to a life-time.

OEHHA concluded that the non-cancer adverse health effects of formaldehyde were largely a manifestation of its irritation of mucous membranes.

4.2.4.3.1 Short-term value

The critical effect that formed the basis of the acute REL was mild to moderate eye irritation.

OEHHA selected the controlled human exposure study originally conducted by Kulle *et al*. (1987), later re-examined by Kulle (1993), as the critical study to derive the acute REL as it considered symptomatic sensory eye irritation, one of the most sensitive effects following acute inhalation exposure to formaldehyde. The eye irritation data were suitable for benchmark concentration (BMC) modelling, which is the OEHHA's preferred method for the derivation of a PoD. A $BMCL_{05}$ for eye irritation of 530 µg/m^3 was derived as the PoD by OEHHA. The $BMCL_{05}$ is defined as the 95% lower confidence limit of the concentration expected to produce a response rate of 5%.

OEHHA noted that the eye irritation was a function of formaldehyde concentration rather than exposure duration; therefore, a time correction factor was not applied. As sensory irritation was not expected to involve large toxicokinetic differences amongst individuals, OEHHA applied an AF of 1 for this consideration. With regards to toxicodynamic differences, OEHHA noted that the respiratory irritant effect of formaldehyde had the potential to exacerbate asthma and differentially impact infants and children compared with adults. OEHHA also considered that formaldehyde might exacerbate the immune response to aeroallergens, which would be of particular concern during the development of the lungs. Therefore, OEHHA applied an AF of 10 for intraspecies toxicodynamic differences. The acute REL was therefore **55 µg/m^3** (OEHHA, 2008).

4.2.4.3.2 8-hour REL

The critical effects that encompassed the 8-hour and chronic RELs comprised nasal obstruction and discomfort, lower airway discomfort and eye irritation.

OEHHA selected an occupational study by Wilhelmssen and Holmström (1992) as the critical study to derive the 8-hour REL. A NOAEC (90 µg/m^3 exposure of the office-worker control population) and LOAEC (260 µg/m^3 exposure of the chemical-plant workers) could be derived from the study, but no other concentration-response information was provided. As the study included only adults, an intraspecies AF of 10 for toxicodynamic variability and developmental susceptibility was applied to give an 8-hour REL of **9 µg/m^3**.

4.2.4.3.3 Chronic REL

OEHHA selected the occupational study by Wilhelmssen and Holmström (1992) as the critical study to derive the chronic REL because it investigated long-term exposure to formaldehyde relatively free of other confounding exposures. As in the 8-hour REL calculation, an AF of 10 was applied to the NOAEC to give a chronic REL of **9 µg/m^3**.

4.2.4.4 World Health Organization (WHO) Guidelines for Indoor Air Quality, Formaldehyde, 2010

The WHO published guidelines for indoor air quality for several pollutants, including formaldehyde, in 2010.

The WHO compiled and reviewed published papers and reports that addressed human exposure and epidemiology, children, animal studies, cell studies and wood dust. More than 120 articles were evaluated in detail (their relevance is discussed by Nielsen and Wolkoff, 2010), but only those that contributed directly to the derivation of the indoor air guidelines were included in the WHO report.

4.2.4.4.1 Human data

The WHO considered that studies in both occupationally exposed workers and controlled human exposures in volunteers clearly demonstrated irritation effects to the

airways, with effects on the eyes noted as being the most sensitive response. Whilst the ATSDR (1999) used data from Holmström *et al*. (1989) to derive guideline values, the WHO considered that this study was not suitable for risk assessment owing to the lack of an exposure-dependent effect in histopathology.

As described previously (Section 4.2.3), the WHO considered the odour thresholds for formaldehyde when reviewing available human studies, given that odour perception can increase the reporting of subjective effects (WHO, 2010). From the evidence it assessed, the WHO concluded that there was no indication that exposures longer than four hours would increase the irritant response or the sensitivity to formaldehyde, because its chemical reaction on the receptor responsible for irritation and inflammatory responses to irritant chemicals is reversible. It also stated that there was no indication of sensitisation by exposure to formaldehyde. The WHO reported a lack of evidence to suggest there was an increased sensitivity to formaldehyde-induced sensory irritation in vulnerable groups, e.g., asthmatics, children and the elderly. However, it noted that people with a personal trait of negative affectivity (e.g., anxiety) might report more symptoms.

One of the epidemiology studies reviewed by WHO was Rumchev *et al*. (2002), which was used by Health Canada to set a long-term guideline value. WHO considered that limitations of this study included potential bias from, for example, gas heating and new materials in the homes, and the difficulty of diagnosing asthma in children. However, the WHO considered a major flaw was the presence of combustion products, as indicated by reported high concentrations in the homes of traffic pollutants that are associated with asthma in children (WHO, 2010).

In summary, the WHO reported that ‘consistent cause-effect and dose-response relationships between formaldehyde and measurable lung effects have not been found in controlled human exposure studies and epidemiological studies below 1 mg/m^3 [1000 µg/m^3]. In general, associations between formaldehyde and lung effects or sensitization in children in homes and schools have not been convincing owing to confounding factors and chance effects [references cited: Liteplo *et al*., 2002; Paustenbach *et al*., 1997; Dales *et al*., 2004]’.

4.2.4.4.2 Animal data

The WHO evaluated animal studies that informed on the carcinogenicity of formaldehyde via inhalation exposure and oral exposure in rats and mice. The study by Rusch *et al*. (1983) in monkeys, was reviewed by the WHO (Nielsen and Wolkoff, 2010) but, as it was not directly used to derive the air quality guidelines, it was not cited in the report. Rusch *et al.* (1983) identified a NOAEC of 1.35 mg/m^3 [1350 µg/m^3], which was slightly higher than the NOAEC of 1.25 mg/m^3 [1250 µg/m^3] in a rat study (with larger group sizes) that the WHO used to derive a long-term value (see below).

4.2.4.4.3 Short-term value

The WHO considered the controlled human exposure study by Lang *et al*. (2008) to be the critical study from which to derive a PoD for formaldehyde inhalation. This was a double-blind, randomised study in which 21 volunteers were subject to 10 exposures of 4 hours each in a controlled chamber, with formaldehyde concentrations ranging from 184 – 737 µg/m^3 over 10 consecutive working days. Peak exposures were included in the study design. Alongside a questionnaire, objective measures were used to evaluate eye and airway irritation and lung function, and adjustments were made for the personal trait of negative affectivity (e.g., anxiety). Lang *et al.* (2008) concluded that the lowest observed effect level (LOEL) was 630 µg/m^3 for four hours without peak exposure for sensory irritation of the eyes (the objective measures of EBF and conjunctival redness). The threshold for subjective sensory irritation was 380 µg/m^3 for four hours. The authors concluded that the NOAEL for both subjective and objective eye irritation would be close to the LOEL at constant exposure, since the effects at the LOEL were weak.

The WHO noted that the LOEL of Lang *et al*. (2008) agreed with the observations of Kulle *et al.* (1987) and Kulle (1993). It also reported that sensory irritation in humans can be predicted from airway responses in mice; the WHO therefore considered that an experimental NOAEC of 380 µg/m^3 in mice (Nielsen *et al.,* 1999; cited in WHO, 2010) provided additional support to the Lang *et al.* estimate.

To derive a short-term value, the WHO adjusted the NOAEL of 630 µg/m^3 for EBF and conjunctival redness by a factor of 5; this factor was derived from the standard deviation of nasal pungency (sensory irritation) thresholds. Therefore, the value was 120 µg/m^3, which was rounded down to **100 µg/m^3**. WHO considered the value to be valid for children.

The WHO recognised that a significant proportion of the general population detects formaldehyde below this suggested short-term (30 minute) air quality guideline value (WHO, 2010).

4.2.4.4.4 Long-term value

The WHO also acknowledged that formaldehyde inhalation causes nasal cancer in experimental animals, with a non-linear dose-response relationship. For long-term effects, WHO identified a NOAEC of 1.25 mg/m^3 [1250 µg/m^3] for increased cell proliferation in the nasal mucosa of rats after long-term exposures (Woutersen *et al*., 1989; Appelman *et al*., 1988). WHO then applied an inter-species assessment factor (AF) of 3 because the effect was local and directly caused by formaldehyde itself; and an intra-species AF of 2 because sensitivity differences were not seen amongst different populations (asthmatics, children and older people). The resultant long-term air quality guideline value was 0.21 mg/m^3 [210 µg/m^3]. As this was above the WHO value derived for acute sensory irritation, the WHO considered the short-term air quality guideline value to be also protective of long-term effects, including cancer.

In conclusion, the WHO used the findings of Lang *et al*. (2008; objective measures of sensory irritation) to set an air-quality guideline value of **0.1 mg/m^3 / 100 µg/m^3** (30-minute average concentration for life-long exposure). Supportive evidence was provided by several reviews that reported sensory irritation at exposure levels between 0.15 – 1.25 mg/m^3 [150 – 1250 µg/m^3]; and 12 controlled, mostly double-blind studies on respiratory effects at exposures of 0.08 – 11.2 mg/m^3 [80 – 11200 µg/m^3].

4.2.4.4.5 Review of the WHO indoor air quality guideline value

Nielsen *et al.* (2013, 2017) re-evaluated the WHO indoor air quality guideline in 2013 and then again in 2017.

Nielsen *et al*. (2013) re-affirmed the suitability of the Lang *et al*. (2008) study for assessing sensory irritation and deriving a short-term value. The authors noted that data published by Mueller *et al*. (2013) to investigate chemosensory effects in hyposensitive and hypersensitive males were in overall agreement with those of Lang *et al*. and supported that the WHO guideline was protective of all subjects. Annesi-Maesano *et al*. (2012) was included in the review, in which it was acknowledged that a cross-sectional study cannot provide causal relationships (this study found a correlation between formaldehyde exposures and rhinoconjunctivitis but not asthma).

In terms of associations of formaldehyde exposure and asthma, Nielsen *et al*. (2013) noted that exposure-response relationships had not been substantiated from lung function effects in controlled chamber studies in either healthy adults or asthmatics at concentrations below 1 mg/m^3 [1,000 µg/m^3]. Nielsen *et al*. (2013) concluded that the new epidemiology studies they assessed had mixed exposures in which the effects of formaldehyde were indistinguishable from effects of other components; and that they did not provide a convincing association between formaldehyde exposures and asthma.

In relation to cancer risk assessment, the authors evaluated new key studies and key cancer cohorts that were published after the WHO (2010) report and compared these with the guideline (Nielsen *et al*., 2013, 2017). They concluded that ‘the credibility of the WHO guideline has not been challenged by new studies’ (Nielsen *et al*., 2017).

4.2.4.5 Scientific Committee on Occupational Exposure Limits (SCOEL) Recommendation for Formaldehyde, 2016

SCOEL was an EU expert committee tasked with recommending occupational exposure limits (OELs).

Given the carcinogenic mode of action, SCOEL noted that the derivation of an OEL based on sensory irritation in humans inherently provides a broad margin of safety in comparison to the induction of URT tumours in rats. SCOEL primarily based its considerations on objective parameters for sensory irritation obtained in studies on human volunteers. It identified Lang *et al*. (2008) and Mueller *et al*. (2013) as two independent but complementary volunteer exposure studies that reported objective signs of eye and URT irritation and were suitable for the setting of an OEL. SCOEL

considered a NOAEC of 0.3 ppm [369 µg/m^3] plus peaks of 0.6 ppm [738 µg/m^3] from 62 volunteers (41 in the Mueller study, which was not available to WHO in 2010 but was considered by Nielsen *et al*. (2013) in a re-evaluation of the WHO value, and 21 in the Lang study) to be sufficiently robust for the derivation of a limit value.

An assessment factor for intra-species variability was not applied because of the irritant nature of the effect on local tissues, with SCOEL noting also that low inter-individual variation was confirmed by older studies. SCOEL also referred to Brüning *et al*. (2014), who concluded that an OEL could be based on a NOAEC from high-quality volunteer studies without an additional safety factor for intra-species variability. SCOEL compared the values obtained from the human volunteer studies with values obtained when extrapolating from animal data. Regarding local irritation effects, SCOEL noted that Brüning *et al*. proposed an inter-species extrapolation factor of 3; because of the existing models of the airway physiology and formaldehyde deposition of rats and humans, SCOEL considered this could be reduced to 2. Starting from a NOAEC of 1 ppm [1230 µg/m^3] in rats for histopathological alterations (Gelbke *et al*., 2014), which was similar to the NOAECs identified in the human volunteer studies. Therefore, SCOEL recommended an 8-hour TWA of **369 µg/m^3** and a short-term exposure level (STEL) of **738 µg/m^3**.

The SCOEL recommendation focused on effects related to workplace exposures, for which the link between formaldehyde and childhood asthma is not relevant; hence, only the following statement addresses this:

*"*Studies reporting induction of asthma have been reviewed by the Deutsche Forschungsgemeinschaft (DFG) (2014) concluding 'that FA [formaldehyde] is responsible for allergic asthmatic conditions only in very rare cases in spite of the wide range of possibilities of exposure' and that a designation as an asthma inducing agent would not be justified*."*

4.2.4.6 UBA and communication from the Committee on Indoor Air Guide Values (AIR), 2016

In 2016, the German Umweltbundesamt's (UBA's) Committee on Indoor Air Guide Values (AIR) confirmed the WHO value as an indoor air guideline value (Fromme *et al*, 2019). Also in 2016, AIR issued a communication 'on the question of the association of formaldehyde with the induction or exacerbation of asthma in children' (Ausschuss für Innenraumrichtwerte, 2016). AIR reviewed the epidemiology studies that were available at the time. In relation to some of the epidemiology studies referred to elsewhere in this report, AIR noted the following.

In addition to the limitations noted above by WHO (2010), AIR considered that the informative value of the Rumchev *et al*. (2002) case-control study was limited by the small sample size, the fact that cases and controls were not obtained at the same institution and that a diagnosis of asthma relied solely upon parental information.

In its review of the cross-sectional study of Krzyzanowski *et al*. (1990), AIR noted that a

relationship between a high prevalence of asthma in children and kitchen formaldehyde levels above 75 µg/m^3 was only applicable if the children were also exposed to second-hand smoke. Furthermore, Krzyzanowski *et al.* (1990) stated that physician-diagnosed asthma was only reported via parental questionnaire; AIR considered that this led to 'considerable uncertainty'.

As a general point, the committee noted the difficulties of diagnosing asthma in epidemiological studies in which a medical diagnosis was not obtained. AIR stated that 'the diagnosis of asthma in children under 6-8 years of age must be scrutinised particularly critically, as reversibility is especially difficult to assess in this age group. This is also applicable to wheezing in this age group'.

Overall, AIR concluded that 'there is no evidence of a conclusive link between exposure to formaldehyde in indoor air and the development or exacerbation of paediatric asthma' (Ausschuss für Innenraumrichtwerte, 2016).

4.2.4.7 ANSES, 2018

In 2018, the French ANSES re-evaluated the toxicological reference values (TRVs) for formaldehyde (ANSES, 2018). Eye irritation was chosen for the establishment of TRVs as it was the first key event and was a precursor of more serious, irreversible effects. Starting from the Lang *et al*. (2008) study and applying an AF of 3 for intra-species differences, ANSES derived a TRV by inhalation of 123 µg/m^3. Noting the concentration-dependent effect of formaldehyde for both acute and chronic irritant effects, ANSES concluded that this value was appropriate for short-term exposure for the protection of the general population for acute and chronic effects. Given this TRV and to maintain consistency with the IAQG proposed by WHO (2010), the French Air Committee proposed a short-term IAQG of **100 µg/m^3**.

With regard to respiratory sensitisation, ANSES noted that the study results were inconsistent. However, following consideration of recent literature reviews that related specifically to the indoor air of homes or occupational environments, ANSES stated that 'respiratory sensitisation caused by formaldehyde was extremely unlikely, in particular at low concentrations. In fact, the associations between formaldehyde and respiratory symptoms may have been due to the influence of co-exposure or confounding factors such as psychosocial factors.'

4.2.4.8 Public Health England (PHE) Indoor Air Quality Guidelines, 2019

In 2019, Public Health England (now the UK Health Security Agency) published indoor air quality guidelines for selected volatile organic compounds including formaldehyde. The guideline value proposed for short-term exposure to formaldehyde was the WHO indoor air quality guideline value of **100 µg/m^3** (30 minutes). In the review of available health-based guidance values (HBGVs), PHE noted that several authoritative bodies had derived long-term HBGVs for formaldehyde based on occupational studies where effects were observed at concentrations lower than the concentration the WHO value is based on and that there was also some evidence to suggest that children might be

susceptible to the effects of long-term exposure to formaldehyde. Therefore, a long-term indoor air quality guideline value of **10 µg/m^3** was proposed. This value was based on the ATSDR chronic MRL of 10 µg/m^3 (PHE, 2019; Shrubsole *et al.*, 2019), which was derived from an occupational study (Holmström *et al*., 1989; see ATSDR section above) in which histopathological changes were recorded from 295 µg/m^3. These values are used by UKHSA to assess general population exposure to formaldehyde in air.

4.2.4.9 ECHA's Committee for Risk Assessment (RAC) Opinion on Formaldehyde and Formaldehyde Releasers, 2020

RAC published its opinion on a proposal to restrict formaldehyde and formaldehyde releasers within the EU in 2020 (ECHA, 2020c). RAC applied the REACH framework for risk assessment and AFs in accordance with ECHA guidance to calculate derived no-effect levels (DNELs). DNELs are used within the UK and EU REACH frameworks to define the level of exposure above which humans should not be exposed.

RAC considered mainly controlled human exposures studies and animal data in deciding upon DNELs for various events. Regarding Lang *et al*. (2008) and Mueller *et al*. (2013), RAC considered that the small numbers of volunteers (in terms of ECHA guidance) and the high variability in EBF reduced the sensitivity of the studies to detect concentration-related effects unless they were marked. RAC also considered the exposures in these studies to be 'rather short', stating that it was not known if the threshold value for EBF would be different if exposure duration and/or frequency were increased. In terms of the animal data, RAC summarised the findings that identified a cascade of pre-cursor events in the development of malignant tumours with prolonged exposure duration, predominantly in rats. Similar effects were reported in studies in monkeys (Rusch *et al*., 1983), although RAC noted that no malignant tumours have been reported in this species (no carcinogenicity studies are available). Overall, RAC concluded that, compared with the information on sensory irritation in humans, the animal data were coherent (consistent across species) and well supported (numerous studies with different durations).

Regarding epidemiology studies, RAC briefly referred to the assessment of formaldehyde by UBA (2016) and highlighted the identified issues with the epidemiological studies (citing Krzyzanowski *et al.*, 1990), which comprised 'small sample sizes (which was much larger than in the studies by Lang *et al.* (2008) and Mueller *et al*. (2013), from implausible formaldehyde concentrations, and the fact that other substances or factors initiating asthma and asthma-like complaints were not adequately considered'. RAC reported that results derived from controlled human exposure studies as well as animal experiments supported this opinion.

In deciding the key effects for DNEL calculation, RAC acknowledged that sensory irritation is one of the most sensitive health effects observed in humans, but questioned whether EBF was an appropriate surrogate for carcinogenic effects after long-term inhalation to formaldehyde. RAC noted that there was no information on other effects

preceding (early) tumour response in humans; and that reliable sensory irritation data were not available for animals. Therefore, RAC concluded that DNELs for long-term effects identified from precursor events in the development of malignant tumours in animals were more appropriate than the derivation of acute DNELs.

4.2.4.9.1 Long-term value

As inter-species differences between humans and monkeys were thought to be minor, RAC placed more weight on the inhalation study in monkeys (Rusch *et al.,* 1983) than on the rat studies. In Rusch *et al*. (1983), metaplasia or hyperplasia was reported in 1/6 male animals at 1250 µg/m^3 and 6/6 male animals at 3700 µg/m^3. The study author identified 1,250 µg/m^3 as a NOAEC, whereas RAC identified this as a LOAEC.

Starting from the LOAEC of 1,250 µg/m^3 in monkeys, and in line with ECHA guidance, RAC corrected for exposure duration (hours/day, days/week) and applied AFs of 2.5 for inter-species differences for local, respiratory effects, 3.16 for intra-species differences in toxicodynamics and 3 for extrapolation from a LOAEC to a NOAEC. This resulted in a DNEL of 50 µg/m^3.

In terms of the rat studies, RAC noted that the NOAEC for cell proliferation (1,240 µg/m^3), the NOAEC for cytotoxicity / inflammation / hyper- / metaplasia (1,240 µg/m^3) and NOAEC for malignant tumours (2,500 µg/m^3) were consistent with those identified by WHO (2010) for long-term effects. However, RAC time-corrected the PoDs and applied different AFs from those applied by WHO to result in DNELs that ranged from 6 to 10 µg/m^3.

Overall, RAC proposed a DNEL for inhalation exposure of consumers of **50 µg/m^3**, starting from the LOAEC for cancer pre-cursor events identified in the study on monkeys (Rusch *et al*., 1983). This also took into account DNELs derived from pre-cursor events in rats, which were in a similar range. RAC considered that the use of a LOAEC instead of a NOAEC as the PoD was more robust, as it took account of remaining uncertainties in relation to dose spacing.

Following discussions of the proposed approach at RAC meetings, ECHA organised an open RAC dialogue with stakeholders to discuss the evidence. Industry proposed to use a NOAEC from the Lang study and to apply an AF of 3 – 5 for inter-individual differences in the general population, noting the WHO (2010) statement that hyper-sensitive groups had not been identified. Consensus on appropriate AFs was not reached, and RAC did not identify a reason to deviate from the default factors (ECHA, 2020c).

4.2.4.10 Environmental Protection Agency (EPA) Integrated Risk Information System (IRIS) Toxicological Review of Formaldehyde, 2024

The EPA's IRIS programme sits within the EPA's Centre for Public Health and Environmental Assessment (CPHEA) in the Office of Research and Development (ORD). The IRIS programme is intended to characterise the health hazards of

chemicals and set toxicity values (Reference Dose (RfD) or Reference Concentration (RfC) values) for health effects resulting from chronic exposure to chemicals that can be used by state or local health agencies, federal agencies, and international health organisations.

The EPA derived candidate RfCs (cRfCs) for health effects relating to formaldehyde inhalation, followed by organ- or system-specific RfCs (osRfCs) based on those cRfCs. The EPA IRIS assessment concluded an overall RfC for the non-cancer inhalation effects of formaldehyde based on these osRfCs (US EPA, IRIS, 2024).

The cRfCs of direct relevance to this report derived by the EPA were for sensory irritation, pulmonary function, respiratory tract pathology and immune effects specifically relating to allergic conditions and asthma prevalence and control.

4.2.4.10.1 Sensory irritation

The EPA identified four critical studies relevant to the derivation of PoDs for sensory irritation, including both controlled human exposure studies (Andersen and Mølhave, 1983; Kulle *et al.,* 1987; Kulle, 1993) and epidemiology studies with continuous exposures (Hanrahan *et al*., 1984; Liu *et al*., 1991).

The epidemiology data came from two complementary studies monitoring formaldehyde exposures in mobile homes (Hanrahan *et al*., 1984; Liu *et al*., 1991). Both studies were conducted in residential populations with comparable continuous exposures and were considered in the IRIS review to be of medium confidence (US EPA, IRIS, 2024). The EPA extracted the main effect data from both studies and selected a benchmark dose response (BMR) representing a 10% extra risk of eye irritation, with an assumed background prevalence of 3% in the absence of formaldehyde exposure. Subsequently, a BMC_{10} of 140.3 µg/m^3 and a $BMCL_{10}$ of 70 µg/m^3 were derived, with the latter serving as the combination PoD for both studies (US EPA, IRIS, 2024). The EPA considered the combined study population to be representative of the US general population, including a range of ages and health conditions (US EPA, IRIS, 2024).

Similarly, PoDs were determined for the two controlled human exposure studies, with both investigating a range of formaldehyde exposures and considered by the EPA to have a medium confidence rating (Andersen and Mølhave, 1983; Kulle, 1993). It is noted the most recent Kulle (1993) paper is a review of the original dataset produced by Kulle *et al.* (1987). A PoD of 440 µg/m^3 from the study by Kulle (1993) was identified as the most appropriate.

The EPA did not derive PoDs for the controlled human exposure studies conducted by Mueller *et al*. (2013) and Lang *et al*. (2008). Despite assigning these studies a high confidence, the EPA IRIS assessment noted concerns over the sample populations being only healthy volunteers, the large inter-individual variabilities hindering adversity cut-offs, and a lack of a clear dose-response trend. The Human Studies Review Board

(US EPA, HSRB, 2023; Goyak and Holm, 2024) provided commentary on the use of controlled chamber studies for quantitative PoD derivation for sensory irritation.

4.2.4.10.2 Pulmonary function

The EPA focused on studies in which exposure settings were most relevant to the derivation of an RfC for chronic exposures (US EPA, IRIS, 2024). Most of these studies comprised occupational exposures, residential or school exposures (adults and children), and medical students' exposure during anatomy classes. Controlled human exposure studies with short-term exposures were determined not to provide clear effects on pulmonary function (US EPA, IRIS, 2024).

The EPA concluded that most epidemiological data provided evidence that long-term formaldehyde exposures were associated with declining pulmonary function.

The EPA considered the study by Krzyzanowski *et al*. (1990) to have the most comprehensive exposure assessment protocol, with repeated PEFR (Peak Expiratory Flow Rate) measurements across a single day. Declines in PEFR were associated with increases in the two-week average indoor formaldehyde concentrations measured, and more notable declines were observed in children (5 – 15) than adults. From this study, a benchmark concentration at 10% increase in prevalence over background (BMC_{10}) was determined. A BMR of 10% reduction in PEFR was used as a cut-off for adversity, based on rationales articulated by the American Thoracic Society (US EPA, IRIS, 2024). As cited in the EPA IRIS report, the American Thoracic Society recommends that "a small transient loss of lung function, by itself, should not automatically be designated as adverse". A 10% functional reduction (from any air pollutant, not specifically formaldehyde) was on the border between mild and moderate adversity at a population level without necessarily "bringing any individual child to a level that is associated with clinically relevant consequences". The EPA also cited the evaluation of the clinical significance of small average declines in pulmonary function across a population, conducted by the American Thoracic Society. Their conclusion was summarised by the EPA as follows: "although the magnitude of the observed declines may not be clinically relevant to an individual, a shift in the population distribution toward lower pulmonary function, assuming the association is causal, may have a large impact on public health" (US EPA, IRIS, 2024). The $BMCL_{10}$ of 21 µg/m^3 was taken as the critical PoD, based on declines in PEFR in children. An AF of 3 for intra-species variability was applied to derive a **cRfC value of 7 µg/m^3**. EPA considered this cRfC to be protective for asthmatic children and other susceptible individuals under continuous inhalation exposure (US EPA, IRIS, 2024). The value was also taken forward as the osRfC for pulmonary function.

Peer review was conducted for the IRIS draft published in 2022; the TSCA Science Advisory Committee on Chemicals (SACC, 2024) provided commentary on the epidemiology data included in EPA (2024). Since human data (including from sensitive populations, i.e., asthmatic children) were used, there were different opinions amongst the SACC membership about appropriate UFs: some committee members considered

that an AF of 3 was appropriate, whilst others believed no AF was needed (SACC, 2024).

4.2.4.10.3 Allergic conditions and asthma

The EPA relied primarily on epidemiology studies in which formaldehyde concentrations were recorded in residential or school settings. The EPA concluded that this dataset provided evidence of an association between formaldehyde concentrations of 40 – 60 µg/m^3 and an increased prevalence of rhinitis or rhinoconjunctivitis, and supported an association between the prevalence of current asthma and formaldehyde exposures >50 µg/m^3 (US EPA, IRIS, 2024).

With respect to allergy-related conditions, e.g., rhinitis or rhinoconjunctivitis, the EPA derived a LOAEC of 40 µg/m^3 and a NOAEC of 24 µg/m^3 for rhinoconjunctivitis prevalence in children from the epidemiology study of Annesi-Maesano *et al*. (2012). The NOAEC from the Annesi-Maesano study was taken forward to derive a cRfC value of 8 µg/m^3 for rhinoconjunctivitis, applying an AF of 3 (US EPA, IRIS, 2024).

The same NOAEC was identified for the risk of current asthma from Annesi-Maesano *et al*. (2012). A NOAEC of 62 µg/m^3 for an increased prevalence of asthma in children was identified from Krzyzanowski *et al*. (1990), which EPA rounded down to 60 µg/m^3. Similarly, a NOAEC of 27 µg/m^3 for degree of asthma control in children was derived by Venn *et al*. (2003). A $BMCL_5$ of 13 µg/m^3 associated with a 5% increase in prevalence of symptoms above that observed in a referent group was calculated.

Application of AFs to the above PoDs (3 for Annesi-Maesano and Venn, 10 for Krzyzanowski) led to cRfC values of 10 µg/m^3, 6 µg/m^3 and 4 µg/m^3. The median cRfC was selected from these three values to give an osRfC for current asthma prevalence of **6 µg/m^3** (Krzyzanowski *et al*., 1990).

4.2.4.10.4 Overall Conclusions

An overall RfC value was selected from the derived osRfCs to reflect continuous exposure within the human population, including potentially sensitive sub-groups, that was likely to be without an appreciable risk of deleterious effects during a lifetime.

The overall RfC for formaldehyde inhalation was selected as 7 µg/m^3, being the median of osRfCs derived for respiratory system-related effects: current asthma prevalence or degree of control at 6 µg/m^3, pulmonary function at 7 µg/m^3, and allergic conditions at 8 µg/m.

The EPA assessment noted the level of conservatism in the approach, with the RfC being without appreciable risk over a lifetime and considered protective for susceptible individuals. It was also noted that the PoDs derived from the available studies were not observed until formaldehyde concentrations reached well above estimated median US indoor air concentrations, noting effects were generally observed at or above 33 µg/m^3 (US EPA, IRIS, 2024).

4.2.4.11 US EPA, Office of Chemical Safety and Environmental Protection Agency Pollution Prevention Human Health Hazard Assessment for Formaldehyde, 2024

In 2024, the EPA has evaluated the risks posed by formaldehyde exposures under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA) and the Toxic Substances Control Act (TSCA). At the time this Agency Technical Report was finalised, the EPA was consulting on an updated draft risk calculation memorandum for formaldehyde [open for public comment until 2 February 2026] (EPA, 2025). In this memorandum, EPA proposed that 'the best available science supports using sensory irritation as the most sensitive endpoint for determining human health effects from inhalation exposures. Managing risks from acute sensory irritation will be protective against other health effects, including cancer.' Therefore, the text below should be read in light of this development.

The 2024 evaluation was a collaborative effort between the Office of Pesticide Programs (OPP) and the Office of Pollution Prevention and Toxics (OPPT), both of which are part of the Office of Chemical Safety and Pollution Prevention (OCSPP). As a result of this collaborative work, multiple federal advisory committees were involved in peer review of the hazard characterisation of formaldehyde, including the National Academies of Sciences, Engineering, and Medicine (NASEM), TSCA Science Advisory Committee on Chemicals (SACC), and the Human Studies Review Board (HSRB) (US EPA, 2024c).

The Office of Pesticide Programs (OPP) and the Office of Pollution Prevention (OPPT) rely on the US EPA IRIS assessment to identify chronic hazards relevant for inhalation exposures to formaldehyde. The OPP and OPPT offices reviewed the available data and identified endpoints and hazard values for dermal, oral, and acute inhalation exposure to formaldehyde.

4.2.4.11.1 Acute Exposures

The OPP and OPPT considered sensory irritation to be the most sensitive endpoint resulting from acute exposures. Therefore, sensory irritation was selected for the basis of acute PoD derivation, as was supported by the HSRB and SACC peer reviews (see Annex 5).

For sensory irritation, PoDs were selected from controlled human exposure studies: Kulle (1993), Lang *et al*. (2008) and Mueller *et al*. (2013). The OPP and OPPT risk evaluation identified NOAECs of 0.5 ppm [620 µg/m^3] from the Kulle and Lang studies and a LOAEC of 0.3 ppm [369 µg/m^3] across 4 hours with a peak at 0.6 ppm [370/740 µg/m^3] for the Mueller study. For all PoDs an AF of 3 was applied to account for inter-individual variation, since these studies were conducted in healthy adult populations. Even though the Mueller study included hypersensitive individuals, the OPP and OPPT applied the same AF because they considered the study population was not representative of the health of the general population. A margin-of-exposure approach was used to estimate acute inhalation risk using the AF of 3 as the benchmark margin.

4.2.4.11.2 Chronic Exposures

The OPP and OPPT risk evaluation used a margin of exposure approach to estimate long-term non-cancer inhalation risk. To align with the IRIS assessment, the OPP and OPPT evaluation used the PoD derived from the draft IRIS RfC, with an AF of 3. The specific $BMDL_{10}$ value (0.021 mg/m^3) was based on reduced pulmonary function in children in the Krzyzanowski study, but was noted to be consistent with the draft RfC derived by IRIS from several studies of effects on the respiratory system.

The SACC had raised concerns in response to the IRIS draft opinion; these primarily centred around the use of epidemiology studies to derive RfC values, and the limited weight of evidence for a causal link between formaldehyde exposures and outcomes other than sensory irritation. For this reason, the SACC recommended that sensory irritation be selected as the most sensitive endpoint. Whilst acknowledging these concerns, the OPP and OPPT aligned with the IRIS evaluation.

4.2.5 Points of departure (PoDs) and health-based guidance values (HBGVs) derived by expert groups

Table 16: Points of departure (PoDs) and health-based guidance values (HBGVs) for formaldehyde proposed by expert groups

Expert Group	Study Type	PoD ($\mu g/m^3$)	Key Effects	Uncertainty Factor	HBGV ($\mu g/m^3$)
ATSDR, 1999	Controlled Human Exposure (Pazdrak *et al.*, 1993)	490 LOAEC	Nasal and eye irritation, changes in nasal lavage fluid.	9 (3 LOAEL, 3 human variability)	50 Acute MRL
	Animal studies (WoE) (Rusch *et al.*, 1983	1210 [0.98 ppm] NOAEC	Nasopharyngeal irritation and lesions in the nasal epithelium.	30 (3 interspecies, 10 human variability)	40 Intermediate MRL
	Occupational Exposure Study (Holmström *et al.*, 1989)	295 [0.24 ppm] LOAEC	Mild irritation of the eyes and upper respiratory tract and mild damage to the nasal epithelium.	30 (3 LOAEL, 10 human variability)	10 Chronic MRL
Health Canada, 2005	Controlled human exposure study (Kulle *et al.*, 1987) (Kulle, 1993)	1230 (LOAEC)	Sensory eye irritation.	10	123 Short-term guideline (1-hour average)
	Epidemiology study	50	Asthma in children.	-	50 Long-term guideline (8-

	(Rumchev *et al.*, 2002)	NOAEC			hour average)
OEHHA, 2008	Controlled human exposure study (Kulle *et al*., 1987) (Kulle, 1993)	530 $BMCL_{05}$	Sensory eye irritation.	10 (10 to account for the potential for asthma exacerbation in children)	55 Acute REL
	Occupational study (Wilhelmssen and Holmström, 1992)	90 NOAEC	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation.	10 (10 to account for the potential for asthma exacerbation in children)	9 8-hour REL
	Occupational study (Wilhelmssen and Holmström, 1992)	90 NOAEC	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation.	10 (10 to account for the potential for asthma exacerbation in children)	9 Chronic REL
WHO, 2010	Controlled human exposure	630 NOAEC	Objective measures of sensory irritation (conjunctival redness,	5 (derived from	100 (30-minute average,

	(Lang *et al.*, 2008)		EBF).	standard deviation of nasal pungency thresholds)	protective of long-term exposures)
SCOEL, 2016	Controlled chamber studies of Lang *et al*. (2008), Mueller *et al*. (2013)	369 with peaks of 738 NOAECs	Objective measures of sensory irritation on eyes and URT.	-	369 OELs of 8-hour TWA 738 STEL
UBA, 2016	Controlled human exposure (Lang *et al.*, 2008)	630 NOAEC	Objective measures of sensory irritation (conjunctival redness, EBF).	5 (derived from standard deviation of nasal pungency thresholds)	100
ANSES, 2018	Controlled human exposure (Lang *et al.*, 2008)	630 NOAEC	Objective measures of sensory irritation (conjunctival redness, EBF).	3 (intra-species difference)	Derived a TRV of 123; Air Committee adopted WHO value of 100
PHE, 2019	Controlled human exposure (Lang *et al.*, 2008)	630 NOAEC	Objective measures of sensory irritation, primarily of the eyes.	5 (derived from standard deviation of nasal pungency	Adopted WHO value of 100 as short-term value (30-minute average)

				thresholds	
	Occupational Exposure Study (Holmström *et al*., 1989)	260 [0.24 ppm] LOAEC	Mild irritation of the eyes and upper respiratory tract and mild damage to the nasal epithelium.	30 (3 LOAEL, 10 human variability)	Adopted ATSDR chronic MRL of 10 as long-term value
ECHA, 2020c	Animal studies (monkey) (Rusch *et al.,* 1983)	1250 LOAEC	Hyperplasia and metaplasia in the nasal epithelium.	23.7 (2.5 and 3.16 for inter and intra species differences, 3 for conversion of LOAEC to NOAEC)	50 DNEL (long-term) for metaplasia/hyperplasia
US EPA, IRIS, 2024	Epidemiology study (Hanrahan *et al.,* 1984) (*Liu et al.,* 1991)	70 $BMCL_{10}$	Eye irritation symptoms (sensory irritation).	3 Intraspecies variability	20 cRfC
	Controlled human exposure (Kulle *et al.,* 1987)	440 $BMCL_{10}$ (99^{th}	Eye irritation symptoms (sensory irritation).	10 Intraspecies variability	40 cRfC

	(Kulle, 1993)	percentile)			
	Controlled human exposure (Andersen and Mølhave, 1983)	120 $BMCL_{10}$ (99^{th} percentile)	Eye irritation symptoms (sensory irritation).	10 Intraspecies variability	10 cRfC
	Epidemiology study (Krzyzanowski *et al.,* 1990)	21 $BMCL_{10}$	Decreased Peak Expiratory Flow rate (PEFR) in children (Pulmonary function).	3 Intraspecies variability	7 cRfC
	Epidemiology study (Annesi-Maesano *et al.,* 2012)	24 NOAEC	Rhinoconjunctivitis prevalence in children (allergic conditions).	3 Intraspecies variability	8 cRfC
	Epidemiology study (Matsunaga *et al.*, 2008)	46 NOAEC	Allergy-related condition -atopic eczema prevalence (allergic conditions).	10 Intraspecies variability 3 and sub-chronic to chronic 3	5 cRfC

	Epidemiology study (Krzyzanowski *et al.,* 1990)	62 (NOAEC)	Asthma prevalence in children, specifically current asthma symptoms or degree of asthma control.	10 Intraspecies variability	6 cRfC
	Epidemiology study (Venn *et al.,* 2003)	13 $BMCL_5$	Asthma control in children with asthma.	3 Intraspecies variability	4 cRfC
	Epidemiology study (Annesi-Maesano *et al*., 2012)	42 NOAEC	Current prevalence of asthma in children.	3 Intraspecies variability	10 cRfC
US EPA, 2024c (OPP and OPPT)	Controlled human exposure (Kulle *et al.,* 1987) (Kulle, 1993)	620 NOAEC	Eye irritation symptoms (sensory irritation).	3 Intraspecies variability	-
	Controlled human exposure (Lang *et al*., 2008)	620 NOAEC	Eye irritation symptoms (sensory irritation).	3 Intraspecies variability	-
	Controlled chamber (Mueller *et al*., 2013)	370 / 740 LOAEC (4 hours with peak)	Eye irritation symptoms (sensory irritation).	3 Intraspecies variability	-

	Epidemiology study (Kryzanowski *et al.*, 1990)	21 $BMDL_{10}$	Asthma prevalence in children, specifically current asthma symptoms or degree of asthma control.	3 Intraspecies variability	-

4.2.6 Summary of human health impacts of formaldehyde inhalation

The toxicity of formaldehyde has been well investigated, with a large and complex dataset. Many studies are available in humans (controlled exposure studies in volunteers, occupational and general population studies) and animals.

Most inhaled formaldehyde gas is retained in the upper airways; very little penetrates to the deep lung or lower airways. Formaldehyde that is adsorbed to particulates might be deposited deeper in the respiratory tract. Inhaled formaldehyde is not expected to be systemically available to a great extent at concentrations relevant to indoor, non-occupational settings.

Formaldehyde is a genotoxic carcinogen that causes DNA damage and proliferative regeneration following cytotoxicity. The risk of cancer associated with formaldehyde levels that do not cause irritation and inflammatory responses appears to be negligible, as stated by, for example, Health Canada (2005), WHO (2010), SCOEL (2016) and ANSES (2018).

The critical health effects following inhalation exposure that are of most relevance to this report, which concerns indoor exposures of the general population, are sensory irritation of the eyes and upper respiratory tract and respiratory conditions. The most relevant data sources to inform on these effects are controlled-exposure studies in volunteers and non-occupational epidemiology studies. Commentary on the suitability of different study designs for identifying PoDs, and concerns about some individual studies, is provided by the Human Studies Review Board (US EPA, HSRB, 2023; Goyak and Holm, 2024) and the TSCA Science Advisory Committee on Chemicals (SACC, 2024).

Sensory irritation is generally recognised as the most sensitive acute effect of inhalation exposure. It is a concentration-dependent effect: brief exposures to a set concentration can elicit the same intensity of responses as longer exposures to the same concentration. Sensory irritation of the eyes and URT is a plausible consequence of exposure to formaldehyde gas. All expert groups identified sensory irritation as the key effect for the derivation of acute guideline values. There are varying expert views on the adversity of sensory-irritation effects, as noted for example by SACC (2024): some committee members commented that mild sensory irritation might not be an adverse effect and is reversible, whilst others considered that irritation of mucous membranes could adversely impact workers or the general population if they were hampered in the safe performance of their occupation or driving, for example. Another factor is that sensory irritation is subjective and can be influenced by odour (although some chamber studies accounted for this through the use of objective measurements and odour-masking chemicals).

The controlled-chamber studies gave largely consistent responses in terms of the reported sensory irritation and the concentrations from which this occurred. From those control-chamber studies most relied upon by expert groups, the lowest PoD of

370 µg/m^3 was identified from Mueller *et al*. (2013), which included subjects that were regarded as being hypersensitive to chemical irritation. The same PoD of 620 µg/m^3 was identified by expert groups from the other key controlled chamber studies (Kulle *et al*., 1987; Kulle, 1993; Lang *et al*., 2008), whilst that identified from Pazdrak *et al*. (1993) was 490 µg/m^3.No impact on lung function was observed in the controlled exposure studies, including those in which subjects included individuals who were asthmatic or had dermal hypersensitivity to formaldehyde (Sauder *et al*., 1987; Green *et al*., 1987; Witek *et al*., 1987). Most studies on formaldehyde-exacerbated asthma have focused on children being a sensitive group. Some observational epidemiology studies have reported an association between respiratory effects and formaldehyde exposure, although they were not able to determine causality specific to formaldehyde. Health Canada (2005) set a long-term guideline value on the basis of asthma exacerbation reported in the study of Rumchev *et al*. (2002), from which a no-effect concentration of 50 µg/m^3 was identified. The US EPA calculated a $BMCL_{10}$ of 21 µg/m^3 from Krzyzanowski *et al*. (1990) for pulmonary function and identified NOAECs ranging from 24 µg/m^3 to 62 µg/m^3 for allergic conditions and asthma from three epidemiology studies (US EPA IRIS, 2024). However, other epidemiology studies did not find respiratory effects or changes in lung function linked to formaldehyde exposure in asthmatic children (Garrett *et al*., 1999; Delfino *et al*., 2003; Tavernier *et al*., 2006), with some such studies not finding a relationship between formaldehyde levels > 100 µg/m^3 and respiratory effects (see Annex 5, Section A.5.1).

The resultant guideline values for exposures of different durations and for different purposes proposed by each expert group were influenced by various factors, as described in Section 4.2.4 above and Section 4.2.7 below.

4.2.7 Limitations and uncertainties in the hazard assessment

- Odour perception of formaldehyde can increase the reporting of subjective sensory irritation effects such as eye/nasal discomfort, olfactory symptoms, and annoyance. Odour thresholds vary for individuals depending on factors such as air purity, smoking status and previous exposure to formaldehyde. Some controlled chamber studies included objective measurements and co-exposure to odour-masking chemicals. Personality traits (anxiety, expectations) can also be a confounding factor.

- There are different opinions on the adversity of sensory irritation. Some experts consider it to be an adverse effect, whilst others regard it as a protective response to exposure to an irritant agent, but not itself adverse. How sensory irritation is interpreted can influence the choice of uncertainty factors, which then impacts the guideline value.

- The role of formaldehyde in the development of respiratory conditions, including asthma and asthma-related symptoms, is not completely understood.

- As noted in Section 3.1.1, activities that generate particles, for example combustion,

cooking, smoking or use of scented candles and incense, might result in a proportion of formaldehyde being present as particulate-adsorbed formaldehyde. Asthma is a disease of the bronchial airways, whereas rhinitis and rhinoconjunctivitis are conditions associated with the nasopharyngeal airways and eyes. Therefore, formaldehyde sources that generate particles might make a greater contribution to asthma exacerbation. This was not addressed in the available epidemiology studies referred to above; i.e., particulate-adsorbed formaldehyde was not measured in the epidemiology studies.

- The application of AFs for intra-species variability might not be needed when the study subjects comprised a sensitive population (i.e., asthmatic children). This impacts the guideline value.

- There is some uncertainty in whether adverse effects from repeated exposures are concentration-dependent or are accumulated dose-dependent effects, which has led to different approaches to the setting of short-term versus long-term guideline values. Some expert groups set separate values for short-term and long-term exposures, whilst others concluded that their short-term values, with sensory irritation as the key effect, were protective of repeated exposures; one expert group explicitly stated that the effects elicited by chronic exposures were concentration dependent.

- Uncertainties around the role of peak exposures from intermittent sources versus steady-state exposure from continuous sources complicate the ability to define dose-response relationships at the exposure levels typically found indoors in domestic settings.

5 Conclusions

5.1 Sources of formaldehyde in indoor air

There is a complex inter-relationship between factors that will contribute formaldehyde to indoor air (sources) and factors that will remove formaldehyde from indoor air (sinks). Other factors that can influence formaldehyde levels include indoor air chemistry and environmental parameters such as temperature and humidity. Depending on the combination of factors, these could increase or decrease the level of formaldehyde that is emitted from various sources. For the purposes of this report, sources have been classified into continuous or intermittent sources.

Continuous sources include building materials and articles such as furnishings, carpets, wall coverings and textiles. Often these sources have a large surface area but emissions will diminish over time as articles age.

Emissions from articles occur in two phases. Initially, emissions appear to be mainly driven by volatility and decline according to a first order exponential process whereas longer term emissions (after around 3 months) may be driven by chemical reactions (hydrolysis) with resins and naturally occurring components such as lignin in wood. This means that environmental parameters such as temperature and humidity will be a key determinant of the scale of longer-term emissions from articles. Given that chamber studies are typically conducted under a standardised set of environmental conditions, it is not possible to extrapolate quantitative emissions' data from chamber studies to emissions that might arise in domestic settings. However, data from chamber tests can be useful to rank articles according to their potential emissions. It is also relevant to note that because chamber tests are typically carried out on newly produced articles, the emissions' data measured in these tests represent a worst-case situation and will not necessarily reflect the scale of emissions that will arise when that article is in situ in someone's home.

For continuous sources, historically, engineered wood boards were a major source of emissions. However, the introduction of the E1 emission standard has resulted in voluntary actions by board producers, including most EU-based and all GB-based producers, to supply lower emitting E1 boards which generate air concentrations of ≤ 124 $\mu g/m^3$ in EN 717-1 chamber tests when new. Boards that do not meet the E1 standard could emit considerably higher concentrations. Information from the WPIF indicates that 98 – 99% of the particleboard and OSB and 83% of the MDF supplied to the GB market originates in GB or the EU and is therefore likely to meet the E1 standard. These percentages do not include the supply of articles made using engineered wood boards such as furniture. HSE does not have data on the market share for different countries of origin for these articles, thus there is uncertainty about

the proportions of high and lower emitting articles that are supplied to the GB market. Other continuous sources such as wallcoverings, mineral wool insulation, foams, paints and textiles appear to emit lower quantities compared with boards.

Intermittent sources are typically point sources that may emit for varying durations ranging from a few minutes to several hours. These include combustion sources (e.g., open fires, wood burners, wood stoves, ethanol fireplaces, high temperature oven cleaning, smoking and vaping, incense burners, scented candles) or use of fragranced personal and household care products. Emissions could be in the form of gaseous or particulate-adsorbed formaldehyde. Where activities generate particles, e.g. combustion, it may be the case that a greater proportion of formaldehyde is present as particulate-adsorbed formaldehyde compared with activities that do not generate particles to the same extent. HSE has not found any experimental data that quantifies the relative amount of gaseous vs particulate-adsorbed formaldehyde from any source. It is also not clear whether the form in which formaldehyde is present in indoor air has relevance for the risk to health that may be created (this may be the case if deposition patterns in the respiratory tract change between gaseous and particulate-adsorbed formaldehyde).

For intermittent sources, combustion processes (e.g., conventional cigarettes, ethanol fireplaces, burning incense sticks, high temperature oven cleaning) generate some of the highest formaldehyde concentrations. Other intermittent sources contributing lower amounts of formaldehyde include fragranced personal and household care products, air fresheners, e-cigarettes and vapes. One study (Dimitroulopoulou *et al.*, 2015a) estimated the daily (24-hr time weighted average) exposure that would arise from typical use patterns for various lower emitting products including all-purpose cleaners, kitchen cleaners, floor cleaners, furniture polish, floor polish, combustible air fresheners, electric air fresheners and perfumes. These data indicate that depending on product use and ventilation rate, use of these household products can result in daily exposures for residents of between 3 and 8 $\mu g/m^3$.

In order to compare sources, Salthammer (2019b) estimated the concentration in air for a standard reference room for a range of continuous and intermittent sources using geometric mean values of emission rates for articles of a defined area or single units of intermittent sources and assuming an air exchange rate of 0.5 h^{-1} (Section 3.2.3.2, Figure 1). This shows that during operation, emissions from intermittent combustion sources can be an order of magnitude higher (or more in some cases) than emissions from continuous sources and household and personal care products. The impact that intermittent sources have on the overall room concentration of formaldehyde will be very dependent on the frequency and duration of operation of each intermittent source.

5.2 Measured formaldehyde concentrations in indoor air

Ambient outdoor concentrations of formaldehyde in European air range from around 1.2 – 27 $\mu g/m^3$ with a geometric mean of 4.3 $\mu g/m^3$. The higher concentrations result

mainly from occasional photochemical reactions in the atmosphere.

Based on a systematic literature review, Clark *et al*. (2023) calculated that the average formaldehyde concentrations in homes in England over the last 25 years ranged between 11 – 49 µg/m^3, maxima ranged from 32 – 187 µg/m^3. Pooling data to obtain a weighted distribution resulted in a geometric mean (GM) of 22.8 µg/m^3 (geometric standard deviation: 2.0 µg/m^3) and 5th and 95th percentiles at 6.5 µg/m^3 and 58.7 µg/m^3, assuming a log-normal distribution. These levels are similar to GMs from pooled studies where indoor monitoring was conducted pre-1998, suggesting little change over the past 25 years. Although Clark *et al.* did not have data for homes in Scotland and Wales, there is no reason to consider that levels in homes in these regions will be significantly different from homes in England.

The currently measured formaldehyde concentrations in English homes are in line with an average of 22 µg/m^3 calculated from data gathered 15 – 20 years ago in the EU AIRMEX and INDEX studies and the GM concentration of 22.2 µg/m^3 reported by Birmili *et al*. (2021) from samples collected in Germany between 2014 – 17. Results from studies carried out in Canada since the early 1990s consistently indicate that formaldehyde concentrations in Canadian homes range between 2.5 and 88 µg/m^3 with an average between 30 and 40 µg/m^3 (Health Canada, 2005). Data collected between 2019/20 in the US indicate that concentrations of formaldehyde may range from 0.27 to 124.2 µg/m^3 (median 19.77 µg/m^3), with 90 percent of homes having concentrations below 41.8 µg/m^3 (US EPA, 2024a). Based on these data, HSE concludes that formaldehyde levels in English (and by extrapolation GB homes) are equivalent to levels found in other high-income countries around the world.

It is noteworthy that in a study of 10 English homes built before 2014 that was conducted to assess the impact of the building regulations, arithmetic mean (AM) formaldehyde concentrations of 34 µg/m^3 (living rooms) and 40 µg/m^3 (bedrooms) were found. Also, a study conducted in Northern Ireland revealed levels up to 1,400 µg/m^3 in three houses constructed to meet Passivhaus standards. This suggests that steps that are taken to improve air tightness and hence the energy efficiency of homes might result in poorer indoor air quality.

Data sets for some indoor air monitoring studies were large enough to investigate the impact of different types of sources on indoor air concentrations. For homes in England, Berry *et al*. (1996) and Raw *et al*. (2004) found that the age of home had a significant impact of indoor formaldehyde levels, with homes built after 1982 having around three times the level of indoor formaldehyde compared with homes built before 1919. This is likely to be the result of changes in building practices (greater use of engineered wood boards and requirements to increase the air tightness of buildings), introduced between the 1970s and 1990s. Similar changes in indoor formaldehyde levels with building age were reported in German studies.

Other factors that contributed to higher indoor formaldehyde levels in GB and German

studies included:

- rural location compared with suburban or urban location;
- detached homes compared with semi-detached, terrace, or flat;
- integral garage compared with detached garage;
- new particleboard furniture and/or flooring;
- frequent use of sanitary cleaners/disinfectants.

The difference for each factor was around 10 – 20 µg/m^3.

Based on data published by Salthammer (2019a), Birmili *et al*. (2021) concluded that:

"Ignoring wood-based materials as a source and taking sinks into account, indoor formaldehyde concentrations in order of 10 – 15 µg/m^3 are expected. We estimate that the inclusion of wood-based materials would roughly double these values."

Birmili *et al*. (2021) also notes that:

"Measurements in a test cabin built from "formaldehyde-free" materials yielded, at an air exchange rate of 0.3 h^{-1}, room concentrations of formaldehyde between 15 and 20 µg/m^3."

These levels are within the range of averages recorded in studies of GB homes.

5.3 Health effects of exposure to formaldehyde in domestic settings

A sensitive response on exposure to formaldehyde in air is sensory irritation, characterised as unpleasant sensations or discomfort in the eyes and nasal passages. Sensory irritation is a non-immunologically driven response, the symptoms of which occur immediately on exposure and which readily resolve when exposure stops; there are different views on whether it reflects an adverse effect, or is a protective response to exposure to an irritant agent. Symptoms of sensory irritation can appear to be similar to asthma-type symptoms. Currently it is not clear if, in addition to sensory irritation, formaldehyde can induce immunologically-mediated responses in the respiratory tract. Although formaldehyde has been identified as a skin sensitiser, there is no evidence that inhalation of formaldehyde triggers the type of IgE-mediated reaction that is typically associated with immunological asthma. This does not exclude the possibility that an immunologically-mediated response might be triggered in the respiratory tract via an alternative mechanism that has yet to be fully understood.

It can be difficult to determine with precision the concentrations in air that trigger sensory irritation. Concentrations of formaldehyde that induce sensory irritation fall within the odour threshold range, and perception of odour can increase the subjective

reporting of sensory irritation effects. For this reason, data from human volunteer studies where participants are exposed under defined conditions can provide a better indication of concentrations triggering sensory irritation compared with larger-scale epidemiological studies conducted in the general population or workplace-exposed individuals where exposure concentrations are less precisely characterised. In controlled chamber studies in human volunteers, the concentration of formaldehyde at which there was no or minimal observed sensory irritation ranged from 370 $\mu g/m^3$ to 620 $\mu g/m^3$.

Some observational epidemiology studies have reported an association between indoor formaldehyde exposure and worsening or increased prevalence of respiratory conditions in children, existing asthmatics and those with impaired lung function. These conditions have included reduced pulmonary function and exacerbation of asthma and other respiratory allergic conditions. However, inconsistent findings are reported and there are differing views on the susceptibility of children to effects of formaldehyde on asthma.

PoDs (NOAECs / BMCs) identified by some expert groups for effects on pulmonary function, allergic conditions and asthma have ranged from 21 $\mu g/m^3$ to 62 $\mu g/m^3$, although such effects were not reported in other epidemiology studies, including ones in which the formaldehyde levels exceeded 100 $\mu g/m^3$.

In addition to respiratory effects, formaldehyde is a known genotoxic carcinogen. *In vivo* data from experimental animals confirms a link between site-of-contact nasal tumours and long-term inhalation exposure. The underlying mode of action is thought to be cell proliferation caused by repeated cycles of cell damage and repair arising from chronic irritation. This in turn leads to hyperplasia or metaplasia and ultimately the development of tumours. As such, this is a local threshold effect. Irritation is a concentration-dependent effect. Exposure to concentrations of formaldehyde that are below the irritant threshold would not cause the initial cell damage that is the precursor event to tumour development. It is also suspected that because intracellular levels of formaldehyde are dominated by endogenous compound, protective DNA repair mechanisms are in place to manage tissue damage. This view is supported by the low incidence of nasal tumours reported in people occupationally exposed to formaldehyde. For these reasons, HSE concludes that tumour development is not likely to arise where exposures are below the threshold for irritation, and the key effects relevant to this report are sensory irritation of the eyes and upper respiratory tract, and respiratory symptoms.

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Further information

This document is available at: https://www.hse.gov.uk/reach/rmoa.htm.

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Annex 1 – Literature search

A literature search using the Proquest platform was carried out in summer 2023 for the period 2018 onwards. The following search terms were used:

Hazard

"Formaldehyde" or "formaldehyde releasing substances" or "formaldehyde releasers" or "formaldehyde releasing substance" or "formaldehyde releaser" or "200-001-8" or "50-00-0" or "methanal" or "formalin" or "paraformaldehyde" or "formol" or "methylene oxide" or "oxomethane" or "paraform" or "oxymethylene" or "formic aldehyde" or "CH2O"

and

irritat* or irritant* or inflam* or dermat* or sensiti* or RADS* or hypersensiti* or breath* or respir* or inhale* or inhalation or allerg* or toxic* or intoxic* or poison* or disease* or illness* or morbid* or mortalit* or neurodegen* or neurotoxic* or neurobehavio* or "nervous system*" or neuropatholog* or brain or derma* or cancer* or carcinogen* or carcinoma* or reproduct* or reprotox* or fertilit* or mutagen* or mutat*or genotoxic* or gene or genes or genetic* or immunotoxic* or immune* or immuni* or hepato* or nephro* or terato* or cell or cells or cytotox* or metabolis* or EDC* or endocrin* or "endocrine disrupt* or skin or absorb* or uptake or transfer or DNA or adduct or crosslink* or nasal or cytotox* or tumour or naso* or pharyn* or eye* or blephar* or * or asthma* or pulmon*

Exposure

"Formaldehyde" or "formaldehyde releasing substances" or "formaldehyde releasers" or "formaldehyde releasing substance" or "formaldehyde releaser" or "200-001-8" or "50-00-0" or "methanal" or "formalin" or "paraformaldehyde" or "formol" or "methylene oxide" or "oxomethane" or "paraform" or "oxymethylene" or "formic aldehyde" or "CH2O"

and

expose* or exposure* or exposing* or consumer* or domestic or monitor* or biological or surveillance or occupation* or paraoccupation* or air or indoor or interior or "aircraft" or cabin or marine or vessel or automo* or car or vehicle or construction or insulation or "wood-based plate materials" or laminat* or "mineral wool" or "Urea Formaldehyde" or "68002-18-6" or "614-201-1" or UF or "Phenol Formaldehyde" or "68610-07-1" or "614-660-8" or PF or plywood or fibreboard or particleboard or MDF or fibreglass or foam *adhesive* or sealant* or paints or lacquer or polish or wax or wallpaper or furniture or mattress* or curtain or textile or carpet or wash* or toy or childcare or mouth* or chew* or clean* or laundry or cosmet* or "personal care" or "air cleaning" or candle or "air freshener" or "air fragrance" or incense or "wood burning" or "ethanol fireplace" or stove

or cook* or tobacco or electronic or “e-cigarette” or vape* or photocopier or printer or 3D or “melamine formaldehyde” or “68002-20-0” or “614-203-2” or MF or “Methylene diphenyl diisocyanate” or “MDI” or “101-68-8” or “202-966-0” or “Polyoxymethy* or POM or “66455-31-0” or “613-936-5” or “1,4-Butanediol” or BDO or “110-63-4” or “203-786-5” or “Pentaerythritol” or Penta or “115-77-5” or “204-104-9” or epoxy or tetrahydrofuran or THF or polybutylene or PBT or alkyd or “electrical appliance”.

Annex 2 – Substances identified by ECHA as formaldehyde releasers

This table provides information on the registration status and hazard classification in GB and the EU for the formaldehyde releasers listed in Table B.2 from the Annex (ECHA, 2020b) to ECHA's restriction dossier (ECHA, 2020a). At this time, the Agency has not sought to identify potential future GB registrants from DUIN data.

Table A.2.1: Information on substance identified by ECHA as formaldehyde releasers

Substance name	CAS number	EC number	Registered under UK REACH/Number of DUINs submitted	GB Tonnage	GB Mandatory Classification	Registered in EU?	EU Tonnage	EU Harmonised Classification
7a-ethyldihydro-1H,3H,5H-oxazolo[3,4-c]oxazole	7747-35-5	231-810-4	Not registered, at least 4 DUINs submitted	N/A	N/A	Yes	100-1,000 t	N/A
1-[1,3-bis(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]-1,3-bis(hydroxymethyl)urea	78491-02-8	278-928-2	Not registered, at least 24 DUINs submitted	N/A	N/A	Yes	100-1,000 t	N/A
1,3-bis(hydroxymethyl)-5,5-dimethylimidazolidine-2,4-dione	6440-58-0	229-222-8	Not registered and at least 50 DUINS submitted	N/A	N/A	Yes	1,000-10,000 t	N/A

Substance name	CAS number	EC number	Registered under UK REACH/Number of DUINs submitted	GB Tonnage	GB Mandatory Classification	Registered in EU?	EU Tonnage	EU Harmonised Classification
N,N''-methylenebis[N'-[3-(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]urea]	39236-46-9	254-372-6	Not registered and at least 24 DUINs submitted	N/A	N/A	Yes	100-1,000 t	N/A
Methenamine (Hexamine)	100-97-0	202-905-8	Yes (4 registrations, 0 DUINs)	> 100 tpa	"Flam. Sol. 2 Skin Sens. 1"	Yes	≥ 10,000 t	"Flam. Sol. 2 Skin Sens. 1"
2,2',2''-(hexahydro-1,3,5-triazine-1,3,5-triyl)triethanol	4719-04-4	225-208-0	Yes (3 registrations, 0 DUINs)	> 1000 tpa	"Acute Tox. 4 * Skin Sens. 1"	Yes	10,000-100,000 t	"Acute Tox. 4 * Skin Sens. 1"
Dimethoxymethane	109-87-5	203-714-2	Not registered, at least 47 DUINs submitted	N/A	N/A	Yes	1,000-10,000 t	N/A
4,4-Dimethyloxazolidine ; 3,4,4-trimethyloxazolidine	81099-36-7 mixture of 51200-87-4 and 75673-43-7	257-048-2	Not registered and at least 31 DUINs submitted	N/A	N/A	Not as a mixture; individual substances: yes for 4,4-Dimethyloxazolidine, no for 3,4,4-Trimethyloxazolidine	N/A	N/A

Substance name	CAS number	EC number	Registered under UK REACH/Number of DUINs submitted	GB Tonnage	GB Mandatory Classification	Registered in EU?	EU Tonnage	EU Harmonised Classification
4-[2-(morpholin-4-ylmethyl)-2-nitro-butyl]morpholine; 4-(2-nitrobutyl)morpholine	37304-88-4; mixture of 1854-23-5 and 2224-44-4	217-450-0 218-748-3	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
2-imidazolidinone, 4,5-dihydroxy-1,3-bis(hydroxymethyl)-, methylated formaldehyde, oligomeric reaction products with 5,5-dimethyl-2,4-imidazolidinedione	68411-81-4; 26811-08-5	270-150-1; 500-052-9	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
4,5-dihydroxy-1,3-bis (hydroxymethyl)-imidazolidin-2-one; 1,3-bis(hydroxymethyl) imidazolidin-2-one	1854-26-8; 136-84-5	217-451-6; 205-264-2	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A

Substance name	CAS number	EC number	Registered under UK REACH/Number of DUINs submitted	GB Tonnage	GB Mandatory Classification	Registered in EU?	EU Tonnage	EU Harmonised Classification
Tetrahydro-1,3-bis(hydroxymethyl)-1*H*-pyrimidin-2-one; 1,3-Bis(hydroxymethyl)-1,3-diazinan-2-one	3270-74-4	221-893-5	Not registered and no DUINS submitted	N/A	N/A	No	N/A	N/A
1,3-Bis(hydroxymethyl) urea	140-95-4	205-444-0	Not registered and at least 3 DUINS submitted	N/A	N/A	Yes	1-10 t	N/A
Hexahydro-1,3,5-triethyl-1,3,5-triazine (b)	7779-27-3 (b)	231-924-4	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
4,5-Dihydroxyimidazolidin-2-one	3720-97-6	223-070-6	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
1-hydroxymethyl-5,5-dimethylhydantoin; 1-hydroxymethyl-5,5-dimethyl-imi-dazolidine-2,4-dione	116-25-6	204-132-1	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
2-Chloro-N -(hydroxymethyl)acetamide	2832-19-1	220-598-9	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
Hydroxymethylurea	1000-82-4	213-674-8	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A

Substance name	CAS number	EC number	Registered under UK REACH/Number of DUINs submitted	GB Tonnage	GB Mandatory Classification	Registered in EU?	EU Tonnage	EU Harmonised Classification
Paraformaldehyde	30525-89-4	608-494-5, 690-727-5	Not registered and at least 30 DUINs submitted	N/A	N/A	No	N/A	N/A
Polyoxymethylene melamine (INCI) or 1,3,5-Triazine-2,4,6-triamine, polymer with formaldehyde	9003-08-1	618-354-5	Not registered and at least 46 DUINs submitted	N/A	N/A	No	N/A	N/A
Polyoxymethylene urea (INCI)	9011-05-6	618-464-3	Not registered and at least 39 DUINs submitted	N/A	N/A	No	N/A	N/A
Formaldehyde dibenzyl acetal	2749-70-4	628-635-4	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
formaldehyde, reaction products with propylene glycol; Propyleneglycol hemiformal	85338-22-3	286-695-3	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
nitromethylidynetrimethanol; 2-(Hydroxymethyl)-2-nitropropane-1,3-diol	126-11-4	204-769-5	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A

Substance name	CAS number	EC number	Registered under UK REACH/Number of DUINs submitted	GB Tonnage	GB Mandatory Classification	Registered in EU?	EU Tonnage	EU Harmonised Classification
hydantoin; Imidazolidine-2,4-dione	461-72-3	207-313-3	Not registered and no DUINs submitted	N/A	N/A	Yes	N/A	N/A
(Hydroxymethyl)-5,5-dimethyl-2-4-imidazolidinedione	27636-82-4	608-120-0	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
3-(Hydroxymethyl)-5,5-dimethylimidazolidine-2,4-dione	16228-00-5	240-352-4	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
2-(Hydroxymethylamino)ethanol	34375-28-5	251-974-0	Not registered, at least 4 DUINs submitted	N/A	N/A	Yes	This substance is registered under the REACH Regulation but is not currently being manufactured in and / or imported to the European Economic Area.	N/A

Substance name	CAS number	EC number	Registered under UK REACH/Number of DUINs submitted	GB Tonnage	GB Mandatory Classification	Registered in EU?	EU Tonnage	EU Harmonised Classification
Polyoxymethilenes (POM)	66455-31-0	613-936-5	Not registered and no DUINs submitted	N/A	N/A	No	N/A	N/A
4,4'-methylenediphenyl diisocyanate (MDI)	101-68-8	202-966-0	8 registrations and at least 220 DUINs submitted	4 (1-10t), 4 (10-100t)	"Carc. 2 Acute Tox. 4 * STOT RE 2 * Eye Irrit. 2 STOT SE 3 Skin Irrit. 2 Resp. Sens. 1 Skin Sens. 1"	Yes	> 100,000 t	"Carc. 2 Acute Tox. 4 * STOT RE 2 * Eye Irrit. 2 STOT SE 3 Skin Irrit. 2 Resp. Sens. 1 Skin Sens. 1"
Butane-1,4-diol (BDO)	110-63-4	203-786-5	8 registrations and at least 181 DUINs submitted	1 (1-10t), 1 (10-100t), 3 (100-1,000t), 3 (1,000+t)	N/A	Yes	≥ 100,000 to < 1,000 000 t	No harmonised classification, a CLH proposal was submitted to ECHA in Dec 2025.
Pentaerythritol (Penta)	115-77-5	204-104-9	5 registrations and at least 341 DUINs submitted	1 (1-10t), 2 (10-100t), 1 (1,000t+), 1 without info	N/A	Yes	≥ 100,000 to < 1,000,000 t	N/A

Annex 3 Emissions and indoor air concentrations of formaldehyde from European studies (Halios *et al.*, 2022)

Table A.3.1: Formaldehyde in European residences: emissions from building and construction materials.

VOC	Study	Adhesives (µg m^{-2} h^{-1})		Ceiling (µg m^{-2} h^{-1})				
		General	Mastic (polyurethane)	General	Tiles	0 months*	6 months*	12 months*
Formaldehyde	Gunschera *et al*. (2013) **TC**				22 (20, 55) [3, 1]			
	Jarnstrom *et al*. (2007) **FLEC**					42 (5, 96) [2]	42 (14, 109) [2]	28 (13, 46) [2]
	Plaisance *et al*. (2017) **TC**		11.6 [3]					
	Plaisance *et al*. (2014b) **TC**				99 [3]			

TC: Test Cell or Chamber; **FLEC**: Field and Laboratory Emission Cell;
[1]: Median, min max; [2]: Average, min max; [3]: Sample value.
*: period after the structure was finished

Table A.3.2: Formaldehyde in European residences: emissions from building and construction materials

	Area-specific emission rate (µg m^{-2} h^{-1})							
VOC	**Study**	**Chipboard**	**Fireboard**	**Composite board**	**Finishing plaster**	**Floor covering (parquet)**		
						0 months*	**6 months***	**12 months***
Formaldehyde	Jarnstrom *et al*. (2007) **FLEC**					7 (5, 10) [2]	5 (5, 6) [2]	5 (5, 8) [2]
	Plaisance *et al*. (2017) **TC**							
	Plaisance *et al*. (2014a)	282.8 [3]			682 [3]			
	Plaisance *et al*. (2014a) **PFS**							
	Plaisance *et al*. (2014b) **TC**	224 (203, 245) [3, 1]		4 [3]	38 (26.7 413) [3, 1]			
	Simon *et al*. (2020) **TC**	83.87[3]	< 0.14 [3]					

TC: Test Cell or Chamber; **PFS**: Passive Flux Sampler; **FLEC**: Field and Laboratory Emission Cell;
[1]: Median, min max; [2]: Average, min max; [3]: Sample value.
*: period after the structure was finished

Table A.3.3: Formaldehyde in European residences: emissions from building and construction materials

VOC	Study	Floor covering				Foam	Furnishing plaster	Glass wool
		Various	PVC: 0 months	PVC: 6 months	PVC: 12 months			
Formaldehyde	Jarnstrom *et al*. (2007) **FLEC**		9 (5,18) [a, 2]	5 (5, 10) [a, 2]	7 (6, 10) [a, 2]			
	Plaisance *et al*. (2017) **TC**						17.5 [a, 3]	1.505 [a, 3]
	Plaisance *et al*. (2014b) **TC**					<3.9 [a, 3]		

TC: Test Cell or Chamber; **MEC**: Miniature Emissions Chamber; **PFS**: Passive Flux Sampler; **FLEC**: Field and Laboratory Emission Cell; [a]: µg m^{-2} h^{-1} (area-specific emission rates); [b]: µg m^{-2}; [1]: Median, min max; [2]: Average, min max; [3]: Sample value.

Table A.3.4: Formaldehyde in European residences: emissions from building and construction materials

VOC	Study	Glue for wallpaper	Gypsum board	Linoleum	MDF	Noise protection panel	OSB
Formaldehyde	Plaisance *et al*., 2017 **TC**		2.6 [a, 2]			5.15 [a, 2]	19.95 [a, 2]
	Plaisance *et al*., 2014a **PFS**						16.4 [a, 2]
	Plaisance *et al*., 2014a **PFS**				133.3 (92.5 , 135.5) [a, 2, 1]		
	Plaisance *et al*., 2014b **TC**	5 [a, 2]	15.5 [a, 2]	<3.9 [a, 2]	193 (92 255) [a, 2, 1]		26.15 (21.3 31) [a, 2, 1]
	Risholm-				3.5 [b, 2]		

	Sundman *et al*., 2007 **PM**						
	Risholm-Sundman *et al*., 2007 **TC**				4.4 [a, 2]		
	Simon *et al*., 2020 **TC**				42.09 [a, 2]		

TC: Test Cell or Chamber; **PFS**: Passive Flux Sampler; **PM**: Perforator Method
[a]: µg m^{-2} h^{-1} (area-specific emission rates); [b]: mg per 100g[1]: Median, min max; [2]: Sample value.

Table A.3.5: Formaldehyde in European residences: emissions from building and construction materials

VOC	**Study**	**Paints**				**Particleboard**	
		General	**Alkyd resin (high airflow rates)**	**Alkyd resin (low airflow rates)**	**Latex on concrete and polyester substrate**	**E0**	**E1**
Formaldehyde	Plaisance *et al*., 2017 **TC**	88 [a, 1]					
	Risholm-Sundman *et al*., 2007 **FM**					2 [b, 1]	4 [b, 1]
	Risholm-Sundman *et al*., 2007 **PM**					2–3 [c, 1]	4.6 [c, 1]
	Risholm-Sundman *et al*., 2007 **TC**					0.8 [a, 1]	2 [a, 1]

TC: Test Cell or Chamber; **FM**: Flask Method; **PM**: Perforator Method
[a]: µg m^{-2} h^{-1} (area-specific emission rates);[b]: mg kg^{-1}; [c]: mg per 100g; [1]: Sample value

Table A.3.6: Formaldehyde in European residences: emissions from building and construction materials

		Plywood						
VOC	**Study**	**General**	**Exterior**	**Exterior (22 mm)**	**exterior (8 mm)**	**Interior**	**Interior (22 mm)**	**Interior (8 mm)**
Formaldehyde	Bohm *et al*., 2012 **TC**			between 0.36 ± 0.02 and 0.85 ± 0.03 [a, 2]	between 0.13 ± 0.01 and 0.72 ± 0.07 [a, 2]		between 1.47 ± 0.19 and 2.65 ± 0.17 [a, 2]	between 1.24 ± 0.04 and 1.66 ± 0.04 [a, 2]
	Plaisance *et al*., 2014b **TC**	8.3 [a, 2]						
	Risholm-Sundman *et al*., 2007 **FM**		1.4 [b, 2]			32 [b, 2]		
	Risholm-Sundman *et al*., 2007 **TC**		0.2 [a, 2]			5.5 [a, 2]		

TC: Test Cell or Chamber; **FM**: Flask Method;
[a]: µg m^{-2} h^{-1} (area-specific emission rates); [b]: mg kg^{-1}; [1]: Median, min max; [2]: Sample value.

Table A.3.7: Formaldehyde in European residences: emissions from building and construction materials

		Area-specific emission rates (µg m^{-2} h^{-1})						
		PVC		Sealing plaster	Silicone	Skirting board	Vapour barriers	Varnish on various substrates
VOC	**Study**	**Only**	**Adhesives**					
Formaldehyde	Plaisance *et al*., 2017 **TC**					17.8 [2]	1.805 [2]	
	Plaisance *et al*., 2014a			43.4 [2]				
	Plaisance *et al*., 2014b **TC**				<3.9 [2]			
	Plaisance *et al*., 2014b **TC**				1.7 [2]			

TC: Test Cell or Chamber; **FLEC**: Field and Laboratory Emission Cell
[1]: Median, min max; [2]: Sample value.

Table A.3.8: Formaldehyde in European residences: emissions from building and construction materials

		Area-specific emission rates (µg m^{-2} h^{-1})							
VOC	**Study**	**Walls**				**Wood stain**			
		0 months*	**12 months***	**6 months***	**Solid wood**	**Solvent based**	**Water based**	**Wooden battens and studs**	**Wooden flooring**
Formaldehyde	Bohm *et al*., 2012 **TC**				between 0.014 ± 0.001 and 0.084 ± 0.009 [1]				
	Jarnstrom *et al*., 2007 **FLEC**	7 (5, 11) [2]	9 (5, 20) [2]	13 (5, 37) [2]					
	Plaisance *et al*., 2017 **TC**							2.26 [3]	134 [3]
	Plaisance *et al*., 2014b **TC**				between 3.9 and 14 [3]				

TC: Test Cell or Chamber; **ECM**: Emission Chamber and Model; **FLEC**: Field and Laboratory Emission Cell;
[1]: Average, SD; [2]: Average, min max; [3]: Sample value. *: period after the structure was finished

Table A.3.9: Formaldehyde in European residences: emissions from consumer products

		Combustible AF				
VOC	**Study**	**General**	**Fragranced candles**	**Unfragranced candles**	**High flow rates***	**Low flow rates****
Formaldehyde	Derudi *et al*., 2012 **TC**	2.91 [d, 1]				
	Manoukian *et al*., 2016 **TC**				2304 (226) [d, 2]	822 (66) [d, 2]
	Manoukian *et al*., 2013 **EH**	1206 (17) [e, 2]				
	Petry *et al*., 2013 **TC**	25.8 (17, 38.1) [e, 3, 1]				
	Petry *et al*., 2014 **TC**		280 (73, 372.2) [b, 3, 1]	22.65 (19.6, 25.7) [b, 3, 1]		
	Petry *et al*., 2014 **TC**		20.7 (1.8, 80.6) [b, 3, 1]	80.6 (0.2, 25.7) [b, 3, 1]		

TC: Test Cell or Chamber; **SV**: Sample Vessel; **FE**: Field Experiment. **EH**: Experimental House
[a]: µg s^{-1} g[product]$^{-1}$; [b]: mg h^{-1}; [c]: µg kg^{-1}; [d]: µg g^{-1}; [e]: µg h^{-1};
[1]: Median, min max; [2]: Average, SD; [3]: Sample value.

***** Air exchange rate 0.25 h^{-1} ****** Air exchange rate 1.5 h^{-1}

Annex 4 – EUROPEAN REFERENCE ROOM (EN 16516) AND INTERNATIONAL TEST PROTOCOLS / METHODS

Loading factor refers to the ratio between the surface of the used product and the total volume of the empty room.

Parameter name	Parameter value	Loading factor (L)
Temperature	23 °C	
Relative humidity	50%	
Air exchange rate (ACH)	0.5 h^{-1}	
Room volume	30 m^3	
Room dimensions	4 x 3 x 2.5 m (1 door, 1 window)	
Surface floor	12 m^2	0.4 m^2/m^3
Surface ceiling	12 m^2	0.4 m^2/m^3
Surface walls	31.4 m^2	1 m^2/m^3 (rounded)
Surface door	1.6 m^2	0.05 m^2/m^3 (rounded)
Surface window	2 m^2	0.05 m^2/m^3 (rounded)
Sealing	0.2 m^2	0.007 m^2/m^3

Test protocols/methods

Emission testing is used to quantify emission of an individual chemical, a group of chemicals or both. Due to regulatory requirements (e.g., USEPA Formaldehyde Emission Standards for Composite Wood Products Rule) formaldehyde is the most often targeted individual chemical for emission testing. Emissions of other volatile organic compounds (VOCs) are often covered by product emission labelling programs.

Each regulation or certification label scheme requires a test protocol to determine emission rates from a specific product (Table A.4.1). Many of the test methods have been developed and maintained by nongovernmental consensus-based organizations (e.g., the International Organization for Standardization (ISO) and ASTM International). The methods have varying degrees of parameter and measurement specificity (e.g., ASTM guides give information or series of options, while ASTM test methods require specific steps that produce a result). Some methods are specific to a product type (e.g., ANSI/BIFMA M7.1 focuses on furniture). Many methods are based in part on or refer to ISO 16000-9 (Indoor air – Part 9: Determination of the emission of volatile organic compounds from building products and furnishing – Emission test chamber method), for example, EN 16516. As described below methods can be either static (sealed chamber) or dynamic (flow-through chamber).

Table A.4.1: Examples of Testing Standards for formaldehyde (from Poppendieck *et al.*, 2022)

Test Method ID	Region	Type	Method Name
ASTM D6007	US	Dynamic	Standard Test Method for Determining Formaldehyde Concentrations in Air from Wood Products Using a Small-Scale Chamber
ASTM E1333	US	Dynamic	Standard Test Method for Determining Formaldehyde Concentrations in Air and Emission Rates from Wood Products Using a Large Chamber
ASTM D5582	US	Static	Standard Test Method for Determining Formaldehyde Levels from Wood Products Using a Desiccator
ANSI/ BIFMA M7.1	US	Dynamic	Testing for VOC Emissions from Office Furniture and Seating (for VOCs and formaldehyde)
UL 2821	US	Dynamic	GREENGUARD Standard for Building Materials, Finishes and Furnishings (for VOCs and Formaldehyde)
EN 717-1	EU	Dynamic	Wood-based Panels – Determination of Formaldehyde Release – Formaldehyde Emission by the Chamber Method
EN 717-3	EU	Static	Wood-based panels – Determination of formaldehyde Release – Formaldehyde Release by the Flask Method
GB/T 17657- 2013	China	Dynamic	Test Methods of Evaluating the Properties of Wood-based Panels and Surface Decorated Wood-based Panels
JIS A 1911: 2015	Japan	Dynamic	Determination of the Emission of Formaldehyde by Building Materials and Building Related Products – Large Chamber Method
JIS A 1460	Japan	Static	Determination of the emission of Formaldehyde from Building Boards – Desiccator Method

Annex 5 – Supplementary information to the human health hazard assessment

Section A.5.1 – Table A.5.1: Summary of key studies investigating the inhalation effects of formaldehyde

Limitations listed in the table below are those identified by study authors and expert groups.

Reference	Population / species	Concentrations / exposures	Study Findings
Epidemiology Studies (general population)			
Hanrahan *et al.*, 1984	61 Teenagers and adults (39% M and 61% F), across 65 mobile homes in Wisconsin, US. 20 (33%) of 61 participants were smokers.	Range <0.10 to 0.80 ppm, median 0.16 ppm [123 to 984 µg/m^3, median 197 µg/m^3]	Indoor air formaldehyde concentrations were measured in 65 randomly selected Wisconsin mobile homes (average of 1-hour measurements taken in 2 rooms; one in the kitchen or living room and one in the bedroom) and the occupants completed questionnaires on current symptoms, including occurrence, since moving into their home. Among the symptoms reported, only burning eyes and eye irritation demonstrated a statistically significant concentration-response relationship to indoor formaldehyde concentration. Adjustments made for potential confounding factors including age, gender, and smoking status. Significant differences in age of respondents were noted, with a higher prevalence of reported ocular discomfort in younger persons. Limitations: multifactorial exposures including odour-mediated sensory irritation

Reference	Population / species	Concentrations / exposures	Study Findings
			biased by the high concentrations of formaldehyde and acrolein from environmental tobacco smoke; the study did not meet its original intent of being representative of Wisconsin mobile homes, as noted by the authors: 'The original intent of our sampling procedure was to correct for these biases [self-referred reports of medical illnesses and symptoms reported in previous investigations] by evaluating a randomly selected and representative cross-section of mobile homes in Wisconsin. However, the participation rate resulted in a sample which could not be considered representative of all Wisconsin mobile homes.'
Krzyzanowski *et al.*, 1990	Cross-sectional study in Arizona, US, 613 adults (ages >15 years) and 298 children (ages 6–15 years), M + F, from 202 households. Selection based on information about potential exposure (age of housing) and potential susceptibility obtained from an initial screening questionnaire.	Average 26 ppb [32 µg/m^3], maximum 140 ppb [172 µg/m^3]	Sampling for formaldehyde involved two one-week samples from subject's kitchen, living room, and bedroom covering two seasons. Peak expiratory flow rates (PEFR) were measured four times daily (morning, noon, early evening and before bed) for 2 weeks. The largest of three test results was recorded for each test period. Data on chronic cough and phlegm, wheeze, attacks of breathlessness, and doctor diagnoses of chronic bronchitis and asthma were collected with self-completed questionnaires. The EPA draft assessment reports that findings were adjusted for potential confounding factors including asthma, smoking, socioeconomic status, nitrogen dioxide levels, episodes of acute respiratory illness and time of day (US EPA, 2024). Analysis was performed separately for ages younger and older than 15 years. In adults, neither respiratory symptoms nor physician-diagnosed chronic bronchitis or asthma were significantly related to formaldehyde concentrations. An association was observed between declining PEFR among adult smokers and increasing average formaldehyde concentrations of between 49 and 172 µg/m^3, but not among the group as a whole (US EPA, 2024).

Reference	Population / species	Concentrations / exposures	Study Findings
			The prevalence of chronic respiratory symptoms in children was not related to formaldehyde levels. The original study used a linear mixed-effects model, which incorporated whether children had asthma or not. A linear relationship was observed between increased formaldehyde exposure and decreased PEFR among children (less than 15 years of age), exposed to average concentrations of 26 ppb [32 µg/m^3]. Overall, the investigators reported a statistically significant decrease of 1.28 ± 0.46 L/minute in PEFR among children per ppb household mean formaldehyde concentration. An increase in the prevalence of physician-diagnosed asthma was observed in children where kitchens had formaldehyde concentrations > 60 ppb [74 µg/m^3] and there was also exposure to environmental tobacco smoke. The investigators established a NOAEL of 62 µg/m^3. Limitations: a relationship between a high prevalence of asthma in children and kitchen formaldehyde levels above 74 µg/m^3 was only applicable if the children were also exposed to second-hand smoke; diagnosis of asthma was by parental questionnaire; potential for confounding by unmeasured indoor contaminants (the only other one determined was environmental tobacco smoke).
Garrett *et al*., 1999	Cross-sectional study in 148 children (7–14 years), including 53 asthmatics.	Median 12.6 ppb [15.8 µg/m^3], maximum 111 ppb [139 µg/m^3]	Cross-sectional survey of 80 Australian homes. Passive residential sampling was performed 4 times between March 1994 and February 1995. Respiratory questionnaires were completed by parents, and skin-prick testing was performed with 12 environmental allergens. No significant increase was observed between the adjusted risk of asthma or respiratory symptoms with increasing formaldehyde concentration. A trend was

Reference	Population / species	Concentrations / exposures	Study Findings
			observed between the formaldehyde exposure category and the proportion of atopic children (also associated with family history). Furthermore, more severe allergic sensitisation was demonstrated with increasing formaldehyde exposure; children suffering from respiratory symptoms, more frequent symptoms were noted in those exposed to higher formaldehyde levels. Limitations: diagnosis of asthma was by parental questionnaire.
Delfino *et al.,* (2003)	21 asthmatic Hispanic children (10–16 years).	Daily air pollution measurements ranged 4.27 ppb [5.25 µg/m^3] to 14.02 ppb [17.25 µg/m^3]	Subjects were 21 asthmatic children living in a Los Angeles community with high traffic density. Subjects recorded daily the severity of asthma symptoms and results of morning and evening peak expiratory flow (PEF) manoeuvres across 3 months. Subjects were followed up weekly at their homes across the 3-month period. The adjusted odds ratio (OR) for bothersome or more severe asthma, with an inter-quartile range (IQR) increase of 0.003 ppm [4 µg/m^3] in formaldehyde, was 1.37 (95% CI 1.04 –1.8) with a 1-day lag. No relationship was observed between formaldehyde concentration and PEFR.
Tavernier *et al*., 2006	200 children (4–17 years old), patients at two primary care facilities in the South of Manchester, UK. 105 asthmatic children, 95 healthy	-	The home environment of 105 asthmatic children and 95 healthy controls, living in the South Manchester area (UK), was investigated. There were no differences in indoor air concentrations of formaldehyde in the homes of the cases and the controls. Limitations: no formaldehyde concentrations are reported, and various other factors are investigated alongside this parameter. Further, the study is based on the expression of asthma symptoms of a wide age range of children. The survey questionnaire was validated against physician diagnoses, but still potentially carries

Reference	Population / species	Concentrations / exposures	Study Findings
	controls.		biases attached to questionnaire-based studies.
Annesi-Maesano *et al.*, 2012	Children in France, M + F, n = 6,590 (aged 9 – 10 years)	Median 28 µg/m^3 and 95th percentile 55 µg/m^3	A cross-sectional study in 401 randomly selected classrooms from 108 primary schools across 6 French cities. Classrooms were passively sampled over a 5-day period for concentrations of fine particles ($PM_{2.5}$), nitrogen dioxide, and 3 aldehydes; formaldehyde, acrolein, and acetaldehyde. A total of 6,683 children completed the entire survey protocol, though analyses were limited to 6,590 for whom all variables of interest were available. Survey protocol required a medical examination, including skin prick testing (SPT) for common allergens and a test for exercise-induced asthma. Parents also completed an enriched version of the International Study of Asthma and Allergies in Childhood (ISAAC). The findings were adjusted for confounding factors including age, gender, passive smoking, maternal and paternal history of asthma and allergic diseases, dampness, gas appliance, ethnicity and socioeconomic status. A high prevalence of rhinoconjunctivitis in the past year was significantly associated with children using classrooms with high levels of formaldehyde (>28 µg/m^3, OR 1.19; 95% CI 1.04 to 1.36). An increased odds ratio was not observed for past-year asthma (OR 0.90), nor was exercise-induced asthma correlated with formaldehyde exposure. Limitations: study authors note that the absence of objective validation may result in the misclassification of asthma and rhinitis. The definition of asthma used in the study may have excluded children who were not aware of their condition owing to not having received a diagnosis. Further, a cross-sectional study cannot establish causal

Reference	Population / species	Concentrations / exposures	Study Findings
			relationships.
Matsunaga *et al.*, 2008	Cross-sectional study of pregnant adult women in Osaka, Japan, n = 998, median 17th week of pregnancy	Median 30 µg/m^3, maximum 161 µg/m^3	Pregnant women were recruited through obstetric clinics and public health nurses. Formaldehyde was measured via 24-hour personal monitor-based sampling and subjects self-reported treatment for asthma, allergic rhinitis or atopic eczema in the past 12 months. The findings were adjusted for age, gestation, parity, family history (of asthma, atopic eczema, allergic rhinitis), smoking status, current passive smoking at home and work, mould in kitchen, indoor domestic pets, dust mite antigen level, family income, education and season. When formaldehyde concentrations were categorised into four groups, there was a tendency for a positive exposure-response relationship between formaldehyde concentration and the prevalence of atopic eczema, although the adjusted odds ratio for highest vs. lowest formaldehyde categories did not reach statistical significance. When formaldehyde concentrations were categorised into two groups to assess the effects of exposure to high levels of formaldehyde on allergic disorders, formaldehyde concentrations of ≥ 58 µg/m^3 were independently associated with an increased prevalence of atopic eczema (adjusted OR 2.25; 95% CI 1.01-5.01). The positive association was more pronounced in women with a negative familial allergic history (OR 2.96, 95% CI 0.87-10.12) than in those with a positive familial allergic history (OR 1.63, 95% CI 0.58-4.57%) at exposures of ≥ 58 µg/m^3. There was also an increased risk of atopic eczema at lower exposures (<58 µg/m^3) in the negative family history group (OR 1.37, 95% CI 1.88-4.21) compared with the positive family group (OR 0.8, 95% CI 0.92-1.45). A NOAEL of 46 µg/m^3 was established. No clear association was found between formaldehyde concentration and the

Reference	Population / species	Concentrations / exposures	Study Findings
			prevalence of asthma or allergic rhinitis. Limitations: the authors found that the odds ratio difference between the lowest and highest tertiles was not statistically significant. Only when the data were collapsed to two groups of highly disparate size (90^{th} percentile cut-off) did the 95^{th} percent lower confidence interval of the OR exceed value of 1.01).
Rumchev *et al.*, 2002	Study included 104 children <3 years of age, and 88 asthmatic children <3 years of age	Formaldehyde measured in air of children's rooms and in another common room.	The cases were children discharged from a hospital emergency department with a diagnosis of asthma (n=88). The controls were community controls without physician-diagnosed asthma (n=104). Formaldehyde exposure was assessed by 8-hour passive sampling in the winter and the summer in the living room and children's bedroom. The mean concentration of formaldehyde in the living room was 27.5 $\mu g/m^3$ and the maximum concentration was 189.7 $\mu g/m^3$. In the bedroom the mean concentration was 30.2 $\mu g/m^3$ and maximum concentration was 224 $\mu g/m^3$. At formaldehyde concentrations ranging from 50 - 59 $\mu g/m^3$ there was a non-significant increased risk of asthma. Children exposed to formaldehyde levels of ≥ 60 mg/m^3 had a 39% increase in odds of having asthma compared with children exposed to formaldehyde levels < 10 mg/m^3. Limitations: potential bias from gas heating, new materials in the homes, cases and controls not obtained at the same institution, and the difficulty of diagnosis in children (based on parental information). An important confounding factor was the presence of combustion products: reported high concentrations of traffic pollutants including benzene, toluene, xylenes, nitrogen dioxide and sulphur dioxide in the homes of the children. Such pollutants are known to be associated with asthma in children. The authors acknowledged the potential for selection bias and the difficulty of

Reference	Population / species	Concentrations / exposures	Study Findings
			differentiating wheezing illnesses in the investigated age group.
Venn *et al.*, 2003	Nested case control. Children 9-11 years. n = 193 persistent wheeze cases, 223 controls.	3-day samples in bedroom; median 22 µg/m^3; median in top quartile 41 µg/m^3.	No association was seen with the prevalence of wheezing during the past year in the case-control analysis and formaldehyde concentration in the children's bedrooms. Among the 193 cases, a two- to three-fold increased risk of frequent symptoms (defined as nocturnal symptoms recorded on ≥10 consecutive days) was seen in the highest quartile of exposure (>32 µg/m^3) compared with <16 µg/m^3, with some evidence of an increased risk at even lower exposures.
Epidemiology studies (occupational)			
Holmström *et al.*, 1989	70 workers in a chemical plant, 100 furniture-factory workers, 34 controls that produced formaldehyde and formaldehyde resins	Estimates of levels were: 0.04-0.4 ppm for the chemical workers [49.6 µg/m^3 – 496 µg/m^3] 0.16-0.4 ppm for the furniture workers [198.4 µg/m^3 – 496 µg/m^3] 0.07-0.13 ppm in the late summer for the office workers	The chemical plant produced formaldehyde and formaldehyde resins; the furniture-factory workers worked with particle-board and glue components. The controls were office workers in the same village as the furniture factories. Recording of histological evidence of mild damage to the nasal epithelial tissue (squamous metaplasia, loss of ciliated cells, goblet cell hyperplasia, and mild dysplasia in biopsied tissue) in formaldehyde-exposed chemical workers. Nasal biopsy sections were scored from 0 (normal) to 8 (carcinoma). Nasal histology scores ranged from 0-4 (mean 2.16, n = 62) for the chemical workers, 0-6 (mean 2.07, n = 89) for the furniture workers and 0-4 (mean 1.46; n=32) for the office workers (statistically significantly different from the chemical workers). No evidence of associations between histological score and duration of exposure, index of accumulated dose or smoking habit. Limitations: the histopathology scores were not exposure-dependent; the estimated

Reference	Population / species	Concentrations / exposures	Study Findings
		[86.8 µg/m^3 – 161.2 µg/m^3]; year-round median 0.07 ppm [86.8 µg/m^3]	exposure for the office workers overlapped with that for the chemical-plant workers; also the same range of scores between these two groups; co-exposure to wood dust for the furniture-factory workers (estimated to be 1-2 mg/m^3).
Wilhelmsson and Holmström, 1992	66 workers in a chemical plant. 36 office workers as controls	Estimated exposures were: 260 µg/m^3 for the chemical-plant workers 90 µg/m^3 for the office workers	66 chemical plant workers (almost exclusively exposed to formaldehyde) exposed to a mean concentration of 260 µg/m^3 compared with a control group of 36 office workers exposed to a mean concentration of 90 µg/m^3. The average exposure duration was 10 years (range 1 – 36 years). The formaldehyde concentrations were measured in the ambient air of the worksite with personal sampling equipment. The critical effects in the study included nasal obstruction and discomfort, lower airway discomfort and eye irritation. The frequency of reported lower airway discomfort and nasal discomfort was significantly higher in the chemical plant workers compared with the control group. In addition, the rate of work-related eye irritation was 20% in the chemical plant workers and non-existent in the office workers. The mean formaldehyde concentration of 90 µg/m^3 for the office workers was considered to be the NOAEL. Limitations: no concentration-response information; exposure of the office-worker control group was high.
Controlled Human Exposure Studies			
Andersen and	16 healthy adults, 11 male and 5 female	300, 500, 1000, 2000 µg/m^3 for 4 –	On four consecutive days, 16 healthy subjects were exposed to different concentrations of formaldehyde for 5-hour periods, preceded by a non-exposed

Reference	Population / species	Concentrations / exposures	Study Findings
Mølhave, 1983	(aged 20 – 33), 5 subjects smoked but only 1 a heavy smoker. No prior exposures to formaldehyde nor history of chronic / acute respiratory distress.	5 hours.	(clean air) period of 2 hours. Measurements were taken during the non-exposed period and after 2-3 hours and 4-5 hours of exposure and included nasal mucociliary flow during exposure and following exposure nasal airflow resistance, forced expiratory vital capacity (FVC), forced expiratory volume in 1 second (FEV1), forced expiratory flow rate between 25 and 75 percent (FEF25-75%) and the odour threshold for “ethyl valeriate”. Subjects were also asked to assess “discomfort” on a 1–100 scale ranging from 1=complete comfort to 100=severe discomfort (scores between 1 and 33 were rated as “slight discomfort”). Average peak discomfort scores for the group generally increased with exposure concentration, but the average discomfort score for the highest exposure concentration (1.6 ppm) never exceeded 18. Numbers of subjects who reported “No discomfort” ratings at the end of exposure periods were 7, 13, 10, and 6, respectively for 0.24, 0.4, 0.81, and 1.61 ppm; respective numbers of subjects reporting “conjunctival irritation and dryness in the nose and throat” were 3, 5, 15, and 15 of the 16 subjects exposed to each respective concentration. A statistical analysis of these data was not reported. A decrease in nasal mucus flow was found but the response did not increase at concentrations above 0.5 mg/m^3 or at prolongation of the exposure period from 3 to 5 hours. Exposure at 2 mg/m^3 during 1-5 hours increased the odour threshold for “ethyl valeriate”, whereas no effect was found at lower concentrations. The frequency of eye blinking was increased at exposures of 2.0 mg/m^3 and above. No significant changes were found in the other airway resistance parameters. Limitations: although findings were consistent with Haber’s law, with eye irritation symptoms reported at low concentrations (0.3–0.5 mg/m^3) and increasing over the course of the 4h exposure (Andersen and Mølhave, 1983), symptoms were not

Reference	Population / species	Concentrations / exposures	Study Findings
			objective and not dose responsive (i.e., mean discomfort scores were higher at 0.3 mg/m^3 relative to 0.5 mg/m^3) and symptoms at higher concentrations (1 and 2 mg/m^3) plateaued or decreased after 3h exposure. Further, the possibility of an odour-mediated bias existed owing to the influence of odour perception on subjective symptom reporting.
Witek *et al*., 1987	15 mild asthmatics.	0 to 2 ppm [2460 $\mu g/m^3$] for 40 minutes.	Volunteers underwent randomised, double-blind exposures to clean air or 2 ppm [2460 $\mu g/m^3$] for 40 minutes in an environmental chamber. Exposures repeated on a separate day during moderate exercise across 10 minutes. Measurements taken before, during, and 4-, 8-, and 24-hours post-exposure. Study authors reported no statistically significant exposure-related effects on acute or sub-acute changes in lung function measurements or in bronchial responsiveness to challenge with methacholine. Sequential measurements of peak flow post-exposure showed no delayed airway response. Symptoms reported by questionnaire with severity score; subjects report bad odour, sore throat, and eye irritation during exposure but infrequently post-exposure.
Sauder *et al.,* 1986	9 healthy non-smoking adults.	0 and 3 ppm [3690 $\mu g/m^3$] for 3 hours.	Clean air on day 1, followed by formaldehyde exposure on day 2, with a 24-hour follow-up on day 3. Intermittent exercise during exposure. Measurements taken daily; pulmonary function, non-specific airway reactivity, and symptoms via questionnaire. Slight statistically significant decrease in FEV_1 and $FEF_{25-75\%}$ after 30 minutes of exposure, however effect no longer present at 60 or 180 minutes. Statistically significant increase in odour perception and nose/throat/eye irritation with exposure.

Reference	Population / species	Concentrations / exposures	Study Findings
			No statistically significant changes in pulmonary function or airway reactivity observed 24 hours after exposure. Authors conclude; acute exposure to 3 ppm [3690 µg/m^3] formaldehyde resulted in small transient decreases in pulmonary function and mild to moderate irritation of the eyes and upper respiratory tract in healthy non-smoking adults.
Sauder *et al*., 1987	9 asthmatic non-smoking adults (aged 26 – 40).	0 and 3 ppm [3690 µg/m^3] for 3 hours.	Clean air followed by formaldehyde exposure, separated by 1 week. Intermittent exercise during exposure period. Measurements taken before and after exposures; pulmonary function, non-specific airway reactivity, and symptoms via questionnaire. No statistically significant changes in pulmonary function observed (FVC, FEV_1, $FEF_{25-75\%}$, SGaw, or FRC). No airway reactivity observed. Statistically significant increase in nose/throat irritation at 30 minutes exposure and in eye irritation at or above 60 minutes of exposure. Authors conclude data suggest asthmatics will not experience significant bronchoconstriction when exposed to 3 ppm [3690 µg/m^3], however eye and upper respiratory tract irritation likely.
Green *et al*., 1987	22 healthy subjects, 16 asthmatics, all non-smoking.	Controlled environment chamber, 0 and 3 ppm [3690 µg/m^3] for 1 hour.	Randomised blinded exposures on 2 days, separated by 1 week. Healthy subjects engaged in intermittent heavy exercise, while the asthmatic subjects engaged in intermittent moderate exercise. Irritation symptoms and pulmonary function assessed during exposure, and non-specific airway reactivity assessed post-exposure. Perceived odour and nose/throat/eye irritation significantly increased during exposure in both groups. Healthy subjects had slight statistically significant decreases in pulmonary function compared to clean air. Asthmatics had no statistically significant

Reference	Population / species	Concentrations / exposures	Study Findings
			change to pulmonary function. Study authors concluded results suggest some individuals demonstrate and increased responsiveness to formaldehyde.
Pazdrak *et al*., 1993	21 non-smoking adults. Group 1: 7 male and 3 female patients occupationally exposed with skin hypersensitivity to formaldehyde. Group 2: 11 healthy males, with no history of allergic disease.	Chamber exposures across 2 hours, at 0 or 500 µg/m^3.	Nasal washings in both groups were performed immediately before and after the 2-hour exposure, then again at 4 and 18 hours after completion of the exposure. Nasal washings were analysed for eosinophil, neutrophil, basophil, and mononuclear cells, and albumin content. Formaldehyde caused transient symptoms of rhinitis; increased itching, sneezing, mucosal congestion, and burning sensations in the eyes or nasal passages. Nasal alterations were also reported; elevated number and proportion of eosinophils, and transient elevated albumin and total protein levels at both 4 and 18 hours post-exposure. No differences in nasal response to formaldehyde were found between patients with skin sensitisation and healthy subjects. The authors concluded the symptoms reported at 500 µg/m^3 likely result from a non-specific, non-allergic process.
Kulle *et al.*, 1987, 1993 Note: the 1993 article reexamines the symptomatic response data in the 1987 paper	19 healthy non-smoking adults with no history of allergy, asthma, hay fever nor upper respiratory infection. Subjects split into Group 1 (n=10) and	Formaldehyde exposures in an environmentally controlled chamber. Group 1: 0, 0.5, 1, and 2 ppm [0, 615, 1230, and 2460 µg/m^3] at rest + 2	Healthy, non-smoking adults received 5 randomised 3-hour exposures (including one clean-air control) to formaldehyde vapour in environmentally controlled chambers. They also separately conducted an 8-minute bicycle ergometer exercise segment every half hour during a 2 ppm [2460 µg/m^3] exposure. Each randomised exposure was separated by 1 week and formaldehyde concentrations were monitored continually using colorimetric monitors. Symptom questionnaires were conducted, asking subjects to rank the severity of eye and nose/throat irritation on a scale of 0 – 3; 0 being none, 1 being mild (present, not

Reference	Population / species	Concentrations / exposures	Study Findings
using additional statistical methodology.	group 2 (n=9).	ppm [2460 µg/m^3] with exercise. Group 2: 0, 1, 2, and 3 ppm [0, 1230, 2460, and 3690 µg/m^3] at rest + 2 ppm [2460 µg/m^3] with exercise.	annoying), 2 being moderate (annoying), and 3 being severe (debilitating). Pulmonary function tests and non-specific airway reactivity by methacholine challenge were also carried out. A statistically significant dose-response relationship in odour and eye irritation was observed, with the frequency of irritation reported increasing with formaldehyde concentration. This effect was not affected by exercise. Nasal flow resistance was increased at 3 ppm [3690 µg/m^3]. No significant decrements in pulmonary function were observed; forced vital capacity (FVC), forced expiratory volume in 1 sec (FEV_1), forced expiratory flow rate between 25 – 75% ($FEF_{25-75\%}$) and specific airway conductance (SGaw). No increases in bronchial reactivity to methacholine. The most sensitive effect was eye irritation, with the 1993 re-examination noting thresholds of ≥0.5 ppm [615 µg/m^3] for odour sensation and eye irritation and 1 ppm [1230 µg/m^3] for nose/throat irritation.
Krakowiak *et al*., 1998	20 Subjects (aged 23 – 52); 10 formaldehyde exposed textile/shoe-manufacturing workers with purported bronchial asthma, and 10 non-exposed healthy subjects.	0 or 500 µg/m^3 in an exposure chamber for 2 hours.	The workers were exposed to formaldehyde during their occupation as textile/shoe-manufacturing workers. The healthy control subjects were not occupationally exposed to formaldehyde. Before and after exposures, spirometry at rest and following bronchial provocation with histamine was recorded. Measurements of formaldehyde-specific serum IgE antibodies and cellular, biochemical, and mediator changes were assessed in nasal lavage before and immediately after exposures, then 4 and 24 hours later. Transient rhinitis observed in both groups. No adverse pulmonary effects were recorded after 2 hours of exposure; no change in FEV_1 (nor in PEFR). No IgE

Reference	Population / species	Concentrations / exposures	Study Findings
			antibodies to formaldehyde were detected in subjects with occupational exposures and no subject with purported occupational asthma developed clinical symptoms of bronchial irritation. Limitations: Single blinding with no randomisation in design and no standard deviation or variability in data reported.
Ezratty *et al*., 2007	12 subjects (aged 18 – 44) non-smokers, with intermittent allergic asthma and allergy to grass pollen.	0 or 500 µg/m^3 for 1 hour at rest.	Double-blind cross-over study; exposures to purified air or formaldehyde were randomised and separated by 2 weeks. After each exposure, an allergen inhalation challenge was performed. Airway responsiveness to methacholine and lower airway inflammation (induced sputum) were assessed 8 hours after allergen challenge. No asthmatic response (no changes in FEV_1 or PEFR) shown after exposure for 1 hour. No impact on bronchial allergen responses to grass pollen or methacholine provocation. Levels of inflammatory markers measured in sputum were similar in subjects exposed to either formaldehyde or purified air. Study authors found exposure to 500 µg/m^3 formaldehyde had no significant deleterious effect on airway allergen responsiveness of patients with intermittent asthma; in fact a trend toward a protective effect was noted.
Lang *et al*., 2008	21 healthy adults (11 males, 10 females) of ages 19 – 39.	Participants exposed across a 10-week period to a randomised sequence of 10 exposure conditions	Double-blind, randomised study aiming to examine occurrence of sensory irritation and subjective symptoms in human volunteers exposed to formaldehyde concentrations relevant to the workplace. Study also investigated effects in the presence and absence of a masking agent, to avoid influence of odour, with ethyl acetate (12 ppm – 16 ppm) added to continuous exposures.

Reference	Population / species	Concentrations / exposures	Study Findings
		on 10 consecutive days, each for a duration of 4 hours. Exposure conditions: 0, 0.15, 0.3, and 0.5 ppm [0, 19, 370, and 615 µg/m^3] continuous. 0.3 or 0.5 ppm [370 or 615 µg/m^3] with peaks of 4 x 0.6 ppm [740 µg/m^3] and 4 x 1.0 ppm [1230 µg/m^3], respectively.	Questionnaires were used to record subjective ratings of discomfort and assess the influence of personality factors on subjective scoring. Objective measurements of conjunctival redness, eye blinking frequency (EBF), nasal resistance and flow, pulmonary function and reaction times also carried out. All measurements were conducted pre- and post-exposures to evaluate the irritating effects of formaldehyde. Results indicated no significant treatment effects on nasal flow and resistance, pulmonary function, nor reaction times. EBF and conjunctival redness (ranging from slight to moderate) were significantly increased by short-term peak exposures of 1 ppm [1230 µg/m^3] at a baseline of 0.5 ppm [615 µg/m^3]. Subjective reporting noted eye and olfactory symptoms at concentrations as low as 0.3 ppm [370 µg/m^3]. Volunteers rating their personality as “anxious” tended to report complaints at a higher intensity, and when “negative affectivity” was used as a covariate, eye and olfactory symptoms at 0.3 ppm [370 µg/m^3] was no longer a significant effect. Even with “negative affectivity” as a covariate, eye and olfactory symptoms remained a significant effect at 0.5 ppm [615 µg/m^3] with peaks of 1 ppm [1230 µg/m^3]. Study authors concluded increased EBF at 615 µg/m^3 was the LOEC, though noted the OAEC for both subjective and objective eye irritation would be close to this threshold at continuous exposures. The odour detection threshold was also determined for the cohort of participants, reported as a range: 190 – 370 µg/m^3. Limitations: RAC considered the group sizes to be small and the variability in EBF to be high, reducing the sensitivity to detect concentration-related effects unless they were marked (ECHA, 2020c).
Mueller *et al*.,	41 healthy male non-	Participants	Subjective pain perception, induced by nasal application of CO_2, was used as an

Reference	Population / species	Concentrations / exposures	Study Findings
2013	smoking adults. Participants were grouped into hyposensitive or hypersensitive based on CO_2 sensitivity measurements in nasal mucosa.	exposed for 5 days (4 hours per day), with clean air or the following conditions: 0.3 ppm [370 µg/m^3] + 4 peaks of 0.6 ppm [740 µg/m^3] 0.4 ppm [490 µg/m^3] + 4 peaks of 0.8 ppm [980 µg/m^3] 0.5 ppm [615 µg/m^3] and 0.7 ppm [860 µg/m^3] Peak exposures were done 4 times per day for 15 minutes.	indicator for sensitivity to sensory nasal irritation, grouping participants into hyposensitive or hypersensitive. Formaldehyde generated through thermal depolymerisation of paraformaldehyde, dynamic chamber conditions with analytical concentrations reported. Before and after exposures, parameters examined were: subjective rating of symptoms and complaints through validated symptom questionnaires (Swedish Performance Evaluation System), conjunctival redness (digital photos), eye-blinking frequency (video, two counters blind to doses), self-reported tear film break-up time (time to first close of eye, staring at mark on wall), and nasal flow rates. Influence of personality factors on the volunteer's subjective scoring was also examined (Positive and Negative Affect Schedule). Exposure to 0.7 ppm [860 µg/m^3] for 4 hours, and 0.4 ppm for 4 hours with 0.8 ppm [980 µg/m^3] peaks caused no significant sensory irritation of the measured conjunctival and nasal parameters. No differences between hypo- and hyper-sensitive subjects were seen. Statistically significant differences noted for olfactory symptoms, notably the "perception of impure air". Subjective complaints were more pronounced in hypersensitive subjects.
Experimental Animal Studies			
Rusch *et al.,* 1983	Cynomolgus monkey (6 males per dose).	Inhalation of cumulative mean exposures of 0, 0,	Histological examination of the lungs, trachea, and nasal turbinates. Monkey – hoarseness, congestion, (nasopharyngeal irritation) and squamous cell

Reference	Population / species	Concentrations / exposures	Study Findings
	Fischer F344 rats (20 males and 20 females per dose). Syrian Golden Hamsters (10 males and 10 females per dose).	0.19, 0.98, and 2.95 ppm [0, 0, 230, 1200, 3600 µg/m^3] of formaldehyde across 22 hr per day, 7 days per week, for 26 weeks.	metaplasia in the nasal turbinates of high-concentration group only (2.95 ppm [3600 µg/m^3]). No signs of toxicity in lower exposure groups. Rat – squamous cell metaplasia in nasal turbinates, decreased body weight and decreased liver weights in the high-concentration group only (2.95 ppm [3600 µg/m^3]). Hamsters – no responses of note to any exposure. No treatment-related mortality was reported during the study. Authors concluded the monkey and rat species were more sensitive to inhalation of formaldehyde than the hamster. It is noted there was no exposure-free time for repair of lesions, nor any analyses on the reversibility of effects.
Appelman *et al.,* 1988	SPF Wistar rats (male), 20 per dose group. Per dose group, 50% of animals had their nasal mucosa damaged by electro-coagulation prior to formaldehyde	Exposure to atmospheres of 0, 0.1, 1, or 10 ppm [0, 123, 1230, or 12300 µg/m^3] formaldehyde vapour for 6 hr/day for 5 days/week across 13 or 52 weeks.	Study designed to investigate the effect of bilateral intranasal electrocoagulation damage on the susceptibility of rats to formaldehyde vapour. Electrocoagulation damage was induced in the anterior third part of the nose. The repair process followed the pattern of wound healing. Loss of turbinates and perforation of the septum were common irreversible findings. After 13 weeks basal cell hyperplasia and squamous metaplasia of the respiratory epithelium, and rhinitis were still visible. After 52 weeks effects attributable to electrocoagulation were slight basal cell hyperplasia and some rhinitis. Major formaldehyde-related adverse effects in the 10 ppm [12300 µg/m^3] group not subjected to electrocoagulation included growth retardation, reduced urine production, and rhinitis accompanied by squamous

Reference	Population / species	Concentrations / exposures	Study Findings
	exposures.		metaplasia of the nasal respiratory epithelium. No adverse effects were seen at 0.1 or 1 ppm [123 or 1230 µg/m^3] in rats with an intact nasal mucosa. The principal untoward effects of formaldehyde in electrocoagulation-treated rats seen after 13 and/or 52 weeks comprised increase in basal cell hyperplasia, squamous metaplasia of the nasal respiratory epithelium, damage to the olfactory epithelium at 10 ppm [12300 µg/m^3], and focal squamous metaplasia of nasal respiratory epithelium at 0.1 and 1 ppm [123 and 1230 µg/m^3]. Major conclusions were: (a) the rat nose damaged by electrocoagulation is more susceptible to the cytotoxic action of formaldehyde than the undamaged nose, (b) 10 ppm [12300 µg/m^3] formaldehyde has no adverse effect on organs remote to the site of entry in rats with an undamaged nose, and (c) 1 ppm [1230 µg/m^3] formaldehyde did not visibly affect the intact nasal epithelium. In groups without pre-damaged nasal mucosa, exposure-related effects were restricted to rhinitis and hyperplasia and metaplasia of the nasal respiratory epithelium in the 10-ppm [12300 µg/m^3] group. Comprehensive histological examination of major tissues and organs in the control and 10-ppm [12300 µg/m^3] groups revealed no other exposure-related lesions. Microscopic examination of nose sections from the 1- and 0.1-ppm [123 and 1230 µg/m^3] groups without electrocoagulation revealed no exposure-related effects. Rats with pre-damaged nasal mucosa were more susceptible to the cytotoxic action of formaldehyde; at 52 weeks, focal squamous metaplasia of the nasal respiratory epithelium was found in rats exposed to 0.1 or 1 ppm [123 and 1230 µg/m^3] formaldehyde.

Reference	Population / species	Concentrations / exposures	Study Findings
Woutersen *et al.,* 1989	Male Wistar rats, 30 animals per group.	Exposed to formaldehyde (generated from paraformaldehyde) in dynamic whole-body chambers. 6 hours/day, 5 days/week for 3 or 28 months. All survivors sacrificed at 28 months. Concentrations were 0, 0.1, 1.0, or 10 ppm [0, 123, 1230, 12300 µg/m^3] per study abstract.	No treatment-related changes at 123−1230 µg/m^3. In the animals exposed for 3 months at 12300 µg/m^3 [11300 µg/m^3 per EPA review] compound-related histopathological changes were found in the nose and comprised of increased incidences of squamous metaplasia of the respiratory epithelium and rhinitis at cross-sectional. In the animals exposed for 28 months to 12300 µg/m^3 [12100 µg/m^3 per EPA review] compound-related histopathological changes were found in the nose and comprised increased incidences of squamous metaplasia and basal cell/pseudo-epithelial hyperplasia (PEH) of the respiratory epithelium at cross-sectional levels II and III, degeneration of the olfactory epithelium characterized by thinning/disarrangement at cross-sectional level, and rhinitis at all sections. Note: This study also evaluated the effects of formaldehyde in a parallel group of rats that had undergone bilateral electrocoagulation 20 to 26 hours prior to the initiation of formaldehyde exposure (not shown).

Section A.5.2 – Burden of Disease

The burden of disease from asthma associated with residential formaldehyde exposures was quantified recently in the published paper by Clark *et al*. (2023), using standard environmental burden of disease (EBD) epidemiological approaches (see Hänninen *et al*. 2020 & Pruss-Ustun *et al.* 2003 for methods). The EBD concept is a way of quantifying the number of new cases of disease, mortalities, and/or some other measure of health burden (i.e., Disability Adjusted Life Years Lost) that are attributable to an exposure being distributed within a specific population over a specific period of time (Plas *et al.* 2019). Disability Adjusted Life Years lost (DALYs) is a measure of health burden taking into account mortality and morbidity burden associated with a disease in a population. To calculate the EBD, first, the exposure of the selected population towards an environmental risk factor is estimated. In a second step, exposure data and information from an exposure-response-function are combined by using the population attributable fraction formula. In a last step, the attributable fraction (percentage) is multiplied by the disease burden resulting from a selected health outcome to estimate the share of disease burden attributable to the environmental risk factor (Plas *et al.* 2019).

Clark *et al*. (2023), focused on children (aged 0-14) as they are a vulnerable group and because of the robust epidemiological evidence for this age group. They estimated a distribution of residential concentrations of formaldehyde across dwellings in England (see Section 3.3.2 for details). They then combined the exposure distribution with exposure-response relationships identified from the two most recently published meta-analyses of epidemiological studies (meta-analysed odds ratio: 1.20) (Lam *et al*. 2021; Liu *et al*. 2023) to estimate a Population Attributable Fraction (PAF) (see Glossary for definition) for childhood asthma diagnosis/self-report. Clark *et al*. (2023) estimated that **formaldehyde concentrations in English residences was associated with contributing to 2.5% (95% confidence interval (CI): 1.3-3.9%) of asthma cases among children in England**, resulting in approximately **800 Disability Adjusted Life Years (DALYs) lost** (95% CI: 246 – 2,021) among this age group in 2019 (8 DALYs per 100,000 children). The authors also noted that the available epidemiological studies used for these quantifications can only provide information on associations with asthma diagnosis or self-report. So, while it is possible that formaldehyde can contribute to the development of asthma, it is also possible that the irritant properties of formaldehyde are triggering symptoms, which then, in turn, can lead to diagnosis.