ANNEX XV RESTRICTION REPORT

PROPOSAL FOR A RESTRICTION

SUBSTANCE NAME: Formaldehyde and formaldehyde releasers

IUPAC NAME: Formaldehyde and formaldehyde releasers

EC NUMBER: 200-001-8

CAS NUMBER: 50-00-0

CONTACT DETAILS OF THE DOSSIER SUBMITTER:

European Chemicals Agency

Annankatu 18, Helsinki, Finland

VERSION NUMBER: 1.1

DATE: 20 March 2019

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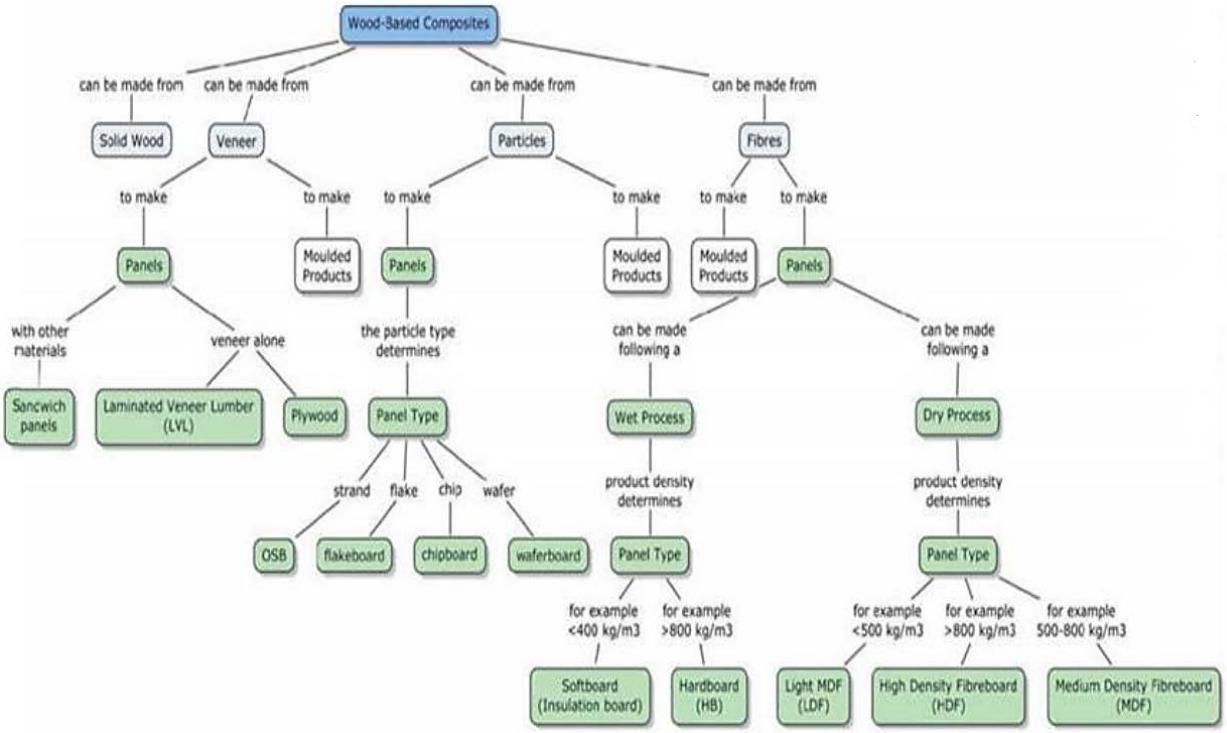
Annex A: Manufacture and uses

A.1. Main types of wood-based panels

According to WPIF/TRADA/TTF (2014), wood-based panel products are panel materials in which wood is predominant in the form of strips, veneers, chips, strands or fibres. Figure 1 provides an overview of the main categories of wood-based panels, which usually comprise plywood, particleboard, oriented strand board (OSB), and fibreboard. The information on the different panel types presented in this Annex are taken directly from WPIF/TRADA/TTF (2014) and information on their application from EPF (2017).

Figure A.1: Types of wood-based panels



Source: Formacare (2018)

Plywood

The term “plywood” includes the true “veneer plywood” and also “blockboard” and “laminboard”. Veneer plywood is generally made from veneers that are peeled from a log. These are bonded together with an adhesive that is appropriate to the end use, with the grain of adjacent veneers generally at right angles to each other. The adhesive is cured by pressing the panel using heated platens. Blockboard and laminboard are produced in a similar fashion to plywood except that the core of the material is made up from strips of solid wood or veneer laid on edge and this core is then faced with two or more veneers on each side. Plywood is a versatile product that can maintain a high performance under a wide variety of environmental conditions. Its construction enables comparatively high strength to weight ratios which are predominantly influenced by the species used. It is available in a range of wood species (both hardwood and softwood), some of which can have an attractive surface appearance, and a range of glue types for interior and exterior conditions (WPIF/TRADA/TTF, 2014).

In 2016, the main applications of plywood were construction (40%) and furniture (28%) followed by transport (14%) and packaging (9%). The remaining 9% was used for other applications (EPF, 2017).

Particleboard

Particleboard made from wood is also known as wood chipboard. Wood chips comprise the bulk of particleboard and are prepared in a mechanical chipper generally from coniferous softwoods, principally spruce (although pine and fir and hardwoods, such as birch, are sometimes used). Particleboards may also incorporate a large proportion from recycled sources. These chips are generally bound together with synthetic resin systems such as urea formaldehyde (UF) or melamine urea formaldehyde (MUF), though phenol formaldehyde (PF) and methylene di-isocyanate (MDI) are used by a few manufacturers. The chips are formed into a mat and are then pressed between heated platens to compress and cure the panel. The finished panels are then sanded and cut to size (WPIF/TRADA/TTF, 2014).

The furniture industry is the largest end-user of particleboard in Europe. In 2016, a share of 66% of the overall particleboard sales in Europe went to the furniture sector. The building industry, including doors and flooring applications, accounted for a share of 22% of the overall particleboard consumption. The remaining 12% of the particleboard consumption went to other applications such as packaging (EPF, 2017).

Oriented strand board (OSB)

OSB is an engineered wood-based panel material in which long strands of wood are bonded together with a synthetic resin adhesive. OSB is usually composed of three layers with the strands of the outer two layers orientated in a particular direction, more often than not in the long direction of the panel. While there is an orientation, it is often hard to see because there is quite a large degree of variability in this orientation among adjacent strands in the panels from any one production line, as well as between panels from different producers (WPIF/TRADA/TTF, 2014).

There are four OSB grades depending on load-bearing and ambient climatic conditions: OSB/1, OSB/2, OSB/3 and OSB/4. OSB/3 panels, i.e. load-bearing panels suitable for structural use in humid conditions, are the major category of OSB produced, accounting for approximately 85% of the European OSB output in 2016. OSB/2 panels, i.e. panels suitable for structural and non-structural use in dry conditions, accounted for 10% of the 2016 production. The remaining 5% of the European OSB production was devoted to the OSB/4 category. These heavy-duty load-bearing panels are suitable for structural use in dry and humid conditions where lots of swell resistance and strength are required. OSB production is mainly sold to the building industry and used in related applications such as sub-flooring, roofing construction and load-bearing applications (walls and ceilings). The remainder of the European OSB production is destined for packaging applications, flooring industry, furniture industry, the do-it-yourself sector, and other uses (EPF, 2017).

Fibreboard

Fibreboard can be further subdivided depending on the basic process method used in its production, i.e. wet process fibreboard and dry process fibreboard. Wet process fibreboard is made by reducing steamed wood into fibres and adding water to form a slurry. This is then formed into a mat on a moving wire mesh. During processing, much of the water is removed by pressing and the final heated pressing promotes bonding of the fibres using the adhesive properties of the natural lignin adhesive present in the wood. Depending upon the degree of

pressing involved and hence the final density of the panel, the product is termed softboard, mediumboard or hardboard. Additives are sometimes included to improve properties. In the case of dry process fibreboard, the wet fibres are dried and an adhesive is added. This is then formed into a mat and pressed in a similar way to particleboard. The resulting product is generally termed medium density fibreboard (MDF), which is not to be confused with the wet process mediumboard (WPIF/TRADA/TTF, 2014).

In 2016, the main applications of hardboard were packaging (27%), furniture applications (20%), and the do-it-yourself sector (20%). Automotive and construction uses accounted for 6% and 5%, respectively, with the remainder going to other uses. Softboard sales consisted mainly of building shells both rigid (45%) and flex (31%). Rigid underlays accounted for 10% and standard boards represented a share of 7% of total softboard sold. The furniture (45%) and laminate flooring applications (32%) sectors remained the main buyers of European MDF panels in 2016. Despite the popularity of renovation and do-it-yourself applications, sales to the building sector amounted to only 16%. The remaining 7% of the European MDF production went to moulding applications and to other applications such as outside panelling, small cabinets for home entertainment, frames, games, toys, garden furniture, etc. (EPF, 2017).

Annex B: Hazard, exposure and risk

B.1. Identity of the substance and physical and chemical properties

Table B.1: Substance identity of formaldehyde

Substance name	CAS number	EC Number	Reg	Source of information
Formaldehyde	50-00-0	200-001-8	> 1000 t	ECHA website

Table B.2: Substances identified as formaldehyde releasers

Substance name	CAS number	EC Number	Reg	Industrial use	Professional use	Consumer use
7a-ethyldihydro-1H,3H,5H-oxazolo[3,4-c]oxazole	7747-35-5	231-810-4	100-1000 t	Formulation for use in leather tanning		
1-[1,3-bis(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]-1,3-bis(hydroxymethyl)urea	78491-02-8	278-928-2	100-1000 t	Formulation		Consumer use not specified (processing aid)
1,3-bis(hydroxymethyl)-5,5-dimethylimidazolidine-2,4-dione	6440-58-0	229-222-8	100-1000 t	Use in manufacture of cosmetic products		Cosmetics
N,N''-methylenebis[N'-[3-(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]urea]	39236-46-9	254-372-6	100-1000 t	Formulation		Consumer use not better specified (processing aids)

Substance name	CAS number	EC Number	Reg	Industrial use	Professional use	Consumer use
methenamine	100-97-0	202-905-8	10000-100000 t	Production of polymers and rubber (curing agent). Use as intermediate in production of explosives Substance for which KEMI (Sweden) has suggested a limit value in construction products. Used in tyres to improve adhesion of rubber to brass coated steel cord or textile and hoses or belts to improve adhesion of rubber to textile. Used as Transported Isolated Intermediate in chemical synthesis of resins and other substances. Used as curing agent in phenolic and epoxy resins. Also used in production of explosives.	Professional use (not specified)	Consumer use (not specified)
2,2',2''-(hexahydro-1,3,5-triazine-1,3,5-triyl)triethanol	4719-04-4	225-208-0	10000-100000 t	Formulation for use in oilfield and treatment of hydro-carbons Scavenger for sulphide in refinery and/or oilfield application. (No free formaldehyde is being formed during this reaction.)		
dimethoxymethane	109-87-5	203-714-2	1000-10000 t	Formulation for use as processing aid and intermediate Used in chemical synthesis (e.g. in the synthesis of cyclic compounds or dimers). Used in the manufacture of chemicals	Professional use as processing aid and inclusion into matrix. Professional use of long life articles with high release (abrasive processing).	Consumer use as processing aid. Inclusion into matrix. Consumer use of long life articles with high release (abrasive processing).

Substance name	CAS number	EC Number	Reg	Industrial use	Professional use	Consumer use
4,4-Dimethyloxazolidine; 3,4,4-trimethyloxazolidine	81099-36-7 (ingred. 75673-43-7 and 51200-87-4)	257-048-2; 616-253-0				
4-[2-(Morpholin-4-ylmethyl)-2-nitro-butyl]morpholine; 4-(2-nitrobutyl) morpholine	37304-88-4 (ingred. 1854-23-5 and 2224-44-4)	218-748-3				
4,5-Dihydroxy-1,3-bis(hydroxymethyl)-imidazolidin-2-one, methylated-5,5-Dimethylimidazolidine-2, 4-dione, formaldehyde	68411-81-4; 26811-08-5	270-150-1; 500-052-9				
4,5-Dihydroxy-1,3-bis (hydroxymethyl)-imidazolidin-2-one; 1,3-Bis(hydroxymethyl) imidazolidin-2-one	1854-26-8; 136-84-5	205-264-2				
1,3-Bis(hydroxymethyl) -1,3-diazinan-2-one	3270-74-4	221-893-5				
1,3-Bis(hydroxymethyl) urea	140-95-4	205-444-0				
(Z)-3-(Bis(2-hydroxyethyl) amino)-2- (2-hydroxyethyl-(hydroxymethyl)amino) prop-2-en-1-ol	77044-78-1					
1,3,5-Triethyl-1,3,5-triazinane (b)	7779-27-3 (b)	231-924-4				
4,5-Dihydroxyimidazolidin-2-one	3720-97-6	223-070-6				

Substance name	CAS number	EC Number	Reg	Industrial use	Professional use	Consumer use
1-(Hydroxymethyl)-5,5-dimethyl-imi-dazolidine-2,4-dione	116-25-6	204-132-1				
2-Chloro-N -(hydroxymethyl)acetamide	2832-19-1	220-598-9				
Hydroxymethylurea	1000-82-4	213-674-8				
Paraformaldehyde	30525-89-4	608-494-5		A source of monomer in condensation polymerisation of aminoplast resins. Use as raw material in chemical reaction at industrial plants		
Polyoxymethylene melamine (INCI)	9003-08-1	618-354-1				
Polyoxymethylene urea (INCI)	9011-05-6	618-464-3				
Formaldehyde dibenzyl acetal	2749-70-4	628-635-4				
Propyleneglycol hemiformal	85338-22-3	286-695-3				
2-(Hydroxymethyl)-2-nitropropane-1,3-diol	126-11-4	204-769-5				
Imidazolidine-2,4-dione	461-72-3	207-313-3		Used as a research and development bonding agent.		
(Hydroxymethyl)-5,5-dimethyl-2-4-imidazolidinedione	27636-82-4	608-120-0				
3-(Hydroxymethyl)-5,5-dimethylimidazolidine-2,4-dione	16228-00-5	240-352-4				

Substance name	CAS number	EC Number	Reg	Industrial use	Professional use	Consumer use
Dimethoxymethane	109-87-5	203-714-2				
2-(Hydroxymethylamino)ethanol	34375-28-5	251-974-0				
Urea formaldehyde resins (UF)	68002-18-6	614-201-1				
Phenol formaldehyde resins (PF)	68610-07-1	614-660-8				
Melamine formaldehyde resins (MF)	68002-20-0	614-203-2				
Polyoxymethilenes (POM)	66455-31-0	613-936-5				
Methylene bis (dephenyl di-isocyanate) (MDI)	101-68-8	202-966-0				
1,4-Butanediol (BDO)	110-63-4	203-786-5				
Pentaerythritol (Penta)	115-77-5	204-104-9				

B.2. Self-classification

Table B.3: Formaldehyde releasers for which self-classification is provided

Substance name	CAS number	EC Number	Harmonised classification (CLP Regulation)	Self-classification	Reg
Formaldehyde	50-00-0	200-001-8	yes	Acute Tox. 3 Skin Corr. 1B Skin Sens. 1 Muta. 2 Carc. 1B	
7a-ethyldihydro-1H,3H,5H-oxazolo[3,4-c]oxazole	7747-35-5	231-810-4	no	Skin Irrit. 2 Skin Sens. 1 Eye Dam. 1 Acute Tox. 4 Aquatic Chronic 3 Eye Irrit. 2	100-1000 t
1-[1,3-bis(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]-1,3-bis(hydroxymethyl) urea	78491-02-8	278-928-2	no	Eye Irrit. 2 Skin Sens. 1 Aquatic Chronic 3 Muta. 2 Skin Irrit. 2 Eye Dam. 1	100-1000 t

Substance name	CAS number	EC Number	Harmonised classification (CLP Regulation)	Self-classification	Reg
1,3-bis(hydroxymethyl)-5,5-dimethylimidazolidine-2,4-dione	6440-58-0	229-222-8	no	Acute Tox. 4 Eye Irrit. 2 Resp. Sens. 1 Skin Sens. 1 Skin Irrit. 2	100-1000 t
N,N''-methylenebis[N'-[3-(hydroxymethyl)-2,5-dioxoimidazolidin-4-yl]urea]	39236-46-9	254-372-6	no	Skin Sens. 1B Eye Irrit. 2 Skin Sens. 1 Carc. 1B	100-1000 t
methenamine	100-97-0	202-905-8	yes	Flam. Sol. 2 Skin Sens. 1	10000-100000 t
2,2',2''-(hexahydro-1,3,5-triazine-1,3,5-triyl)triethanol	4719-04-4	225-208-0	yes	Acute Tox. 4 Skin Sens. 1	10000-100000 t
dimethoxymethane	109-87-5	203-714-2	no	Flam. Liq. 2 Acute Tox. 4 STOT SE 2 Acute Tox. 1 Skin Irrit. 2 Eye Irrit. 2	1000-10000 t
4,4-Dimethyloxazolidine; 3,4,4-trimethyloxazolidine	81099-36-7 (ingred. 75673-43-7 and 51200-87-4)	257-048-2; 616-253-0	no	Flam. Liq. 3 Acute Tox. 4 Skin Irrit. 2 Eye Dam. 1	
4-[2-(Morpholin-4-ylmethyl)-2-nitro-butyl]morpholine; 4-(2-nitrobutyl) morpholine	37304-88-4 (ingred. 1854-23-5 and 2224-44-4)	218-748-3	no	Acute Tox. 4 Skin Irrit. 2 Skin Sens. 1 Eye Dam. 1 Aquatic Acute 1	
4,5-Dihydroxy-1,3-bis(hydroxymethyl)-imidazolidin-2-one, methylated-5,5-Dimethylimidazolidine-2, 4-dione, formaldehyde	68411-81-4; 26811-08-5	270-150-1; 500-052-9	no	no	
4,5-Dihydroxy-1,3-bis (hydroxymethyl)-imidazolidin-2-one; 1,3-Bis(hydroxymethyl) imidazolidin-2-one	1854-26-8; 136-84-5	205-264-2	no	Acute Tox. 4 Eye Irrit. 2 Skin Sens. 1 Carc. 2	
1,3-Bis(hydroxymethyl)-1,3-diazinan-2-one	3270-74-4	221-893-5	no	no	

Substance name	CAS number	EC Number	Harmonised classification (CLP Regulation)	Self-classification	Reg
1,3-Bis(hydroxymethyl) urea	140-95-4	205-444-0	no	Eye Irrit. 2 Acute Tox. 4 Skin Sens. 1 Resp. Sens. 1 Skin Irrit. 2 Eye Irrit. 2	
(Z)-3-(Bis(2-hydroxyethyl)amino)-2- (2-hydroxyethyl-(hydroxymethyl)amino) prop-2-en-1-ol	77044-78-1		no		
1,3,5-Triethyl-1,3,5-triazinane (b)	7779-27-3 (b)	231-924-4	no	Acute Tox. 4 Skin Irrit. 2 Eye Irrit. 2 Eye Dam. 1	
4,5-Dihydroxyimidazolidin-2-one	3720-97-6	223-070-6	no	Skin Irrit. 2 Eye Irrit. 2	
1-(Hydroxymethyl)-5,5-dimethyl-imidazolidine-2,4-dione	116-25-6	204-132-1	no	Acute Tox. 4 Skin Sens. 1 Skin Irrit. 2 Eye Irrit. 2	
2-Chloro-N -(hydroxymethyl)acetamide	2832-19-1	220-598-9	no	Skin Corr. 1B Eye Dam. 1 Acute Tox. 4 Skin Irrit. 2 Eye Irrit. 2 Skin Sens. 1 Muta. 2 Aquatic Chronic 3	
Hydroxymethylurea	1000-82-4	213-674-8	no	no	
Paraformaldehyde	30525-89-4	608-494-5	no	Flam. Sol. 2 Acute Tox. 4 Skin Irrit. 2 Skin Sens. 1 Eye Dam. 1 Resp. Sens. 1	
Polyoxymethylene melamine (INCI)	9003-08-1	618-354-1	no		
Polyoxymethylene urea (INCI)	9011-05-6	618-464-3	no		
Formaldehyde dibenzyl acetal	2749-70-4	628-635-4	no	Eye Irrit. 2 Aquatic Acute 1	
Propyleneglycol hemiformal	85338-22-3	286-695-3	No	Not classified	
2-(Hydroxymethyl)-2-nitropropane-1,3-diol	126-11-4	204-769-5	no	Acute Tox. 4 Aquatic Acute 1 Aquatic Chronic 1 Skin Irrit. 2 Eye Irrit. 2 STOT SE 3	

Substance name	CAS number	EC Number	Harmonised classification (CLP Regulation)	Self-classification	Reg
Imidazolidine-2,4-dione	461-72-3	207-313-3	no	not classified	
(Hydroxymethyl)-5,5-dimethyl-2-4-imidazolidinedione	27636-82-4	608-120-0	no	not classified	
3-(Hydroxymethyl)-5,5-dimethylimidazolidine-2,4- dione	16228-00-5	240-352-4	no	Skin Irrit. 2 Eye Irrit. 2 STOT SE 3 Acute Tox. 4	
Dimethoxymethane	109-87-5	203-714-2	no	Flam. Liq. 2 Acute Tox. 4 STOT SE 2 Skin Irrit. 2 Eye Irrit. 2	
2-(Hydroxymethylamino)ethanol	34375-28-5	251-974-0	no	Acute Tox. 4 Skin Sens. 1 Eye Irrit. 2 Eye Dam. 1 Skin Irrit. 2 STOT SE 3	
Urea formaldehyde (UF) resins	68002-18-6	614-201-1	No	Aquatic Chronic 4 Aquatic Chronic 2 Skin Corr. 1C	
Phenol formaldehyde (PF) resins	68610-07-1	614-660-8	No	Skin Sens. 1 Eye Irrit. 2 Aquatic Chronic 3 Aquatic Chronic 1 Eye Dam. 1 Acute Tox. 4 Skin Irrit. 2 STOT SE 3 Skin Sens. 1B	
Melamine formaldehyde (MF) resins	68002-20-0	614-203-2	No	Aquatic Chronic 3 Skin Sens. 1 Acute Tox. 4 Carc. 2 Aquatic Chronic 3 Flam. Liq. 3 Eye Dam. 1	
Polyoxymethilenes (POM)	66455-31-0	613-936-5	No	Not classified	
Methylene bis (dephenyl di-isocyanate) (MDI)	101-68-8	202-966-0	STOT SE 3; Resp. Sens. 1; Skin Irrit. 2; Eye Irrit. 2	STOT SE 3; Resp. Sens. 1; Skin Irrit. 2; Eye Irrit. 2	
1,4-Butanediol (BDO)	110-63-4	203-786-5	No	Acute Tox. 4 STOT SE 3 Skin Irrit. 2	
Pentaerythritol (Penta)	115-77-5	204-104-9	No	Eye Irrit. 2	

B.3. Human health assessment

B.3.1. Endogenous formaldehyde

In humans (as in animals) formaldehyde is an essential metabolic intermediate in all cells. It is produced endogenously from serine, glycine, methionine and choline, and it is generated in the demethylation of N-, O- and S-methyl compounds. It is an essential intermediate in the biosynthesis of purines, thymidine and certain amino acids (IARC, 1995).

The endogenous concentration of formaldehyde in the blood of human subjects not exposed to formaldehyde was 2.61 ± 0.14 µg/g of blood (mean ± SE; range, 2.05-3.09 µg/g) (Heck et al., 1985), i.e. about 0.1 mmol/L. This concentration represents the total concentration of endogenous formaldehyde in the blood, both free and reversibly bound (IARC, 1995). In rats the concentrations of free and acid labile formaldehyde were 12.6, 6.03, 8.4, and 2.9 µg/g wet tissue weight in the nasal mucosa, liver, testes, and brain, respectively (Heck et al., 1982).

Human exhaled air contains formaldehyde in concentrations in the order of 0.001 to 0.01 mg/m^3, with an average value of about 0.005 mg/m^3 (WHO, 2010).

B.3.2. Toxicokinetics (absorption, distribution, metabolism and excretion)

B.3.2.1. Absorption and distribution

Due to the high water solubility and reactivity, airborne formaldehyde is absorbed mainly in the upper respiratory tract, the site of first contact. The localisation of uptake in each species is determined by nasal anatomy, mucus coating and clearance mechanisms. At an exposure concentration of 1 ppm, predicted formaldehyde nasal uptake was 99.4%, 86.5%, and 85.3% in the rat, monkey, and human, respectively. At an exposure concentration of 1 ppb (0.001 ppm), predicted nasal uptake was 17.5% and 42.8% in the rat and monkey (Schroeter et al., 2014).

In biological systems, formaldehyde first reacts reversibly with water to form an acetal (methanediol). At physiological temperature and pH, > 99.9% of formaldehyde is present as methanediol, with < 0.1% as free formaldehyde (Andersen et al., 2010; Golden, 2011).

Formaldehyde reacts at the site of first contact virtually instantaneously with primary and secondary amines, thiols, hydroxyls and amides to form methylol derivatives. Due to its electrophilic properties, formaldehyde also reacts with macromolecules such as DNA, RNA and protein to form reversible adducts or irreversible cross-links (WHO, 2010).

The concentration of formaldehyde in the blood was not increased immediately after the exposure period in humans exposed to 1.9 ppm formaldehyde for 40 minutes, in rats exposed to 14.4 ppm for 2 hours (Heck et al., 1985), or in monkeys exposed to 6 ppm for 4 weeks (6 h/day, 5 days/week) (Casanova et al., 1988).

Formaldehyde is poorly absorbed following dermal application. Absorption appears to be limited to cell layers immediately adjacent to the point of contact and formaldehyde is rapidly metabolised at the initial site of contact. Due to rapid metabolism, distribution of formaldehyde molecules to other more distant organs is not likely, except from exposure to high concentrations (Lyapina et al., 2012).

B.3.2.2. Metabolism

The simplified metabolism of formaldehyde (acetal) involves (Andersen et al., 2010; Golden, 2011; Tulpule and Dringen, 2013; WHO, 2010):

1. reduction to methanol by alcohol dehydrogenase 1;
2. oxidation to formate by aldehyde dehydrogenase 2;
3. spontaneous reaction with glutathione (GSH) to form S-hydroxymethyl GSH, which is subsequently oxidised by alcohol dehydrogenase 3 (also known as formaldehyde dehydrogenase) to the intermediate S-formyl GSH, which is metabolised by S-formylglutathione hydrolase to formate and reduced glutathione.

Due to high circulating concentrations of glutathione in human blood, the S-hydroxymethyl GSH is the major form of formaldehyde seen *in vivo* (Sanghani et al., 2000).

Formate is oxidised to 10-formyl tetrahydrofolate (THF) by methylene tetrahydrofolate dehydrogenase 1; 10-formyl THF is either metabolised to CO_2 by 10-formyl THF dehydrogenase or further metabolised within the one-carbon metabolism pathway that is centred around folate (Tulpule and Dringen, 2013).

B.3.2.3. Elimination

Formaldehyde disappears from the plasma with a half-time of about 1 to 1.5 minutes, most of it being converted to CO_2 and exhaled via the lungs shortly after exposure. Smaller amounts are excreted in the urine as formate salts and several other metabolites (WHO, 2010).

The fate of inhaled formaldehyde was studied by Heck et al. (1983) in Fischer 344 rats exposed to ^{14}C-formaldehyde (at 0.63 or 13.1 ppm [0.8 or 16.0 mg/m^3]) for 6 hours. About 40% of the inhaled ^{14}C was eliminated as expired ^{14}C-carbon dioxide over a 70-hour period; 17% was excreted in the urine, 5% was eliminated in the faeces and 35% to 39% remained in the tissues and carcass. Elimination of radioactivity from the blood of rats after exposure by inhalation to 0.63 ppm or 13.1 ppm ^{14}C-formaldehyde is multiphasic. After inhalation, the terminal half-time of the radioactivity in the plasma was approximately 55 hours (IARC, 2006).

B.3.2.4. Gene expression

Andersen et al. (2010) examined the concentration and exposure duration transitions in formaldehyde mode of action (MOA) with pharmacokinetic (PK) modelling for tissue formaldehyde acetal and glutathione (GSH) and with histopathology and gene expression in nasal epithelium from rats exposed to 0, 0.7, 2, 6, 10, or 15 ppm formaldehyde (6 h/day) for 1, 4, or 13 weeks. Patterns of gene expression varied with concentration and duration. At 2 ppm, sensitive response genes (SRGs) – associated with cellular stress, thiol transport/reduction, inflammation, and cell proliferation – were upregulated at all exposure durations. At 6 ppm and greater, gene expression changes showed enrichment of pathways involved in cell cycle, DNA repair, and apoptosis. ERBB, EGFR, WNT, TGF-*b*, Hedgehog, and Notch signalling were also enriched. Benchmark doses for significantly enriched pathways were lowest at 13 weeks. Transcriptional and histological changes at 6 ppm and greater corresponded to dose ranges in which the PK model predicted significant reductions in free GSH and increases in formaldehyde acetal. Genomic changes at 0.7 to 2 ppm likely represent changes in extracellular formaldehyde acetal and GSH.

B.3.2.5. DNA-protein crosslinks

In rats exposed to 0.3, 0.7, 2, 6, or 10 ppm ^{14}C-formaldehyde for 6 hours, DNA-protein crosslinks (DPX) occurred at all concentrations. The formation of crosslinks was interpreted in terms of a nonlinear pharmacokinetic model incorporating oxidation of inhaled formaldehyde as a defence mechanism. The slope of the fitted concentration-response curve at 10 ppm is 7.3-fold greater than at 0.3 ppm, and the detoxification pathway is half-saturated at an airborne concentration of 2.6 ppm (Casanova et al., 1989).

In rhesus monkeys exposed to 0.7, 2, or 6 ppm ^{14}C-formaldehyde for 6 hours, DPX concentration was highest in the mucosa of the middle turbinates; lower concentrations were produced in the anterior lateral wall/septum and nasopharynx. Very low concentrations were found in the larynx/trachea/carina and in the proximal portions of the major bronchi of some monkeys exposed to 6 ppm but not to 0.7 ppm. No cross-links were detected in the maxillary sinuses or lung parenchyma (Casanova et al., 1991).

B.3.2.6. DNA adducts

Lu et al. (2010) used a very sensitive method to differentiate DNA adducts and DNA-DNA crosslinks originating from endogenous and inhalation-derived formaldehyde exposure. Exposure of rats to 10 ppm [$^{13}CD_2$]-formaldehyde for 1 to 5 days (6 h/day) induced mainly labile DNA (N^2-hydroxymethyl-dG; dG) monoadducts, but not N^6-HO$^{13}CD_2$-deoxyadenosie (dA) monoadducts. Such adducts were found only in the respiratory nasal mucosa but not at sites remote to the port of entry. In contrast, endogenous formaldehyde dG and dA monoadducts were present in all tissues examined.

In a further experiment Lu et al. (2011) exposed rats to 0.7, 2, 5.8, 9.1, or 15 ppm [$^{13}CD_2$]-formaldehyde for 6 hours and investigated N^2-hydroxymethyl-dG adducts in nasal DNA. The number of exogenous N^2-hydroxymethyl-dG adducts induced was 0.039 ± 0.019, 0.19 ± 0.08, 1.04 ± 0.24, 2.03 ± 0.43, and 11.15 ± 3.01 adducts/10^7 dG for 0.7, 2.0, 5.8, 9.1, and 15.2 ppm [$^{13}CD_2$]-formaldehyde, respectively. Thus, the exogenous adducts were formed in a highly nonlinear fashion, as demonstrated by the fact that a 21.7-fold increase in exposure (0.7 to 15.2 ppm) formed 286-fold higher amounts of exogenous DNA adducts in rat nasal epithelium. In contrast, the amount of endogenous N^2-hydroxymethyl-dG in nasal DNA of rats calculated for all rats combined was 4.7 ± 1.8 adducts/10^7 dG.

B.3.2.7. Protein adducts

Binding of formaldehyde to free amino groups of proteins (albumin) was reversible within the first 60 minutes (Bogdanffy et al., 1987).

Edrissi et al. (2017) investigated the formation of the protein adduct N^6-formyllysine in rats exposed to 2 ppm [$^{13}CD_2$]-formaldehyde for 7, 14, 21, and 28 days (6 h/day). The results showed formation of exogenous protein adducts in nasal epithelium and to some extent in trachea but not in distant tissues of lung, bone marrow, or white blood cells, with a 2-fold increase over endogenous N^6-formyllysine over a 3-week exposure period. Post-exposure analyses indicated a bi-exponential decay of N^6-formyllysine in proteins extracted from different cellular compartments, with half-lives of ~25 and ~182 hours for the fast and slow phases, respectively, in cytoplasmic proteins.

B.3.3. Acute toxicity

Formaldehyde is acutely toxic following ingestion, dermal and inhalation exposure and has the following classifications: Acute Tox. 3; H331; Acute Tox. 3; H311; Acute Tox. 3; H301.

In the Chemical Safety Report (BASF, 2017) the LC_{50} of formaldehyde is reported with < 463 ppm. The test was performed in the year 2015 following OECD Guideline 403 in rats with 4 hours whole-body exposure. All animals died on study day 1 or 2. Consequently, the registrant self-classified formaldehyde as Acute Tox. 2 (H330, fatal if inhaled).

B.3.4. Irritation

B.3.4.1. Experimental animals

In concentrations between 5 and < 25 %, formaldehyde has irritating properties: Skin Irrit. 2; H315: 5 % ≤ C < 25 %; Eye Irrit. 2; H319: 5 % ≤ C < 25 %.

Formaldehyde is also irritating to the respiratory tract: STOT SE 3; H335: C ≥ 5 %.

B.3.4.2. Human data

The most sensitive effects in humans following inhalation exposure to formaldehyde is sensory irritation. This reaction is initiated by an interaction of local irritants with receptors of the nervous system (e.g., trigeminal nerve endings) and a downstream cascade of reflexes and defence mechanisms (e.g., eye blinking, coughing). While the first stages of this pathway are thought to be completely reversible, high or prolonged exposure can lead to neurogenic inflammation and subsequently tissue damage. The second, "tissue irritation" pathway starts with the interaction of the local irritant with the epithelial cell layers of the eyes and the upper respiratory tract. Adaptive changes are the first response on that pathway followed by inflammation and irreversible damages (Bruening et al., 2014).

High quality studies in volunteers are available examining sensory irritation under controlled exposure to formaldehyde, as described by SCOEL (2016):

"*In itself, an odour cue can increase reporting of symptoms (e.g. headache, nausea, and eye and throat irritation) due to stress-related perceptions, triggered by belief about potential toxicological risks; this is especially prominent among individuals with "environmental worry" and "negative affectivity", but symptom reporting may also be influenced by belief about (positive, neutral or negative) health effects of an odour.*

Studies with the controlled exposure of volunteers must be distinguished from epidemiological studies of persons exposed at the workplace or under certain environmental conditions. The most reliable data are obtained in controlled studies with volunteers. Studies of persons exposed at the workplace are less suitable for making quantitative statements, mainly because of uncertain levels of exposure. Approximately 150 scientific papers (animal studies, human volunteer and occupational studies) on FA effects were evaluated by a panel of independent experts convened by the Industrial Health Foundation (IHF) (Paustenbach et al., 1997). The data were indicative of a relatively wide individual susceptibility to irritation from FA. Data available for eye irritation from a total of 17 volunteer studies had been compiled and evaluated. The experts concluded that between 0 and 0.3 ppm there is no increase in eye irritation above the general background level of about 10-20%, and irritation below 0.3-0.5 ppm FA was too unreliable to attribute the irritation solely to FA. A concentration-effect curve was constructed showing that at 0.5-1 ppm, exposure for up to 6 hours can produce eye irritation in 5–25% of the exposed persons, although responses below 20% were often not

considered attributable to FA alone. It was concluded, based on the controlled human and epidemiological studies, that at 0.3 ppm or less no irritation attributable to FA should occur, if people are exposed up to 8 hours per day. Significant increases in eye irritation are reported, however, only at concentrations of at least 1 ppm, which is the reason that this concentration is often regarded as a ceiling value (Paustenbach et al., 1997). Similar reviews with a partly overlapping database were carried out by Bender (2002) and Arts et al. (2006) basically coming to the same conclusions. It must be taken into consideration that apart from one study all the others reviewed only relied on reporting of subjective symptoms for sensory eye irritation.

The question of a threshold for chemosensory irritation was experimentally addressed by (Lang et al., 2008). Twenty-one volunteers (11 males, 10 females) were examined over a 10-week period using a repetitive design. Each subject was exposed to 10 exposure conditions on 10 consecutive working days, each for 4 hours. FA exposures were 0 (control), 0.15, 0.3 and 0.5 ppm, respectively. Also, a group with 0.3 ppm FA exposure with 4 peaks, each with a duration of 15-min, at 0.6 ppm and a group exposed at 0.5 ppm with 4 peaks at 1 ppm were included. Furthermore, ethyl acetate was used to mimic or mask the odour of FA. Thus, ethyl acetate alone (another control group), and 0.3 and 0.5 ppm FA groups were added ethyl acetate, as was a group with 0.5 ppm FA with peaks at 1 ppm. The ethyl acetate concentrations were 12-16 ppm. During exposure, subjects had to perform three cycle ergometer units at 80 watts for 15 min. Apart from reporting of subjective symptoms for irritation, measurements were related to objective effects of FA exposures as conjunctival redness, blinking frequency, nasal flow and resistance, pulmonary function and reaction times. Blinking frequency and conjunctival redness (ranging from slight to moderate) were significantly increased at 0.5 ppm with peak exposures, but no increase was observed at 0.5 ppm alone. FA had no effect on the other objective parameters. Results of subjective ratings (score for total symptom, eye irritation, nasal irritation, olfactory symptoms, respiratory irritation, and annoyance) were highly variable as indicated from the SDs and the maximum scores; the prerequisite (normal distribution) for the ANOVA testing was not reported. The total symptom score was increased only at 0.5 ppm with peaks at 1 ppm. The eye irritation score was increased at 0.3 and 0.5 ppm FA compared to the 0 ppm FA group; the mean symptom rating was below "slight". However the increases were not exposure-dependent and they were similar to that in the ethyl acetate (odour) control group. The 0.5 ppm group with peak exposures had significantly higher score than the two control groups; eye irritation was on average less than "somewhat". Nasal irritation was similar in the FA groups, 0.3, 0.3 with peaks and 0.5 ppm alone, and the ethyl acetate (odour) control group and not different from the 0 ppm control group; the 0.5 ppm FA group with peaks had a significantly higher score than the two control groups. An exposure-dependent significant respiratory irritation score was only reported at the 0.5 ppm with peaks, but this was not significantly different from the ethyl acetate (odour) control group; the mean symptom rating was below "slight". Olfactory symptom scores were increased in ≥ 0.3 ppm FA exposure groups compared with the 0 ppm control group. The ratings in the 0.3 group with peaks, the 0.5 group alone and the 0.5 ppm group with peaks were similar to the ethyl acetate control group. Annoyance was increased in the 0.3 group with peaks, the 0.5 group and the 0.5 ppm group with peaks compared with the 0 ppm control group. When negative affectivity was introduced as a covariate, the level of 0.3 ppm was no longer an effect level, but 0.5 ppm with peaks of 1.0 ppm was. The authors concluded that eye irritation was the most sensitive parameter recorded, and that the no-observed-adverse-effect level for objective eye irritation was 0.5 ppm. The similar value was observed for subjective eye irritation if odour bias and negative affectivity were included in the evaluation. The LOAEC was 0.5 ppm with peaks at 1 ppm. No sex differences were noted.

In view of open questions resulting from this study, a new exposure study in volunteers was conducted to examine chemosensory effects of FA in so-called "hyposensitive" and "hypersensitive" persons (Mueller et al., 2013). Forty-one male volunteers (aged 32 years ± 9.6) were exposed for 5 days (4 hours per day) in a randomised schedule to the control condition (0 ppm) and to FA concentrations of 0.5 and 0.7 ppm and to 0.3 ppm with peak exposures of 0.6 ppm, and to 0.4 ppm with peak exposures of 0.8 ppm, respectively. Peak exposures were carried out four times a day over a 15-min period. During exposure, subjects had to perform four cycle-ergometer units at 80 watts for 15 min. Subjective pain perception induced by nasal application of carbon dioxide (CO_2) served as indicator for sensitivity to sensory nasal irritation. The division between "hypersensitive" and "hyposensitive" subjects was based on the median in sensitivity towards the irritating effect of CO_2. The following parameters were examined before and after exposure: subjective rating of symptoms and complaints (Swedish Performance Evaluation System, SPES), conjunctival redness, eye-blinking frequency, self-reported tear film break-up time and nasal flow rates. In addition, the influence of personality factors on the volunteer's subjective scoring was examined (Positive And Negative Affect Schedule, PANAS). FA exposures to 0.7 ppm for 4 hours and to 0.4 ppm for 4 hours with peaks of 0.8 ppm for 15 min caused no significant sensory irritation of the measured conjunctival and nasal parameters (conclusion by the authors). In all groups, the mean sum score of the individual symptoms, the eye irritation score and the nasal irritation score were within a range of less than 2.5 mm on a 100-mm Visual Analogue Scale (VAS). No differences between hypo- and hypersensitive subjects were seen. Statistically significant differences were noted for olfactory symptoms, especially for the "perception of impure air". These subjective complaints were more pronounced in hypersensitive subjects. But after a detailed analysis the authors concluded that these effects were mainly induced by unpleasant smell and the situational and climatic conditions in the exposure chamber. FA concentrations of 0.7 ppm for 4 hours and of 0.4 ppm for 4 hours with peaks of 0.8 ppm for 15 min did not cause adverse effects related to irritation, and no differences between hypo- and hypersensitive subjects were observed (Mueller et al., 2013). Interestingly, Lang et al. (2008) observed subjective symptoms of eye irritation at concentrations upward of 0.3 ppm, but not Mueller et al. (2013). This was explained by differences in the study populations because the PANAS score for negative affectivity in the Lang study was significantly higher ($p < 0.02$) as compared to that in the Mueller study. This finding underlines in as much subjective symptoms may be influenced by personality factors like expectation or anxiety.

The study was accompanied by satellite investigations (Zeller et al., 2011). The results indicated that despite large differences in CO_2 sensitivity (see above), the susceptibility towards nasal irritation was not related to the induction of genotoxic effects (DPX, SCEs) in peripheral blood or the protection of blood cells against FA-induced effects (expression of FDH, repair capacity for FA-induced DPX). There was no correlation between CO_2 sensitivity and the expression of FDH. There was also no close correlation between the various indicators of cellular sensitivity towards FA-induced genotoxic effects, and no subgroups were identified with particular mutagen sensitivity towards FA (Zeller et al., 2011). Moreover, investigations of potential individual susceptibility of human blood cells towards FA-induced genotoxicity indicated no biologically relevant differences with regard to various indicators of cellular sensitivity to genotoxic effects along with the expression of FDH and genetic polymorphisms of the glutathione S-transferases GSTT1 and GSTM1 (Zeller et al., 2012). The authors suggested that a low scaling factor to address possible human inter-individual differences in FA-induced genotoxicity could be reasonable. This is also supported by field studies investigating polymorphisms of glutathione S-transferases (Jiang et al., 2010; Santovito et al., 2011)."

A study by Berglund et al. (2012) determined the average (P50) absolute odour threshold (corrected for "false alarm") of formaldehyde to 0.1 ppm (range: 0.02-0.5 ppm). Overall, the odour response of formaldehyde occurs below observed toxicological effects (SCOEL, 2016).

B.3.5. Corrosivity

Formaldehyde has corrosive properties and has the classification: Skin Corr. 1B; H314, with a concentration limit C ≥ 25 %.

B.3.6. Sensitisation

B.3.6.1. Skin sensitisation

Formaldehyde is a known skin sensitiser, which has the classification: Skin Sens 1; H317. The concentration limit for mixtures for skin sensitisation is 0.2%.

Related to skin sensitisation, the registration dossier (BASF, 2017) clearly sets out that formaldehyde is a strong skin sensitiser with positive results in several studies including Local Lymph Node Assay (LLNA). Formaldehyde solution is a primary skin sensitiser inducing allergic contact dermatitis Type IV and may induce contact urticaria Type I (WHO, 1989). The EC3 value (3-fold stimulation of proliferation as an index of the relative potency of a contact allergen) was 0.93% formalin or 0.35% formaldehyde. No induction was detected at 0.04% formaldehyde and first sensitising effects were seen at 0.2% (BASF, 2017). This is consistent with the special concentration limit in CLP for substances in mixtures. Concentrations leading to elicitation of effects are lower than the concentrations leading to induction.

The biocidal assessment for formaldehyde (ECHA, 2017) concluded: *"However, the currently available methodology is not considered suitable for derivation of an acceptable exposure level protecting from sensitisation by formaldehyde which is relevant to human health. Nevertheless, the available data is in support of the current legal classification limit for formaldehyde formulations of ≥ 0.2% (w/w) with regard to its sensitising properties and the resulting labelling provisions with EUH208 at ≥ 0.02% (w/w)."*

B.3.6.2. Respiratory sensitisation

Formaldehyde might also lead to respiratory sensitisation. However, against the background of a widespread use, respiratory sensitisation has been reported only in single cases (DFG, 2010).

During the last decade a number of human exposure studies in children and adults have been carried out with lung function testing. From such studies WHO (2010) concluded 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. In general, associations between formaldehyde and lung effects or sensitisation in children in homes and schools have not been convincing owing to confounding factors and chance effects. Well known confounders for asthma are e.g. dust mites, cockroach allergen, pets or mould.

The German Umweltbundesamt (UBA, 2016) also reviewed the results from epidemiological studies investigating if there is an association between formaldehyde exposure and the induction or exacerbation of asthma in children. UBA concluded, that 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.

B.3.7. Repeated dose toxicity

B.3.7.1. Animal data

The repeated dose toxicity studies with inhalation exposure are summarised by SCOEL (2016):

"*In rats exposed to FA concentrations of 10 ppm, daily for 6 hours on 5 days a week, rhinitis, hyperplasia and squamous metaplasia of the respiratory epithelium of the nasal mucosa were described in all studies. In rats exposed to 1.0 ppm for 2 years no histopathological changes were observed (no observed adverse effect concentration, NOAEC; Woutersen et al. (1989)). From concentrations of 2 ppm, rhinitis, epithelial dysplasia and even papillomatous adenomas and squamous metaplasia of the respiratory epithelium of the nose were found, from 6 ppm squamous cell carcinomas (Kerns et al., 1983; Swenberg et al., 1980). At this concentration also the cell proliferation rate in the nasal mucosa was increased transiently, and from 10 ppm increased permanently (Monticello et al., 1996).*

Uninterrupted exposure of rats for 8 hours/day ("continuous") was compared with 8 exposures for 30 minutes followed by a 30-minute phase without exposure ("intermittent") in two 13-week studies with the same total dose. Effects were seen only after intermittent exposure to FA concentrations of 4 ppm, but not after continuous exposure to 2 ppm. The authors concluded that the toxicity in the nose depends on the concentration and not on the total dose (Wilmer et al., 1989).

In mice exposed to FA concentrations of 2.0, 5.6 or 14.3 ppm for 2 years (6 hours/day, 5 days/week), rhinitis and epithelial hyperplasia was observed, from 5.6 ppm dysplasia, metaplasia and atrophy. Squamous cell carcinomas were observed only after concentrations of 14.3 ppm (Kerns et al., 1983).

In hamsters exposed to FA concentrations of 10 ppm (5 hours/day, 5 days per week) for life, survival was reduced and the incidence of hyperplasia and metaplasia (4/88, 5%) was slightly increased, but not that of tumours (Dalbey, 1982).

In Cynomolgus monkeys exposed almost continuously to FA concentrations of 0.2, 1 or 3 ppm for 26 weeks, metaplasia and hyperplasia were observed in 1/6 and 6/6 animals of the 1 and 2 ppm groups, respectively. In the animals exposed to concentrations of 0.2 ppm, no histopathological changes were found (Rusch et al., 1983).

Reduced body weight gains were reported in rats exposed to FA concentrations from 10 ppm for 6 hours a day in a 13-week inhalation study (Woutersen et al., 1987) and in those exposed to concentrations from 5.6 ppm in a 2-year inhalation study (Kerns et al., 1983; Swenberg et al., 1980). In mice, reduced body weight gains were found in a 13-week inhalation study only at concentrations from 20 ppm. Other systemic effects were not observed in these studies. Only in a 26-week inhalation study with continuous exposure (22 hours a day, 7 days a week) were reduced absolute and relative liver weights observed from concentrations as low as 3 ppm (in addition to reduced body weight gain and lesions in the nasal region) (Rusch et al., 1983).

The findings in rats were reconfirmed after exposure of male F344 rats to concentrations of 0, 0.5, 1, 2, 6, 10 and 15 ppm (6 h/d, 5 d/week over 4 weeks). At 10 or 15 ppm clear site-

specific pathological changes (focal epithelial degeneration, inflammation and squamous metaplasia) were observed in a decreasing gradient (anterior to posterior) (Speit et al., 2011).

A study related to the possible induction of lympho-haematopoetic neoplasms has been carried out in Fischer-344 rats and B6C3F1 mice at exposure concentrations between 0.5 and 15 ppm over 4 weeks (Kuper et al., 2011). Nasopharynx-associated lymphoid tissues (NALT) and upper-respiratory tract-draining lymph nodes were studied by standard histopathology and immunohistochemistry for cell proliferation. The only effect noted was simple hyperplasia and increased proliferation rate of the lympho-epithelium of rats at 15 ppm. Therefore the study did not support the hypothesis that FA may induce such systemic neoplasms by reaction with local lymphoid cells."

Table B.4 summarises the key events.

Table B.4: Repeated dose toxicity studies

FA (ppm)		Effects	Species, exposure	References
0.2	NOAEC	No metaplasia or hyperplasia	Monkeys, 26 week inhalation exposure	Rusch et al. (1983)
1.0	NOAEC	No histopathological effects in the nose	Rats, 2 year inhalation exposure	Kerns et al. (1983), Swenberg et al. (1980), Woutersen et al. (1989)
2.0	LOAEC	Rhinitis, epithelial dysplasia, metaplasia		
6.0		Squamous cell carcinoma		
		Cell proliferation increased transiently		Monticello et al. (1996)
10		Cell proliferation increased permanently		

B.3.8. Mutagenicity

Formaldehyde has the following harmonised classification: Muta. 2; H341.

This classification is based on genotoxic effects observed *in vivo* in somatic cells at the site of contact. No evidence of an effect on germ cells by a relevant route of exposure is available (RAC, 2012).

SCOEL (2016) summarised the data: "*There is consistent evidence for the genotoxicity of FA in in vitro systems, laboratory animals and exposed humans. DNA-protein crosslinks have been reproducibly detected in the nasal mucosa of rats and monkeys exposed to FA and provide a useful marker of genotoxicity. The biphasic behaviour of the dose-response curve for this genotoxic endpoint points to a steeper slope at 2-3 ppm in Fischer 344 rats; for rhesus monkeys the slope is less well defined. At concentrations above 6 ppm of FA, genotoxicity is greatly amplified by cell proliferation, resulting in a marked increase of malignant lesions in the nasal passages (IARC, 2006).*"

The most sensitive effects in the nose and upper respiratory tract following inhalation formaldehyde exposure are DNA adducts and DNA-protein crosslinks.

DNA adducts (N^2-hydroxymethyl-dG adducts) were detected in the nasal DNA of rats exposed to 0.7, 2, 5.8, 9.1 or 15 ppm [$^{13}CD_2$]-formaldehyde for 6 hours. The number of exogenous N^2-

hydroxymethyl-dG adducts induced was 0.039 ± 0.019, 0.19 ± 0.08, 1.04 ± 0.24, 2.03 ± 0.43 and 11.15 ± 3.01 adducts/10^7 dG for 0.7, 2.0, 5.8, 9.1 and 15.2 ppm [$^{13}CD_2$]-formaldehyde, respectively (Lu et al., 2011). The concentration of endogenous N^2-hydroxymethyl-dG adducts was 4.7 ± 1.8 adducts/10^7 dG. Therefore, the exogenous N^2-hydroxymethyl-dG adducts formed following 0.7 ppm formaldehyde exposure were less than 1% of the endogenous N^2-hydroxymethyl-dG adducts.

DNA-protein-crosslinks (DPX) – the covalent linkage of proteins with a DNA strand – are one of the most deleterious and understudied forms of DNA damage, posing as steric blockades to transcription and replication. If not properly repaired, these lesions can lead to mutations, genomic instability, and cell death (Heck and Casanova, 2004). Endogenously, DPX are commonly derived through reactions with aldehydes, as well as through trapping of various enzymatic intermediates onto the DNA. Proteolytic cleavage of the protein moiety of a DPX is a general strategy for removing the lesion. This can be accomplished through a DPX-specific protease and/or proteasome-mediated degradation. Nucleotide excision repair and homologous recombination are each involved in repairing DPX, with their respective roles likely dependent on the nature and size of the adduct (Klages-Mundt and Li, 2017).

DPX have been identified in the nasal mucosa of rats and in the upper respiratory tract of monkeys exposed to formaldehyde but not in the bone marrow of rats exposed to ^3H and ^{14}C-formaldehyde at concentrations as high as 15 ppm. DPX formation in the nose was identified still at the lowest formaldehyde concentrations tested of 0.3 ppm in rats (Casanova et al., 1989) and 0.7 ppm in rhesus monkeys (Casanova et al., 1991).

In summary, taking into account the relatively high endogenous concentrations of formaldehyde and the endogenous mechanisms to repair DNA adducts and DPX formed by endogenous formaldehyde, exogenous formaldehyde concentrations that do not lead to a significant increase in endogenous formaldehyde levels are not expected to lead to a significant contribution in genotoxic effects.

B.3.9. Carcinogenicity

Formaldehyde has the harmonised classification Carc. 1B; H350.

The classification is mainly based on nasal tumours (site of contact) observed in rats of both sexes exposed to formaldehyde at concentrations of 2 ppm and higher for ≥ 24 months. Details on the data are reported in RAC (2012).

In Table B.5, nasal epithelial squamous cell carcinomas (SCC) in combined groups of male and female rats from long-term inhalation studies with formaldehyde exposures (Kamata et al., 1997; Kerns et al., 1983; Monticello et al., 1996; Sellakumar et al., 1985) are presented according to Nielsen et al. (2017):

Table B.5: Nasal epithelial squamous cell carcinomas (SCC) in rats

Formaldehyde (ppm)	Rats with SCC/group size (% with SCC)
0	0/453 (0)
0.3	0/32 (0)
0.7	0/90 (0)
2	0/364 (0) (apparent NOAEC)
6	3/325 (0.9) (apparent LOAEC)
10	20/90 (22)
14	102/232 (44)
15	120/278 (43)

Source: Nielsen et al. (2017)

B.3.9.1. U.S. EPA

U.S. EPA (1990) has derived a unit risk for human respiratory cancer of 1.3 x 10^{-5} per µg/m^3 based linear extrapolation from squamous cell carcinoma in male F344 rats (Kerns et al., 1983). U.S. EPA (2010) re-evaluated the cancer risk resulting in a unit risk of 0.13 per ppm (1.1 x 10^{-4} per µg/m^3). The inhalation unit risk is defined as the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/m^3 in air.

B.3.9.2. RAC

RAC (2012) concluded the following:

"Overall, the carcinogenicity of formaldehyde is well established in rats by inhalation with induction of tumours at the site of contact. Formaldehyde is highly cytotoxic and irritant and nasal tumours are observed only at doses producing chronic irritation, as evidenced by the accompanying inflammatory, hyperplastic and metaplastic responses. Among species, the degree of sensitivity to nasal irritation is associated with the degree of sensitivity to nasal tumour induction. Localisation of damage to the nasal epithelium also corresponds with tumour site and distribution is attributable to regional dosimetry and/or local tissue susceptibility.

A consistent database provides evidence that regenerative cell proliferation (RCP) secondary to cytolethality highly correlates with tumour incidence and regional distribution (of nasal tumours). RCP is observed at 10 and 15 ppm with 6 ppm being a borderline concentration (Casanova et al., 1994; Meng et al., 2010; Monticello et al., 1996). Besides, Woutersen et al. (1989) have demonstrated that nasal mucosa damage induced by pre-exposure to electrocoagulation treatment contributes to tumour induction.

Modelling studies (Conolly et al., 2004) have discussed the induction of proliferation in response to cytotoxicity and formation of DPX to explain the mechanism of nasal tumour induction and its particular dose-response relationship. At low doses, a delay in replication by DPX formation may induce a decrease in cellular proliferation, as supported by the observed J-shaped dose-response (Conolly et al., 2004), and it may allow the repair of DNA damage to occur. A delay in cell replication at low dose was, however, not confirmed by the findings of

(Meng et al., 2010) who observed a dose-related increase in cell proliferation which was statistically significant from 10 ppm. As discussed in the mutagenicity section, at low doses the incremental DNA damage may be repaired due to cell proliferation not being elevated. Therefore, the genotoxic potential of formaldehyde is not expected to give rise to mutagenicity at low doses.

At higher doses, cytolethality is followed by RCP. An increased rate of cell proliferation is associated with a larger probability of fixing a primary DNA lesion as a mutation and a decrease in the time available for DNA repair. The observed hyperplastic and metaplastic changes strongly support the hypothesis of a mechanism driven by regenerative proliferation accompanied by an inflammatory response that may also result in secondary amplification of the high-dose genotoxic effects of formaldehyde. A steep increase in tumour induction is therefore expected at doses exerting cytotoxicity and RCP, as has been observed experimentally. It is also consistent with the induction of chromosomal aberrations at the site of contact at high doses (Dallas et al., 1992). Besides, saturation of the glutathione-mediated detoxification of formaldehyde may contribute to the non-linearity of the dose response (McGregor et al., 2006).

Experimental results and mechanistic data therefore support the existence of a threshold type dose-response for induction of nasal tumours, with regenerative cell proliferation being the predominant feature in the carcinogenic process. The genotoxicity of formaldehyde is also expected to play a role above this threshold.

Overall, there is no convincing evidence of a carcinogenic effect at distant sites or via routes of exposure other than inhalation […]

Overall, the database for low-dose effects is limited. The fact that the responses of key events below 2 ppm are non-significant, albeit dose-related, may lead to consideration of the possibility of a threshold mode of action. However, the data does not allow a firm conclusion on a threshold-mode of action or the identification of a threshold. Extrapolation from 2 ppm formaldehyde to lower concentrations may be linear or non-linear and no firm conclusion whether the carcinogenic response is primarily caused by a genotoxic or a cytotoxic mechanism is possible."

B.3.9.3. SCOEL

SCOEL (2016) concluded the following

"Due to the high water solubility and the high reactivity of FA, it shows intrinsic hazardous properties predominantly with respect to local effects. In addition, directly induced systemic effects of inhalation at concentrations relevant for the workplace are considered unlikely. The following key effects were considered as being relevant for the protection of workers and in particular the OEL derivation:

a) the potential of the substance to produce respiratory irritation and chemosensory effects, both in humans and animals, and

b) the local carcinogenicity in studies with experimental animals exposed by inhalation.

Ad (a): Sensory irritation has been investigated in experimental animals, in exposed workers, and most importantly also under controlled exposures in volunteers.

Ad (b): Tumour induction of the upper respiratory tract has been studied in experimental animals including mechanistic investigations on events that will trigger carcinogenesis, like

DNA-protein crosslinks (DPX), DNA-adducts and sustained cytotoxicity leading to cell proliferation. In addition, several high quality epidemiological studies are available on exposed workers. A review by RAC (2012) concluded that these data would not provide sufficient evidence to classify FA as a human carcinogen but a classification as Cat. 1B carcinogen (H350 "May cause cancer"; based on CLP criteria) would be appropriate.

Mechanistic studies have provided strong evidence that tumour induction in the nasal mucosa of rats and mice is the result of chronic proliferative processes caused by the cytotoxic effects of the substance in combination with DNA alterations by endogenous and exogenous FA. The dose-response relationships for all parameters investigated, such as damage to the nasal epithelium, cell proliferation, tumour incidence, the formation of DPX and DNA-adducts, is very flat for low level exposures and becomes much steeper at higher concentrations. For these endpoints no-effect concentrations were demonstrated with the exception of the formation of DPX and DNA-adducts. However, at the lowest concentrations investigated so far (0.7 ppm), adducts caused by the endogenous, physiological FA by far exceeded the amounts caused by exogenous FA. The background incidence of nasal tumours in rodents and of nasopharyngeal tumours in humans is very low in spite of the appreciable amount of endogenous DNA adducts. One of the reasons may be the low physiological proliferation rate of the respiratory epithelium, and as long as this is not increased (which requires exposure to concentrations of more than 2 ppm), the probability of tumour formation also is low. At prolonged exposure at 2 ppm in rats, the half-life of the most sensitive biomarker of DNA-adducts, N^2-hydroxymenthy-dG, was 7 days. At 2 days of exposure in monkeys, the biomarker was estimated to be by a factor of 5-11 lower for the exogenous adduct than that of the endogenous adduct in the nasal epithelium. Comparing short term exposures, the relationship of exogenous/endogenous DNA–adducts was by a factor of about 5-fold lower for monkeys than for rats, suggesting monkeys being a less sensitive species than rats. Taking into consideration the strong non-linearity of the dose response curve after a single exposure at lower exposure concentrations, the ratio between exogenous/endogenous adducts will at low exposures be dominated by the endogenous adducts, but the ration will increase disproportionately with increasing FA concentrations. Also in the low dose range, cell proliferation is not increased. It has therefore been considered that the genotoxicity of FA plays no or at most a minor role in a potential carcinogenic effect at this exposure-range.

Therefore SCOEL considers FA as a group C carcinogen (genotoxic carcinogens for which a limit value derived from mode-of-action based threshold is supported).

Experimental studies support that the local carcinogenesis at the portal-of-entry is pivotal. In the sensitive rat species, the apparent LOAEC was 6 ppm, and the apparent NOAEC was 2 ppm for nasal cancer. Experimentally, the histopathological NOAEC for nasal effects of FA in rats and monkeys is 1 ppm and the NOAEC for regenerative cell replication 2 ppm. At these NOAECs, the FA-DNA adducts were less in monkeys than in rats as was the relationship of exogenous/endogenous DNA adducts, which is in line with the assumption that humans should be a less sensitive species. The new studies confirm that local FA-DNA adducts show a highly non-linear relationship with external FA exposures. At ≤ 2 ppm FA, the FA DNA-adducts induced by external exposures comprise a minor portion of the total FA-DNA adducts, which were driven mainly by internal (naturally generated) FA. This is supported by considerations on toxicokinetics, concluding that the intracellular FA concentration increases only slightly, and the intracellular glutathione concentration decreases only slightly in this range and that the homeostasis within the epithelial cells would not be affected. Therefore, the apparent NOAEC of 1 ppm can be considered a mode-of-action based NOAEC for carcinogenic effects at the portal-of-entry.

Ad (a): Preventing histopathological effects, like irritation, inflammation and regenerative cell replication caused by cytotoxic irritation, will also prevent nasal cancer as at such low exposure concentrations (< 1 ppm) the total intracellular FA concentration is dominated by the internal (natural) FA. This experimentally derived paradigm, namely the avoidance of cell proliferation in the upper respiratory tract being critical to prevent local carcinogenicity, also holds valid for humans. Ideally the lower sensitivity against cytotoxic irritation of humans as compared to rats should be taken into consideration. While cytotoxic irritation cannot be investigated in humans, mainly for ethical reasons, there is a broad database available for sensory irritation from volunteer studies under controlled exposure conditions. By derivation of limit values for sensory irritation of eye and upper respiratory tract in humans also the critical effects of irritation-induced local cell proliferation and subsequent possible carcinogenesis shall be covered (Bruening et al., 2014)."

B.3.9.4. German UBA

For the assessment of the cancer risk of inhaled formaldehyde, UBA (2016) used a non-linear approach due to the results of the animal studies showing an exponential increase of the risk curve: the additional theoretical cancer risk of a non-smoker following a continuous (80 years) inhalative exposure to 0.1 mg formaldehyde per cubic meter is assumed to be 3×10^{-7}. In conclusion the indoor air guide value for formaldehyde is also protective against cancer risk of inhaled formaldehyde.

B.3.9.5. Conclusion

Formaldehyde is a genotoxic carcinogen for which a threshold for its carcinogenic effect in the nose is very likely. SCOEL (2016) in its opinion has recommended an Occupational Exposure Limit Value (OEL) of 0.3 ppm (8h TWA) with a short term exposure limit of 0.6 ppm. This is based on their assessment that formaldehyde is a genotoxic carcinogen for which a mode-of-action based limit value can be derived.

As described by SCOEL (2016) the endogenous formaldehyde concentrations are relatively high with an appreciable amount of endogenous DNA adducts formed, whereas the background incidence of nasal tumours in rodents and of nasopharyngeal tumours in humans is very low. One of the reasons may be the low physiological proliferation rate of the respiratory epithelium, and as long as this is not increased, the probability of tumour formation also is low. Tumour induction in the nasal mucosa of rats and mice is the result of chronic proliferative processes caused by the cytotoxic effects of the substance in combination with DNA alterations by endogenous and exogenous formaldehyde. The dose-response relationships for all parameters investigated, such as damage to the nasal epithelium, cell proliferation, tumour incidence, the formation of DPX and DNA adducts, is very flat for low level exposures and becomes much steeper at higher concentrations. For these endpoints no-effect concentrations were demonstrated with the exception of the formation of DPX and DNA adducts. At the lowest concentrations investigated so far (0.7 ppm), adducts were still detected. However, adducts caused by endogenous, physiological formaldehyde by far exceeded the amounts caused by exogenous formaldehyde. At 0.3 ppm no sensory irritation in humans, which is considered the most sensitive endpoint, was observed (Lang et al., 2008; Mueller et al., 2013).

In summary, the inhalation cancer risk opposed by formaldehyde in the air at the OEL for workers of 0.3 ppm (0.369 mg/m^3) recommended by SCOEL and at the WHO Guideline for Indoor Air Quality for formaldehyde of 0.1 mg/m^3 (0.08 ppm; see Annex B.3.11.2) can be considered as negligible in relation to the endogenous formaldehyde concentrations.

Related to dermal exposure and carcinogenesis, formaldehyde is poorly absorbed through intact skin; rapid metabolism makes systemic effects unlikely following dermal exposure. In dermal initiation/promotion studies, formaldehyde did not initiate or promote skin tumorigenesis in mice. From a mouse skin painting study, no skin tumours were observed in 16 male and 16 female mice with topical application of 200 µg formaldehyde twice a week at the end of the study after 60 weeks (Iversen, 1986).

B.3.10. Reproductive toxicity

Formaldehyde is not classified for toxicity to reproduction.

Multiple studies have been published on reproductive and developmental effects of formaldehyde in human and animal studies. Epidemiological studies focus for example on male and female fertility, pre-term birth or abortions, and birth weights. Animal studies focus on male and in few studies on female fertility as well as on developmental toxicity with different routes of administration including, inhalation, oral administration, intraperitoneal, intravenous or subcutaneous injections or dermal administration.

Collins et al. (2001) performed a review of adverse pregnancy outcomes and formaldehyde exposures in humans and in animal studies and summarised that "*Formaldehyde is unlikely to reach the reproductive system in humans in concentrations sufficient to cause damage since it is rapidly metabolized and detoxified upon contact with the respiratory tract. While there are effects seen in in vitro studies or after injection, there is little evidence of reproductive or developmental toxicity in animal studies under exposure levels and routes relevant to humans. Most of the epidemiology studies examined spontaneous abortion and showed some evidence of increased risk (meta-relative risk=1:4, 95% CI 0.9-2.1). We found evidence of reporting biases and publication biases among the epidemiology studies and when these biases were taken into account, we found no evidence of increased risk of spontaneous abortion among workers exposed to formaldehyde (meta-relative risk=0:7, 95% CI 0.5-1.0). The small number of studies on birth defects, low birth weight, and infertility among formaldehyde workers; the limitations in the design of these studies; and the inconsistent findings across these studies make it difficult to draw conclusions from the epidemiology data alone. However, information from experimental studies and studies of metabolism indicate reproductive impacts are unlikely at formaldehyde exposures levels observed in the epidemiology studies.*"

A different conclusion was reached in a systematic review by Duong et al. (2011) including meta-analyses. The authors concluded the following: "*The mostly retrospective human studies provided evidence of an association of maternal exposure with adverse reproductive and developmental effects. Further assessment of this association by meta-analysis revealed an increased risk of spontaneous abortion (1.76, 95% CI 1.20-2.59, p = 0.002) and of all adverse pregnancy outcomes combined (1.54, 95% CI 1.27-1.88, p < 0.001), in formaldehyde-exposed women, although differential recall, selection bias, or confounding cannot be ruled out. Evaluation of the animal studies including all routes of exposure, doses and dosing regimens studied, suggested positive associations between formaldehyde exposure and reproductive toxicity, mostly in males. Potential mechanisms underlying formaldehyde-induced reproductive and developmental toxicities, including chromosome and DNA damage (genotoxicity), oxidative stress, altered level and/or function of enzymes, hormones and proteins, apoptosis, toxicogenomic and epigenomic effects (such as DNA methylation), were identified.*"

Nielsen et al. (2013) critically evaluated the review by Duong et al. (2011) considering the effects observed in human and animal studies in quantitative terms and in relation to the

general toxicity of formaldehyde. With respect to epidemiological studies on females, the authors concluded that the review by Duong et al. (2011) describes 18 human studies, but only one study (Zhou et al., 2006) was published after the review of Collins et al. (2001); this study did not find differences in 'preterm birth', 'small for gestation age' and 'major malformations'. Nielsen et al. (2013) also found that the results from the meta-analysis by Collins et al. (2001) and the first meta-analysis by Duong et al. (2011) are not substantially different. No significant increase was observed in studies with low recall bias. A somewhat increased meta-relative risk observed in both studies can be explained by the lack of confounder control. Thus, no convincing effect of formaldehyde was observed in pregnant women. With respect to epidemiological studies on males, Nielsen et al. (2013) commented that although the effect of formaldehyde exposure on male reproduction has been studied only to a limited extent, there is no convincing indication that it is affected. The lack of effects on female and male reproduction is in agreement with the toxicokinetic studies indicating that formaldehyde does not reach the internal organs.

With respect to testicular effects observed in male animals, several studies are reported by Duong et al. (2011) and Nielsen et al. (2013). After exposure of male rats for 4 and 13 weeks to 10 and 20 ppm formaldehyde (5 days/week, 8 h/day), reduced body weight gains, reduced testes weights and changed concentrations of trace elements including copper, zinc and iron were reported (Ozen et al., 2002). Thirteen week exposure to 5 and 10 ppm led to reduced testosterone levels, reduced diameters of seminiferous tubules and immunohistochemical changes in the testes (Ozen et al., 2005). Two week formaldehyde exposure of male rats to 10 mg/m^3 (8 ppm, 12 h/day) led to reduced testicular weights and histopathological changes in the testes such as atrophication of seminiferous tubules, decreased spermatogenic cells, seminiferous epithelial cells disintegrated and shed into lumina, edematous interstitial tissue with vascular dilatation and hyperemia, azoospermia of the lumina (Zhou et al., 2006). Exposure to 2.46 mg formaldehyde/m^3 (2 ppm) for 60 consecutive days resulted in significantly decreased sperm quantity and quality, decreased testicular seminiferous tubular diameter, reduction in the activities of superoxide dismutase and glutathione peroxidase, increased levels of malondialdehyde, atrophy of seminiferous tubules, decreases of spermatogenic cells and the lumina were oligozoospermic. No effects were reported at 0.5 mg/m^3 (0.4 ppm) (Zhou et al., 2011).

Nielsen et al. (2013) indicated that none of the inhalation studies reviewed by Duong et al. (2011) interpreted the formaldehyde-induced testicular effects in the context of known biological effects of formaldehyde. The prominent clinical symptoms reported at 5 ppm included unsteady breathing, an increase in nose cleaning, excessive licking, frequent sneezes and haemorrhage in nasal mucosa (Ozen et al., 2005) and are in agreement with expected occurrence of more severe irritation-induced stress. Also, decreased food consumption may reasonably explain the observed decrease in body weight gain. The reduced testicular levels of zinc and copper may be due to one or more of the potential indirect mechanisms causing testicular damage; these include stress from irritation, hypoxia and reduced intake of food. The latter may cause insufficient supply of the metals. The increased iron (Ozen et al., 2002) would be in line with an increase in hyperaemia in the testes, which was observed after. The LOAEL of 2 ppm for testicular effects in rats (Zhou et al., 2011) was a level that causes moderate sensory irritation-induced stress and hypoxia-induced stress (20% decrease in respiratory minute volume); higher levels caused exposure dependent increase in testicular effects. At the LOAEL, no increase is expected in formaldehyde absorption. The NOAEL for testicular effects in rats was 0.4 ppm (Zhou et al., 2011) where neither sensory irritation nor decreased respiratory minute volume was observed; no effect was observed in the absence of sensory irritation, which is the case at the indoor air guideline value. Nielsen et al. (2013)

further commented, that recent toxicokinetic studies do not support that formaldehyde reaches the sexual organs.

Nielsen et al. (2013) also reviewed the studies on developmental toxicity in animals. In a developmental toxicity study in 25 rats per group with formaldehyde exposure to 5, 10, 20 or 40 ppm on gestational days 6 to 20 (6 h/day), a decreased body weight gain was observed in the dams at the highest exposure level of 40 ppm (LOAEC) with no effects observed at 20 ppm (NOAEC). A slight foetotoxic effect (reduced weight in male foetuses) was observed ≥ 20 ppm with a NOAEC at 10 ppm (Saillenfait et al., 1989). No data were reported on clinical signs or local effects; however, local irritant effects are to be expected at ≥10 ppm.

Another developmental toxicity study was conducted in 25 rats per group exposed to 2, 5 or 10 ppm formaldehyde for 6 h/day from gestational day 6 to 15. This study showed an NOAEC for maternal toxicity at 5 ppm with reduced food consumption at 10 ppm and no relevant developmental effect up to 10 ppm (Martin, 1990).

Nielsen et al. (2013) also referred to several Russian inhalation studies with formaldehyde exposures from 0.01 to 1.2 ppm in female rats that showed adverse reproductive and developmental outcomes. However, with unusual methods. These results are inconsistent with the above-reviewed studies, which showed no teratogenic effect up to 40 ppm in spite of potential pain-induced and hypoxia-induced stress.

In summary, there is no convincing evidence that formaldehyde would lead to reproductive or developmental effects in human or in experimental animals at concentrations in the air that do not lead to irritation in the respiratory tract.

B.3.11. Derivation of DNEL(s) / DMEL(s)

For the purpose of this restriction, mainly DNELs for long-term inhalation exposure, local effects are relevant.

B.3.11.1. Workers

For workers, the registrant of the REACH dossier has derived a DNEL of 0.375 mg/m3 for long-term inhalation exposure, local effects (BASF, 2017) with the following justification: “For histopathological lesions a NOAEL of 1 ppm was found for rats and monkeys. Monkeys were continuously exposed over 26 weeks (22 h/d, 7 d/week) (Rusch et al., 1983) without any exposure free time for repair of lesions in contrast to exposures at the workplace. Therefore, workplace exposure conditions would most probably lead to a higher NOAEL in monkeys. For the NOAEL of 1 ppm in rats it has to be taken into consideration that the lesions observed at the LOAEL (2 ppm) cannot be regarded as prestages to tumor development, neither by their histopathological features nor by their location (Gelbke et al., 2014). Applying the AF of 3 to the histopathological NOAEL would lead to a DNEL of 0.3 ppm. This DNEL is in agreement with the 8-hour TWA of 0.3 ppm (0.369 mg/m^3) recommended by SCOEL. This recommendation is based on studies in volunteers examining sensory irritation following 4 hour daily exposure for 5 days with 15 minutes peak exposure (Lang et al., 2008; Mueller et al., 2013). From such studies, SCOEL derived a NOAEC of 0.3 ppm with peak exposure of 0.6 ppm (SCOEL, 2016).

B.3.11.2. General population

For the general population, the registrant of the REACH dossier has derived a DNEL of 0.1 mg/m^3 for long-term inhalation exposure, local effects (BASF, 2017).

This DNEL is in agreement a recommendation from German BfR (2006) that considered 0.1 ppm formaldehyde as "safe level".

This value is also in agreement with the WHO Guideline for Indoor Air Quality for formaldehyde of 0.1 mg/m^3 (WHO, 2010). This guidance is based on the NOAEC of 0.6 mg/m^3 for eye blinking response and is adjusted by using assessment factor 5 derived from the standard deviation of nasal pungency (sensory irritation) threshold, leading to a value of 0.12 mg/m^3, which has been rounded down to 0.1 mg/m^3. The guidance is a short-term (30-minutes) value. Neither increased sensitivity nor sensitisation is considered plausible at such indoor concentrations in adults and children. For long-term effects, including cancer, WHO calculated a guideline of 0.21 mg/m^3, starting from the NOAEL for cell proliferation of 1.25 mg/m^3 and applying assessment factor 3 for interspecies variability and assessment factor 2 for inter-individual variation. WHO calculated an alternative approach using biologically motivated models. Their assessment led to a predicted additional risk of 2.7 x 10^{-8} for continuous lifetime exposure to 0.125 mg/m^3 and a predicted additional risk of 10^{-6} or less for non-smokers continuously exposed to 0.25 mg/m^3. WHO concluded that the use of the short-term (30-minute) guideline of 0.1 mg/m^3 will also prevent long-term health effects, including nasopharyngeal cancer.

Nielsen et al. (2017) re-evaluated the WHO Guideline for Indoor Air Quality for formaldehyde of 0.1 mg/m^3 and concluded that the credibility of the WHO guideline of 0.1 mg/m^3 has not been challenged.

Also the German Umweltbundesamt (UBA, 2016) has confirmed an indoor guideline value of 0.1 mg formaldehyde/m^3.

In 2018, also the French ANSES (2018) re-evaluated the reference values for formaldehyde and concluded on an indoor air value of 100 µg/m^3 (0.1 mg/m^3) in line with the WHO guideline value.

JRC (2005) performed a "Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU". With respect to formaldehyde, JRC concluded that "*Due to being ubiquitous pollutant in indoor environments and to the increasing evidence indicating that children may be more sensitive to formaldehyde respiratory toxicity than adults it is considered a chemical of concern at levels exceeding 1 µg/m^3, a concentration more or less corresponding with the background level in rural areas.*" The value proposed by JRC of 1 µg/m^3 is based on studies investigating respiratory symptoms and pulmonary function in children and adults, e.g. Krzyzanowski et al. (1990). Effects were reported at formaldehyde concentrations as low as 37 µg/m^3 (0.037 mg/m^3). However, UBA (2016) performed a review of epidemiological studies investigating the association between formaldehyde exposure and the induction or exacerbation of asthma in children. On the basis of the current data, UBA concluded that 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.

B.3.11.3. Conclusion

A long-term inhalation DNEL, local effects, for the general population of 0.1 mg/m^3, as recommended by WHO, is considered appropriate to protect the general population including children from local formaldehyde-related effects.

B.4. Exposure assessment

B.4.1. Estimation of consumer exposure from mixtures

To estimate consumer exposure to formaldehyde (inhalation and dermal) from the use of certain types of mixtures, the Dossier Submitter used the Consexpo[1] web tool version 1.0.5 developed by the Dutch National Institute for Public Health and the Environment (RIVM).

Calculations were performed for a worst case scenario assuming a formaldehyde concentration of 0.09% w/w (just below the classification limit of 0.1%) for a number of mixtures (such as cleaning products, water borne paints and glues) used by consumers. Additional calculations were performed under the same conditions but for more realistic formaldehyde concentrations in mixtures of 0.05% w/w and 0.02% w/w. Information on the concentrations of formaldehyde in mixtures for consumer use attributable to biocidal and non-biocidal uses is not available. Therefore, as a worst case scenario, it has been assumed that the total concentration of formaldehyde in a mixture for consumer use is due to non-biocidal uses of formaldehyde or formaldehyde releasers. Default conditions of use as set in Consexpo (described in the RIVM Fact Sheets) have been assumed. Furthermore, the calculations were performed under the following assumptions:

- No further dilution applied
- Use temperature: 20 °C
- Vapour pressure of the substance;[2] 16 Pa (0.09%)-15.9 Pa (0.05% and 0.02%)

Table B.6 includes the references to the uses analysed and related exposure scenarios.

Assumptions made by Consexpo are conservative (e.g. small room volume for some exposure scenarios, low air exchange, high mass transfer from product to air, and long exposure time). Moreover, the Dossier Submitter assumed that no additional risk management measures have been taken (e.g. special container design or substances to reduce the volatility or to prevent dermal contact with hazardous substances). Such measures are commonly used in mixtures for consumer use.

In Table B.7, estimated concentrations in indoor air as daily mean (24 hours average concentration) are reported for the analysed exposure scenarios. For the more realistic formaldehyde concentrations in mixtures of 0.02% w/w and 0.05% w/w, consumer exposure is reduced by a factor of two and four, respectively, if compared to mixtures containing 0.09% w/w of formaldehyde.

Calculated dermal exposure during application phase is low in all scenarios (< 0.1 mg/kg/d as dose).

Based on the exposure estimation it is possible to conclude that the WHO Guideline for Indoor Air Quality for formaldehyde of 0.1 mg/m^3 is not exceeded for any of the assessed uses even when an unlikely worst case formaldehyde concentration in the mixture of 0.09% is assumed.

[1] https://www.rivm.nl/en/consexpo [Accessed 7 January 2019]

[2] The vapour pressure of diluted formaldehyde has been calculated using the Lacey equation as modified by Walker (1964)

Table B.6: Uses and use conditions assumed for mixtures for use by consumers

Exposure scenario source	Product database	Product categories	Default products	Duration of the exposure
RIVM: Cleaning Products Fact Sheet[3]	Cleaning and washing	All-purpose cleaning liquid	Application cleaning	240 min
		Floor cleaning liquid	Application cleaning	
		Furniture polishing liquid	Application polishing	120 min
RIVM: Paint Products Fact Sheet[4]	Painting products	Brush and roller painting water borne paint	Application	132 min
		Brush and roller painting water borne wall paint	Application	132 min
RIVM: Do-It-Yourself Products Fact Sheet[5]	Do it yourself product	Glues	Bottled glue – universal/wood glue	240 min
		Glues	Two-component glue	240 min

Table B.7: Estimated formaldehyde concentrations in air per type of use

Type of product/application	Mean concentration on day of exposure (mg/m³)		
	0.09% w/w	0.05% w/w	0.02% w/w
Application of all-purpose cleaning liquid	0.024	0.014	0.005
Application of floor cleaning liquid	0.023	0.013	0.005
Application of furniture polishing liquid	0.059	0.033	0.013
Brush and roller painting water borne paint	0.014	0.008	0.003
Brush and roller painting water borne wall paint	0.041	0.023	0.009
Application of bottled glue	0.022	0.012	0.005
Application of two component glue	0.036	0.020	0.008

B.4.2. Permanent formaldehyde emission sources

Table B.8 gives an overview of a broad range of permanent formaldehyde releasing sources in indoor air together with information on measured emission rates and/or steady-state concentrations, the test method used for obtaining the measurements as well as the source of the information. The information contained in Table B.8 covers the following types of products:

Solid wood

Formaldehyde is a decomposition product of lignin and is therefore released in small quantities from solid wood products. Formaldehyde emission rates from solid wood (oak, pine, beech, poplar, birch, spruce, and douglas fir) have been measured between 3 and 7 µg/(m²h) (Salthammer and Gunschera, 2017). Böhm et al. (2012) measured formaldehyde emission

[3] https://www.rivm.nl/bibliotheek/rapporten/2016-0179.pdf [Accessed 7 January 2019]

[4] https://www.rivm.nl/bibliotheek/rapporten/320104008.pdf [Accessed 7 January 2019]

[5] https://www.rivm.nl/bibliotheek/rapporten/320104007.pdf [Accessed 7 January 2019]

values from 0.014 to 0.084 mg/(m^{2}h) for various wood species, where the highest emissions originated from beech and spruce.

Wood-based products

Wood-based panels used as construction material and/or in finished articles, such as furniture and flooring, are a major formaldehyde emission source in indoor air (Marquart et al., 2013). These materials are usually covered with layers (e.g. primer, gypsum board, paint) that significantly reduce emissions of formaldehyde (Salthammer and Gunschera, 2017). A number of formaldehyde-based resins are used in the manufacturing process of plywood, particleboard, and medium density fibreboard (MDF), and in a variety of agents used in the treating process of wood surfaces depending on the desired properties of the finished product:

- 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^{2}h) (range 8.6-1 580 µg/(m^{2}h)) (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^{2}h) (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).

Salem et al. (2012) determined formaldehyde emissions and content from different kinds of wood-based panels and flooring materials using different test methods: European small-scale chamber (according to EN 717-1), gas analysis (EN 717-2), the American small-scale chamber (ASTM D 6007-02), and the perforator (EN 120) method. The tested materials included particleboard, medium and high density fibreboard (MDF and HDF), plywood and different flooring materials including HDF laminate, solid wood, solid bamboo and polyvinyl chloride (PVC). Measured data showed a good correlation among the four test methods for particleboard of 16 mm thickness. For this type of product, similar formaldehyde content/emissions as well as similar behaviour were observed with all test methods. Formaldehyde emissions from particleboard of different type and thickness varied from 0.4 to 2.52 mg/(m^{2}h), as measured with EN 717-2 method. The two highest emission rates were obtained from veneered particleboard with the thickness of 15 mm and 22 mm. The three lowest emission rates were obtained from panels of 8 and 10 mm thickness (uncoated and laminated particleboard). For MDF and laminated MDF, measured formaldehyde emissions were in the range of 0.20 to 0.73 mg/(m^{2}h) with the boards' thickness strongly affecting the emission rates. Emissions from plywood materials were in the range of 0.15 to 0.36 mg/(m^{2}h).

Flooring materials were tested using the EN 717-1 method measuring formaldehyde concentration in a standard chamber under specific conditions. The highest value – 0.125 mg/m^3 – was obtained for solid wood flooring (spruce, multi-layer, of 15.5 mm thickness). Thinner solid wood flooring generated concentrations in the test chamber of 0.035 mg/m^3 and oak multi-layer solid wood flooring of 0.021 and 0.041 mg/m^3. The

formaldehyde concentration of HDF laminate flooring (7, 8, 11 and 12 mm) ranged from 0.042 to 0.123 mg/m^3. Formaldehyde emissions from PVC flooring with UV-curable layer were within a range of 0.003 to 0.008 mg/m^3 (Salem et al., 2012).

Yrieix et al. (2010) used the test chamber method EN ISO 16000-9 for sampling formaldehyde emissions from a wood-based panel made from maritime pine particleboard glued with a UF resin. Emissions were measured after three and 28 days in six different laboratories. The main aldehydes, including formaldehyde, were analysed according to EN ISO 16000-3. This resulted in a mean formaldehyde concentration of 57.6 µg/m^3 after 28 days and a specific emission rate of 58.5 µg/(m^{2}h). A reduction of about 6% in the test chamber's formaldehyde concentration was observed between emissions after three and 28 days in the chamber test.

Böhm et al. (2012) performed measurements of formaldehyde emissions from various materials (solid wood, plywood, flooring, and blockboard used for building and furnishing materials) by using EN 717-1 chamber test and EN 717-2 gas analysis. The results showed that the wood species, plywood type and the thickness affected the results from EN 717-2. The steady state formaldehyde concentrations ranged from 0.006 mg/m^3 (engineered flooring with PVA coating) to 0.048 mg/m^3 (painted birch blockboard).

Yu and Kim (2012), using the EN 717-1 test method, investigated formaldehyde emission rates from different wood-based wardrobe panels. Steady-state formaldehyde emission rates ranged from 0.007 mg/(m^{2}h) for PVC laminated MDF to 0.096 mg/(m^{2}h) for uncoated raw MDF. Uncoated and coated particleboard exhibited similar emission rates of 0.04 mg/(m^{2}h) and 0.041 mg/(m^{2}h), respectively.

In its 2014 enforcement project, the Swedish Chemicals Agency (KEMI, 2015) measured formaldehyde emissions from 18 different wood-based panels covering plywood, particleboard, OSB and MDF mainly bonded by UF resins (see Section 2.4.3 in the main report). The samples were first tested with EN 717-2 as screening method and subsequently in a test chamber according to EN 717-1, if the initial screening indicated that emissions could exceed the E1 limit value. The initial screening (EN 717-2) showed emission rates between 0.1 mg/(m^{2}h) and 4.7 mg/(m^{2}h), with an average of 1.9 mg/(m^{2}h). The highest emissions were associated with particleboard and MDF bonded with UF or unknown resins. The lowest emissions were measured for plywood bonded with PF or MUF resins. Seven boards were followed up with chamber testing (EN 717-1), including one UF-bonded plywood, three particleboards (two UF-bonded, one unknown), and three MDF (two UF-bonded, one unknown). Formaldehyde concentrations in the test chamber were between 0.04 mg/m^3 (plywood) and 0.2 mg/m^3 (UF-bonded particleboard), with an average of 0.11 mg/m^3.

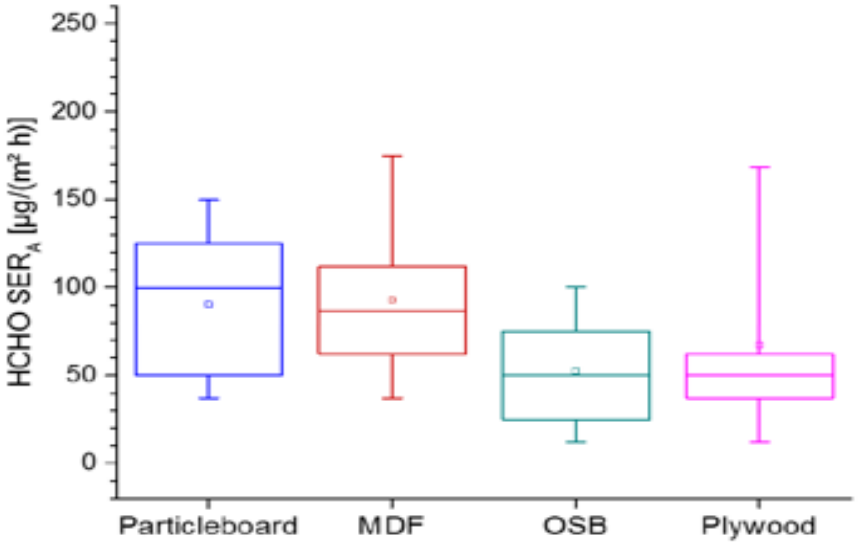
Kolarik et al. (2012) determined formaldehyde emissions for 22 different specimens prepared from purchased products and consumer products including wood-based panels, insulation materials, carpets, textiles, paints and detergents. MDF and chipboard were identified as the strongest formaldehyde sources, the highest steady-state concentrations were 0.101 mg/m^3 and 0.098 mg/m^3, respectively. All of the tested samples fulfilled the Danish requirements of formaldehyde concentrations of less than 124 µg/m^3 when measured in a standard test chamber.

Salthammer and Gunschera (2017) include in their report data on area specific emission rates for different types of raw (i.e. uncovered) wood-based panels which are taken from a research project of the Deutsches Institut für Bautechnik (DiBt). The measurements were carried out according to EN 717-1 and converted into µg/(m^{2}h) under assumption of steady-state conditions. The median emission rates are 99.5 µg/(m^{2}h) for particleboard, 87 µg/(m^{2}h) for

MDF, 62 µg/(m²h) for OSB and 50 µg/(m²h) for plywood, though there are large variations within each panel type (Figure B.1).

By covering the raw wood-based panels with different coating materials (e.g. gypsum board, wallpaper, primer, paint), formaldehyde emissions can be reduced markedly. According to experiments reported in Salthammer and Gunschera (2017), emission reductions from covering range from 70% to 98% depending on the number and type of coating materials used.

Figure B.1: Calculated area specific emission rates of wood-based panels



Note: Box-Whisker plots show P10, P25, P50 (median), P75, P90 and arithmetic mean (□).

Source: Salthammer and Gunschera (2017)

Salthammer and Gunschera (2017) also investigated formaldehyde release from laminates using unpublished chamber test data from Fraunhofer WKI's database. Formaldehyde emissions from laminate products are significantly reduced compared to raw materials as a result of lamination. For laminate produced in Europe, steady-state concentrations and emission rates ranged from < 3.8 to 32.5 µg/m³ and from < 3 to 28 µg/(m²h), respectively. A study by the Centers for Disease Control and Prevention reported considerably higher emission rates for laminate wood flooring products made in China with a geometric mean of 41.7 µg/(m²h) and a maximum value of 350 µg/(m²h) (CDC, 2016b).

Furniture

Wood-based panels are not only used in construction but also feature prominently in the production of furniture which might also contribute to indoor air formaldehyde concentrations. Veneering and preparation of furniture with acid-curing lacquer may also cause long-term emissions of formaldehyde (Jensen et al., 2001). Formaldehyde used as a fumigant and preservative in fabrics and foams applied in the furniture is an additional source of formaldehyde emissions (Andersen et al., 2016) but is not covered in the scope of the restriction.

In Denmark, Andersen et al. (2016) tested formaldehyde emissions of 20 different pieces of furniture made from wood-based panels. High emission rates were detected for a stool (0.18 mg/(m²h)) and a kitchen front door (0.15 mg/(m²h)). The latter product, in particular,

can be expected to be widely used covering large surface areas potentially contributing to high formaldehyde concentrations. Further, two bookcases were found to have a large impact on the formaldehyde concentration in the loaded standard room. Even though their area specific emission rates were found to be moderate (0.02 mg/(m^2h)), their large surface areas resulted in a formaldehyde release that impacted the concentration in the standard room significantly.

Wallcoverings

There has been a substantial decline in the release of formaldehyde from wallcoverings over the years. While in the past the basic material used for wallcoverings was paper (simplex or duplex) and the layers were assembled with glue, nowadays formaldehyde-free fleece is commonly applied as the backing material of wallcoverings. Data presented in Salthammer and Gunschera (2017) show that the majority of emission rates measured in test chambers between 2011 and 2016 were below the detection limit of 1 µg/(m^2h) (107 out of 144 samples after 3 days, 89 out of 97 samples after 28 days). After 28 days, seven samples showed emissions in the range of 1-10 µg/(m^2h) and only one measurement showed emissions in the range of 11-30 µg/(m^2h).

Paints

Some polymers used in paints and lacquers are manufactured with small percentages of monomers containing methanol groups, which may release small amounts of formaldehyde. Acid curing lacquers made of modified UF resins, which are considered a potentially high emitting source, have almost completely been replaced (Formacare, 2018). Photocatalytic indoor wall paints contain modified TiO_2, which is used as a catalyst under indoor daylight or artificial light. Organic binders like acrylic blends, vinyl acetate, styrene and unsaturated fatty acids are also typical constituents of wall paints. Formaldehyde might be formed from degradation of the paint ingredients during irradiation (Salthammer and Gunschera, 2017).

Salthammer and Fuhrmann (2007) studied the release of formaldehyde from photocatalytic paints. The experiments were carried out in a 1 m^3 glass chamber with an air exchange rate of 0.4 h^{-1} and a sunlight simulating lamp was used for irradiation. Results showed clearly that photocatalytic paint, when irradiated, was a strong source of formaldehyde emissions. Kolarik et al. (2012) measured formaldehyde concentrations of two paints in a test chamber according to EN 717-1. The initial concentrations were 0.023 mg/m^3 and 0.043 mg/m^3 and steady-state concentrations were 0.01 mg/m^3 and < 0.01 mg/m^3. On the basis of chamber concentrations taken from a study by Horn et al. (2007), Salthammer and Gunschera (2017) calculated area specific emission rates of 1-8 µg/(m^2h) after 10 days and 1-5 µg/(m^2h) after 28 days for different paints and lacquers.

Mineral wool

Mineral wool is used for insulation purposes in walls, floorings and house tops. Inorganic rock or slag is the main component (typically 97%) of stone wool. The remaining 3% is generally a thermosetting resin binder and oil. Glass wool is made from sand and recycled glass, lime-stone and soda ash. It usually contains 95-96% inorganic material. Urea-modified PF resins are used as binders, producing low emissions of formaldehyde during use (Salthammer and Gunschera, 2017).

Wiglusz et al. (2000) reported results from an inter-laboratory comparison experiment on the formaldehyde emissions from mineral wool board using a small test chamber (1 m^3, T = 23 °C, RH = 50 %, ACH = 1 h^{-1} and L = 1 m^2/m^3). Eleven laboratories took part and formaldehyde

emissions after 24-28 hours of testing ranged from 22 µg/(m²h) to 225 µg/(m²h). Mean values for different runs were 70-76 µg/(m²h).

Foams

UF foams as insulation material are used today only in gaps with good ventilation or when the foam is placed into closed cavities. Open-cell, tempered foams from MF resins are used for specific applications, e.g. seats in airplanes or noise insulation in concert halls. Also PF resins are used extensively to manufacture foams. However the formaldehyde emissions have been detected to be extremely low from these resins (Formacare, 2018).

Textiles (curtains and carpet)

Formaldehyde is commonly used in textile production processes. For example, after treatment of substantive dyeing, hardening of casein fibres, as a wool protection agent, anti mould and above all as a cross linking agent in resin finishing. According to chamber experiments carried out by Aldag et al. (2017), steady-state concentrations for curtains ranged from 1.0 µg/m³ to 5.3 µg/m³. Emission rates ranged from < 0.4 µg/(m²h) to 5 µg/(m²h).

Katsoyiannis et al. (2008) investigated the formaldehyde release from different carpets in test chambers with calculated area specific emission rates ranging from 3.5 to 17.5 µg/(m²h). The presence of ozone increased formaldehyde emissions from carpets in a study performed by Abbass et al. (2017). The emission rates without ozone were generally between 3 and 16 µg/(m²h) and with ozone between 13 and 29 µg/(m²h). Polyester and poly-triexta carpet samples were among the highest emitters.

Kolarik et al. (2012) measured formaldehyde concentrations for different textile samples, including two types of carpet, a roller blind (made of cotton) and a curtain (made of 50% cotton and 50% polyester). Measured in a test chamber according to EN 717-1, the initial concentrations were 0.009 and 0.011 mg/m³ for the carpets, 0.690 mg/m³ for the roller blind and 0.011 mg/m³ for the curtain. Steady-state concentrations were however < 0.01 mg/m³ for all the textile samples, except for the roller blind (0.047 mg/m³).

Table B.8: Steady state concentrations / emission rates for formaldehyde releasing products

Product	Min	Average (GM, AM)	Max	Used method	Reference
Solid wood					
Solid wood (six different wood species)	0.004 mg/m³ / 0.014 mg/(m²h)		0.008 mg/m³ / 0.084 mg/(m²h)	EN 717-1 / EN 717-2	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)
Blockboard, painted	0.025 mg/m³		0.037 mg/m³	EN 717-1	Böhm et al. (2012)
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		< 0.01 mg/m³		EN 717-1	Kolarik et al. (2012)
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	0.07 mg/m³ / 2.4 mg/(m²h)		0.20 mg/m³ / 4.7 mg/(m²h)	EN 717-1 / EN 717-2	KEMI (2015)
Particleboard (chipboard)	0.042 mg/m³		0.098 mg/m³	EN 717-1	Kolarik et al. (2012)
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)

Product	Min	Average (GM, AM)	Max	Used method	Reference
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)
OSB, UF		1.0 mg/(m²h)		EN 717-2	KEMI (2015)
OSB	< 0.01 mg/m³		0.042 mg/m³	EN 717-1	Kolarik et al. (2012)
MDF, UF and unknown	0.10 mg/m³ / 3.1 mg/(m²h)		0.13 mg/m³ / 3.6 mg/(m²h)	EN 717-1 / EN 717-2	KEMI (2015)
MDF	< 0.01 mg/m³		0.101 mg/m³	EN 717-1	Kolarik et al. (2012)
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)
Flooring laminate	0.006 mg/m³		0.018 mg/m³	EN 717-1	Böhm et al. (2012)
Laminate wood flooring products made in China		41.7 µg/(m²h)	350 µg/(m²h)		CDC (2016a)
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)
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					
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)
Shelf, MUF		1.1 mg/(m²h)		EN 717-2	KEMI (2015)

Product	Min	Average (GM, AM)	Max	Used method	Reference
Bookcase and drawer (chipboard)	0.027 mg/m^3		0.046 mg/m^3	EN 717-1	Kolarik et al. (2012)
Wallcoverings					
Wallcoverings (N = 144)	107 samples < 1 µg/(m^{2}h)		3 samples 31-60 µg/(m^{2}h)	Chamber test, unpublished data	Salthammer and Gunschera (2017)
Wallcoverings (N = 97)	89 samples < 1 µg/(m^{2}h)		1 samples 11-30 µg/(m^{2}h)	Chamber test, unpublished data	Salthammer and Gunschera (2017)
Paints					
Paints and lacquers	1 µg/(m^{2}h) (10 days); 1 µg/(m^{2}h) (28 days)		8 µg/(m^{2}h) (10 days); 5 µg/(m^{2}h) (28 days)	Chamber test	Horn et al. (2007) as reported in Salthammer and Gunschera (2017)
Paints	< 0.01 mg/m^3		0.010 mg/m^3	EN 717-1	Kolarik et al. (2012)
Paints	8.1 µg/(m^{2}h)		9.8 µg/(m^{2}h)		Salthammer et al. (2010)
Photocatalytic paints (lights on)			Peak value of 76 µg/m^3	Chamber test	Salthammer and Fuhrmann (2007)
Mineral wool, insulating materials					
Insulating materials	< 0.01 mg/m^3		0.011 mg/m^3	EN 717-1	Kolarik et al. (2012)
Mineral wool board	22 µg/(m^{2}h)	70-76 µg/(m^{2}h)	225 µg/(m^{2}h)	Chamber test	Wiglusz et al. (2000)
Textiles					
Carpet	3 µg/(m^{2}h); 13 µg/(m^{2}h)		16 µg/(m^{2}h); 29 µg/(m^{2}h)	Without O_3; with O_3	Abbass et al. (2017)
Carpet	3.5 µg/(m^{2}h)		17.5 µg/(m^{2}h)	Chamber test	Katsoyiannis et al. (2008)
Carpet	< 0.01 mg/m^3		< 0.01 mg/m^3	EN 717-1	Kolarik et al. (2012)
Curtain	1.0 µg/m^3	2.5 µg/m^3	5.3 µg/m^3	EN 717-1	Aldag et al. (2017)
Curtain		< 0.01 mg/m^3		EN 717-1	Kolarik et al. (2012)
Roller blind		0.047 mg/m^3		EN 717-1	Kolarik et al. (2012)

B.4.3. Temporary formaldehyde emission sources (combustion sources)

The information on temporary formaldehyde emission sources presented below is taken from Salthammer and Gunschera (2017).

Burning candles

Candle wax, usually a mix of hydrocarbons, is decomposed to carbon dioxide and water. The formation of reactive compounds and radicals lead to numerous by-products, including formaldehyde.

Ahn et al. (2015) studied six different types of candles and their VOC emissions before lighting and when lit. The mass loss was in the range between 0.041 g/min and 0.082 g/min. Kim et al. (2016) provided mass related formaldehyde emission rates, ranging from 0.59 µg/g to 95.7 µg/g for the six types of candles. Combining mass loss and mass related emission rates, time related values in the range of 3 to 310 µg/h can be derived. In an unpublished Fraunhofer WKI study, a mass loss of 4 g/h and a mass related emission rate of 96 µg/g was determined. This can be converted to a time related value of 384 µg/h. Derudi et al. (2012) measured formaldehyde emission rates between 2 µg/g and 3 µg/g from scented candles but did not determine the mass loss.

Burning incense

Incense sticks are known to release high levels of pollutants and particles, which is due to the combustion of ingredients like frankincense, myrrh, spices, herbs, wood, bark, fragrances, etc. (Lee and Wang, 2004).

Formaldehyde is a common by-product of incense combustion. Lee and Wang (2004) investigated the release of formaldehyde from 10 types of incense sticks in an 18.26 m^3 stainless-steel chamber at T = 23 ºC, RH = 5% and ACH = 0.5 h^{-1}. The average burn time was between 25 min and 51 min. Chamber concentrations were widely spread and ranged from approximately 20 µg/m^3 to 300 µg/m^3. Mass related formaldehyde emission rates ranged from approximately 400 µg/g to 1 700 µg/g.

Cooking and cooking related activities

Formaldehyde is also produced as the result of cooking, although a clear distinction has to be made between emissions from the cooking fuel and the cooking process itself.

Peng et al. (2017) studied effects of cooking method, cooking oil and food type on aldehyde emissions in cooking oil fumes. The formaldehyde concentrations in the oil fumes were between 4 µg/m^3 and 27 µg/m^3, depending on the cooking oil (palm rapeseed, sunflower and soybean) and the cooking method (pan-frying, deep-frying, stir-frying).

Fortmann et al. (2006) carried out experiments in a test house that allow a comparison of formaldehyde indoor concentrations with gas and electric ranges and for different cooking activities. The highest concentrations were measured during oven cleaning, both with the gas and electric range. The average formaldehyde concentrations during the 5 hour long oven-cleaning events, which involved baking the surfaces at high temperatures, were 416 µg/m^3 and 224 µg/m^3 for the gas and electric ranges, respectively. Formaldehyde concentrations were also elevated in the kitchen during broiling of fish, while cooking of a pork roast in the oven was associated with substantially lower formaldehyde concentrations.

Bednarek et al. (1997) performed a study on human exposure to air pollutants during a dinner. Seven adults volunteered in a 55 m^3 room at ACH = 0.29 h^{-1}. During the cooking phase (indoor barbecue) the formaldehyde concentration increased from 23 µg/m^3 to 58 µg/m^3 within two hours.

Ethanol fireplaces

Decorative fireplaces are usually operated with liquid ethanol or gel-type fuel and have no fume extraction system. Therefore, all of the emitted gases from combustion, VOCs and particulate matter are released into the room. This makes these devices strong sources of pollutants with considerable influence on indoor air quality. Schripp et al. (2014) studied a variety of fireplaces in a 48 m^3 emission test chamber under typical living room environmental conditions. The measured maximum value of formaldehyde was 456 ppb. Guillaume et al. (2013) also measured high formaldehyde concentrations between 0.4 mg/m^3 and 0.9 mg/m^3 in the exhaust gas of four decorative ethanol fireplaces.

Wood combustion

Wood combustion takes place more or less incompletely, which may cause undesirable by-products such as carbonyl compounds (formaldehyde and acetaldehyde).

Different researchers have studied the release of formaldehyde from residential wood combustion and found that the emission factors were between 113 mg/kg and 1 772 mg/kg, depending on the wood type and wood humidity. For the combustion of softwood pellets Reda et al. (2015) measured lower emission factors with formaldehyde values between 3 mg/kg and 4 mg/kg. Lévesque et al. (2001) investigated 31 Canadian homes and found that there was no difference in the formaldehyde concentrations in relation to the sampling location nor in relation to whether a combustion appliance was present or not. In a study performed by Salthammer (2014), seven private homes were investigated before and during the operation of wood burning fireplaces. In one case, the results showed a significant increase of the formaldehyde concentration from 16 ppb to 55 ppb.

Smoking

Tobacco smoking is known to be one of the strongest emission sources for formaldehyde, other organic and inorganic compounds and particulate matter in the indoor environment.

Baek and Jenkins (2004) measured an average formaldehyde concentration of 234 µg/m^3 when six cigarettes were smoked in a 30 m^3 chamber under almost static conditions. Schripp et al. (2013) compared formaldehyde concentrations during consumption of electronic and conventional cigarettes in an 8 m^3 stainless steel chamber at ACH = 0.3 h^{-1}. A clear increase of the formaldehyde concentration could be observed when the conventional cigarette was smoked. A study by Baker (2006) with 13 experimental cigarettes (saccharides added) gave emission rates between 30 µg and 57 µg per cigarette smoked. In the study by Singer et al. (2003) distinctly higher formaldehyde emission rates of 950-1 310 µg per cigarette smoked were measured. Maroni et al. (1995) reported 70-100 µg formaldehyde in the undiluted mainstream smoke of nonfilter cigarettes and 0.2 mg per cigarette in side stream smoke. According to Baker et al. (2006) formaldehyde is mainly generated from the pyrolysis of saccharides used as tobacco ingredients.

B.4.4. Furnishing scenarios

The Danish EPA developed three typical furnishing scenarios in a study on formaldehyde emissions from furniture (Andersen et al., 2016). The three furnishing scenarios, which result in loading factors of 0.75 m^2/m^3, 0.88 m^2/m^3 and 0.72 m^2/m^3, are shown in Table B.9. For the exposure scenario described in Section 1.3.6.5 of the main report, the central scenario, i.e. furnishing scenario 1, resulting in a loading factor of 0.75 m^2/m^3 has been chosen.

Table B.9: Typical furnishing scenarios

Furniture item	Item surface area [m^2]	Number of items	Total surface area [m^2]	Room volume [m^3]	Loading factor [m^2/m^3]
Furnishing scenario 1					
Stool	1.21	1	1.21	30.00	0.04
Armchair	2.60	2	5.20	30.00	0.17
Chest of drawers	10.00	1	10.00	30.00	0.33
Bookcase	6.00	1	6.00	30.00	0.20
		Total	***22.41***	***30.00***	***0.75***
Furnishing scenario 2					
Dining chair	0.77	6	4.62	30.00	0.15
Chest of drawers	10.00	1	10.00	30.00	0.33
Dining table	5.80	1	5.80	30.00	0.19
Bookcase	6.00	1	6.00	30.00	0.20
		Total	***26.42***	***30.00***	***0.88***
Furnishing scenario 3					
Dining chair	0.77	6	4.62	30.00	0.15
Kitchen front door	11.16	1	11.16	30.00	0.37
Dining table	5.80	1	5.80	30.00	0.19
		Total	***21.58***	***30.00***	***0.72***

Source: Andersen et al. (2016)

B.4.5. Monte Carlo simulations

Indoor air formaldehyde concentrations have been simulated for 100 000 rooms equipped according to the exposure scenario laid out in Section 1.3.6.5 of the main report. The purpose of this Annex is to provide additional explanations on the approach taken.

As a first step, emission rates (or concentrations in the case of outdoor air) were simulated for all formaldehyde emission sources relevant for the exposure scenario. This means that for each source 100 000 values (i.e. one per room) were drawn from a log-normal distribution of emission rates/concentrations.[6] This type of distribution is frequently used to represent environmental data in statistical analysis. For each of the emission sources, the input parameters for the log-normal distribution, i.e. geometric mean (GM) and geometric standard deviation (GSD), were taken from Salthammer and Gunschera (2017), which in turn are based on a review of the formaldehyde emission literature (Table B.10).

[6] For window, the concentration was fixed at 2 µg/m^3 rather than drawing values from a log-normal distribution. This is because formaldehyde concentrations related to wood-based windows, as reported in Salthammer and Gunschera (2017), were below the detection limit (< 2 µg/m^3).

Table B.10: Input parameters per emission source

Source	GM	GSD	Measure [1]	Unit
Particleboard	79.00	1.37	SER_A	µg/(m²h)
Paint	2.30	1.56	SER_A	µg/(m²h)
Laminate	8.50	1.80	SER_A	µg/(m²h)
Furniture	17.80	2.54	SER_A	µg/(m²h)
Textiles	1.90	1.38	SER_A	µg/(m²h)
Door	18.20	2.70	SER_A	µg/(m²h)
Window	2.00		Concentration	µg/m³
Outdoor air	3.49	2.11	Concentration	ppb
Indoor chemistry	40.05	1.65	SER_U	µg/h

1. SER_A = area specific emission rate, SER_U = unit specific emission rate

Source: Salthammer and Gunschera (2017)

Table B.11 gives an example of simulated emission rates/concentrations for each of the formaldehyde emission sources for three of the 100 000 rooms.

Table B.11: Example of simulated emission rates/concentrations

Source	Room 1	Room 2	Room 3	Measure [1]	Unit
Particleboard	66	73	129	SER_A	µg/(m²h)
Paint	3	5	2	SER_A	µg/(m²h)
Laminate	10	5	3	SER_A	µg/(m²h)
Furniture	17	35	19	SER_A	µg/(m²h)
Textiles	3	2	2	SER_A	µg/(m²h)
Door	24	4	23	SER_A	µg/(m²h)
Window	2	2	2	Concentration	µg/m³
Outdoor air	2	1	2	Concentration	ppb
Indoor chem.	76	40	18	SER_U	µg/h

1. SER_A = area specific emission rate, SER_U = unit specific emission rate

Next, the simulated emission rates were used to calculate reference room concentrations for the different formaldehyde emission sources. Assuming steady-state conditions, reference room concentrations can be calculated as (Salthammer and Gunschera, 2017):

- $C = SER_A \cdot L/ACH$ from area specific emission rates (Eq. 1)
- $C = SER_U/(V \cdot ACH)$ from unit specific emission rates (Eq. 2)

where:

- C [µg/m³] ... reference room concentration (calculated)
- SER_A [µg/(m²h)] ... area specific emission rate (simulated)
- SER_U [µg/h] ... unit specific emission rate (simulated)
- L [m²/m³] ... loading factor (defined in the exposure scenario)
- V [m³] ... volume of the reference room (= 30 m³)
- ACH [h^{-1}] ... air exchange rate in the reference room (= 0.5 h^{-1})

Eq. 1 and Eq. 2, together with assumptions on loading factors and emission reduction from covering of particleboard, were used to get from simulated emission rates to reference room concentrations.

Finally, the reference room concentrations obtained for the different sources as well as the contribution of outdoor air were combined and the sink effect taken into account to arrive at the simulated indoor air formaldehyde concentrations for the 100 000 rooms.

Table B.12 shows an example of formaldehyde concentrations in three rooms that were derived from the simulated emission rates/concentrations in Table B.11 under the assumptions of sub-scenario B (see Table 8 in the main report). The rightmost column provides information on the calculations.

Table B.12: Example of reference room concentrations for sub-scenario B (µg/m³)

Source	Room 1	Room 2	Room 3	Notes on calculation
Wall 1	20	22	39	Eq. 1 with simulated SER_A for particleboard from Table B.11, L = 0.6, 75% reduction from covering
Ceiling 1	13	15	26	Eq. 1 with simulated SER_A for particleboard from Table B.11, L = 0.4, 75% reduction from covering
Wall 2	5	10	4	Eq. 1 with simulated SER_A for paint from Table B.11, L = 1
Ceiling 2	2	4	2	Eq. 1 with simulated SER_A for paint from Table B.11, L = 0.4
Flooring	8	4	3	Eq. 1 with simulated SER_A for laminate from Table B.11, L = 0.4
Furniture	25	52	29	Eq. 1 with simulated SER_A for furniture from Table B.11, L = 0.75
Textiles	2	1	1	Eq. 1 with simulated SER_A for textiles from Table B.11, L = 0.3
Door	2	0	2	Eq. 1 with simulated SER_A for door from Table B.11, L = 0.05
Window	2	2	2	Already a concentration (µg/m³), no further calculations needed
Outdoor air	3	1	2	Concentration in Table B.11 multiplied by 1.24 to get from ppb to µg/m³
Indoor chem.	5	3	1	Eq. 2 with simulated SER_U for indoor chemistry from Table B.11
Sink	-22	-29	-28	25% of the sum of the other sources
Total	***65***	***86***	***84***	Sum of all sources minus sink effect

The simulations were carried out in R version 3.5.0 using the following code:

```
########## Input parameters ##########################################################################

# Create data frame of emission sources with their respective GM and GSD
mcinputs <- data.frame(source = c("pb", "paint", "laminate", "furniture", "textiles",
                                  "door", "window", "outdoorair", "indoorchem"),
                       mu = c(79.00, 2.30, 8.50, 17.80, 1.90, 18.20, 2.00, 3.49, 40.05),
                       sigma = c(1.37, 1.56, 1.80, 2.54, 1.38, 2.70, NA, 2.11, 1.65))

########## Monte Carlo simulation of emission rates ##################################################

# Initialize matrix for simulated emission rates
m <- 100000 #Number of rooms
n <- length(mcinputs$source) #Number of sources
mcsim <- matrix(NA, nrow = m, ncol = n)

# Set seed for reproducibility of simulated emission rates
set.seed(123)

# For each source draw m emission rates from log-normal distribution with above input parameters
# Use constant value for window rather than drawing from log-normal distribution
for (i in 1:n) {
        if (mcinputs$source[i] != "window") {
                mcsim[, i] <- rlnorm(m, log(mcinputs$mu[i]), log(mcinputs$sigma[i]))
        }
        else {
                mcsim[, i] <- rep(mcinputs$mu[i], m)
        }
}

# Transform matrix of simulated emission rates into a data frame
colnames(mcsim) <- mcinputs$source
mcsim <- as.data.frame(mcsim)

########## Reference room concentrations ##############################################################

# Set the air exchange rate
ACH <- 0.5

# Initialize the sub-scenarios
snames <- c("A", "B", "C")
scenario <- list()
for (i in snames) {
        scenario[[i]] <- data.frame(wall1 = numeric(m),
                                    ceiling1 = numeric(m),
                                    wall2 = numeric(m),
                                    ceiling2 = numeric(m),
                                    flooring = numeric(m),
                                    furniture = numeric(m),
                                    textiles = numeric(m),
                                    door = numeric(m),
                                    window = numeric(m),
                                    outdoorair = numeric(m),
                                    indoorchem = numeric(m),
                                    sink = numeric(m))
}

# Calculate reference room concentrations for sub-scenario A: PB ceiling
scenario[[1]]$wall1 <- 0 #Non-FA emitting material used
scenario[[1]]$ceiling1 <- 0.25*(mcsim$pb*0.4/ACH) #PB, L = 0.4, Covering: -75%
scenario[[1]]$wall2 <- mcsim$paint*1/ACH #Paint, L = 1
scenario[[1]]$ceiling2 <- mcsim$paint*0.4/ACH #Paint, L = 0.4
scenario[[1]]$flooring <- mcsim$laminate*0.4/ACH #Laminate, L = 0.4
scenario[[1]]$furniture <- mcsim$furniture*0.75/ACH #L = 0.75
scenario[[1]]$textiles <- mcsim$textiles*0.3/ACH #L = 0.3
scenario[[1]]$door <- mcsim$door*0.05/ACH #L = 0.05
scenario[[1]]$window <- mcsim$window #Constant value of 2 ug/m3
scenario[[1]]$outdoorair <- mcsim$outdoorair*1.24 #Times 1.24 to get from ppb to ug/m3
scenario[[1]]$indoorchem <- mcsim$indoorchem/(30*ACH) #C = SER/(V*ACH) to get from ug/h to ug/m3
scenario[[1]]$sink <- 0.25*(apply(scenario[[1]][, 1:11], 1, sum) - scenario[[1]][, 12]) #Sink: -25%

# Calculate reference room concentrations for sub-scenario B: PB ceiling + PB in two walls
scenario[[2]]$wall1 <- 0.25*(mcsim$pb*0.6/ACH) #PB, L = 0.6, Covering: -75%
scenario[[2]]$ceiling1 <- 0.25*(mcsim$pb*0.4/ACH) #PB, L = 0.4, Covering: -75%
scenario[[2]]$wall2 <- mcsim$paint*1/ACH #Paint, L = 1
scenario[[2]]$ceiling2 <- mcsim$paint*0.4/ACH #Paint, L = 0.4
scenario[[2]]$flooring <- mcsim$laminate*0.4/ACH #Laminate, L = 0.4
scenario[[2]]$furniture <- mcsim$furniture*0.75/ACH #L = 0.75
scenario[[2]]$textiles <- mcsim$textiles*0.3/ACH #L = 0.3
scenario[[2]]$door <- mcsim$door*0.05/ACH #L = 0.05
scenario[[2]]$window <- mcsim$window #Constant value of 2 ug/m3
scenario[[2]]$outdoorair <- mcsim$outdoorair*1.24 #Times 1.24 to get from ppb to ug/m3
scenario[[2]]$indoorchem <- mcsim$indoorchem/(30*ACH) #C = SER/(V*ACH) to get from ug/h to ug/m3
scenario[[2]]$sink <- 0.25*(apply(scenario[[2]][, 1:11], 1, sum) - scenario[[2]][, 12]) #Sink: -25%
```

```
# Calculate reference room concentrations for sub-scenario C: PB ceiling + PB in all walls
scenario[[3]]$wall1 <- 0.25*(mcsim$pb*1/ACH) #PB, L = 1, Covering: -75%
scenario[[3]]$ceiling1 <- 0.25*(mcsim$pb*0.4/ACH) #PB, L = 0.4, Covering: -75%
scenario[[3]]$wall2 <- mcsim$paint*1/ACH #Paint, L = 1
scenario[[3]]$ceiling2 <- mcsim$paint*0.4/ACH #Paint, L = 0.4
scenario[[3]]$flooring <- mcsim$laminate*0.4/ACH #Laminate, L = 0.4
scenario[[3]]$furniture <- mcsim$furniture*0.75/ACH #L = 0.75
scenario[[3]]$textiles <- mcsim$textiles*0.3/ACH #L = 0.3
scenario[[3]]$door <- mcsim$door*0.05/ACH #L = 0.05
scenario[[3]]$window <- mcsim$window #Constant value of 2 ug/m3
scenario[[3]]$outdoorair <- mcsim$outdoorair*1.24 #Times 1.24 to get from ppb to ug/m3
scenario[[3]]$indoorchem <- mcsim$indoorchem/(30*ACH) #C = SER/(V*ACH) to get from ug/h to ug/m3
scenario[[3]]$sink <- 0.25*(apply(scenario[[3]][, 1:11], 1, sum) - scenario[[3]][, 12]) #Sink: -25%

########## Results ###############################################################################

# Aggregate data
c_bysource <- list()
c_total <- matrix(NA, nrow = m, ncol = length(snames)); colnames(c_total) <- snames
c_aboveWHO <- numeric(length(snames)); names(c_aboveWHO) <- snames
for (i in 1:length(snames)) {
        c_bysource[[snames[i]]] <- round(t(apply(scenario[[i]], 2,
                                                 quantile, probs = c(0.5, 0.95))), 0)
        c_total[, i] <- rowSums(scenario[[i]][, 1:11]) - scenario[[i]][, 12]
        c_aboveWHO[i] <- length(c_total[c_total[, i] > 100, i])/m
}

# Table 9: Summary of simulated formaldehyde concentration in 100 000 rooms
round(apply(c_total, 2, quantile, probs = c(0.5, 0.75, 0.9, 0.95)), 0)
round(100*c_aboveWHO, 1)

# Table 10: Simulated reference room concentration by source
c_bysource
```

Annex C: Baseline

C.1. Voluntary agreements and commitments in other industries

C.1.1. Furniture industry

In September 2015, the European furniture industry, represented by the European Furniture Industries Confederation (EFIC), has joined the European Panel Federation (EPF) in calling for a common EU-wide legislation for the production, import and marketing of wood-based panels and of products made from them at a formaldehyde emission limit that is in line with the E1 emission class (EPF/EFIC, 2015).

According to EFIC (2017), *"mandatory E1 in the EU would:*

- *Guarantee an equal level of protection to all consumers at a level deemed safe;*
- *Starting from a safe level, leave to the consumer the choice of going further (e.g. Ecolabel and other environmental labels);*
- *Guarantee a level-playing-field in the EU, imposing equal requirements to all European producers and all international importers;*
- *Provide certain and clear regulation to furniture producers and their suppliers;*
- *Eliminate costs for testing complex articles as furniture, and not raise unbearable cots for the furniture and panel industry: European panel producers will be supplying furniture manufacturers with products complying with E1 standards only and will provide related certification."*

C.1.2. Automotive industry

According to information received from the European Automotive Industry Association (ACEA), manufacturers have been monitoring formaldehyde release to the indoor vehicle air for more than 15 years. Test methods and a voluntary approach in reducing the amount of formaldehyde released from vehicle interiors have been implemented in order to work towards the following objectives:

1. Harmonisation of measurement standards
2. Implementation of voluntary limit values for formaldehyde in vehicle indoor emissions

ISO 12219-1 to 9 "Interior air of road vehicles" are the relevant standards describing the test methods for determining vehicle indoor air emissions.

In 2017, the UNECE has harmonised the standard ISO 12219-1 (ISO, 2012) with existing national testing standards to propose a UN mutual resolution for indoor air measurement methods (UNECE, 2017). This recommendation states "*This Mutual Resolution contains the provisions and harmonized test procedure for the measurement of interior air emission from interior materials, concerning the protection of passengers and driver from toxic emissions emitted from interior materials used for the construction of vehicles*".

It furthermore states that the analytical equipment used for the determination of volatile organic compounds (VOCs) and carbonyl compounds or formaldehyde alone shall be in accordance with ISO 16000-6 (VOCs) or ISO 16000-3 (carbonyl compounds), respectively

(ISO, 2011b; ISO, 2011c). Carbonyl compounds (formaldehyde, acetaldehyde and acrolein) are to be measured according to ISO 16000-3.

ACEA outlined that the formaldehyde concentration for European vehicles should not exceed 100 µg/m^3 when tested according to the ambient mode of ISO 12219-1 or UNECE (2017).

To achieve these limit values, vehicle manufacturers have various strategies such as formaldehyde limits for vehicle components, materials or the application of air conditioning/filter strategies.

C.2. Breakdown into class E1 and E2 wood-based panels

For both EU-manufactured and imported wood-based panels, the Dossier Submitter arrived at the E1/E2 breakdown by applying estimated shares of E1 and E2 panels for the year 2017 provided by EPF to Eurostat (2018b) and FAO (2018) data on the quantities of wood-based panels produced in and imported into the EU for the year 2016.

For EU-manufactured wood-based panels, the estimated E1/E2 shares provided by EPF are shown in Table C.1 by Member State for the main panel types (excluding hardboard and softboard as these generally don't use formaldehyde-based resins). EPF derived the E1/E2 split under the following assumptions:

- All EPF members produce only class E1 panels.
- Companies that are not EPF members are assumed to produce 50% class E1 panels and 50% class E2 panels.
- The estimates take into account that some EU Member States have restrictions on producing class E2 panels (see Section 1.5.1 in the main report), with the exception of Greece, because it is not clear whether the legislation is respected.

The share of class E2 panels in the EU production is estimated at 3% for plywood, 4% for particleboard, 0% for OSB, and 2% for MDF. This means that around 3% of the EU's total wood-based panel production is expected to be class E2.

For extra-EU imports, the estimated E1/E2 shares provided by EPF for the year 2017 are shown in Table C.2 by trading partner for the main panel types (excluding hardboard and softboard as these generally don't use formaldehyde-based resins). The EPF calculations of extra-EU imports take into account that some Member States have legislation in place that prohibits the placing on the market of class E2 panels. However, even for those countries it is assumed that 5% of imported wood-based panels could be class E2.

The estimated share of class E2 panels in extra-EU imports is 35% for plywood, 31% for particleboard, 25% for OSB, and 38% for MDF. Overall, around one-third (32%) of the total imported wood-based panels volume are estimated to fall into the E2 emission class.

Table C.1: Share of E1 and E2 panels in EU production of wood-based panels, 2017

Member State	Plywood		Particleboard		OSB		MDF	
	E1	E2	E1	E2	E1	E2	E1	E2
Austria			100%	0%			100%	0%
Belgium			100%	0%	100%	0%	100%	0%
Bulgaria	50%	50%	84%	16%	100%	0%		
Croatia			100%	0%				
Cyprus								
Czech Republic	100%	0%	94%	6%	100%	0%	100%	0%
Denmark			100%	0%				
Estonia	67%	33%	100%	0%				
Finland	100%	0%	100%	0%				
France	100%	0%	99%	1%	100%	0%	100%	0%
Germany	100%	0%	100%	0%	100%	0%	100%	0%
Greece	100%	0%	100%	0%			50%	50%
Hungary			50%	50%	100%	0%	100%	0%
Ireland					100%	0%	100%	0%
Italy	100%	0%	100%	0%	100%	0%	100%	0%
Latvia	98%	2%	100%	0%	100%	0%		
Lithuania			75%	25%				
Luxembourg					100%	0%	100%	0%
Malta								
Netherlands								
Poland	99%	1%	100%	0%	100%	0%	91%	9%
Portugal	67%	33%	96%	4%			95%	5%
Romania	50%	50%	78%	22%	100%	0%	100%	0%
Slovakia	57%	43%	74%	26%				
Slovenia							100%	0%
Spain	100%	0%	100%	0%			100%	0%
Sweden	100%	0%	100%	0%				
United Kingdom			100%	0%	100%	0%	100%	0%
Total	***97%***	***3%***	***96%***	***4%***	***100%***	***0%***	***98%***	***2%***

Source: EPF estimations

Table C.2: Share of E1 and E2 panels in extra-EU imports of wood-based panels, 2017

Trading partner	Plywood		Particleboard		OSB		MDF	
	E1	E2	E1	E2	E1	E2	E1	E2
Argentina			50%	50%	100%	0%	50%	50%
Australia			100%	0%	100%	0%	100%	0%
Belarus	50%	50%	75%	25%	75%	25%	50%	50%
Bosnia and Herzegovina			75%	25%	100%	0%	75%	25%
Brazil	100%	0%	50%	50%	100%	0%	50%	50%
Cameroon			50%	50%	100%	0%	50%	50%
Canada	100%	0%	100%	0%	100%	0%	100%	0%
Chile	100%	0%	75%	25%	100%	0%	75%	25%
China	50%	50%	75%	25%	50%	50%	50%	50%
Côte D'Ivoire			25%	75%	100%	0%	25%	75%
Ecuador			50%	50%	100%	0%	50%	50%
Equatorial Guinea			50%	50%	100%	0%	50%	50%
Gabon	75%	25%	50%	50%	100%	0%	50%	50%
Ghana	50%	50%	25%	75%	100%	0%	75%	25%
Hong Kong			50%	50%	100%	0%	50%	50%
India			50%	50%	100%	0%	50%	50%
Indonesia	54%	46%	50%	50%	100%	0%	50%	50%
Israel			50%	50%	100%	0%	50%	50%
Japan			100%	0%	100%	0%	100%	0%
Korea			75%	25%	100%	0%	50%	50%
Malaysia	50%	50%	50%	50%	100%	0%	50%	50%
Mauritius			100%	0%	100%	0%	100%	0%
Morocco			25%	75%	100%	0%	25%	75%
Nepal			50%	50%	100%	0%	50%	50%
New Zealand			100%	0%	100%	0%	100%	0%
Norway	100%	0%	100%	0%	100%	0%	100%	0%
Pakistan			50%	50%	100%	0%	50%	50%
Paraguay			50%	50%	100%	0%	50%	50%
Russia	55%	45%	50%	50%	75%	25%	50%	50%
Serbia	70%	30%	25%	75%	100%	0%	25%	75%
South Africa			100%	0%	100%	0%	100%	0%
Switzerland	100%	0%	100%	0%	100%	0%	100%	0%
Taiwan			50%	50%	100%	0%	50%	50%
Thailand			50%	50%	100%	0%	50%	50%
Tunisia			50%	50%	100%	0%	50%	50%
Turkey	57%	43%	50%	50%	100%	0%	50%	50%
Ukraine	51%	49%	50%	50%	100%	0%	25%	75%
United Arab Emirates			50%	50%	100%	0%	50%	50%
United States	100%	0%	100%	0%	100%	0%	75%	25%
Uruguay	100%	0%	25%	75%	100%	0%	25%	75%
Venezuela			25%	75%	100%	0%	25%	75%
Vietnam			50%	50%	100%	0%	50%	50%
Total	***65%***	***35%***	***69%***	***31%***	***75%***	***25%***	***62%***	***38%***

Source: EPF estimations

C.3. EU production and extra-EU trade of wood-based panels

Table C.3: EU production of wood-based panels, 2016 (1 000 m³)

Member State	Plywood	Particleboard	Of which: OSB	Fibreboard	Of which: MDF	*Total*
Austria	291	2 350	0	644	556	*3 285*
Belgium	24	1 600	350	300	300	*1 924*
Bulgaria	71	834	205	64	8	*969*
Croatia	2	158	0	0	0	*160*
Cyprus	0	0	0	0	0	*0*
Czech Republic	212	1 112	645	32	32	*1 356*
Denmark	0	346	0	3	0	*349*
Estonia	53	215	0	74	0	*341*
Finland	1 139	92	0	15	0	*1 246*
France	250	3 627	390	1 256	1 062	*5 133*
Germany	114	7 016	1 398	5 399	1 502	*12 530*
Greece	21	240	0	75	70	*336*
Hungary	49	389	20	355	336	*792*
Ireland	0	284	284	490	420	*774*
Italy	280	2 600	50	930	930	*3 810*
Latvia	280	1 040	598	0	0	*1 320*
Lithuania	44	728	0	68	0	*840*
Luxembourg	0	210	210	160	160	*370*
Malta	0	0	0	0	0	*0*
Netherlands	0	0	0	29	0	*29*
Poland	462	5 417	920	4 523	3 616	*10 402*
Portugal	47	700	0	405	405	*1 152*
Romania	285	4 480	1 580	655	650	*5 421*
Slovakia	420	595	0	0	0	*1 015*
Slovenia	86	0	0	130	130	*216*
Spain	369	1 755	2	1 629	1 520	*3 754*
Sweden	60	550	0	0	0	*610*
United Kingdom	0	2 349	345	684	684	*3 033*
Total	***4 559***	***38 687***	***6 997***	***17 920***	***12 381***	***61 166***

Source: FAO (2018)

Table C.4: Extra-EU imports of wood-based panels, 2016 (1 000 m³)

Member State	Plywood	Particleboard	Of which: OSB	Fibreboard	Of which: MDF	*Total*
Austria	19	14	1	7	1	*39*
Belgium [1]	442	0	0	7	6	*450*
Bulgaria	48	24	0	22	21	*95*
Croatia	6	11	0	2	2	*19*
Cyprus	5	0	0	1	1	*5*
Czech Republic [1]	44	21	0	3	1	*68*
Denmark	119	6	0	1	0	*126*
Estonia	74	6	0	22	5	*102*
Finland	75	5	0	7	3	*87*
France	173	6	1	36	13	*215*
Germany	675	219	2	240	108	*1 134*
Greece [1]	269	18	0	37	37	*325*
Hungary	27	34	0	7	4	*68*
Ireland	44	1	0	17	8	*62*
Italy [1]	264	64	1	46	19	*375*
Latvia [1]	68	7	0	2	1	*76*
Lithuania	57	141	8	27	18	*225*
Luxembourg	0	0	0	0	0	*0*
Malta [1]	3	0	0	1	1	*4*
Netherlands [1]	227	7	6	8	6	*241*
Poland	229	942	2	208	177	*1 379*
Portugal	33	3	0	8	8	*44*
Romania	53	91	5	79	50	*224*
Slovakia	20	32	32	3	0	*55*
Slovenia	8	5	0	1	0	*13*
Spain	57	1	0	12	6	*70*
Sweden	63	100	3	34	15	*196*
United Kingdom	1 200	26	5	50	12	*1 277*
Total	***4 303***	***1 784***	***67***	***887***	***523***	***6 974***

1. Data refer to 2015.

Source: Eurostat (2018b)

Table C.5: Extra-EU exports of wood-based panels, 2016 (1 000 m³)

Member State	Plywood	Particleboard	Of which: OSB	Fibreboard	Of which: MDF	*Total*
Austria	103	306	4	195	194	*605*
Belgium [1]	17	38	10	3	3	*58*
Bulgaria	21	217	111	10	1	*249*
Croatia	4	59	0	4	4	*67*
Cyprus	0	0	0	0	0	*0*
Czech Republic [1]	23	37	13	3	2	*63*
Denmark	24	59	7	4	0	*87*
Estonia	16	6	0	18	0	*40*
Finland	192	4	0	6	0	*202*
France	9	67	2	78	7	*154*
Germany	94	435	186	1 012	296	*1 540*
Greece [1]	2	18	0	8	2	*28*
Hungary	4	49	4	37	34	*90*
Ireland	0	5	0	14	9	*19*
Italy [1]	45	154	21	222	213	*421*
Latvia [1]	54	144	137	16	0	*214*
Lithuania	2	7	0	5	4	*14*
Luxembourg	:	:	:	:	:	*0*
Malta [1]	0	0	0	0	0	*0*
Netherlands [1]	11	14	1	18	16	*43*
Poland	23	101	42	391	155	*515*
Portugal	60	67	0	147	108	*274*
Romania	29	1 108	483	240	189	*1 377*
Slovakia	16	2	2	1	0	*18*
Slovenia	22	3	0	32	22	*57*
Spain	60	232	0	436	373	*728*
Sweden	9	19	3	38	33	*65*
United Kingdom	4	27	23	5	4	*36*
Total	***842***	***3 177***	***1 052***	***2 944***	***1 670***	***6 963***

1. Data refer to 2015.

Source: Eurostat (2018b)

Annex D: Impact assessment

D.1. Other Union-wide risk management options than restriction

The possibility to address the risks posed by the use of formaldehyde under other REACH regulatory measures, existing EU legislation and other possible Union-wide RMOs was examined. These measures were assessed as inappropriate to address *all* of the sectors and products contributing to risk.

Possible Union-wide risk management measures other than a restriction are outlined in Table D.1 below. However, it is concluded that none of these are realistic, effective and balanced means of solving the problem and none of these other risk management options have been analysed further.

Table D.1: Possible other Union-wide options discarded at this stage

Option	Reason for discarding this option
Non-legislative measures	
Voluntary industry agreements	The European wood-based panels industry adopted an internal agreement to produce only class E1 panels and to no longer place higher formaldehyde emitting class E2 panels on the EU market (see Section 1.5.1 in the main report). Voluntary agreements or commitments with respect to limiting formaldehyde emissions exist also in the European furniture and automotive industries (see Annex C.1). Articles that are not compliant with the voluntary agreements can however still be placed on the EU market, due to non-compliant EU producers and/or extra-EU imports.
Legislation other than REACH	
Construction Products Regulation (EU) 305/2011 (CPR)	The CPR requires a CE marking for construction products before they can be placed on the internal market. Construction products for which a harmonised European standard exists must comply with the relevant standard to obtain the required CE marking. The harmonised European standard for wood-based panels (EN 13986) defines two formaldehyde classes and requires formaldehyde containing wood-based panels to be tested and classified as either E1 or E2. E1/E2 emission classes are also defined in harmonised standards for other construction products (see Table D.2). The placing on the market of higher formaldehyde emitting class E2 products is however not restricted.
Biocidal Products Regulation (EU) 528/2012 (BPR)	The BPR requires an authorisation for all biocidal products before they can be placed on the market in the EU and the active substances contained in the products have to be previously approved. Biocidal products are defined as products intended to protect humans, animals, materials or articles against harmful organisms like pests or bacteria. Formaldehyde and some formaldehyde releasers are under review under BPR for certain product-types (PT). Wood treatment (PT8) is not included in the review implying that this use is not permitted in the EU. BPR does not apply to treated articles imported from non-EU countries and to articles releasing formaldehyde from substances used for other purposes than biocide.
Cosmetic Products Regulation (EU) 1223/2009	The Cosmetic Products Regulation is the main regulatory framework for finished cosmetic products placed on the EU market. Annex V to the regulation provides a list of preservatives allowed in cosmetic products including the conditions under which these substances can be used. Preservatives are defined in the regulation as substances which are exclusively or mainly intended to inhibit the development of micro-organisms in the cosmetic product. The annex specifies that all finished products containing formaldehyde or substances identified as formaldehyde releasers must be labelled with the warning “contain formaldehyde” if the concentration in the finished products is > 0.05%. The Cosmetic Products Regulation has a limited scope and it does not apply to non-cosmetic use of mixtures and articles.

Textile Regulation (EU) 1007/2011	The Textile Regulation sets out the general obligation to state the full fibre composition of textile products and a requirement to indicate the presence of non-textile parts of animal origin. The regulation does not have any provisions with respect to human or environmental safety of chemicals used in textiles.
Toy Safety Directive 2009/48/EC	This directive aims to ensure a high level of protection of children. Part III (Chemical properties) of Annex II to the Directive states that toys shall comply with the relevant Community legislation relating to certain categories or products or to restrictions for certain substances and mixtures. Moreover, the same part prohibits the use of CMR category 1A, 1B or 2 substances in toys, in components of toys or in micro-structurally distinct parts of toys. However, some exceptions to this provision exists, e.g. that these substances and mixtures are inaccessible to children in any form, including inhalation. The Toy Safety Directive has a limited scope as it only applies to toys.
Taxation on formaldehyde content	Taxation in general is not a harmonised measure across the EU. Therefore, whilst it might be effective in encouraging substitution, it is not likely that all Member States would introduce relevant taxes and thereby not all EU citizens will be protected. This is likely to lead to a non-harmonised situation where different Member States apply different tax rates (if at all).
Other REACH processes	
REACH Authorisation process	Authorisation may apply to formaldehyde (Carc. 1B). However, a number of formaldehyde releasers (including some formaldehyde-based resins) are not classified as CMR category 1A or 1B nor are they identified as PBTs or vPvBs nor have they been identified as substances of equivalent concern. Therefore authorisation cannot be used as a risk management measure for them. Also, this regulatory risk management measure does not apply to articles.
REACH Art. 68.2	REACH Article 68(2) stipulates that substances that are CMR categories 1 or 2 can be subject to a proposal from the Commission to inclusion in Annex XVII for consumer uses without using the procedures in article 69-73 in the REACH Regulation. While formaldehyde is classified Carc. 1B, a number of formaldehyde releasers (including formaldehyde-based resins) are not classified as CMR categories 1 or 2, therefore REACH Article 68(2) does not apply to them.

D.2. Evaluated restriction options

RO1 would certainly reduce risks as it would eliminate inhalation exposure to formaldehyde from articles and mixtures. Such a far-reaching measure would however not be consistent with the risk assessment, as underlined by the fact that measured formaldehyde indoor air concentrations are in most cases below the WHO guideline value and that this measure will not affect the contribution of external and temporary sources to formaldehyde concentration in indoor air. Furthermore, whilst the exposure estimates in Section 1.3.6.5 of the main report indicate that exceedances of the WHO guideline value are possible, specific concurrent circumstances are required for this to materialise. The risk reduction potential in relation to mixtures would be limited as these are already considered low risk (see Section 1.3.6.2 of the main report). RO1 is considered not proportionate to the risk. Its implementation would carry high costs given the large scale use of formaldehyde and formaldehyde-based resins and the unavailability of suitable substitutes for all uses (Global Insight, 2007). RO1 is considered enforceable in principle because of available methods to determine formaldehyde emissions but not practicable as it is not implementable and manageable in the foreseeable future owing to the substantial adjustments this measure would entail. Based on these considerations, RO1 is disregarded.

RO2 is associated with a number of uncertainties following from the difficulty of linking a concentration limit for formaldehyde and known (to date) formaldehyde releasers to formaldehyde emissions. Such a link is hard to establish as formaldehyde emissions do not

only result from the release of free formaldehyde but can also be generated through other mechanisms (e.g. hydrolysis) or through reactions of other substances. In some cases the mechanism through which formaldehyde is formed and released may be very complex and depending on a number of external factors (temperature, humidity, degradation of substances, chemical reactions occurring in nature, etc.) that are not always predictable. As a result RO2's effectiveness in reducing risks from consumer articles as well as its proportionality to the risk are uncertain. The risk reduction potential in relation to mixtures would be limited as these are already considered low risk (see Section 1.3.6.2 of the main report). Whilst a concentration limit is in principle considered enforceable, there are uncertainties with regard to the practicability of such a measure, which depends on the substance(s) the limit applies to. RO2 is disregarded due to its inherent uncertainties and the closer link of an emission limit to inhalation exposure and hence to the actual risk.

RO3 is considered consistent with the risk assessment as wood-based panels are the major (permanent) source of formaldehyde emissions to indoor air. The measure would effectively reduce risks as it would prevent high formaldehyde emitting class E2 wood-based panels from being placed on the EU market or used in the EU. RO2 is considered proportionate as costs to the EU society are expected to be limited because of a voluntary industry agreement based on which the vast majority of EU manufacturers already today produce only class E1 panels. For the same reason, RO3 is considered implementable and manageable. It is also considered enforceable as test methods for the determination of formaldehyde emissions exist. While RO3 would ensure that only class E1 panels are used for the manufacturing of finished products such as furniture or laminate flooring in the EU, high formaldehyde emitting articles made from non-compliant panels could still be imported from outside the EU. RO3 is therefore disregarded in favour of RO4.

RO4 extends the emission limit described in RO3 to all articles, including, but not limited to, wood-based panels. In setting a minimum standard for all consumer articles, RO4 represents a further precaution against producing and importing additional formaldehyde emitting articles, in particular wood-based products such as furniture and laminate flooring.[7] As such, RO4's risk reduction potential is considered somewhat higher than that of RO3, while it is expected to be similar to RO3 with respect to impact and efficiency considerations.

Under both options, RO3 and RO4, an emission limit lower than the one defined by the E1 emission class would not be consistent with the risk assessment. Not only are measured indoor air formaldehyde concentrations in most cases below the WHO guideline value, but the assessment in Section 2.5 of the main report also finds that the E1 emission class ensures the WHO guideline value under reasonable worst case assumptions. A lower emission limit would also lead to additional production costs and possibly additional investment costs, at least for some producers, depending on the level of such a limit (Nwaogu et al., 2013b). Compared to the E1 emission limit, a lower emission limit is not supported by the available information from a proportionality point of view.

[7] In a 2016 study, for instance, the Centers for Disease Control and Prevention found high formaldehyde emissions from Chinese-produced laminate flooring imported into the United States (CDC, 2016a; CDC, 2016b).

D.3. Formaldehyde testing

Nowadays, two standardised chamber methods for the determination of formaldehyde from construction products exist in Europe:

- **EN 717-1**: Wood-based panels – Determination of formaldehyde release – Part 1: Formaldehyde emission by the chamber method (CEN, 2004a)
- **EN 16516**: Construction products – Assessment of release of dangerous substances – Determination of emissions into indoor air (CEN, 2017)

EN 717-1 was developed to specifically determine formaldehyde emissions from wood-based panels and it is the reference method for determining the formaldehyde emission classes E1 and E2 of wood-based panels defined in EN 13986 (CEN, 2004b). EN 717-1 has also been successfully used, over the years, to measure formaldehyde emissions from a wide range of articles, such as flooring materials, furniture, textiles and insulation materials (see column "Used method" in Table B.8). Furthermore, it used to define the E1/E2 formaldehyde emission classes in other harmonised European standards as well (Table D.2).

Table D.2: Standards with formaldehyde emission classes based on EN 717-1

Standard	Title
EN 13986	Wood-based panels for use in construction – Characteristics, evaluation of conformity and marking
EN 14041	Resilent, textile and laminate floor coverings – Essential characteristics
EN 14080	Timber structures – Glue laminated timber and glued solid timber – Requirements
EN 14279	Laminated veneer lumber – Definitions, classification and specification
EN 14342	Wood flooring – Characteristics, evaluation of conformity and marking
EN 14374	Timber structures – Structural laminated veneer lumber – Requirements
EN 14915	Solid wood panelling and cladding – Characteristics, evaluation of conformity and marking
EN 15479	Structural finger jointed solid timber – Performance requirements and minimum production requirements
EN 16351	Timber structures – Cross laminated timber – Requirements

Source: Marutzky (2018)

The horizontal standard EN 16516 was published in 2017. Unlike EN 717-1, it is not specific to formaldehyde but has a broader scope and is applicable to volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), and some volatile carbonyl compounds, including formaldehyde. The standard defines a chamber test for the determination of area specific emission rates for these substances under defined test conditions and their expression as concentrations in a reference room. EN 16516 also defines this so-called European Reference Room (see Table 7 in the main report), which is not a test room but rather serves as exposure scenario to which all test results are calculated back (Marutzky, 2018; Oppl, 2014).

EN 717-1 and EN 16516 do not only differ in scope – formaldehyde from wood-based panels versus VOCs from construction products – but also in terms of test conditions (Table D.3). In particular, different specifications of relative humidity, air exchange rate and loading factors will yield different test results. The choice of a reference test method is therefore an important element with respect to ensuring compliance with the proposed restriction.

Table D.3: Test chamber parameters for EN 717-1 and EN 16516

Parameter	EN 717-1	EN 16516
Temperature	23 ± 0.5 °C	23 ± 1 °C
Relative humidity	45 ± 3%	50 ± 5%
Air exchange rate	1.0 ± 0.05 h^{-1}	**Test chamber**: 0.25-1.5 h^{-1} (± 5%) **Reference room**: 0.5 h^{-1}
Loading factor	1.0 ± 0.02 m^2/m^3	**Test chamber**: 50-200% of loading factor specified in reference room, max 2.0 m^2/m^3 **Reference room**: see Table 7 in the main report
Volume	**Option 1**: Large test chamber with minimum net volume of 12 m^3 **Option 2**: 1 m^3 **Option 3**: 0.225 m^3	**Test chamber**: Minimum 20 l **Reference room**: see Table 7 in the main report

Source: Adapted from Marutzky (2018) and Oppl (2014)

In response to the Call for Evidence launched by ECHA in January 2018, the German Environment Agency (Umweltbundesamt) provided a comment concerning the two chamber methods. In Germany, the Chemicals Prohibition Ordinance ("Chemikalien-Verbotsverordnung") sets a limit of formaldehyde emissions from wood-based panels of 0.1 ppm (= 0.124 mg/m^3). Germany is planning to replace the current test procedure, based on EN 717-1, with a new method based on EN 16516, the main argument being that the air exchange rate of 0.5 h^{-1} under EN 16516 is deemed more realistic in present-day dwellings than the 1 h^{-1} under EN 717-1. Germany proposes that testing in accordance with EN 717-1 should also be possible in the future but that the measured formaldehyde concentrations should be adjusted by a factor of two, i.e. doubled, to bring the results in line with EN 16516. An adjustment factor of two would effectively mean a halving of the emission limit permissible in the current definition of E1. This means that some wood-based panels which would be classified as E1 when tested under EN 717-1 conditions may be above the E1 emission limit when tested according to EN 16516 (or when the adjustment factor of two is applied to EN 717-1 test results).

The following considerations have led the Dossier Submitter to conclude that EN 717-1 is, among existing test methods, the most reliable and suitable method for the determination of formaldehyde emissions from articles:

- **Basing the proposed emission limit on EN 16516 would not be consistent with the risk assessment**: A literature review of indoor air measurements of formaldehyde in Europe shows that the WHO guideline value is exceeded only in rare cases. The main reasons for such exceedances have been attributed to the use of high-emitting materials (e.g. non E1 compliant wood-based panels), high loading factors, and/or the presence of temporary emission sources generating peak formaldehyde concentrations even when all used materials are in line with the E1 emission class. In addition, the estimated formaldehyde concentrations under realistic worst case assumptions would not warrant to effectively halve the proposed emission limit by basing it either on EN 16516 or by multiplying formaldehyde concentrations measured under EN 717-1 conditions by a factor of two.

- **EN 717-1 is considered more robust for the determination of formaldehyde emissions**: Formaldehyde has a different emission profile compared to other volatile compounds. Formaldehyde emissions from wood-based panels bonded with UF resins,

for instance, are declining over time but not to a value near zero. Instead, after some days of testing under constant test conditions they reach a constant emission rate defined as "steady-state concentration". The steady-state concentration declines as well over the course of months and years but at a much slower rate than the initial rapid decline (see Section 1.3.6.3 in the main report). The reason for this behaviour has been explained by the fact that in addition to free formaldehyde released from the formaldehyde-based resins used in production of wood-based panels, new formaldehyde is formed by hydrolysis of formaldehyde-based products contained in the panels when in contact with air. Wood-based panels typically contain formaldehyde-based resins in an amount of 5-15% of the mass of the board and new formaldehyde is formed from this pool. EN 717-1 defines a procedure to determine steady-state concentrations of formaldehyde, which is not the case for EN 16516 (Marutzky, 2018).

It follows from the above described emission behaviour that temperature and relative humidity have a direct impact on the measured emissions. EN 717-1 is considered to produce more reliable results as the tolerances allowed for temperature and humidity shown in Table D.3 are narrower compared to EN 16516.

- **EN 717-1 and its derived methods are deemed more suitable for ensuring compliance**: Derived test methods exist for EN 717-1, namely EN 717-2 (gas analysis method)[8] and EN 120 (perforator method)[9] which are also clearly recognised in the in the definition of the E1 emission class for wood-based panels in EN 13986. These simpler methods correlate with EN 717-1 (Marutzky and Meyer, 1993; Salem et al., 2012; Schwarz et al., 1992) and are cheaper and require less time. This is already now important for producers of wood-based panels who use these derived tests on a routine basis to determine the formaldehyde emission class of their production and to ensure compliance with national formaldehyde emission limits. As demonstrated in an enforcement project carried out in Sweden in 2014 (see Section 2.4.3 in the main report), authorities can use these derived methods also for screening purposes to identify potentially non-compliant articles which can subsequently be tested with the more expensive and time consuming EN 717-1 reference test method.

- **Stakeholders have experience with EN 717-1 and its derived test methods**: Both industry, as demonstrated by the need to classify wood-based panels and other products into formaldehyde emission classes, and enforcement authorities, at least in countries with national formaldehyde emission limits, have experience with EN 717-1 and its derived test methods. This limits any additional burden associated with the restriction proposal in terms of testing by companies to ensure compliance and enforcement activities by national authorities.

- **Analytical methods in EN 717-1 and EN 16516 are equally accurate but the latter requires more sophisticated and expensive equipment**: The standard EN 717-1 allows for different analytical methods for formaldehyde determination. Analytical methods include the reaction of formaldehyde with ammonium ions and acetylacetone (Hantzsch reaction) to form a yellow compound called

[8] EN 717-2: Wood-based panels – Determination of formaldehyde release – Part 2: Formaldehyde release by the gas analysis method. This standard has been replaced by ISO 12460-3 in 2008 (ISO, 2015).

[9] EN 120: Wood-based panels – Determination of formaldehyde release – Extraction method (called perforator method). This standard has been replaced by ISO 12460-5 in 2011 (ISO, 2011a).

diacetyldihydrolutidine (DDL). This reaction is specific to formaldehyde. DDL is determined photometrically (with a detection limit of 3 µg/m^3) or fluorimetrically (detection limit 1.5 µg/m^3). The standard allows also the determination of formaldehyde using the DNPH method according to standard ISO 16000-3 (ISO, 2011b). The DNPH method uses 2-4 dinitrophyhadazine (DNPH) to form hydrazones with carbonyl compound in acidic solution. This reaction is not specific to formaldehyde and therefore a chromatographic separation of formed hydrazones by means of high performance liquid chromatography (HPLC) with a photometric detector (detection limit 1 µg/m^3) is required. EN 16516 allows only the DNPH method for the determination of formaldehyde. Both methods described above allow for accurate determination of formaldehyde in the range of 5-150 µg/m^3. However, while the DDL method (EN 717-1) requires standard equipment and simple analytical procedures, the DNPH method requires more sophisticated and expensive HPLC equipment. In addition, as EN 717-1 allows also the use of the DNPH method along with the DDL method, it provides for more flexibility than EN 16516, which only allows the DNPH method (Marutzky, 2018).

D.4. Alternatives to UF resins

Notwithstanding temporary emission sources, external sources and naturally occurring reactions, the use of formaldehyde-based resins (in particular UF and MUF resins) in the production of articles (particularly wood-based panels) represents the most relevant source of formaldehyde exposure for consumers. The analysis of alternatives presented in this Annex therefore focuses on these products and is based on the risk management option analysis prepared by France on formaldehyde (ANSES, 2016) and on industry information (Nwaogu et al., 2013a).

Considering potential alternatives is important since any proposed risk management measure may instigate a shift to such alternatives. Availability, technical and economic feasibility, as well as effects on the environment and human health of alternatives are important aspects to consider in determining the most appropriate risk management option and in developing a restriction proposal.

The research of alternatives to formaldehyde-based resins includes technologies, processes and materials aiming at minimising the release of formaldehyde from articles. This includes the research and development of formaldehyde free resins as well as lower formaldehyde emitting resins (such as PF, MF, MUF and PRF resins).

UF and MUF resins are mostly used in the production of wood-based panels. UF resins are very economical and fast curing but are not suitable for damp conditions and are typically used for panels intended for non-structural use such as particleboard and hardwood plywood. UF adhesives are also non-staining and therefore do not blemish the high quality expensive face veneers used for hardwood panels for interior finish applications. Because the formaldehyde component of UF adhesives is not completely chemically fixed by the urea, some formaldehyde is free to dissipate and, as such, UF resins are associated with the highest releases of formaldehyde when compared with other formaldehyde-based resins (IARC, 2006). Other formaldehyde-based resins (PF, MF, MUF, RF, and PRF), which release little to no formaldehyde from the cured product, can be considered substitutes for UF resins (Nwaogu et al., 2013a). Due to limited information on availability, cost and performance of formaldehyde free products as alternatives to UF resins, a level of uncertainty remains. Table D.4 provides an overview of currently available alternatives to UF resins along with considerations on technical and economic feasibility as well as environmental and health considerations for each proposed

alternative. Alternatives include low emission formaldehyde-based resins, formaldehyde free products (chemically synthetized) and natural products.

ANSES (2016) contains details on availability, technical feasibility and costs of alternatives to MF, PF and POM resins. Some of these products are used in the production of consumer articles, including wood-based panels (e.g. plywood). However, as discussed in Annex B.4.2 (Wood-based products), formaldehyde exposure to consumer from the use of MF, PF and POM resins in consumer products is much lower compared to UF resins. For this reason, alternatives to MF, PF and POM resins are not further investigated here.

Table D.4: Alternatives to UF resins

Alternative	End use	Availability	Technical feasibility	Economic feasibility	Environmental / health considerations
Formaldehyde-based resins					
Phenol formaldehyde resins (PF)	Plywood, particleboard, OSB, MDF	Proven availability across Europe but widespread substitution of UF would require additional capacity investment or conversion of existing equipment where possible	• Good weather resistance, durability, adhesive properties and stability • Suitable for exterior, structural grade boards • Requires high temperature curing and long press times • Suitable for existing equipment and processes • Dark colour	• More expensive than UF (double to triple the price) but cheaper than other formaldehyde-based resins • Increased adhesive consumption required • Expected loss of production capacity	• Low/no formaldehyde emissions form cured product; no risks to consumer of formaldehyde emissions • Extent of actual risk reduction for workers uncertain due to continued use of formaldehyde • Concern for worker health due to risks when manufacturing/using phenol • Environmental concerns when using phenol
Melamine formaldehyde (MF) and melamine urea formaldehyde (MUF) resins	Plywood, particleboard, MDF		• Good weather and water resistance, clear and strong • Suitable for interior and semi-exterior panels • Suitable for existing equipment and processes • Similar to UF in terms of processing and applications	• MF is more expensive than UF (melamine about three times more expensive than urea) • MUF is cheaper than MF (but more expensive than UF) depending on the quantity of melamine used • Melamine capacity to meet demand of wood-based panels industry is uncertain	• Low/no formaldehyde emissions from cured products; no risks to consumer of formaldehyde emissions • Extent of actual risk reduction for workers uncertain due to continued use of formaldehyde
Resorcinol formaldehyde (RF) and phenol resorcinol formaldehyde (PRF) resins			• Good weather resistance, durable, curing at room temperature • Suitable for interior, exterior and humid environments • Suitable for existing equipment and processes • Produces dark colouration	• Expensive due to high cost of resorcinol; around four times the price of UF resins • Supplies of resorcinol may not be sufficient to meet the needs of the wood-based panels industry	• Low/no formaldehyde emissions from cured product; no risks to consumer of formaldehyde emissions • Resorcinol on CoRAP for evaluation in 2014 • Extent of actual risk reduction for workers uncertain due to continued use of formaldehyde • Worker health concerns regarding both phenol and resorcinol

Synthetic substances					
Polymeric methylene diphenyl diisocyanate (p-MDI)	Plywood, particleboard, OSB, MDF, beams	High variability in availability due to dependency on other applications like foam production for insulation and automotive production; due to stringent regulation no further p-MDI production capacity will be available in Europe in future	• Excellent strength, heat, water and humidity resistance • Suitable for exterior grade boards • Not suitable to existing plant and equipment	• More expensive than UF and PF (around four times the costs of UF) • Smaller dosage required • Major supply issues; cannot meet demand of wood-based panels industry • Costs of achieving suitable plant and equipment • Additional costs of maintaining safe operations in plants due to hazards	• No formaldehyde emissions from cured product; no risks to consumers of formaldehyde emissions • Potential worker exposure to isocyanates (risk of occupational asthma) • Worker health risks due to contents of p-MDI, particularly MDI
Emulsion polymer isocyanates (EPI)	Solid wood lamination, laminated beams; not for plywood, particleboard, MDF	Low availability	• High dry/wet strength, durable bonds, cold cured and fast setting seeds • Short pot life • May be suitable for use with existing equipment • Additional process steps and equipment required for mixing and metering and to manage tackiness of EPI • Sticks to metals	• High costs • Additional processing steps and equipment required	• No threat to the environment • No formaldehyde emissions and inert when properly hardened • Potential worker exposure to isocyanate during manufacture
Polyurethanes	Solid wood lamination, laminated beams; not for particleboard, MDF	Low availability, actually just serving a small proportion of solid wood lamination	• High wet/dry strength • Resistance to water and damp atmospheres • Curing at room temperatures • Sticks to press platens, stains easily	• High costs, may be prohibitive • Additional release agent required to avoid sticking to the press platens	• Potential worker exposure to isocyanates such as MDI • IARC group 3 substance
Epoxy adhesives	Special bonding application between wood/wood-based panels and other materials	Insufficient availability for high volume wood-based panels production	• Excellent moisture and weather resistance and strong bonds • Additional metering and mixing equipment required • Can be difficult to use and require long cure times	• High market price • Typically used at greater weights per bonded surface	• Cured epoxy resins are inert • Potential for health risks to workers as many components are toxic or irritants • Potential environmental concerns

PVA and EVA	Solid wood lamination, coating of particleboard with veneers and finish foils; no known application for particleboard, MDF	Sufficient availability	• Good dry strength and easy to use • Poor moisture resistance and thermoplastic • Lack of technical characteristics required for use in wood-based panels	• Significantly more expensive than UF	• Environmentally friendly • No health risks; low/no VOCs and solvent free
Natural/bio-based adhesives					
Protein glues	Particleboard, MDF	So far no industrial production	• Poor water/mould resistance and limited durability • Uncross-linked glues generally lack required technical properties • Requires chemical cross-linker (usually formaldehyde) to be technically viable	• Generally low cost • Critical supply problems are likely to exist for blood and casein	• No formaldehyde emissions from final product • Environmentally safe • Health and safety concerns exist over the use of blood and in relation to additional cross-linkers needed to produce technically suitable boards
Tannins	Plywood, particleboard, MDF	Tannins are only available outside Europe, with some very small volumes imported to Europe for niche markets; product can only be shipped in powder form, which needs additional energy for spray drying	• Low performance • Inconsistency of the material makes manufacturing with consistent properties difficult • Short pot life and weak bond formation • Requires chemical cross-linker (usually formaldehyde) to be technically viable	• Expensive • Limited supply	• No health/environmental concerns for uncross-linked tannin adhesives • Extent of actual risk reduction when cross-linked using formaldehyde is unclear
Lignin adhesives	Plywood, particleboard, MDF	Lignin as such is available in large volumes, but original lignin from the pulp and paper industry cannot be used without pre-treatment limiting the actual availability; homogeneity and purity are limiting factors for usage	• Long cure times and high cure temperature • Can be corrosive to machinery • Requires chemical cross-linker (usually formaldehyde) to be technically viable	• Available in large quantities • Low cost	• No health/environmental concerns for uncross-linked lignin adhesives • Extent of actual risk reduction when cross-linked using formaldehyde is unclear

Source: ANSES (2016), Nwaogu et al. (2013a)

D.5. Enforcement costs

Enforcement costs are administrative costs incurred by Member States' enforcement agencies to ensure that economic actors on the EU-28 market comply with EU regulations. By evaluating data reported from European studies on inspection/enforcement costs of REACH restrictions (Milieu, 2012), ECHA assessed the administrative burden of enforcement for new restriction proposals. ECHA concluded that based on data reported by Member States, the average administrative cost of enforcing a restriction is approximately €55 600 per year.

This value is estimated based on numbers of controls over the period 2010-2014 reported by Member States (reporting under REACH art. 117 / CLP art.46). The calculation is based on an average cost per control (inspection) and an average number of controls per restriction. ECHA notes that while the average enforcement costs may remain fairly similar over time, as they are driven by budgetary constraints, the costs for individual restrictions would likely vary. It is often the practice that enforcement campaigns focus on newer restrictions or high-risk restrictions considered a priority by Member States, and fewer resources are allocated to restrictions industry is already familiar with.

For the purpose of the current assessment, the value of €55 600 per year, rounded up to €60 000, should be seen as only illustrative in terms of the order of magnitude of the cost.

D.6. Human health impacts

D.6.1. Average dwelling size in the EU

Table D.5: Average size of dwelling by tenure status, 2012 (m^2)

Member State	Total population	Owner		Tenant	
		without mortgage	with mortgage	market price	reduced price or free
Austria	99.7	125.3	130.2	66.6	81.0
Belgium	124.3	139.0	145.5	85.7	91.0
Bulgaria	73.0	75.0	76.3	53.7	60.9
Croatia	81.6	82.7	87.6	57.7	72.8
Cyprus	141.4	156.5	177.6	91.9	112.3
Czech Republic	78.0	80.7	92.9	59.1	63.1
Denmark	115.6	141.4	146.6	79.6	117.1
Estonia	66.7	68.0	83.4	44.3	53.3
Finland	88.6	99.4	109.8	54.3	55.6
France	93.7	110.1	108.9	66.7	71.3
Germany	94.3	121.4	127.7	69.2	74.3
Greece	88.6	93.4	100.3	70.6	79.1
Hungary	75.6	77.9	81.2	49.8	56.2
Ireland	80.8	83.0	98.9	63.7	58.4
Italy	93.6	99.6	98.6	73.9	82.0
Latvia	62.5	64.3	85.1	44.7	48.6
Lithuania	63.2	64.4	70.9	42.5	47.6
Luxembourg	131.1	156.4	147.6	83.2	106.4
Malta [1]	:	:	:	:	:
Netherlands	106.7	133.1	127.8	78.0	113.2
Poland	75.2	80.4	88.1	45.7	52.5
Portugal	106.4	110.5	123.5	77.6	82.8
Romania	44.6	44.9	44.7	32.4	34.5
Slovakia	87.4	89.2	95.4	63.1	76.5
Slovenia	80.3	86.0	93.6	47.6	66.1
Spain	99.1	103.3	101.4	81.0	92.8
Sweden	103.3	105.1	125.3	69.7	131.4
United Kingdom [1]	:	:	:	:	:
Total	***95.9***	***96.8***	***119.7***	***74.5***	***78.7***

1. Unreliable data

Source: Eurostat (2016) based on Eurostat 2012 ad-hoc module 'Housing Conditions' (ilc_hcmh01)

D.6.2. Average household size in the EU

Table D.6: Average household size, 2016

Member State	Household members	Member State	Household members
Austria	2.2	Italy	2.3
Belgium	2.3	Latvia	2.4
Bulgaria	2.5	Lithuania	2.2
Croatia	2.8	Luxembourg	2.5
Cyprus	2.7	Malta	2.6
Czech Republic	2.4	Netherlands	2.2
Denmark	2.0	Poland	2.8
Estonia	2.2	Portugal	2.5
Finland	2.0	Romania	2.7
France	2.2	Slovakia	2.8
Germany	2.0	Slovenia	2.5
Greece	2.6	Spain	2.5
Hungary	2.3	Sweden	2.0
Ireland	2.7	United Kingdom	2.3
		Total	***2.3***

Source: Eurostat (2018a)

D.7. Average costs of new dwelling

Table D.7: Average transaction price of a new dwelling in selected Member States, 2016

Member State	Transaction price [€/m²]	Price for EU average dwelling size (= 96 m²) [1 000 €]
Austria	2 553	245
Belgium	2 261	217
Czech Republic	2 162	208
Denmark	2 554	245
France	4 103	394
Germany[1]	3 242	311
Hungary	1 164	112
Ireland	3 458	332
Italy	2 334	224
Netherlands[2]	2 306	221
Poland	1 321	127
Portugal[3]	1 068	103
Slovenia	2 150	206
Spain	2 030	195
United Kingdom	4 397	422

1. Bid price rather than transaction price.
2. Older dwellings rather than new dwellings.
3. All dwellings (old and new).

Source: Deloitte (2017)

Annex E: Assumptions, uncertainties and sensitivities

E.1. EU housing stock and share of dwellings built/completed

Table E.1: Number of dwellings in the EU, 2015 (or nearest)

Member State	Total number of dwellings	Year
Austria	4 506 000	2015
Belgium [1]	5 083 960	2015
Bulgaria	3 935 105	2015
Croatia	1 923 522	2013
Cyprus	441 251	2013
Czech Republic	4 756 572	2011
Denmark	2 628 338	2015
Estonia	657 791	2011
Finland	2 934 440	2015
France	34 923 000	2014
Germany	41 446 300	2015
Greece	6 371 901	2011
Hungary	4 420 296	2015
Ireland	2 022 000	2015
Italy	31 208 161	2011
Latvia	1 022 570	2011
Lithuania	1 308 671	2014
Luxembourg	227 326	2014
Malta	223 850	2011
Netherlands	7 588 000	2015
Poland	13 983 000	2014
Portugal	5 926 286	2015
Romania	8 722 398	2011
Slovakia	1 941 176	2011
Slovenia	845 415	2015
Spain	25 208 623	2011
Sweden	4 637 636	2015
United Kingdom	28 073 000	2014
Total	***246 966 588***	
Member States without "E1 legislation" [2]	***147 271 581***	

1. Estimated as 2/3 of dwellings in the Netherlands based on difference in population size.
2. Total minus Austria, Denmark, Germany, Greece, Italy, Lithuania, Netherlands and Sweden.

Source: OECD (2016) and own calculations

Table E.2: Share of dwellings built/completed in EU housing stock, 2015 (or nearest)

Member State	Number of dwellings built/completed in the year	Total number of dwellings	Dwellings built/completed as a share of total number of dwellings	Year
Austria	44 000	4 506 000	0.98%	2015
Bulgaria	7 806	3 935 105	0.20%	2015
Croatia	10 090	1 923 522	0.52%	2013
Cyprus	3 833	441 251	0.87%	2013
Czech Republic	25 238	4 756 572	0.53%	2011
Denmark	14 352	2 628 338	0.55%	2015
Estonia	2 079	657 791	0.32%	2011
Finland	28 672	2 934 440	0.98%	2015
France	413 600	34 923 000	1.18%	2014
Germany	247 722	41 446 300	0.60%	2015
Hungary	7 612	4 420 296	0.17%	2015
Ireland	12 666	2 022 000	0.63%	2015
Latvia	2 201	1 022 570	0.22%	2011
Lithuania	7 624	1 308 671	0.58%	2014
Luxembourg [1]	2 642	227 326	1.16%	2013
Netherlands	48 000	7 588 000	0.63%	2015
Poland [1]	148 000	13 983 000	1.06%	2015
Portugal	6 687	5 926 286	0.11%	2015
Slovakia [2]	15 100	1 941 176	0.78%	2013
Slovenia	5 498	844 656	0.65%	2011
Spain	179 351	25 208 623	0.71%	2011
Sweden	37 549	4 637 636	0.81%	2015
United Kingdom [1]	138 000	28 073 000	0.49%	2013
Total	***1 408 322***	***195 355 559***	***0.72%***	

Note: Data on dwellings built/completed missing for Belgium, Greece, Italy, Malta and Romania.
1. The total number of dwellings refers to 2014 and differs from the year of construction/completion.
2. The total number of dwellings refers to 2011 and differs from the year of construction/completion.

Source: OECD (2016) and own calculations

Annex F: Stakeholder information

F.1. Call for evidence

In parallel with the publication of the intention to prepare this Annex XV restriction dossier, ECHA launched a call for evidence with the aim to gather information from stakeholders on the use of formaldehyde and formaldehyde releasers in articles and mixtures (in concentration < 0.1%) by consumers. The consultation started on 11 January 2018 and ended on 11 April 2018. In total, 21 comments were received from industry associations (including downstream users of formaldehyde-based products), private companies, Member State authorities, and individuals. Information was provided on the use of formaldehyde and formaldehyde releasers in articles and mixtures for consumer use, the release of formaldehyde from articles, consumer exposure, test methods, and enforcement activities performed by individual Member States. In addition, information was provided on the use of formaldehyde-based resins in the woodworking industry, in construction products, and on the use of formaldehyde releasers as preservatives in some mixtures. More information is available in the background note included in the call for evidence: https://echa.europa.eu/previous-calls-for-comments-and-evidence/-/substance-rev/18151/terml [Accessed 7 January 2019]

F.2. Discussions with industry

In addition to launching the call for evidence, ECHA held a meeting in January 2018 including representatives of the chemical industry (represented by the European Chemical Industry Council – Cefic), manufacturers of formaldehyde and formaldehyde-based products (represented by the formaldehyde sector group – Formacare), and the wood-based panels industry (represented by the European Panels Federation – EPF). The aim of the meeting was to clarify the scope of the restriction proposal and to gather information on substances, uses and emissions.

A follow up meeting was held at the ECHA premises in June 2018. Stakeholders attending the meeting included the chemical industry, represented by the lead registrant (BASF) and the relevant industry association (Cefic/Formacare), the wood-based panels industry, represented by the European Panel Federation (EPF), the Competent Authority of the Netherlands (RIVM), and Professor Tunga Salthammer from Fraunhofer WKI. Professor Salthammer has played a major role, over the years, in studying formaldehyde exposure in indoor environments and was responsible for the preparation of a report on consumer exposure to formaldehyde (Salthammer and Gunschera, 2017), which served as a major source of information for the preparation of the present Annex XV restriction report. The purpose of the meeting was to discuss the Salthammer and Gunschera (2017) report, to gather additional information and clarifications from both industry and Professor Salthammer, and to provide information on the Substance Evaluation work carried out by the Netherlands as well as ECHA's restriction work related to formaldehyde.

Following the discussion in the June 2018 meeting, EPF provided ECHA with specific information on the wood-based panels market (including production and import quantities, a breakdown of these quantities into emission classes E1 and E2, and price information), on the use of formaldehyde-based resins in the production of wood-based panels, and on test methods for the determination of formaldehyde emissions by involving Professor Rainer Marutzky, former Director of the Fraunhofer Institute for Wood Research (Fraunhofer WKI).

Furthermore, in the course of 2018, ECHA exchanged information via phone and email with a number of other relevant industry groups. To gather additional information on the use of

formaldehyde and formaldehyde releasers in mixtures, ECHA contacted the International Association for Soaps, Detergents and Maintenance Products (AISE), the Association of the European Adhesive and Sealant Industry (FEICA), and the European Council of the Paint, Printing Ink and Artists' Colour Industry (CEPE). ECHA was also in contact with the European Furniture Industries Confederation (EFIC). Additional discussions were held with the European Automobile Manufacturers' Association (ACEA) in the second half of 2018, to gather information on the use of formaldehyde and formaldehyde releasers in the automotive industry and on measures to control consumer exposure to formaldehyde emissions in car interiors (e.g. from foams used in car seats, plastics and textiles).

F.3. Consultations with authorities

This Annex XV restriction dossier was prepared in parallel with the work carried out by the Competent Authority of the Netherlands (RIVM) on Substance Evaluation of formaldehyde covering risks for consumers. ECHA and RIVM had numerous exchanges via phone and email throughout 2018 on consumer uses and exposure related to formaldehyde. Representatives of the RIVM also participated in the June 2018 meeting at ECHA with industry and Professor Salthammer.

ECHA also consulted the Swedish Chemicals Agency (KEMI) regarding an enforcement project on formaldehyde emissions from wood-based panels carried out in Sweden in 2014. KEMI provided ECHA with further details on the project, including on costs and human resources.

ECHA also had discussions with the French Competent Authority (ANSES) who carried out the Substance Evaluation of formaldehyde addressing risks for workers. ECHA also kept the European Commission informed along the various phases of dossier preparation to obtain feedback, provide clarifications, and to discuss further information needs.

References

Abbass, O. A., Sailor, D. J., Gall, E. T. (2017). Effect of fiber material on ozone removal and carbonyl production from carpets. *Atmospheric Environment,* 148, 42-48.

Ahn, J.-H., Kim, K.-H., Kim, Y.-H., Kim, B.-W. (2015). Characterization of hazardous and odorous volatiles emitted from scented candles before lighting and when lit. *Journal of Hazardous Materials,* 286, 242-251.

Aldag, N., Gunschera, J., Salthammer, T. (2017). Release and absorption of formaldehyde by textiles. *Cellulose,* 24(10), 4509-4518.

Andersen, H. V., Klinke, H. B., Funch, L. W., Gunnarsen, L. (2016). *Emission of Formaldehyde from Furniture*. Copenhagen: The Danish Environmental Protection Agency.

Andersen, M. E., Clewell, H. J., 3rd, Bermudez, E., Dodd, D. E., Willson, G. A., Campbell, J. L., Thomas, R. S. (2010). Formaldehyde: integrating dosimetry, cytotoxicity, and genomics to understand dose-dependent transitions for an endogenous compound. *Toxicol Sci,* 118(2), 716-31.

ANSES (2016). *Analysis of the most appropriate risk management option (RMOA)*. Maisons-Alfort: Agence nationale de sécurité sanitaire de l'alimentation, de l'environnement et du travail.

ANSES (2018). OPINION of the French Agency for Food, Environmental and Occupational Health & Safety on the revision of ANSES's reference values for formaldehyde: occupational exposure limits (OELs), derived no-effect levels (DNELs) for professionals, toxicity reference values (TRVs) and indoor air quality guidelines (IAQGs).

Arts, J. H., Rennen, M. A., de Heer, C. (2006). Inhaled formaldehyde: evaluation of sensory irritation in relation to carcinogenicity. *Regul Toxicol Pharmacol,* 44(2), 144-60.

BAAQMD (2012). *Compliance Advisory Wood Products Coatings: Formaldehyde Emissions Estimates*. San Francisco: Bay Area Air Quality Management District.

Baek, S.-O., Jenkins, R. A. (2004). Characterization of trace organic compounds associated with aged and diluted sidestream tobacco smoke in a controlled atmosphere—volatile organic compounds and polycyclic aromatic hydrocarbons. *Atmospheric Environment,* 38(38), 6583-6599.

Baker, R. R. (2006). The generation of formaldehyde in cigarettes—Overview and recent experiments. *Food and Chemical Toxicology,* 44(11), 1799-1822.

Baker, R. R., Coburn, S., Liu, C. (2006). The pyrolytic formation of formaldehyde from sugars and tobacco. *Journal of Analytical and Applied Pyrolysis,* 77(1), 12-21.

BASF (2017). *Chemical Safety Report: Formaldehyde*.

Bednarek, M., Fuhrmann, F., Meyer, B., Rohde, D., Salthammer, T., Schulz, M., Uhde, E. (1997). Human exposure to air pollutants during a dinner. *In:* Woods, J. E., Grimsrud, D. T., Boschi, N. (eds.) *Healthy Buildings.* Washington DC, 209-214.

Bender, J. (2002). The use of noncancer endpoints as a basis for establishing a reference concentration for formaldehyde. *Regul Toxicol Pharmacol,* 35(1), 23-31.

Berglund, B., Hoglund, A., Esfandabad, H. S. (2012). A Bisensory Method for Odor and Irritation Detection of Formaldehyde and Pyridine. *Chemosensory Perception,* 5(2), 146-157.

BfR (2006). *Toxikologische Bewertung von Formaldehyd; Stellungnahme des BfR Nr. 023/2006 vom 30. März 2006*. Bundesamt für Risikobewertung.

Bogdanffy, M. S., Morgan, P. H., Starr, T. B., Morgan, K. T. (1987). Binding of formaldehyde to human and rat nasal mucus and bovine serum albumin. *Toxicol Lett,* 38(1-2), 145-54.

Böhm, M., Salem, M. Z., Srba, J. (2012). Formaldehyde emission monitoring from a variety of solid wood, plywood, blockboard and flooring products manufactured for building and furnishing materials. *J Hazard Mater,* 221-222, 68-79.

Bruening, T., Bartsch, R., Bolt, H. M., Desel, H., Drexler, H., Gundert-Remy, U., Hartwig, A., Jackh, R., Leibold, E., Pallapies, D., Rettenmeier, A. W., Schluter, G., Stropp, G., Sucker, K., Triebig, G., Westphal, G., van Thriel, C. (2014). Sensory irritation as a basis for setting occupational exposure limits. *Arch Toxicol,* 88(10), 1855-79.

Casanova, M., Deyo, D. F., Heck, H. D. (1989). Covalent binding of inhaled formaldehyde to DNA in the nasal mucosa of Fischer 344 rats: analysis of formaldehyde and DNA by high-performance liquid chromatography and provisional pharmacokinetic interpretation. *Fundam Appl Toxicol,* 12(3), 397-417.

Casanova, M., Heck, H. D., Everitt, J. I., Harrington, W. W., Jr., Popp, J. A. (1988). Formaldehyde concentrations in the blood of rhesus monkeys after inhalation exposure. *Food Chem Toxicol,* 26(8), 715-6.

Casanova, M., Morgan, K. T., Gross, E. A., Moss, O. R., Heck, H. A. (1994). DNA-protein cross-links and cell replication at specific sites in the nose of F344 rats exposed subchronically to formaldehyde. *Fundam Appl Toxicol,* 23(4), 525-36.

Casanova, M., Morgan, K. T., Steinhagen, W. H., Everitt, J. I., Popp, J. A., Heck, H. D. (1991). Covalent binding of inhaled formaldehyde to DNA in the respiratory tract of rhesus monkeys: pharmacokinetics, rat-to-monkey interspecies scaling, and extrapolation to man. *Fundam Appl Toxicol,* 17(2), 409-28.

CDC (2016a). *Laminate Flooring Test Results - Health Issues and Solutions* [Online]. Centers for Disease Control and Prevention. Available: https://www.cdc.gov/nceh/laminateflooring/ [Accessed 20 November 2016].

CDC (2016b). *Possible health implications from exposure to formaldehyde emitted from laminate flooring tested by the Consumer Product Safety Commission*. Atlanta: Centers for Disease Control and Prevention.

CEN (2004a). *EN 717-1 Wood-based panels - Determination of formaldehyde release - Part 1: Formaldehyde emission by the chamber method*. Brussels: European Committee for Standardization.

CEN (2004b). *EN 13986 Wood-based panels for use in construction - Characteristics, evaluation of conformity and marking*. Brussels: European Committee for Standardization.

CEN (2017). *EN 16516 Construction products: Assessment of release of dangerous substances - Determination of emissions to indoor air*. Brussels: European Committee for Standardization.

Collins, J. J., Ness, R., Tyl, R. W., Krivanek, N., Esmen, N. A., Hall, T. A. (2001). A review of adverse pregnancy outcomes and formaldehyde exposure in human and animal studies. *Regul Toxicol Pharmacol,* 34(1), 17-34.

Conolly, R. B., Kimbell, J. S., Janszen, D., Schlosser, P. M., Kalisak, D., Preston, J., Miller, F. J. (2004). Human respiratory tract cancer risks of inhaled formaldehyde: dose-response

predictions derived from biologically-motivated computational modeling of a combined rodent and human dataset. *Toxicol Sci,* 82(1), 279-96.

Dalbey, W. E. (1982). Formaldehyde and tumors in hamster respiratory tract. *Toxicology,* 24(1), 9-14.

Dallas, C. E., Scott, M. J., Ward, J. B., Jr., Theiss, J. C. (1992). Cytogenetic analysis of pulmonary lavage and bone marrow cells of rats after repeated formaldehyde inhalation. *J Appl Toxicol,* 12(3), 199-203.

Deloitte (2017). *Property Index: Overview of European Residential Markets. 6th edition, July 2017.* Deloitte.

Derudi, M., Gelosa, S., Sliepcevich, A., Cattaneo, A., Rota, R., Cavallo, D., Nano, G. (2012). Emissions of air pollutants from scented candles burning in a test chamber. *Atmospheric Environment,* 55, 257-262.

DFG (2010). Formaldehyde [MAK Value Documentation, 2010]. *In:* Hartiwig, A. (ed.) *The MAK-Collection for Occupational Health and Safety.* Weinheim: Wiley-VCH.

Duong, A., Steinmaus, C., McHale, C. M., Vaughan, C. P., Zhang, L. (2011). Reproductive and developmental toxicity of formaldehyde: a systematic review. *Mutat Res,* 728(3), 118-38.

ECHA (2017). *Assessment Report: Formaldehyde Product-type 02 (Disinfectants and algaecides not intended for direct application to humans or animals)*. Helsinki: European Chemicals Agency.

Edrissi, B., Taghizadeh, K., Moeller, B. C., Yu, R., Kracko, D., Doyle-Eisele, M., Swenberg, J. A., Dedon, P. C. (2017). N(6)-Formyllysine as a Biomarker of Formaldehyde Exposure: Formation and Loss of N(6)-Formyllysine in Nasal Epithelium in Long-Term, Low-Dose Inhalation Studies in Rats. *Chem Res Toxicol,* 30(8), 1572-1576.

EFIC (2017). *EFIC comments to the decree on the labelling of furniture products regarding their emissions of volatile pollutants notified to the EU under Directive (EU) 2015/1535* [Online]. Available: http://www.efic.eu/public/documents/EFIC%20comments%20to%20French%20draft%20regulation%20on%20VOC%20labelling.pdf [Accessed 7 December 2018].

EPF (2017). *Annual Report 2016-2017*. Brussels: European Panel Federation.

EPF/EFIC (2015). *Announcement on project "Compulsory E1" related to furniture products* [Online]. Available: http://www.efic.eu/public/documents/E1%20project.pdf [Accessed 7 December 2018].

Eurostat (2016). *Average size of dwelling by tenure status, 2012* [Online]. Available: https://ec.europa.eu/eurostat/statistics-explained/index.php?title=File:Average_size_of_dwelling_by_tenure_status,_2012.png [Accessed 19 November 2018].

Eurostat (2018a). *Average household size - EU-SILC survey [ilc_lvph01]* [Online]. Eurostat. Available: http://ec.europa.eu/eurostat/data/database [Accessed 9 November 2018].

Eurostat (2018b). *Sawnwood and panels [for_swpan]* [Online]. Eurostat. Available: http://ec.europa.eu/eurostat/data/database [Accessed 4 January 2019].

FAO (2018). *Forestry Production and Trade* [Online]. Food and Agriculture Organization of the United Nations. Available: http://www.fao.org/faostat/en/#data/FO [Accessed 4 January 2019].

Formacare (2018). *Response from Formacare to the call for evidence in relation to the Annex XV dossier on formaldehyde and formaldehyde releasers*. Formacare.

Fortmann, R., Kariher, P., Clayton, R. (2006). *Indoor Air Quality: Residential Cooking Exposures*. Sacramento, CA: California Air Resources Board.

Global Insight (2007). *Socio-Economic Benefits of Formaldehyde to the European Union (EU 25) and Norway. Prepared for Formacare*. Lexington: Global Insight.

Golden, R. (2011). Identifying an indoor air exposure limit for formaldehyde considering both irritation and cancer hazards. *Crit Rev Toxicol,* 41(8), 672-721.

Guillaume, E., Loferme-Pedespan, N., Duclerget-Baudequin, A., Raguideau, A., Fulton, R., Lieval, L. (2013). Ethanol fireplaces: Safety matters. *Safety Science,* 57, 243-253.

Heck, H. A., Chin, T. Y., Schmitz, M. C. (1983). Distribution of [^{14}C]formaldehyde in rats after inhalation exposure. *In:* Gibson, J. E. (ed.) *Formaldehyde toxicity.* Washington DC: Hemisphere, 26-37.

Heck, H. D., Casanova-Schmitz, M., Dodd, P. B., Schachter, E. N., Witek, T. J., Tosun, T. (1985). Formaldehyde (CH2O) concentrations in the blood of humans and Fischer-344 rats exposed to CH2O under controlled conditions. *Am Ind Hyg Assoc J,* 46(1), 1-3.

Heck, H. D., Casanova, M. (2004). The implausibility of leukemia induction by formaldehyde: a critical review of the biological evidence on distant-site toxicity. *Regul Toxicol Pharmacol,* 40(2), 92-106.

Heck, H. D., White, E. L., Casanova-Schmitz, M. (1982). Determination of formaldehyde in biological tissues by gas chromatography/mass spectrometry. *Biomed Mass Spectrom,* 9(8), 347-53.

Horn, W., Jann, O., Kasche, J., Bitter, F., Müller, D., Müller, B. (2007). *Umwelt- und Gesundheitsanforderungen an Bauprodukte - Ermittlung und Bewertung der VOC-Emissionen und geruchlichen Belastungen.* Dessau: Umweltbundesamt.

IARC (1995). Formaldehyde. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans.* Lyon: Word Health Organisation, 217-374.

IARC (2006). Formaldehyde. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans.* Lyon: World Health Organisation, 37-326.

ISO (2011a). *ISO 12460-5 Wood-based panels - Determination of formaldehyde release - Part 5: Extraction method (called the perforator method)*. Geneva: International Organization for Standardization.

ISO (2011b). *ISO 16000-3 Indoor air - Part 3: Determination of formaldehyde and other carbonyl compounds in indoor air and test chamber air - Active sampling method*. Geneva: International Organization for Standardization.

ISO (2011c). *ISO 16000-6 Indoor air - Part 6: Determination of volatile organic compounds in indoor and test chamber air by active sampling on Tenax TA sorbent, thermal desorption and gas chromatography using MS or MS-FID*. Geneva: International Organization for Standardization.

ISO (2012). *ISO 12219-1 Interior air of road vehicles - Part 1: Whole vehicle test chamber - Specification and method for the determination of volatile organic compounds in cabin interiors*. Geneva: International Organization for Standardization.

ISO (2015). *ISO 12460-3 Wood-based panels - Determination of formaldehyde release - Part 3: Gas analysis method*. Geneva: International Organization for Standardization.

Iversen, O. H. (1986). Formaldehyde and skin carcinogenesis. *Environment International,* 12(5), 541-544.

Jensen, L. K., Larsen, A., Molhave, L., Hansen, M. K., Knudsen, B. (2001). Health evaluation of volatile organic compound (VOC) emissions from wood and wood-based materials. *Arch Environ Health,* 56(5), 419-32.

Jiang, S. F., Yu, L. Q., Cheng, J., Leng, S. G., Dai, Y. F., Zhang, Y. S., Niu, Y., Yan, H. F., Qu, W. D., Zhang, C. Z., Zhang, K., Yang, R. J., Zhou, L. H., Zheng, Y. X. (2010). Genomic damages in peripheral blood lymphocytes and association with polymorphisms of three glutathione S-transferases in workers exposed to formaldehyde. *Mutation Research-Genetic Toxicology and Environmental Mutagenesis,* 695(1-2), 9-15.

JRC (2005). *Final Report The INDEX project Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU*. Ispra, Iraly: Joint Research Center.

Kamata, E., Nakadate, M., Uchida, O., Ogawa, Y., Suzuki, S., Kaneko, T., Saito, M., Kurokawa, Y. (1997). Results of a 28-month chronic inhalation toxicity study of formaldehyde in male Fisher-344 rats. *J Toxicol Sci,* 22(3), 239-54.

Katsoyiannis, A., Leva, P., Kotzias, D. (2008). VOC and carbonyl emissions from carpets: a comparative study using four types of environmental chambers. *J Hazard Mater,* 152(2), 669-76.

KEMI (2015). *Formaldehyd i träskivor. Tillsynsprojekt 2014*. Sundbyberg: Swedish Chemicals Agency.

Kerns, W. D., Pavkov, K. L., Donofrio, D. J., Gralla, E. J., Swenberg, J. A. (1983). Carcinogenicity of formaldehyde in rats and mice after long-term inhalation exposure. *Cancer Res,* 43(9), 4382-92.

Kim, K.-H., Szulejko, J. E., Ahn, J.-H. (2016). Response to the comment on characterization of hazardous and odorous volatiles emitted from scented candles before lighting and when lit. *Journal of Hazardous Materials,* 303, 172-173.

Klages-Mundt, N. L., Li, L. (2017). Formation and repair of DNA-protein crosslink damage. *Science China Life Sciences,* 60(10), 1065-1076.

Kolarik, B., Gunnarsen, L., Logadottir, A., Funch, L. W. (2012). Concentrations of Formaldehyde in new Danish Residential Buildings in Relation to WHO Recommendations and CEN Requirements. *Indoor and Built Environment,* 21(4), 552-561.

Krzyzanowski, M., Quackenboss, J. J., Lebowitz, M. D. (1990). Chronic respiratory effects of indoor formaldehyde exposure. *Environmental Research,* 52(2), 117-125.

Kuper, C. F., van Oostrum, L., Ma-Hock, L., Durrer, S., Woutersen, R. A. (2011). Hyperplasia of the lymphoepithelium of NALT in rats but not in mice upon 28-day exposure to 15 ppm formaldehyde vapor. *Exp Toxicol Pathol,* 63(1-2), 25-32.

Lang, I., Bruckner, T., Triebig, G. (2008). Formaldehyde and chemosensory irritation in humans: a controlled human exposure study. *Regul Toxicol Pharmacol,* 50(1), 23-36.

Lee, S.-C., Wang, B. (2004). Characteristics of emissions of air pollutants from burning of incense in a large environmental chamber. *Atmospheric Environment,* 38(7), 941-951.

Lévesque, B., Allaire, S., Gauvin, D., Koutrakis, P., Gingras, S., Rhainds, M., Prud'Homme, H., Duchesne, J.-F. (2001). Wood-burning appliances and indoor air quality. *Science of The Total Environment,* 281(1), 47-62.

Lu, K., Collins, L. B., Ru, H., Bermudez, E., Swenberg, J. A. (2010). Distribution of DNA adducts caused by inhaled formaldehyde is consistent with induction of nasal carcinoma but not leukemia. *Toxicol Sci,* 116(2), 441-51.

Lu, K., Moeller, B., Doyle-Eisele, M., McDonald, J., Swenberg, J. A. (2011). Molecular dosimetry of N2-hydroxymethyl-dG DNA adducts in rats exposed to formaldehyde. *Chem Res Toxicol,* 24(2), 159-61.

Lyapina, M., Kisselova-Yaneva, A., Krasteva, A., Tzekova–Yaneva, M., Dencheva-Garova, M. (2012). Allergic contact dermatitis from formaldehyde exposure. *J of IMAB,* 18(4), 255-262.

Maroni, M., Seifert, B., Lindvall, T. (1995). Indoor Air Quality. *Indoor Air Quality.* Amsterdam: Elsevier.

Marquart, H., Verbist, K., Dieperink-Hertsenberg, S. (2013). *Analysis of consumer exposure associated with the use of products and articles containing formaldehyde-based resins*. Zeist: TNO Triskelion BV.

Martin, W. J. (1990). A teratology study of inhaled formaldehyde in the rat. *Reprod Toxicol,* 4(3), 237-9.

Marutzky, R. (2018). *Comparison of suitability of EN 717-1 and EN 16516 chamber test methods to determine formaldehyde emissions from wood-based panels*. Expertise No. 0106-2018 ordered by the European Panel Federation.

Marutzky, R., Meyer, B. (1993). Emissionen aus Holzfaserformteilen und Textilvliesen für den Automobil-Innenraum. *ATZ Automobiltechnische Zeitschrift,* 95(6).

McGregor, D., Bolt, H., Cogliano, V., Richter-Reichhelm, H. B. (2006). Formaldehyde and glutaraldehyde and nasal cytotoxicity: case study within the context of the 2006 IPCS Human Framework for the Analysis of a cancer mode of action for humans. *Crit Rev Toxicol,* 36(10), 821-35.

Meng, F., Bermudez, E., McKinzie, P. B., Andersen, M. E., Clewell, H. J., 3rd, Parsons, B. L. (2010). Measurement of tumor-associated mutations in the nasal mucosa of rats exposed to varying doses of formaldehyde. *Regul Toxicol Pharmacol,* 57(2-3), 274-83.

Milieu (2012). *Implementation and Enforcement of Restrictions under Title VIII and Annex XVII to REACH in the Member State*. Brussels: Milieu Ltd.

Monticello, T. M., Swenberg, J. A., Gross, E. A., Leininger, J. R., Kimbell, J. S., Seilkop, S., Starr, T. B., Gibson, J. E., Morgan, K. T. (1996). Correlation of regional and nonlinear formaldehyde-induced nasal cancer with proliferating populations of cells. *Cancer Res,* 56(5), 1012-22.

Mueller, J. U., Bruckner, T., Triebig, G. (2013). Exposure study to examine chemosensory effects of formaldehyde on hyposensitive and hypersensitive males. *International Archives of Occupational and Environmental Health,* 86(1), 107-117.

Nielsen, G. D., Larsen, S. T., Wolkoff, P. (2013). Recent trend in risk assessment of formaldehyde exposures from indoor air. *Archives of Toxicology,* 87(1), 73-98.

Nielsen, G. D., Larsen, S. T., Wolkoff, P. (2017). Re-evaluation of the WHO (2010) formaldehyde indoor air quality guideline for cancer risk assessment. *Arch Toxicol,* 91(1), 35-61.

Nwaogu, T., Bowman, C., Marquart, H., Postle, M. (2013a). *Analysis of the most appropriate risk management option for formaldehyde*. TNO Triskelion BV and Risk & Policy Analysts.

Nwaogu, T., Bowman, C., Marquart, H., Postle, M. (2013b). *Analysis of the most appropriate risk management option for formaldehyde. Annex 2*. TNO Triskelion BV and Risk & Policy Analysts.

OECD (2016). *Affordable Housing Databse* [Online]. Available: http://www.oecd.org/social/affordable-housing-database.htm [Accessed 27 June 2018].

Oppl, R. (2014). New European VOC emissions testing method CEN/TS 16516 and CE marking of construction products. *Gefahrstoffe - Reinhaltung der Luft,* 74(3), 62-68.

Ozen, O. A., Akpolat, N., Songur, A., Kus, I., Zararsiz, I., Ozacmak, V. H., Sarsilmaz, M. (2005). Effect of formaldehyde inhalation on Hsp70 in seminiferous tubules of rat testes: an immunohistochemical study. *Toxicol Ind Health,* 21(10), 249-54.

Ozen, O. A., Yaman, M., Sarsilmaz, M., Songur, A., Kus, I. (2002). Testicular zinc, copper and iron concentrations in male rats exposed to subacute and subchronic formaldehyde gas inhalation. *J Trace Elem Med Biol,* 16(2), 119-22.

Paustenbach, D., Alarie, Y., Kulle, T., Schachter, N., Smith, R., Swenberg, J., Witschi, H., Horowitz, S. B. (1997). A recommended occupational exposure limit for formaldehyde based on irritation. *J Toxicol Environ Health,* 50(3), 217-63.

Peng, C.-Y., Lan, C.-H., Lin, P.-C., Kuo, Y.-C. (2017). Effects of cooking method, cooking oil, and food type on aldehyde emissions in cooking oil fumes. *Journal of Hazardous Materials,* 324, 160-167.

RAC (2012). *Opinion proposing harmonised classification and labelling at EU level of Formaldehyde*. Helsinki: Committee for Risk Assessment.

Reda, A. A., Czech, H., Schnelle-Kreis, J., Sippula, O., Orasche, J., Weggler, B., Abbaszade, G., Arteaga-Salas, J. M., Kortelainen, M., Tissari, J., Jokiniemi, J., Streibel, T., Zimmermann, R. (2015). Analysis of Gas-Phase Carbonyl Compounds in Emissions from Modern Wood Combustion Appliances: Influence of Wood Type and Combustion Appliance. *Energy & Fuels,* 29(6), 3897-3907.

Rusch, G. M., Clary, J. J., Rinehart, W. E., Bolte, H. F. (1983). A 26-week inhalation toxicity study with formaldehyde in the monkey, rat, and hamster. *Toxicol Appl Pharmacol,* 68(3), 329-43.

Saillenfait, A. M., Bonnet, P., de Ceaurriz, J. (1989). The effects of maternally inhaled formaldehyde on embryonal and foetal development in rats. *Food Chem Toxicol,* 27(8), 545-8.

Salem, M., Böhm, M., Barcík, Š., Beránková, J. (2011). Formaldehyde Emission from Wood-Based Panels Bonded with Different Formaldehyde-Based Resins. *Drvna industrija,* 62(3), 177-183.

Salem, M. Z. M., Böhm, M., Srba, J., Beránková, J. (2012). Evaluation of formaldehyde emission from different types of wood-based panels and flooring materials using different standard test methods. *Building and Environment,* 49, 86-96.

Salthammer, T. (2014). Release of Organic Compounds and Particulate Matter from Products, Materials, and Electrical Devices in the Indoor Environment. *In:* Pluschke, P., Schleibinger, H. (eds.) *Indoor Air Pollution. The Handbook of Environmental Chemistry, Vol 64.* Springer Berlin Heidelberg, 1-35.

Salthammer, T., Fuhrmann, F. (2007). Photocatalytic Surface Reactions on Indoor Wall Paint. *Environ Sci Technol,* 41(18), 6573-6578.

Salthammer, T., Gunschera, J. (2017). *Information requirements on formaldehyde given in the ECHA decision letter "Decision on substance evaluation pursuant to Article 46(1) of regulation (EC) No 1907/2006, for formaldehyde, CAS No 50-00-0 (EC No 200-001-8)"*. Braunschweig: Fraunhofer WKI.

Salthammer, T., Mentese, S., Marutzky, R. (2010). Formaldehyde in the Indoor Environment. *Chemical Reviews,* 110(4), 2536-2572.

Sanghani, P. C., Stone, C. L., Ray, B. D., Pindel, E. V., Hurley, T. D., Bosron, W. F. (2000). Kinetic mechanism of human glutathione-dependent formaldehyde dehydrogenase. *Biochemistry,* 39(35), 10720-10729.

Santovito, A., Schiliro, T., Castellano, S., Cervella, P., Bigatti, M. P., Gilli, G., Bono, R., DelPero, M. (2011). Combined analysis of chromosomal aberrations and glutathione S-transferase M1 and T1 polymorphisms in pathologists occupationally exposed to formaldehyde. *Archives of Toxicology,* 85(10), 1295-1302.

Schripp, T., Markewitz, D., Uhde, E., Salthammer, T. (2013). Does e-cigarette consumption cause passive vaping? *Indoor Air,* 23(1), 25-31.

Schripp, T., Salthammer, T., Wientzek, S., Wensing, M. (2014). Chamber Studies on Nonvented Decorative Fireplaces Using Liquid or Gelled Ethanol Fuel. *Environmental Science & Technology,* 48(6), 3583-3590.

Schroeter, J. D., Campbell, J., Kimbell, J. S., Conolly, R. B., Clewell, H. J., Andersen, M. E. (2014). Effects of endogenous formaldehyde in nasal tissues on inhaled formaldehyde dosimetry predictions in the rat, monkey, and human nasal passages. *Toxicol Sci,* 138(2), 412-24.

Schwarz, A., Marutzky, R., Hoferichter, E., Scheithauer, M. (1992). Prüfverfahren und Materialgrenzwerte zur Formaldehydabgabe von Finishfolien. *HK Holz- und Möbelindustrie,* 10, 1111-1113.

SCOEL (2016). *SCOEL/REC/125 Formaldehyde. Recommendation from the Scientific Committee on Occupational Exposure Limits*. Brussels: European Commission.

Sellakumar, A. R., Snyder, C. A., Solomon, J. J., Albert, R. E. (1985). Carcinogenicity of formaldehyde and hydrogen chloride in rats. *Toxicol Appl Pharmacol,* 81(3 Pt 1), 401-6.

Singer, B. C., Hodgson, A. T., Nazaroff, W. W. (2003). Gas-phase organics in environmental tobacco smoke: 2. Exposure-relevant emission factors and indirect exposures from habitual smoking. *Atmospheric Environment,* 37(39), 5551-5561.

Speit, G., Schutz, P., Weber, I., Ma-Hock, L., Kaufmann, W., Gelbke, H. P., Durrer, S. (2011). Analysis of micronuclei, histopathological changes and cell proliferation in nasal epithelium cells of rats after exposure to formaldehyde by inhalation. *Mutat Res,* 721(2), 127-35.

Swenberg, J. A., Kerns, W. D., Mitchell, R. I., Gralla, E. J., Pavkov, K. L. (1980). Induction of squamous cell carcinomas of the rat nasal cavity by inhalation exposure to formaldehyde vapor. *Cancer Res,* 40(9), 3398-402.

Tulpule, K., Dringen, R. (2013). Formaldehyde in brain: an overlooked player in neurodegeneration? *J Neurochem,* 127(1), 7-21.

U.S. EPA (1990). *IRIS Assessment Formaldehyde CASRN 50-00-0*. Washington DC: U.S. Environmental Protection Agency.

U.S. EPA (2010). *IRIS Toxicological Review of Formaldehyde (Inhalation) EPA/635/R-10/002A*. Washington DC: U.S. Environmental Protection Agency.

UBA (2016). On the Question of an Asthma-triggering and/or worsening Potential of Formaldehyde in the Indoor Air in Children Notification of the Committee on Interior Guideline (IGC). *Bundesgesundheitsblatt-Gesundheitsforschung-Gesundheitsschutz,* 59(8), 1028-1039.

UNECE (2017). *Proposal for a new Mutual Resolution (M.R.3) of the 1958 and the 1998 Agreements concerning Vehicle Interior Air Quality (VIAQ). ECE/TRANS/WP.29/GRPE/2017/10*. Prepared by the Informal Working Group on Vehicles Interior Air Quality (VIAQ).

Walker, J. F. (1964). Formaldehyde (3rd ed.). *American Chemical Society Monograph Series No. 159.* New York: Reinhold Publishing Company.

WHO (1989). *Formaldehyde. Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization*. Geneva: World Health Organization.

WHO (2010). Formaldehyde. *WHO Guidelines for Indoor Air Quality: Selected Pollutants.* Geneva: World Health Organisation, 103-156.

Wiglusz, R., Jarnuszkiewicz, I., Sitko, E., Nikel, G. (2000). Interlaboratory comparison experiment on the determination of formaldehyde emitted from mineral wool board using small test chambers. *Building and Environment,* 35, 53-57.

Wiglusz, R., Sitko, E., Nikel, G., Jarnuszkiewicz, I., Igielska, B. (2002). The effect of temperature on the emission of formaldehyde and volatile organic compounds (VOCs) from laminate flooring - case study. *Building and Environment,* 37, 41-44.

Wilmer, J. W., Woutersen, R. A., Appelman, L. M., Leeman, W. R., Feron, V. J. (1989). Subchronic (13-week) inhalation toxicity study of formaldehyde in male rats: 8-hour intermittent versus 8-hour continuous exposures. *Toxicol Lett,* 47(3), 287-93.

Woutersen, R. A., Appelman, L. M., Wilmer, J. W., Falke, H. E., Feron, V. J. (1987). Subchronic (13-week) inhalation toxicity study of formaldehyde in rats. *J Appl Toxicol,* 7(1), 43-9.

Woutersen, R. A., van Garderen-Hoetmer, A., Bruijntjes, J. P., Zwart, A., Feron, V. J. (1989). Nasal tumours in rats after severe injury to the nasal mucosa and prolonged exposure to 10 ppm formaldehyde. *J Appl Toxicol,* 9(1), 39-46.

WPIF/TRADA/TTF (2014). *Panel Guide Version 4*. Wood Panel Industries Federation, Timber Research and Development Association, Timber Trade Federation.

Yrieix, C., Dulaurent, A., Laffargue, C., Maupetit, F., Pacary, T., Uhde, E. (2010). Characterization of VOC and formaldehyde emissions from a wood based panel: results from an inter-laboratory comparison. *Chemosphere,* 79(4), 414-9.

Yu, C. W. F., Kim, J. T. (2012). Long-term Impact of Formaldehyde and VOC Emissions from Wood-based Products on Indoor Environments; and Issues with Recycled Products. *Indoor and Built Environment,* 21(1), 137-149.

Zeller, J., Hogel, J., Linsenmeyer, R., Teller, C., Speit, G. (2012). Investigations of potential susceptibility toward formaldehyde-induced genotoxicity. *Archives of Toxicology,* 86(9), 1465-1473.

Zeller, J., Ulrich, A., Mueller, J. U., Riegert, C., Neuss, S., Bruckner, T., Triebig, G., Speit, G. (2011). Is individual nasal sensitivity related to cellular metabolism of formaldehyde and susceptibility towards formaldehyde-induced genotoxicity? *Mutat Res,* 723(1), 11-7.

Zhou, D., Zhang, J., Wang, H. (2011). Assessment of the potential reproductive toxicity of long-term exposure of adult male rats to low-dose formaldehyde. *Toxicol Ind Health,* 27(7), 591-8.

Zhou, D. X., Qiu, S. D., Zhang, J., Tian, H., Wang, H. X. (2006). The protective effect of vitamin E against oxidative damage caused by formaldehyde in the testes of adult rats. *Asian J Androl,* 8(5), 584-8.