Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Phthalate	BBP BBzP BzBP	Derived no effect level (DNEL) for general population (0.78mg/m^3) via inhalation route for repeated dose toxicity. [179]	Phthalate esters are endocrine disruptors. They decrease foetal testis testosterone production and reduce the expression of steroidogenic genes by decreasing mRNA expression. Animal studies have shown that these effects disrupt reproductive development and can cause a number of malformations in affected young [180] IARC Group 3: Not classifiable as to its carcinogenicity to humans [180]
Phthalate	DBP DnBP	Irritation to the eyes and upper respiratory system [181] DNEL for general population (0.02 mg/m^3) via inhalation route for developmental toxicity/teratogenicity [182] Workers exposed to di-n-butyl phthalate for 0.5–19 years at concentrations of 1.7–66 mg/m^3 experienced neurological symptoms (pain, numbness, spasms, weakness) and exhibited reflex disturbances, elevated thresholds for pain sensitivity and olfactory stimulation, and depression of vestibular function. [183] No studies regarding reproductive and respiratory effects in humans following inhalation exposure to di-n-butyl phthalate were located. [183]	Studies in rats calculate acute inhalation LC50 values for rats of 25 g/m^3 (NTP 1995) and 4.25 g/m3 (NTP 1995), and for mice of 25 g/m^3/2H (NTP 1995). [183] Only one study reported an increase in relative lung weight in rats exposed to 50mg/m^3 DNBP 6 hours/day for 6 months however lung weight not altered in 0.5mg/m^3 group and significance difficult to assess as histopathology not examined [183] In rats, a statistically significant increase in brain weight as a percent of body weight was observed following exposure to 50mg/m^3 for 6 months. However, a significant decrease in body weight gain was reported for this dose group and absolute brain weight increase was small. [183]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Phthalate	DEHP	Irritation to respiratory tract (eyes, mucous membrane, cough, sore throat) [184] DNEL for general population (0.13 mg/m^3) via inhalation route for developmental toxicity/teratogenicity [185] Epidemiological studies note an association between DEHP urinary metabolite levels and high blood pressure however the results are not consistent. [186] Data relating to neurological effects in studies of prenatal and/or early postnatal exposure included cognitive, mental and psychomotor development; behaviour and emotional development; social development. [186] An association is reported between increased frequency of nonoptimal reflexes in male infants and sum of DEHP metabolites in maternal urine samples [186] There were also 26 studies of 13 cohorts however they were not comparable due to study differences. However, three studies suggest an association between poor performance on the mental development intake at 6 months, 223-26 months and 2-3 years of age and prenatal exposure to DEHP. [186]	Group of 5 males and 5 female rats were exposed nose only for 4 hours to clean air (control group) or DEHP in concentrations of 3.39, 6.82 or 10.62mg/l. All treated animals showed slightly unkempt appearance for 1-2 days and highest dose had a yellowish staining on the fur. They also had a reduced body weight gain on the second day but returned to the normal pattern. In all groups (case and control), dark red foci and patches were observed in the lung. [184] IARC Group 2B Possibly carcinogenic to humans [184]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Epidemiology studies evaluated depression in relation to urinary levels and found no association. [186] Clinical symptoms of neurotoxicity (headaches, fatigue, dizziness, muscle weakness, nausea and vomiting) reported in occupational environment when workers exposed to levels between 233 and 707μg/m^3 DEHP [186]. Chronic and/or high levels of DEHP exposure may cause reproductive and developmental damage. This includes damaged sperm, delayed sexual maturity, and deficiencies in the development of male babies. DEHP may also cause liver and kidney damage, and is potentially carcinogenic [184] Endocrine studies including those examining thyroid hormone levels in pregnant women and cross-sectional studies of other populations were inconsistent. [186] DEHP has been classified as a human carcinogen by the US Environmental Protection Agency (US EPA) and classified as B2 carcinogen (probable human carcinogen by the International Agency for Research on Cancer (IARC) [184] There is some epidemiologic evidence for an association between the concentrations of	

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		phthalates in indoor dust and/or the occurrence indoors of plasticised products such as PVC and allergic symptoms in the airways (e.g., asthma), nose and skin in children (Bornehag et al. 2004 [80]; Jaakkola et al. 2000 [49]; Kolarik et al. 2008 [48]). These are detailed in the health table (Supplementary table S6)	
Phthalate	DiBP	DNEL for general population (0.02 mg/m^3) via inhalation route for developmental toxicity/teratogenicity [187]	Reproductive toxicity, Endocrine Disrupting. [187] Phthalate esters are endocrine disruptors. They decrease foetal testis testosterone production and reduce the expression of steroidogenic genes by decreasing mRNA expression. Animal studies have shown that these effects disrupt reproductive development and can cause a number of malformations in affected young [188]
Phthalate	DMP	Acute (short-term) exposure to dimethyl phthalate, via inhalation in humans and animals, results in irritation of the eyes, nose, and throat. No information is available on the chronic (long-term), reproductive, developmental, or carcinogenic effects of dimethyl phthalate in humans. [189]	Animal studies have reported slight effects on growth and on the kidney from chronic oral exposure to the chemical. [189] Phthalate esters are endocrine disruptors. Animal studies have shown that they disrupt reproductive development and can cause a number of malformations in affected young, such as reduced anogenital distance (AGD), cryptorchidism, hypospadias, and reduced fertility. [189] LCLo Cat (inhalation) 9300mg/m^3/6.5H [189] EPA has classified dimethyl phthalate as a Group D, not classifiable as to human carcinogenicity [189]
Flame retardant	BDE 17	May cause respiratory tract irritation [190]	

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Flame retardant	BDE 196	Higher risk of acute lymphoblastic leukaemia in 0-7 years for those exposed to carpet dust with higher level of BDE-196 in their room. [191]	
Flame retardant	BDE 197	Reproductive toxicity [192]	
Flame retardant	BDE 203	Reproductive toxicity [193] Higher risk of acute lymphoblastic leukaemia in 0-7 years for those exposed to carpet dust with higher level of BDE-203 in their room. [191]	
Flame retardant	BDE 206	Minimum risk level: Intermediate inhalation: 0.006mg/m^3 [194] Higher risk of acute lymphoblastic leukaemia in 0-7 years for those exposed to carpet dust with higher level of BDE-206 in their room. [191]	May affect the thyroid gland and liver. Animal studies have also shown that PBDEs can cause neurobehavioural alterations and affect the immune system. [194]
Flame retardant	BDE 207	Higher risk of acute lymphoblastic leukaemia in 0-7 years for those exposed to carpet dust with higher level of BDE-207 in their room. [191]	
Flame retardant	BDE 209	Symptoms of exposure can include irritation of eyes, mucous membrane and upper respiratory tract. [195] Long term exposure reports possible effects on the thyroid [195] Minimum risk level: Intermediate inhalation: 0.006mg/m^3 [195]	May affect the thyroid gland and liver. Animal studies have also shown that PBDEs can cause neurobehavioural alterations and affect the immune system. [195]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Higher BDE 209 concentration in dust at home were associated with increased odds of having papillary thyroid cancer [196].	
Flame retardant	BDE 28	May cause respiratory tract irritation [197]	
Flame retardant	BDE 47	Altered hormone levels in men were associated with BDE 47 and 99 exposures estimated as concentrations in house dust. Exposure pathway was not specified. [61]	
Flame retardant	BDE 99	Minimum risk level: Intermediate inhalation: 0.006mg/m^3 [198] Occupational health assessment for 6 weeks observed a higher-than-normal prevalence of primary hypothyroidism and a significant reduction in sensory and fibula motor velocities [198] Altered hormone levels in men were associated with BDE 47 and 99 exposures estimated as concentrations in house dust. Exposure pathway was not specified. [61]	May affect the thyroid gland and liver. Animal studies have also shown that PBDEs can cause neurobehavioural alterations and affect the immune system. [198]
Flame retardant	HBBz	May cause respiratory tract (eyes and mucous membrane) irritation [199]	Acute inhalation toxicity was evaluated in groups of 10 male Manor Farms rats and 10 male ChR-CD rats exposed to hexabromobenzene at a concentration that was not reported for a period of 4 hours. Mortality and the LC50 were not reported. Clinical observations for both strains of rats included salivation, irregular respiration, lacrimation, exophthalmos, a reddish nasal discharge, and tremors. Gross necropsy revealed necrotic tracheitis and slight

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
			testicular degeneration accompanied by suppressed spermatogenesis in the Manor Farms rats and necrotic and diphtheritic tracheitis, bronchiolitis, bronchitis, and murine pneumonia in the ChR-CD rats. [199]
Flame retardant	mmp-TMPP	Chronic toxicity where simultaneous inhalation and cutaneous absorption, main symptoms were hyperhidrosis, hypotension, general fatigue, affective irritability and paraesthesia in limbs [200] Tricresyl phosphate (TMPP) could cause the organophosphate-induced delayed neuropathy. Moreover, PFR levels in house dust were associated with the altered hormone levels and decrease of semen quality [167] Dust in mothers' mattress (2 months after birth) contained lower levels of mmp-TMPP among children developed asthma at 4 or 8 years old [53].	Cats inhaled atmosphere containing 27mg/L/7 H, there was irritation of mucous membrane and animals were restless, but this supervened after 6 hours. [200]
Flame retardant	PBB	No studies were located regarding respiratory and cardiovascular effects or cancer in humans after inhalation exposure to PBBs. [201] Occupational exposure of twenty-five workers (exposure duration and levels not reported) displayed mean scores on tests of memory and learning that were typical for people of their age, and educational, occupational, and cultural backgrounds, even though they had an elevated mean PBB concentration in adipose tissue (9.33	Slight dyspnoea was observed in five males and five female rats that were exposed to decabromobiphenyl dust mixture at 5000mg/m^3 concurrently with starch dust (6mg/m^3) for 6 hours/day, 5 days/week for 4 weeks. This effect was not observed at 500mg/m^3 and lower concentrations or in air only exposed controls and no changes in pulmonary resistance and compliance in urethane-anesthetized rats, blood gases and lung histology at any of the exposure levels. [201]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		ppm). Workers with the highest concentrations of PBBs in adipose tissue showed no evidence of memory dysfunction in these tests. [201]	No studies were located regarding neurological or cardiovascular effects in animals after inhalation exposure to PBBs. [201] IARC 2A Probably carcinogenic to humans [201]
Flame retardant	pTBX	May case respiratory tract irritation [202]	
Flame retardant	TBE	Inhalation symptoms reported include cough, sore throat, headaches, and dizziness [203] Irritation and central nervous system depressant causing bromism due to bromide ion [203] Occupational exposure to 1-2ppm/7.5 hours and 16ppm/10 minutes reported near fatal liver injury with initial symptoms including headache. [203] No indication of carcinogenicity to humans and not listed by IARC. [203]	Toxicological studies report LC50 (rat) from inhalation at a dose of 549mg/m^3/4 hours [203]
Flame retardant	TBP	May cause respiratory tract irritation and nausea and headache. [204]	LC50 rat inhalation - 28gm/m^3/1H LC50 mouse inhalation – 1300mg/m^3 LClo Cat inhalation – 24510mg/m^3/5H – dyspnoea and cyanosis [204] Irritant, suspected to be carcinogenic, in inhalation studies, serum cholinesterase activity is depressed on the day after exposure and if inhalation is prolonged there can be general poisoning with paralysis. [204]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Flame retardant	TXP	May cause respiratory tract and mucous membrane irritation [205]	Toxic to reproduction [206]
Flame retardant	β-HBCD β-HBCDD	DNEL for general population (0.719 mg/m^3) via inhalation route for developmental toxicity/teratogenicity. Most sensitive endpoint: developmental toxicity/teratogenicity. No threshold effect and/or no dose-response information available. [207] May cause respiratory tract irritation [208]	No information was available for respiratory sensitisation. HBCD is not a skin sensitiser and no cases of respiratory sensitisation have been reported. [207]
PAH	Benz[a]anthracene	May cause irritation and inflammation of the skin and lung tissue. [209] No studies located regarding cardiovascular, or neurological effects in humans following inhalation exposure [210] Qualitative evidence of the potential for mixture containing PAHs such as benz[a]anthracene to cause cancer in humans. [210]	No studies located regarding cardiovascular, respiratory or neurological effects in animals following inhalation exposure [210] IARC B2 Possible human carcinogen [210]
PAH	Benzo[a]pyrene	One occupational study investigated respiratory health and noted radiographic abnormalities including patch opacities, prominent bronchiovascular markings and pleural effusions. Other symptoms included bloody vomit, breathing problems, chest pains, chest irritations, throat irritations and cough. [210]	Group of 40 Fischer – 344 rats exposed nose only to BaP (7.7mg/m^3) 2 hours/day, 5 days/week for 4 weeks. Nasal and left lung sections examined histopathologically. No treatment-related lesions were noted in the lungs or nasal cavities of the animals exposed to BaP. [210] No studies located regarding cardiovascular effects in animals following inhalation exposure [210]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		These symptoms were associated with those with the highest exposure to particulate matter and BaP. No attempt was made to separate the effects of exposure to BaP and particulate matter or to identify other possible chemicals exposed. [210] No studies located regarding cardiovascular effects in humans following inhalation exposure [210]. Ischemic heart disease was observed to increase in a dose-dependent manner in occupational exposure to BaP. Mean exposure for cohort was 273ng/m^3 and exposure at or above this level was associated with a 1.64-fold greater risk of ischemic heart disease mortality compared to those exposed to below 68ng/m^3 [70] There is evidence to suggest that BaP may cause developmental neurotoxic effects. Human studies of prenatal environmental PAH exposure (determined by personal air monitoring and measuring BaP-DNA adduct levels in cord blood) have reported neurodevelopmental effects including impaired cognitive ability, impaired neuromuscular function and increased attention problems and anxious/depressed behaviour. [70]	Lifetime inhalation studies in hamsters gave dose-response related increases in papilloma and squamous cell-carcinomas in the upper respiratory and upper digestive tracts. [70] Several inhalation studies where animals given BaP noted an increase in all lung tumors and a dose-dependent increase in malignant lung tumors for mice exposed to PAH enriched exhausts containing 0.05 or 0.09mg/m^3 BaP. [210] Inhalation exposure to benzo[a]pyrene has been associated with squamous cell neoplasia in the larynx, pharynx, trachea, nasal cavity, oesophagus, and forestomach of male Syrian golden hamsters exposed for up to 130 weeks [70] In animals, inhalation exposure to benzo[a]pyrene has been shown to result in developmental and reproductive toxicity (decreased embryo/foetal survival and nervous system effects in offspring and decreased testes weight and sperm count in adult animals [70]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		IARC classified BaP as Group 1: carcinogenic to humans and contributes to the genotoxic and carcinogenic effects resulting from occupational exposure to complex PAH mixtures. Robust animal evidence and consistent and coherent mechanistic evidence from experimental and human studies provide biological plausibility to support the overall classification. [70] Symptoms of exposure include mucous membrane irritation, dermatitis, bronchitis, cough, dyspnoea, conjunctivitis, photosensitization, pulmonary oedema, reproductive effects and leukaemia [211] Carcinogenic to humans based on strong and consistent evidence in animals and humans [212] BaP is carcinogenic at multiple tumour sites (alimentary tract, liver, kidney, respiratory tract, pharynx and skin) by all routes of exposure [212]	
PAH	Benzo[b]fluoranthene	May cause irritation and inflammation of the skin and lung tissue. [213] No studies located regarding cardiovascular or neurological effects in humans following inhalation exposure. [210] Qualitative evidence of the potential for mixture containing PAHs such as	No studies located regarding cardiovascular, respiratory or neurological effects in animals following inhalation exposure [210] IARC 2B possibly human carcinogen [210]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		benzo[b]fluoranthene to cause cancer in humans. [210]	
PAH	Chrysene	No studies located regarding cardiovascular or neurological effects in humans following inhalation exposure [210] Qualitative evidence of the potential for mixture containing PAHs such as chrysene to cause cancer in humans. [210] PAHs are carcinogens and have been associated with the increased risk of skin, respiratory tract, bladder, stomach, and kidney cancers. They may also cause reproductive effects and depress the immune system [213] Acute exposure to PAHs causes irritation and inflammation of the skin and lung tissue [213]	No studies located regarding cardiovascular, respiratory or neurological effects in animals following inhalation exposure [210] IARC B2 possibly carcinogenic to humans [210]
PAH	dibenz[a,h]anthracene	No studies located regarding cardiovascular or neurological effects in humans following inhalation exposure. [210] Qualitative evidence of the potential for mixture containing PAHs such as dibenz[a,h]anthracene to cause cancer in humans. [210] May cause irritation and inflammation of the skin and lung tissue. [214]	No studies located regarding cardiovascular or neurological effects in animals following inhalation exposure [210] IARC Group 2A probably carcinogenic to humans [214]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Pesticide	Aldrin	Inhalation symptoms include convulsions, dizziness, headache, nausea, muscle twitching. Target organs include developmental, endocrine and neurological. [215] Occupational studies have conflicting outcomes for respiratory effects, where one noted significant increased incidence of pneumonia and other pulmonary diseases, another found no evidence of exposure-related pulmonary disease or deterioration of existing pulmonary disease. All studies were noted to be limited due to small sample sizes and potential for exposure to other chemicals/pesticides. [216] Very limited information available in regard to cardiovascular effects in humans. Suggestive evidence of an association between dieldrin and hypertension where a slight but significant association between serum cholesterol and dieldrin levels was observed in exposed workers. However, other studies did not support the correlation of hypertension with dieldrin exposure. Studies are limited by small size and exposure to multiple pesticides. [216] Central nervous system excitation culminating in convulsions was the principal adverse effect noted in occupational studies of workers employed either in the application or manufacturer of aldrin or dieldrin. [216]	Cats, guinea pigs, rats, rabbits and mice exposed to aldrin vapours and particles (by sublimating at 200^{o}C) exhibited symptoms indicative of mucous membrane irritation, however exposure levels were not reported. [216] No information regarding respiratory effects following inhalation exposure to dieldrin. [216] No studies were located regarding cardiovascular effects in animals after inhalation exposure to aldrin or dieldrin. [216] No inhalation studies for neurological effects in animals identified [216] EPA has classified Aldrin as Group B2 (probable human carcinogen) based on sufficient evidence in animals (significantly increased liver tumors in three strains of male and female mice and noted that tumor induction was also observed in structurally related chemicals, including dieldrin, a metabolite. [216]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Other central nervous system symptoms reported by workers involved in the manufacturer or application of aldrin and/or dieldrin included headaches, dizziness, hyperirritability, general malaise, nausea and vomiting, anorexia, muscle twitching and myoclonic jerking. More severe symptoms were accompanied by electroencephalogram (EEG) patterns with bilateral spike and wave complexes [216].	
Pesticide	Atrazine	Symptoms of exposure includes irritation eyes, dyspnea (breathing difficulty), lassitude (weakness, exhaustion), incoordination, salivation [217] Target organs include developmental, endocrine and reproductive [217]	LC50 rat (inhalation) 5200mg/m^3/4H [217] IARC Group 3: Not classifiable as to its carcinogenicity to humans [217]
Pesticide	cypermethrin	May cause respiratory tract irritation and inhalation symptoms include burning sensation, cough, dizziness, headache, nausea and shortness of breath. Muscle twitching, reduced energy, and changes in awareness can also result from inhalation [218] It is suspected of damaging fertility or the unborn child. [218]	Group C Possible human carcinogen [218] LC50 rat (inhalation) 7889mg/m^3/4h [218]
Pesticide	Diazinon	The substance is mildly irritating to the eyes and skin. The substance may cause effects on the nervous system. Cholinesterase inhibition. This may result in convulsions and respiratory depression. The effects may be delayed. Long	Nasal discharge was observed in Sprague-Dawley rats exposed to 2330mg/m^3 for four hours in an inhalation chamber. [220]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		term: Cholinesterase inhibition. Cumulative effects are possible. This substance is probably carcinogenic to humans. [219]	A statistically significant increase in lung to body weight was observed in hybrid female rats exposed to 0.46 and 1.57mg/m^3 (nose only) for 6 hrs/day, 5 days/week for 3 weeks. This was not seen in male rats or in female rats exposed to 11.gmg/m^3 so its toxicological significance is unclear.[220] No gross or histological evidence of treatment-related damage to the heart was observed in hybrid rats (10/sex) exposed to 11.6 mg/m^3 diazinon (nose-only) 6 hours/day, 5 days/week for 3 weeks. [220] No gross or histological evidence of treatment-related damage to the adrenal gland was observed in hybrid rats (10/sex) exposed to 11.6 mg/m^3 diazinon (nose-only) for 6 hours/day, 5 days/week for 3 weeks. [220]
Pesticide	Dieldrin	Inhalation symptoms include convulsions, dizziness, headache, nausea, and muscle twitching. The target organs are the central nervous system, liver, kidneys and skin. [221] Occupational studies have conflicting outcomes for respiratory effects, where one noted significant increased incidence of pneumonia and other pulmonary diseases, another found no evidence of exposure-related pulmonary disease or deterioration of existing pulmonary disease. All studies were noted to be limited due to small sample sizes and potential for exposure to other chemicals/pesticides. [216]	LC50 rat (inhalation) 13mg/m^3/4H LC50 cat (inhalation) 80mg/m^3/4H – somnolence, convulsions, excitement [221] Cats, guinea pigs, rats, rabbits and mice exposed to aldrin vapours and particles (by sublimating at 200°C) exhibited symptoms indicative of mucous membrane irritation, however exposure levels were not reported. [216] No information regarding respiratory effects following inhalation exposure to dieldrin. [216] No studies were located regarding cardiovascular effects in animals after inhalation exposure to aldrin or dieldrin. [216]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Very limited information available in regard to cardiovascular effects in humans. Suggestive evidence of an association between dieldrin and hypertension where a slight but significant association between serum cholesterol and dieldrin levels was observed in exposed workers. However, other studies did not support the correlation of hypertension with dieldrin exposure. Studies are limited by small size and exposure to multiple pesticides. [216] Central nervous system excitation culminating in convulsions was the principal adverse effect noted in occupational studies of workers employed either in the application or manufacturer of aldrin or dieldrin. (ATSDR) Other central nervous system symptoms reported by workers involved in the manufacturer or application of aldrin and/or dieldrin included headaches, dizziness, hyperirritability, general malaise, nausea and vomiting, anorexia, muscle twitching and myoclonic jerking. More severe symptoms were accompanied by electroencephalogram (EEG) patterns with bilateral spike and wave complexes [216].	No inhalation studies for neurological effects in animals identified [216] EPA has classified Aldrin as Group B2 (probable human carcinogen) based on sufficient evidence in animals (significantly increased liver tumors in three strains of male and female mice and noted that tumor induction was also observed in structurally related chemicals, including dieldrin, a metabolite. [216] IARC Group 2A: Probably carcinogenic to humans [221]
Pesticide	Endrin	Population based study found no association between hypertension and serum endrin levels. [222]	Degenerative lesions of the brain were observed in two rabbits that died after exposure to 0.36ppm for 165 days (5 days/week); these lesions were not observed in a mouse that died after similar exposure. Seizures were not observed prior to death. [222]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		An occupational health survey reported 17 acute cases of convulsion over a 9-year period. Acute high exposure, without personal protective equipment accounted for all cases, however the exposure levels were not reported. [222] Several epidemiological studies have examined the potential relationship between environmental endrin exposure and thyroid hormone levels and have observed only weak (if any) relationship and inconsistent findings [222] Inhalation symptoms include dizziness, weakness, headache, nausea, convulsions and target organs is neurological. [221]	EPA has classified endrin in Group D, and IARC has classified it in Group 3, (not classifiable as to its carcinogenicity in humans) [222] Date on potential endocrine effects is limited and solely based on oral studies. (ATSDR)
Pesticide	Metolachlor	Inhalation symptoms include headache and nausea [223] Health effects reported include CNS depression, dizziness, dyspnoea, cardiovascular failure, and adverse reproductive effects but pathway not stipulated [223]	LD50 rat, inhalation > 1750mg/m^3/4 hr [223] LC50 Rat, inhalation > 2.02mg/L/4 hr air [223] LC50 Rat inhalation >4.33 mg/L/4 hr [223] Possible human carcinogen (Group c) based on appearance of proliferative liver lesions at highest dose (3000ppm) in female rats [223]
Pesticide	4,4'-DDT	Short term exposure effects include upper respiratory irritation (cough) and possible effects on the central nervous system which can also be an effect of long-term exposure. [224] Inconsistent evidence for associations between serum levels of DDT or DDE and measures of lung function. In one study serum levels were	No inhalation studies identified to assess respiratory, neurological and cardiovascular health effects [225] Animal studies report oral route of exposure as this is the presumed principal route of exposure in all of the cancer studies however it was noted that dermal or inhalation could not be excluded. [225]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		inversely associated with forced vital capacity and forced expiratory volume in 1 second and were not statistically significant. However, in another study maternal serum levels of DDE showed no association with reduced lung function but a positive association with airway obstruction was noted in offspring at 20 years of age. [225] In a single controlled exposure study, volunteers were exposed by inhalation to an aerosol containing DDT and moderate irritation of the nose, throat and eyes were reported however this may be attributable to the method of dispersing the aerosol. [225] Several epidemiological studies have examined associations between serum or adipose levels of DDT, DDD or DDE and cardiovascular outcomes and results provide inconsistent evidence for associations. [225] Epidemiological serum levels of thyroid studies provide inconsistent evidence for associations between levels of DDT and serum levels of thyroid levels. [225]	B2 (Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals - Liver tumours, benign and malignant) [224]
Pesticide	PBO	May cause respiratory tract irritation [226] IARC Cancer classification: Group 3 not classifiable as carcinogenic to humans. [226]	

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Pesticide	PCP	May cause respiratory tract irritation. Dust can irritate mucous membranes, causes coughing and sneezing. Inhalation symptoms can also include dizziness, drowsiness, headache, fever, laboured breathing, and sore throat. [227] Target organs include developmental, endocrine and reproductive [227] Chronic high dose (not specified) occupational exposure causes inflammation of the upper respiratory tract and bronchitis (possible exposure to other compounds). [228] Three human studies evaluated potential association between PCP and thyroid disease/function and in one no association was observed and the other two did report associations however there were study design limitations and exposure to other chemicals involved [228] There is limited data on neurotoxicity in humans from inhalation of PCP and are based on occupational exposure. Symptoms reported include lethargy, tachypnea, distractibility, and low mood. The evidence is only suggestive due to exposure to other compounds and the size of samples. [228]	LC50 rat (inhalation) 355mg/m^3 - excitement, muscle contractions and dyspnoea. [227] LC50 mouse (inhalation) 225 mg/m^3 - excitement, muscle contractions and dyspnoea. [227] IARC concluded that PCP is carcinogenic to humans (Group 1) [228]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		A study where elevated blood levels of PCP and/or lindane was identified in women with a history of habitual abortion, unexplained fertility, menstrual disorders, and onset of menopause. Exposure period was between 4.6 to 10 years and off gassing from walls, ceilings, carpets and treated leather upholstery). The relationship for this health effect is uncertain due to exposure with other compounds and study design limitations. [228]	
Pesticide	Permethrin	Respiratory tract irritation (cough) [229] Sudden bronchospasm, swelling of oral and laryngeal mucous membranes, and anaphylactoid reactions have been reported after inhalation. [229] Neurotoxic [229]	Chronic health hazard assessment for noncarcinogenic effects – inhalation exposure not evaluated [230] Carcinogen risk from inhalation exposure not assessed [230] Permethrin has elicited neurotoxic behaviour among laboratory animals [230] Hermant et al., 2018 [72] reports chronic exposure to permethrin has been associated with cancers and/or endocrine disorders. US-EPA classified probably carcinogenic to humans. IARC classified as group 3 (not classifiable) LD50 mouse (inhalation) 685mg/m^3 [229]
Pesticide	α-endosulfan	Limited information is available regarding health effects in human after sole inhalation exposure. [231]	Limited information is available regarding health effects in animals after sole inhalation exposure. [231] Irregular respiration was observed in male and female rats after 4-hour nose only inhalation exposure. Dyspnea was observed at the lowest concentration tested (12.3 and

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		No studies were located regarding respiratory, cardiovascular effects in humans after inhalation exposure. [231] Neurotoxicity is the primary effect observed in human following occupational exposure. Convulsions were reported by nine individuals and other effects reported prior to their onset of convulsions included malaise, nausea, vomiting, dizziness, confusion and/or weakness. [231] Case control study of occupational exposures to various suspected estrogenic chemicals and occurrence of breast cancer, noted that breast cancer OR was not elevated above unity (OR=0.8; 95% CI 0.2-3.2) compared to unexposed control however sample size was very small and other unreported chemicals co-exposure also occurred. [231] A cohort also examined possible associations between level of endosulfan in bone marrow and acute haematological malignancies in children. The report results were questionable due to small sample size and lack of control for potential confounders. [231] Symptoms of exposure includes tremor, convulsion, headache) and target organs	3.6mg/m^3) Necropsy of the rats that died showed dark-red pinhead foci in the lungs, although unclear if effects were direct effects on respiratory tissues or secondary to central nervous system effects on respiratory function. [231] No treatment related effects were revealed by examination of nasal cavity, trachea, lungs, heart, aorta and neurotoxicity of male and female rats exposed (nose only) to concentrations of up to 2mg/m^3 for 6 hours/day 5 days/week for a total of 21 out of 29 days (ATSDR) Nose only exposure of rats to aerosolized technical endosulfan at concentrations of 3.6mg/m^3 in females and 12.3mg/m^3 in males resulted in trembling and ataxia. [231] Not likely to be carcinogenic to humans. [231] LC50 rat (inhalation) 80mg/m^3/4 Hr [232] LC50 Cat (inhalation) 90mg/m^3/4 Hr [232]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		include endocrine (glands and hormones and neurological (nervous system) [232]	
Pesticide	α-HCH	No studies were located regarding respiratory effects in humans after exposure to α-HCH [233]	No neurological effects of α-HCH were located [233] EPA lists as a probable human carcinogen based on sufficient evidence of carcinogenicity in animals and inadequate data in humans [233]
Pesticide	β-HCH	Cross sectional study of vegetable farmers showed a significant positive association between serum levels of ß-HCH and respiratory symptoms (cough, phlegm, and wheezing) [233] Epidemiological studies have been undertaken for hypertension/peripheral artery disease and biomarkers of ß-HCH with mixed results. [233] There is a suggestion of an association for biomarkers of ß-HCH and serum thyroid hormone levels, but the results were inconsistent. [233]	EPA lists as a possible human carcinogen based on evidence of benign liver tumours in mice but inadequate data in humans. [233]
Pesticide	γ-HCH (Lindane)	Cross sectional study of vegetable farmers showed no association between serum levels of γ-HCH and respiratory symptoms (cough, phlegm, and wheezing) [233] Mucous membrane irritation of the nose and throat was observed after acute exposure to HCH products by an overheated γ-HCH vaporizer however exposure levels were not reported. [233]	No respiratory, cardiac or endocrine histopathology effects were observed in rats exposed levels up to 5mg/m^3 for 6 hours/day for 90 days or in mice similarly exposed for 14 weeks. [233] Rats exposed to various concentrations of 99.6% *γ*-HCH aerosol via nose-only inhalation for 4 hours exhibited concentration-related neurological effects when observed for up to 22 days after exposure. Slight-to-moderate sedation was observed after exposure to 101 mg/m^3; slight-to-severe sedation was noted after exposure to 378

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Cohort study involving occupational exposure to γ-HCH was associated with increased odds of hypothyroid disease [233] IARC has determined this compound as Group 1 (carcinogenic to humans) due to available in vivo and in vitro data [234] Cardiovascular effects have been reported in human exposed to HCH, exposure concentrations were not reported but electrocardiogram abnormalities in 15% of 45 factory workers reported. [233] Paraesthesia of the face and extremities, headache and vertigo have been reported in a group of 45 workers occupationally exposed to HCH for several years. [233] Target organs include eyes, skin, respiratory system, central nervous system and symptoms of exposure includes irritation eyes, skin, nose, sore throat, coughing, headache, nausea, muscle convulsions, respiratory difficulty, cyanosis, aplastic anaemia, muscle spasm. [234]	mg/m^3; restlessness, excitation, and ataxia were seen after exposure to 642 and 2,104 mg/m^3; and spasms were also noted at the highest concentration (2,104 mg/m^3) [233] Rats exposed to 0.02-5mg/m^3 γ-HCH aerosol for 90 days exhibited a "slightly disturbed general condition" beginning at day 15 where mice similarly exposed for 14 days showed no clinical signs of neurotoxicity [233] No respiratory effects were observed in rats exposed to up to 603mg/m^3 *Y*-HCH aerosol for 4 hours or when exposed to levels up to 5mg/m^3 for 6hours/day for 90 days or in mice similarly exposed for 14 weeks. [233] Rats exposed to 0.02-5mg/m^3 γ-HCH aerosol for 90 days exhibited a "slightly disturbed general condition" beginning at day 15 where mice similarly exposed for 14 days showed no clinical signs of neurotoxicity [233] TCLo human (inhalation) 400µg/kg/3D – headache, nausea LC50 rat (inhalation) 690mg/m^3/4H [234] LCLo mouse (inhalation) 120mg/m^3/6H – changes in white blood cell count [234]
PCB	PCB 101	PCBs can also cause endocrine disruption by altering the production of thyroid hormones and binding to oestrogen receptors, which can stimulate the growth of certain cancer cells and	

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		produce other estrogenic effects, such as reproductive dysfunction. [235] Chronic exposure is reported to cause headache, dizziness, depression, nervousness, irregular menstrual cycles [235] IARC Class 1: Carcinogenic to humans [235]	
PCB	PCB 118	PCBs can also cause endocrine disruption by altering the production of thyroid hormones and binding to oestrogen receptors, which can stimulate the growth of certain cancer cells and produce other estrogenic effects, such as reproductive dysfunction. [235] Chronic exposure is reported to cause headache, dizziness, depression, nervousness, irregular menstrual cycles [235] IARC Class 1: Carcinogenic to humans [235]	
PCB	PCB 128	Short term exposure: inhalation: may produce irritation to nose, throat, and lungs. The vapours can cause coughing and/or difficulty in breathing. Levels above 10 mg/cu m are reported to be unbearable. Inhalation may contribute significantly to all symptoms of long-term exposure. [235] Chronic exposure is reported to cause headache, dizziness, depression, nervousness, irregular menstrual cycles [235]	US EPA Classes B2 Probable human carcinogen. [235]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
PCB	PCB 153	PCBs can also cause endocrine disruption by altering the production of thyroid hormones and binding to oestrogen receptors, which can stimulate the growth of certain cancer cells and produce other estrogenic effects, such as reproductive dysfunction. [235] Chronic exposure is reported to cause headache, dizziness, depression, nervousness, irregular menstrual cycles [235] IARC Class 1: Carcinogenic to humans [235]	
PFAS	10:2 FTAC	May cause respiratory tract irritation [236]	
PFAS	6:2 FTMAC	May cause respiratory tract irritation [237]	
PFAS	6:2 FTOH	May cause respiratory tract irritation [238]	
PFAS	8:2 FTAC	May cause respiratory tract irritation [239]	
PFAS	FOSA	May cause respiratory tract irritation [240] Single study in the general population (n=10859 adults) did find an association of an increased risk of cardiovascular disease. However, increases in the intima media thickness in the common carotid artery was associated with serum FOSA levels in a study of 70-year-old men and women [241]	No laboratory animal studies examining potential respiratory tract effects were identified for FOSA [241] No studies assessing cardiovascular effects in laboratory animals were identified for PFNA. [241] There are no identified laboratory animal inhalation studies of PFOS and carcinogenic health outcomes. [241]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		No epidemiological studies examining endocrine health outcomes have been identified. [241] No epidemiological studies examining potential neurological effects were found for FOSA. [241] An increased breast cancer risk was identified for FOSA but based on a single study. [241]	
PFAS	MeFOSE	May cause respiratory tract irritation [242]	
PFAS	PFDA	May cause respiratory tract irritation [243] General population studies for PFDA have not consistently found increases in the risk of coronary artery or cardiovascular disease. [241] Most general studies did not find an association between serum PFDA levels and Thyroid Stimulating Hormone (TSH), T3 or T4 levels except in studies of pregnant women where a positive association or inverse association wit T3 or TSH levels were reported. [241] No epidemiological studies examining potential neurological effects were found for PFDA. [241] An increased breast cancer risk was identified for PFDA but based on a single study. No associations with prostate cancer identified. However, an association was found among men	Laboratory animal studies did not find increases in the incidence of histological alterations in the heart following exposure to PFDA. [241] There are no identified laboratory animal inhalation studies of PFDA and endocrine health outcomes. [241] Based on oral pathway studies, the pituitary, parathyroid, thyroid, and adrenal glands do not appear to be sensitive targets following exposure to PFDA. [241] There are no identified laboratory animal inhalation studies of PFDA and carcinogenic health outcomes. [241]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		with a first degree relative with prostate cancer [241] Suspected of causing cancer and reproductive toxicity [243]	
PFAS	PFDoA	May cause respiratory tract irritation [243]	
PFAS	PFHxS	General population studies for PFHxS have not consistently found increases in the risk of coronary artery disease or cardiovascular disease. [241] Associations between serum PFHxS and TSH, T3, or T4 levels or thyroid disease have been found however the results are not consistent across studies and a larger number of studies have found no association. [241] Study examined associations of prenatal serum concentrations with foetal and postnatal growth with PFHxS (median 1.6ng/mL) being detected 100% of samples and girls of upper tertile weighed 108g less in adjusted models [244] There has also been a study on association between levels of PFHxS and semen volume, concentration, total count, mobility and morphology. It concluded that cells were 35% lower for the third tertile as compared with the first. [244]	Laboratory animal studies did not find increases in the incidence of histological alterations in the heart following exposure to PFHxS. [241] There are no identified laboratory animal inhalation studies of PFHxS and endocrine health outcomes. [241] There are no identified laboratory animal inhalation studies of PFHxS and neurological health outcomes. [241] There are no identified laboratory animal inhalation studies of PFHxS and carcinogenic health outcomes. [241]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Limited number of studies on neurological health outcomes and mixed results. Decrease in the risk of self-reported memory loss (OR 0.89 (0.79-0.99)) was observed in older adults' participants in health study (n=21024 older adults; >50 years of age). No association for neurological health outcomes in a general population study (n=1766 older adults aged 60-<85 years). [241] No consistent associations were observed for breast cancer and no associations with prostate cancer identified. However, an association was found among men with a first degree relative with prostate cancer [241]	
PFAS	PFNA	May cause respiratory tract irritation [245] General population studies for PFNA have not consistently found increases in the risk of cardiovascular disease.[241] Associations between serum PFNA and TSH, T3, or T4 levels or thyroid disease have been found however the results are not consistent across studies and a larger number of studies have found no association. [241] Limited number of studies on neurological health outcomes and mixed results. Self-reported memory loss was shown to be inversely associated (OR 0.86 (0.78-0.96) with	Laboured breathing during and after a 4-hour nose only exposure to 590mg/m^3 PFNA dust was reported in rats. [241] No studies assessing cardiovascular or endocrine effects in laboratory animals were identified for PFNA. [241] Reported to be endocrine disrupting [245] There are no identified laboratory animal inhalation studies of PFNA and carcinogenic health outcomes. [241]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		serum PFNA level in health study (n=21024 older adults; >50 years of age). However, another study (n=1766 older adults aged 60-<85 years) did not find an association. [241] No consistent associations were observed for breast cancer and no associations with prostate cancer was identified. [241]	
PFAS	PFOA	Respiratory tract irritant (sore throat, cough) [246] Long term exposure reports possibly carcinogenic to humans and may cause toxicity to human reproduction or development [241] Epidemiological studies examining respiratory effects are only available for PFOA. No alterations in lung function were observed in workers at a PFOA facility but increases in respiratory illnesses (chronic bronchitis and shortness of breath) were observed in residents living near the PFOA facility. [241] Residents living near a PFOA facility reported increased risks of self-reported cardiovascular disease (angina, myocardial infarction, and stroke). However, another community study of residents did not find an increased risk of coronary artery disease. [241]	Studies in laboratory animals have examined the potential for perfluoroalkyls to induce histological lesions in the lungs following inhalation [241] Inhalation exposure to PFOA dusts (male and female rates to 18,600mg/m^3) have resulted in red nasal discharge and dry rales [241] Laboratory animal studies did not find increases in the incidence of histological alterations in the heart following rats exposed intermittently head-only to 84mg/m^3 APFO dust for 2 weeks. [241] One study report repeated intermittent head only exposure of male rats up to 84mg/m^3 APFO dust for 2 weeks and gross or microscopic alterations in the thyroid and adrenal gland were not observed. [241] Based on oral pathway studies, the pituitary, parathyroid, thyroid, and adrenal glands do not appear to be sensitive targets following exposure to PFOA. [241]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Associations between serum PFOA and thyroid stimulating hormone (TSH), triiodothyronine (T3), or thyroxine (T4) levels or thyroid disease have been found however the results are not consistent across studies and a larger number of studies have found no association. [241] Three epidemiological studies were identified, with mixed results. One study (n=21,024 older adults > 50 years of age) found a decreased risk of self-reported memory loss in older adults with serum PFOA levels (OR 0.88 (0.79 – 0.97)), with the risk of memory loss being higher among diabetics (P=0.014). A general population study (n=126) of 55-74 years old participants reports higher memory and learning scores (association p=0.03) and inverse association in perseverative errors and responses (p=0.04, p=0.03). However, another study reports no association between serum PFOA and self-reported memory difficulty in a cohort aged 60 to <85 years old. [241] Occupational and community exposure studies have found increases in the risk of testicular and kidney cancer associated with exposure to PFOA. No consistent epidemiological evidence identified for other cancer types. [241] No associations with prostate cancer identified. However, an association was found among men	Exposure to 18,600mg/m^3 APFO dust for 1 hour induced excessive salivation in rats. [241] Laboratory animal studies did not find increases in the incidence of histological alterations in the brain following rats exposed intermittently head-only to 84mg/m^3 APFO dust for 2 weeks. [241] IARC classes PFOA as Group 2B: Possibly carcinogenic to humans [246]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		with a first degree relative with prostate cancer [241]	
PFAS	PFOS	General population studies for PFOS have not consistently found increases in the risk of cardiovascular disease. [241] Three studies have evaluated the possible association between PFOS and heart disease. No association was identified between serum PFOS and physician diagnosed coronary artery disease, angina and/or heart attack. One study found an association between serum PFOS levels and carotid intima media thickness in a general population study and was able to identify a subpopulation that could be at risk. [241] An increased risk of hypertension associated with serum PFOS levels were observed in female adults in one study. It also found associations for systolic and diastolic blood pressure in males and females combined and in females only. [241] Associations between serum PFOS and TSH, T3, or T4 levels or thyroid disease have been found however the results are not consistent across studies and a larger number of studies have found no association. [241]	Inhalation exposure to PFOS dusts (1890 to 45970 mg/m^3) have resulted in nasal discharge rates, and/or laboured breathing in laboratory animals [241] Laboratory animal studies did not find increases in the incidence of histological alterations in the heart following exposure to PFOS. [241] There are no identified laboratory animal inhalation studies of PFOS and endocrine health outcomes. [241] Based on oral pathway studies, the pituitary, parathyroid, thyroid, and adrenal glands do not appear to be sensitive targets following exposure to PFOS[241] There are no identified laboratory animal inhalation studies of PFOS and neurological health outcomes. [241] There are no identified laboratory animal inhalation studies of PFOS and carcinogenic health outcomes. [241]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
		Limited number of studies on neurological health outcomes and mixed results. Three studies examined serum PFOS on self-reported memory in older adults. One study (n=21,024 older adults >50 years of age) found an inverse association for a risk of memory loss (OR 0.86 (0.78 – 0.96)), however a second cohort study (n=1766 older adults aged 60-<85 years) found no association for difficulty remembering/confusion. Another general population study (n=126 older adults, aged 55-74 years) found an association between serum PFOS levels memory and learning scores (association p=0.04) and visual and spatial function scores (association p=0.05). [241] One occupational exposure study reported an increase in bladder cancer, but this result was not replicated in other occupational studies. General population studies have not consistently reported increases in malignant tumours. [241] No associations with prostate cancer identified. However, an association was found among men with a first degree relative with prostate cancer [241]	
PFAS	PFPA	Respiratory tract irritation [247]	

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Bisphenol	BPA	Derived no effect level for general population for repeat dose toxicity (1 mg/m^3) via inhalation route. [14] May cause respiratory tract irritation (cough, sore throat) and reproductive toxicity [248]	LC, mice inhalation >1700mg/m^3/2h (dyspnoea) Slight and transient nasal tract epithelial damage was observed in rats exposed to bisphenol A dust at 170 mg/m^3 for 6 hours. Slight local inflammatory effects in the upper respiratory tract were observed in rats exposed to 50 mg/m^3 and 150 mg/m^3 of Bisphenol A in 2 and 13-week repeat inhalation studies but were not observed at 10 mg/m^3 in the same studies. Taken together with anecdotal human evidence, these data suggest bisphenol A has a limited respiratory irritation potential. [14] Groups of 30 male and 30 female rats were exposed to 0, 10, 50, or 150 mg/m^3 aerosolised bisphenol A via whole body exposure under dynamic airflow conditions for 6 hours per day, 5 days per week, for 13 weeks. The authors concluded that inhalation exposure to BPA did not result in any lower respiratory tract effects at any concentration tested. [14] No inhalation or dermal carcinogenicity studies were available, although in repeat exposure inhalation toxicity studies, bisphenol A did not exhibit properties that raised concern for potential carcinogenicity. Overall, bisphenol A has no carcinogenic potential. [14] Low doses (not specified) of bisphenol A can mimic the body's own hormones, possibly causing negative health effects. There is thus concern that long term low dose exposure to bisphenol A may induce chronic toxicity in humans [248]

Group	Compound	Human (Inhalation pathway)	Animal (Inhalation pathway)
Bisphenol	BPF	May cause respiratory tract irritation [249]	
alkylphenol	4-NP		Endocrine Disrupting [250]
alkylphenol	OP	Epidemiological studies about associations between prenatal exposure and foetal growth are extremely limited and further studies are required [252]	Endocrine Disrupting [251] LD50 Rat inhalation </= 116 mg/L/ 24 hours /89% purity/ [252]
alkylphenol	4-n-octylphenol	Inhalation symptoms include burning sensation, cough, sore throat, laboured breathing, and shortness of breath [253]	
Phenol	PTBP	Irritating to eyes, nose and throat. If inhaled will cause breathing difficulties [254]	LCLo inhalation rat, dose was 5600mg/m^3 over 4 hours. Effects reported include respiratory depression [254]
Phenol	TCS		under assessment as endocrine disruptor [255]