# **Firefighting - Correlation of non-chemical pathogens and exposures with factors in the Statements of Principles**

The RMA has considered the activities and exposures that are associated with the occupation of firefighting and has reviewed the SOPs to see which diseases or injuries those activities or exposures may be implicated in.

**Table 2: Firefighting – Correlation of non-chemical pathogens and exposures with factors in the Statements of Principles**

Please note: This table has been prepared to assist stakeholders including claimants. It is not to be seen as prescriptive or definitive. In particular each potential claimant has to consider their own individual circumstances to see what factors may be relevant to their particular kind of disease or injury.

| **Non-chemical pathogen or exposure** | **SOP factor RH and [BOP]** | **Statement of Principles** |
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| Firefighting  | firefighting for a cumulative period of at least 1 000 hours before the clinical onset of mesothelioma, where the first exposure occurred at least 10 [15 BOP] years before the clinical onset of mesothelioma;***firefighting*** means being involved in the direct combat of fires, including activities to control, extinguish, mop-up or prevent fires, or participating in training activities involving fires. | Mesothelioma (104, 105/2015) |
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| Stressors | experiencing a category 1A stressor …..***category 1A stressor*** means one of the following severe traumatic events: (a) experiencing a life-threatening event; | Acute stress disorder (41, 42/2014)Adjustment disorder (23, 24/2016)Alcohol use disorder (102, 103/2014)Anxiety disorder (102, 103/2014) Bipolar disorder (53, 54/2018)Depressive disorder (83, 84/2015)Eating disorder (13, 14/2016)Panic disorder (55, 56/2018)Personality disorder (17, 18 of 2018)Posttraumatic stress disorder (82, 83/2014)Schizophrenia (83, 84/2016)Substance use disorder (59, 60/2017)Suicide and attempted suicide (65, 66/2016)Adrenal insufficiency (71, 72 of 2018)Cardiomyopathy (Takotsubo) (85, 86/2015)Cerebrovascular accident (65/2015)Chronic insomnia disorder (37, 38/2019)Chronic multisymptom illness (3/2020)Female sexual dysfunction (95, 96/2016)Gastric ulcer and duodenal ulcer (61/2015)Gingivitis (17/2022)Inflammatory bowel disease (90/2020)Inflammatory bowel disease - worsening only (91/2020)Multiple sclerosis - worsening only (11, 12/2020)Psoriasis (13/2021)Chronic fatigue syndrome (105/2021)Fibromyalgia (107/2021)Irritable bowel syndrome (65/2019) |
| experiencing a category 1B stressor …..***category 1B stressor*** means one of the following severe traumatic events:(b) being an eyewitness to a person being killed or critically injured;(d) participating in the clearance of a corpse or a critically injured casualty; or(e) viewing a corpse or a critically injured casualty as an eyewitness.***corpse*** means the human remains or body parts of one or more persons who have met a violent or horrific death. Note: Examples of a violent or horrific death may include death due to suicide, gunshot, improvised explosive devices, natural and technological disasters, terrorist attacks or motor vehicle accidents. Seeing a closed body bag or viewing a body in an open-casket coffin are excluded from this definition.***eyewitness*** means a person who experiences an incident first-hand and can give direct evidence of it. This excludes persons exposed only to public broadcasting or mass media coverage of the incident. | Acute stress disorder (41, 42/2014)Adjustment disorder (23, 24/2016)Alcohol use disorder (102, 103/2014)Anxiety disorder (102, 103/2014)Bipolar disorder (53, 54/2018)Depressive disorder (83, 84/2015)Eating disorder (13, 14/2016)Panic disorder (55, 56/2018)Personality disorder (17, 18 of 2018)Posttraumatic stress disorder (82, 83/2014) Schizophrenia (83, 84/2016)Substance use disorder (59, 60/2017)Suicide and attempted suicide (65, 66/2016)Cardiomyopathy (Takotsubo) (85, 86/2015)Cerebrovascular accident (65/2015)Chronic insomnia disorder (37, 38/2019)Chronic multisymptom illness (3/2020)Female sexual dysfunction (95, 96/2016)Gastric ulcer and duodenal ulcer (61/2015)Gingivitis (17/2022)Inflammatory bowel disease (90/2020)Inflammatory bowel disease - worsening only (91/2020)Multiple sclerosis - worsening only (11, 12/2020)Psoriasis (13/2021)Ischaemic heart diseases (1,2/2016)Chronic fatigue syndrome (105/2021)Irritable bowel syndrome (65/2019) |
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| Shift work | undertaking night shift work on at least 250 occasions over a continuous period, for at least the five years before the clinical onset/worsening of ischaemic heart disease;***night shift work*** means working for at least three hours between 12 AM and 5 AM. | Ischaemic heart disease(1/2016) |
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| Strenuous exercise | for angina, acute myocardial infarction or sudden death from ischaemic heart disease only: (a) undertaking physical activity of five METs [6 METs BOP] or more within the 24 hours [12 hours BOP] before the clinical onset of ischaemic heart disease;for acute myocardial infarction or sudden death from ischaemic heart disease only:(a) undertaking physical activity of five METs [6 METs BOP] or more within the 24 hours [12 hours BOP] before the clinical worsening of ischaemic heart disease;***MET*** means a unit of measurement of the level of physical exertion. 1 MET = 3.5 ml of oxygen/kg of body weight per minute, 1.0 kcal/kg of body weight per hour or resting metabolic rate. | Ischaemic heart disease(1,2/2016) |
| experiencing a stimulus from Specified List 1 of stimuli within the 24 hours before the clinical worsening of sickle-cell disorder;***Specified List 1 of stimuli*** means: (d) strenuous exercise. | Sickle-cell disorder(40, 41/2017) |
| experiencing a specified physical stimulus immediately before the clinical onset of an epileptic seizure***a specified physical stimulus*** means a specified activity or physical process from the following list: (d) strenuous exercise; | Epileptic seizure(77, 78/2013) |
| undertaking weight bearing exercise involving jumping or repeated flexion and extension of the affected knee, at a minimum intensity greater than six METs for at least four hours [six hours BOP] per week, for at least the four weeks before the clinical onset/worsening of patellar tendinopathy;[***Note***: *Similar exercise factors variously worded causing injury to different parts of the musculoskeletal system are included in a range of SOPs, including chondromalacia patella, iliotibial band syndrome, Achilles tendinopathy and bursitis, and trochanteric bursitis or gluteal tendinopathy.*] | Patellar tendinopathy(21, 22/2020) |
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| Diesel exhaust | being heavily exposed to diesel engine exhaust for a cumulative period of at least 10 000 [15 000 BOP] hours, at least five [ten BOP] years before the clinical onset of malignant neoplasm of the lung; | Malignant neoplasm of the lung (92, 93/2014) |
| inhaling vapour, gas, dust or fumes of a substance from the specified list in an enclosed space:(i) for a cumulative period of at least 10 000 hours, before the clinical onset of chronic obstructive pulmonary disease; and(ii) where that exposure has ceased, the clinical onset/worsening of chronic obstructive pulmonary disease has occurred within 20 years of cessation; ***a substance from the specified list*** means: (h) diesel engine exhaust; | Chronic obstructive pulmonary disease (37/2014) |
| being heavily exposed to diesel engine exhaust for a cumulative period of at least 15 000 hours, before the clinical onset of malignant neoplasm of X;***being heavily exposed to diesel engine exhaust***means:(a) being an occupant in an enclosed diesel-powered vehicle cabin contaminated with diesel fumes;(b) working in an enclosed space where diesel-powered engines or motors are being operated; or(c) repairing and/or servicing diesel engines; | Malignant neoplasm of the bladder (83/2019)Malignant neoplasm of the renal pelvis and ureter (92/2019) |
| inhaling a vapour, gas, dust or fumes produced by a substance from the specified list of substances, or smoke from fire, in an enclosed space:(a) for a cumulative period of at least 5,000 hours before the clinical onset/worsening of fibrosing interstitial lung disease; and (b) if that exposure has ceased before the clinical onset/worsening of fibrosing interstitial lung disease, then that onset/worsening occurred within 20 years of cessation;***specified list of substances*** means: diesel engine exhaust; | Fibrosing interstitial lung disease (85, 86/2021) |
| for reactive airways dysfunction syndrome only, inhaling very high concentrations of a substance with irritant properties, where such inhalation has resulted in acute toxic lower respiratory tract effects, within the 24 hours before the clinical onset of reactive airways dysfunction syndrome;Note 1: Irritant substances that cause reactive airways dysfunction syndrome include:(d) fumes (including **diesel exhaust** and fire smoke);  | Asthma(31, 32/2021) |
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| Thermal, electrical or chemical burn | having exposure to infrared radiation sufficient to cause erythema at the affected area of the body at the time of the clinical onset of external burn;having exposure to a heat source sufficient to cause erythema at the affected area of the body at the time of the clinical onset of external burn;having contact with vesicant or corrosive chemicals sufficient to cause erythema at the affected area of the body within the 48 hours before the clinical onset of external burn; | External burn (110, 111/2015) |
| having a thermal burn or electrical injury within the 30 days before the clinical onset of peripheral neuropathy;***a thermal burn*** means: (a) a full thickness thermal burn to at least ten percent of the total body surface area; or (b) a partial thickness thermal burn to at least 20 percent of the total body surface area; | Peripheral neuropathy(74, 75/2014) |
| having an external burn to the affected arm requiring hospitalisation, within the five years [two years BOP] before the clinical onset/worsening of ulnar neuropathy at the elbow; | Ulnar neuropathy at the elbow (65, 66/2017) |
| having trauma or surgery to the affected wrist or hand before the clinical onset/worsening of carpal tunnel syndrome, which: (a) alters the normal contour of the carpal tunnel; or (b) damages the flexor tendons within the carpal tunnel;***trauma to the affected wrist or hand*** means a discrete event involving the application of significant physical force to or through the affected wrist or hand, that causes:(a) damage to the wrist or hand; and (b) the development, within 24 hours of the event occurring, of symptoms and signs of pain and tenderness, and either altered mobility or range of movement of the wrist or hand. In the case of sustained unconsciousness or the masking of pain by analgesic medication, these symptoms and signs must appear on return to consciousness or the withdrawal of the analgesic medication; and(c) the persistence of these symptoms and signs for a period of at least 7 days following their onset, save for where medical intervention for the trauma to that wrist or hand has occurred and that medical intervention involves one of the following: (i) immobilisation of the wrist or hand by splinting or similar external agent;(ii) injection of a corticosteroid or local anaesthetic into that wrist or hand; or (iii) surgery to that wrist or hand.Note: Examples of trauma include laceration, sprain, crush injury, fracture, dislocation, and a **thermal, electrical or chemical burn**. | Carpal tunnel syndrome(93, 94/2021) |
| having a severe thermal or chemical burn to the affected eye before the clinical onset/worsening of acquired cataract;***severe thermal or chemical burn*** means a burn involving at least one of the following clinical features:(a) corneal anaesthesia; (b) corneal opacification; (c) symptoms and signs lasting at least three days and requiring medical attention; or(d) for chemical burns only, penetration of the chemical into the anterior chamber. | Acquired cataract(87, 88/2016) |
| having a traumatic injury to the affected trigeminal nerve within the three months before the clinical onset/worsening of trigeminal neuropathy;***traumatic injury*** means a mechanical injury caused by compression, crush, transection, stretching, or a chemical or thermal burn. | Trigeminal neuropathy(79, 80/2015) |
| having trauma to the hand, wrist or forearm of the affected side, within the one year before the clinical onset/worsening of Dupuytren disease; Note 2: Examples of trauma include, but are not limited to, laceration, sprain or crush injury, fracture or surgery, and a **thermal, electrical or chemical burn**. | Dupuytren disease(9, 10/2019) |
| for squamous cell or undifferentiated carcinoma of the oesophagus only: (b) having a caustic burn of the oesophagus, at least ten years before the clinical onset of malignant neoplasm of the oesophagus; | Malignant neoplasm of the oesophagus(120, 121/2015) |
| having a thermal or chemical burn within the seven days before the clinical onset/worsening of herpes simplex at or close to the site of the burn;having a severe thermal burn within the 30 days before the clinical onset/worsening of herpes simplex;***severe thermal burn*** means a burn injury caused by the application of heat to body tissue, including inhalational burn, which is of sufficient severity to warrant hospital admission as an inpatient. | Herpes simplex(39, 40/2018) |
| for thyrotoxicosis only, having trauma involving the thyroid gland within the 14 days before the clinical onset/worsening of thyrotoxicosis;***trauma involving the thyroid gland*** means:(a) a blunt injury or **burn injury** resulting in soft tissue damage adjacent to or overlying the thyroid gland;  | Hyperthyroidism and thyrotoxicosis(5, 6/2022) |
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| Smoke from fires | inhaling, ingesting or having cutaneous contact with a chemical agent that causes oxidation of haemoglobin, within the two days before the clinical onset/worsening of methaemoglobinaemia;Note: Chemicals that cause methaemoglobinaemia include, but are not limited to, aniline and its derivatives (for example, found in dyes); naphthalene (for example, found in mothballs); nitrobenzenes (for example, found in solvents and paints); **nitrogen oxide (for example, found in smoke from fires**); paraquat (for example, found in herbicides); and chlorates, nitrates and nitrotoluenes (for example, found in explosives). | Methaemoglobinaemia (17, 18/2019) |
| inhaling airborne dusts, smoke from fires, or fumes or vapours from fuel or a chemical agent within the 48 hours before the clinical onset/worsening of sinusitis; | Sinusitis (73/2018) |
| inhaling vapours, gases or fumes of a chemical agent from the specified list of chemical agents:(a) resulting in signs and symptoms of severe acute lower respiratory damage requiring medical attention within 48 hours after exposure; and(b) the persistence of respiratory symptoms and signs for at least one week after exposure, within the five [two BOP] years before the clinical onset/worsening of bronchiectasis;***specified list of chemical agents*** means: (e) smoke from fires. | Bronchiectasis (30, 31/2017) |
| inhaling a respiratory tract irritant from the specified list:(i) resulting in signs and symptoms of severe acute lower respiratory damage requiring medical attention within 48 hours after exposure; and(ii) the persistence of respiratory symptoms and signs for at least one week after exposure, within the ten [five BOP] years before the clinical onset/worsening of chronic obstructive pulmonary disease; ***a respiratory tract irritant from the specified list*** means: (i) smoke from fires;inhaling smoke from the combustion of wood, charcoal, coal or other biomass or fossil fuel, in an enclosed space:(i) for a cumulative period of at least 5 000 [10 000 BOP] hours, before the clinical onset/worsening of chronic obstructive pulmonary disease; and(ii) where that exposure has ceased, the clinical onset/worsening of chronic obstructive pulmonary disease has occurred within 20 [ten BOP] years of cessation;  | Chronic obstructive pulmonary disease (37, 38/2014) |
| inhaling a vapour, gas, dust or fumes produced by a substance from the specified list of substances, or smoke from fire, in an enclosed space:(a) for a cumulative period of at least 5,000 hours before the clinical onset/worsening of fibrosing interstitial lung disease; and (b) if that exposure has ceased before the clinical onset/worsening of fibrosing interstitial lung disease, then that onset/worsening occurred within 20 years of cessation;inhaling a toxic gas or fumes resulting in:(a) signs and symptoms of severe acute lower respiratory damage requiring medical attention within 48 hours after exposure; and (b) the persistence of respiratory signs and symptoms for at least 1 week after exposure, within the 1 year before the clinical onset of fibrosing interstitial lung disease;Note: Types of toxic gases or fumes include anhydrous ammonia fumes, **smoke**, oxides of sulphur, oxides of nitrogen, chlorine, phosgene, humidifier detergents, perfluoroisobutylene (may be released in fires of military vehicles containing Teflon) and zinc chloride smoke from smoke bombs. | Fibrosing interstitial lung disease (85, 86/2021) |
| inhaling smoke from the combustion of wood, charcoal or coal while in an enclosed space, on more days than not for at least ten years or for a cumulative period of at least 7500 hours, before the clinical onset of malignant neoplasm of the nasopharynx; | Malignant neoplasm of the nasopharynx (9/2020) |
| inhaling smoke from the combustion of biomass or fossil fuels whilst in an enclosed space for at least 5 000 hours before the clinical onset of malignant neoplasm of the larynx, where inhaling such smoke commenced at least five years before the clinical onset of malignant neoplasm of the larynx; | Malignant neoplasm of the larynx (61/2013) |
| inhaling smoke from the combustion of coal, wood, charcoal or another solid biomass fuel while in an enclosed space with a visible smoke haze:(i) for a cumulative period of at least 7 500 [15 000 BOP] hours before theclinical onset of malignant neoplasm of the lung; and(ii) where the first inhalation of smoke commenced at least five [ten BOP] years before the clinical onset of malignant neoplasm of the lung;  | Malignant neoplasm of the lung (92, 93/2014) |
| for reactive airways dysfunction syndrome only, inhaling very high concentrations of a substance with irritant properties, where such inhalation has resulted in acute toxic lower respiratory tract effects, within the 24 hours before the clinical onset of reactive airways dysfunction syndrome;Note 1: Irritant substances that cause reactive airways dysfunction syndrome include:(d) fumes (including diesel exhaust and **fire smoke**);  | Asthma(31, 32/2021) |
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| Highly polluted air | for angina, acute myocardial infarction or sudden death from ischaemic heart disease only: (a) inhaling ambient highly polluted air as specified for at least two hours within the seven days before the clinical onset of ischaemic heart disease;for acute myocardial infarction or sudden death from ischaemic heart disease only:(a) inhaling ambient highly polluted air as specified for at least two hours within the seven days before the clinical worsening of ischaemic heart disease;***ambient highly polluted air as specified*** means air with 24 hour average concentrations of: (a) particulate matter with an aerodynamic diameter of < 2.5 µm (PM2.5) exceeding 25 µg/m3; or(b) particulate matter with an aerodynamic diameter of < 10 µm (PM10) exceeding 50 µg/m3. | Ischaemic heart disease (1,2/2016) |
| inhaling ambient polluted air as specified, for a cumulative period of at least 100 hours, within the seven days before the clinical onset of cerebrovascular accident;***ambient polluted air as specified*** means air with (annual average) levels of:(a) particulates with an aerodynamic diameter <2.5 μm (PM2.5) exceeding 30 μg/m3; or(b) particulates with an aerodynamic diameter <10 μm (PM10) exceeding 150 μg/m3. | Cerebrovascular accident (65/2015) |
| inhaling ambient polluted air as specified: (i) for a cumulative period of at least 70 000 hours before the clinical onset/worsening of chronic obstructive pulmonary disease; and (ii) where that exposure has ceased, the clinical onset/worsening of chronic obstructive pulmonary disease has occurred within 20 years of cessation;***ambient polluted air as specified*** means air with annual average levels of: (a) particulates with an aerodynamic diameter <2.5 μm (PM2.5) exceeding 30 μg/m3; (b) particulates with an aerodynamic diameter <10 μm (PM10) exceeding 150 μg/m3; (c) nitrogen dioxide exceeding 18.0 μg/m3; or (d) nitrous oxides exceeding 31.5 μg/m3. | Chronic obstructive pulmonary disease(37/2014) |
| inhaling ambient polluted air as specified:(i) for a cumulative period of at least 35 000 hours [70 000 hours BOP] before the clinical onset of malignant neoplasm of the lung; and (ii) where the first inhalation of ambient polluted air occurred at least five years [ten years BOP] before the clinical onset of malignant neoplasm of the lung;***ambient polluted air as specified*** means air with annual average levels of:(a) particulates with an aerodynamic diameter <2.5 μm (PM2.5) exceeding 30 μg/m3; or (b) particulates with an aerodynamic diameter <10 μm (PM10) exceeding 50 μg/m3; | Malignant neoplasm of the lung (92, 93/2014) |
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| Chemical irritant | having the affected area of skin exposed to an irritant within the three days before the clinical onset/worsening of irritant contact dermatitis;***irritant*** means a chemical substance or physical agent which, when it comes into contact with an area of skin, is capable of producing direct tissue injury and inflammation. It does not include heat, cold, friction, solar radiation or other forms of radiation. Examples of irritants include:(a) cleansers (including soap and alcohol based hand rubs); (b) detergents (including sodium lauryl sulphate); (c) oils and coolants; (d) fuels; (e) solvents; (f) food (including citrus fruits);(g) alkalis (including wet cement or lime); (h) pesticides (including insect repellent); (i) adhesives (including epoxy resins); (j) tear gas or smokescreens; (k) fibreglass; (l) plant sap (including spurge); (m) bodily fluids of the rove beetle; and(n) urine or faecal incontinence. | Irritant contact dermatitis (3, 4/2021) |
| having an irritant substance exposure to the region of the affected eye at the time of the clinical onset/worsening of blepharitis;***irritant substance*** means a chemical which causes an inflammatory effect on living tissue by chemical action at the site of contact, leading to irritant contact dermatitis. | Blepharitis (29, 30/2019) |
| having ocular or periocular exposure to an irritant substance within the 24 hours before the clinical onset of conjunctivitis;***irritant substance*** means a chemical which causes an inflammatory effect on living tissue by chemical action at the site of contact; | Conjunctivitis (76, 77/2020) |
| inhaling a drug or irritant substance which results in:(a) acute nasal symptoms or signs within 48 hours of the inhalation; and(b) scarring or erosion of the nasal or sinus mucosa; before the clinical onset/worsening of sinusitis;Note 2: Examples of irritant substances include gases (ammonia, chlorine, mustard, nitrogen dioxide, sulphur dioxide), powdered solids (aspirin, baking soda, levamisole, capsules, tablets, pills) and lewisite.Note 3: Examples of acute nasal symptoms or signs include rhinorrhoea, and the inflammation, oedema, ulceration or haemorrhage of the nasal mucosa | Sinusitis (73, 74/2018) |
| being exposed to an immunologic or non-immunologic stimulus within the 24 hours before the clinical onset/worsening of asthma;***immunologic or non-immunologic stimulus*** means an allergenic or irritant substance, an activity or an environment that can cause inflammation of the airways and bronchial hyperresponsiveness.Note: Examples of immunologic or non-immunologic stimuli include:(a) air pollutants; (b) cereal dusts;(c) chemical fumes; (d) cold air; (e) drugs; (f) exercise; (g) irritant gases (including mustard gas); (h) metals; (i) moulds; (j) proteins derived from animals, insects and fish;(k) respiratory infections; and (l) wood dusts.for reactive airways dysfunction syndrome only, inhaling very high concentrations of a substance with irritant properties, where such inhalation has resulted in acute toxic lower respiratory tract effects, within the 24 hours before the clinical onset of reactive airways dysfunction syndrome;Note 1: Irritant substances that cause reactive airways dysfunction syndrome include:(a) acids (including acetic and hydrochloric acids);(b) alkalis (including ammonia and hydrazine);(c) biocides (including formalin and fumigating agents);(d) fumes (including diesel exhaust and fire smoke); (e) gases (including chlorine and sulphur dioxide); (f) halogenated derivatives (including trifluoromethane and chlorofluorocarbons);(g) solvents (including perchloroethylene); and(h) sprays (including paints and floor sealant). | Asthma (31, 32/2021) |
| inhaling a respiratory tract irritant from the specified list:(i) resulting in signs and symptoms of severe acute lower respiratory damage requiring medical attention within 48 hours after exposure; and(ii) the persistence of respiratory symptoms and signs for at least one week after exposure, within the ten [five BOP] years before the clinical onset/worsening of chronic obstructive pulmonary disease; ***a respiratory tract irritant from the specified list*** means: (a) ammonia; (b) chlorine; (c) Lewisite; Page 5 of 7 of Instrument No. 37 of 2014 (d) oxides of nitrogen; (e) oxides of sulphur; (f) phosgene; (g) phthalic anhydride; (h) smoke from fires; (i) sulphur mustard (mustard gas); or (j) another respirable agent which causes comparable tissue damage; | Chronic obstructive pulmonary disease (37, 38/2014) |
| inhaling high concentrations of a substance with irritant properties, where:(a) the inhalation has resulted in signs and symptoms of acute damage to the lower respiratory tract within the 48 hours after the inhalation; and(b) the clinical onset/worsening of bronchiolitis obliterans organising pneumonia occurs within the 30 days following the inhalation of the substance; | Bronchiolitis obliterans organising pneumonia (79, 80/2018) |
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| Asbestos fibres | inhaling respirable asbestos fibres in an enclosed space:(i) at the time material containing asbestos fibres was being applied, removed, dislodged, cut or drilled; and(ii) the first such inhalation of asbestos fibres occurred at least five [ten BOP] years before the clinical onset of pleural plaque; orinhaling respirable asbestos fibres for a cumulative period of at least 1 000 hours in an open environment:(i) at the time material containing asbestos fibres was being applied, removed, dislodged, cut or drilled; and(ii) the first such inhalation of asbestos fibres occurred at least five [ten BOP] years before the clinical onset of pleural plaque;  | Pleural plaque (45, 46/2014) |
| inhaling respirable asbestos fibres in an enclosed space at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 1,000 hours [1,500 hours BOP] before the clinical onset of asbestosis; and(b) where the first inhalation of asbestos fibres commenced at least 5 years [10 years BOP] before the clinical onset of asbestosis;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an enclosed space may result in exposure to respirable asbestos fibres.(2) inhaling respirable asbestos fibres in an open environment at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 3,000 hours [5000 hours BOP] before the clinical onset of asbestosis; and(b) where the first inhalation of asbestos fibres commenced at least 5 years [10 years BOP] before the clinical onset of asbestosis;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an open environment may result in exposure to respirable asbestos fibres.inhaling respirable asbestos fibres at the time material containing respirable asbestos fibres was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 1,000 hours [1,500 hours BOP] before the clinical worsening of asbestosis; and (b) within the 2 years before the clinical worsening of asbestosis;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an enclosed space or an open environment may result in exposure to respirable asbestos fibres. | Asbestosis (59, 60/2021) |
| inhaling respirable asbestos fibres in an enclosed space, at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 1,000 hours [1,500 hours BOP] before the clinical onset of fibrosing interstitial lung disease; and (b) where the first inhalation of asbestos fibres commenced at least 5 years [10 years BOP] before the clinical onset of fibrosing interstitial lung disease;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an enclosed space may result in exposure to respirable asbestos fibres. inhaling respirable asbestos fibres in an open environment, at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 3,000 hours [5000 hours BOP] before the clinical onset of fibrosing interstitial lung disease; and (b) where the first inhalation of asbestos fibres commenced at least 5 years [10 years BOP] before the clinical onset of fibrosing interstitial lung disease;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an open environment may result in exposure to respirable asbestos fibres.inhaling respirable asbestos fibres at the time material containing respirable asbestos fibres was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 1,000 hours [1,500 hours BOP] before the clinical worsening of asbestosis; and (b) within the 2 years before the clinical worsening of asbestosis;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an enclosed space or an open environment may result in exposure to respirable asbestos fibres. | Fibrosing interstitial lung disease (85, 86/2021) |
| inhaling respirable asbestos fibres in an enclosed space, at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 1,000 hours [2,000 hours BOP] before the clinical onset of malignant neoplasm of the colon or rectum; and(b) where the first inhalation of asbestos fibres commenced at least 5 years [10 years BOP] before the clinical onset of malignant neoplasm of the colon or rectum;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an enclosed space may result in exposure to respirable asbestos fibres.inhaling respirable asbestos fibres in an open environment, at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 3,000 hours [6,000 hours BOP] before the clinical onset of malignant neoplasm of the colon or rectum; and(b) where the first inhalation of asbestos fibres commenced at least 5 years [10 years] before the clinical onset of malignant neoplasm of the colon or rectum;Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an open environment may result in exposure to respirable asbestos fibres. | Malignant neoplasm of the colon and rectum (19, 20/2022) |
| inhaling respirable asbestos fibres in an enclosed space at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 1,000 hours before the clinical onset of malignant neoplasm of the oral cavity, oropharynx or hypopharynx; and (b) where the first inhalation of asbestos fibres commenced at least 5 years before the clinical onset of malignant neoplasm of the oral cavity, oropharynx or hypopharynx; Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an enclosed space may result in exposure to respirable asbestos fibres.inhaling respirable asbestos fibres in an open environment at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 3,000 hours before the clinical onset of malignant neoplasm of the oral cavity, oropharynx or hypopharynx; and (b) where the first inhalation of asbestos fibres commenced at least 5 years before the clinical onset of malignant neoplasm of the oral cavity, oropharynx or hypopharynx; Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an open environment may result in exposure to respirable asbestos fibres. | Malignant neoplasm of the oral cavity, oropharynx and hypopharynx (65/2021) |
| inhaling respirable asbestos fibres in an enclosed space:(a) for a cumulative period of at least 1 000 [2 000 BOP] hours before the clinical onset of malignant neoplasm of the oesophagus; and(b) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(c) the first inhalation of respirable asbestos fibres commenced at least five [ten BOP] years before the clinical onset of malignant neoplasm of the oesophagus;inhaling respirable asbestos fibres in an open environment:(a) for a cumulative period of at least 3 000 [6 000 BOP] hours before the clinical onset of malignant neoplasm of the oesophagus; and(b) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(c) the first inhalation of respirable asbestos fibres commenced at least five [ten BOP] years before the clinical onset of malignant neoplasm of the oesophagus; | Malignant neoplasm of the oesophagus (120, 121/2015) |
| inhaling respirable asbestos fibres in an enclosed space:(i) for a cumulative period of at least 1 000 hours before the clinical onset of malignant neoplasm of the stomach; and(ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(iii) the first inhalation of respirable asbestos fibres commenced at least five years before the clinical onset of malignant neoplasm of the stomach; orinhaling respirable asbestos fibres in an open environment:(i) for a cumulative period of at least 3 000 hours before the clinical onset of malignant neoplasm of the stomach; and(ii)at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(iii) the first inhalation of respirable asbestos fibres commenced at least five years before the clinical onset of malignant neoplasm of the stomach; | Malignant neoplasm of the stomach (58/2014) |
| inhaling respirable asbestos fibres in an enclosed space:(i) for a cumulative period of at least 1 000 hours before the clinical onset of malignant neoplasm of the bile duct; and(ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(iii) where the first inhalation of respirable asbestos fibres commenced at least five years before the clinical onset of malignant neoplasm of the bile duct; orinhaling respirable asbestos fibres in an open environment:(i) for a cumulative period of at least 3 000 hours before the clinical onset of malignant neoplasm of the bile duct; and(ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (iii) where the first inhalation of respirable asbestos fibres commenced at least five years before the clinical onset ofmalignant neoplasm of the bile duct;  | Malignant neoplasm of the bile duct (69/2015) |
| inhaling respirable asbestos fibres in an enclosed space:(i) for a cumulative period of at least 1 000 [2 000 BOP] hours before the clinical onset of malignant neoplasm of the larynx; and(ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(iii) the first inhalation of respirable asbestos fibres commenced at least five [ten BOP] years before the clinical onset of malignant neoplasm of the larynx; orinhaling respirable asbestos fibres in an open environment:(i) for a cumulative period of at least 2 000 [4 000 BOP] hours before the clinical onset of malignant neoplasm of the larynx; and(ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(iii) the first inhalation of respirable asbestos fibres commenced at least five [ten BOP] years before the clinical onset of malignant neoplasm of the larynx;  | Malignant neoplasm of the larynx (61, 62/2013) |
| inhaling respirable asbestos fibres in an enclosed space:(i) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(ii) where the first inhalation of respirable asbestos fibres commenced at least five [ten BOP] years before the clinical onset ofmalignant neoplasm of the lung; orinhaling respirable asbestos fibres in an open environment:(i) for a cumulative period of at least 1 000 hours before the clinical onset of malignant neoplasm of the lung; and(ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(iii) where the first inhalation of respirable asbestos fibres commenced at least five [ten BOP] years before the clinical onset ofmalignant neoplasm of the lung;  | Malignant neoplasm of the lung (92, 93/2014) |
| inhaling asbestos fibres:(a) at the time material containing asbestos fibres was being applied, removed, dislodged, cut or drilled; and(b) the first inhalation of asbestos fibres occurred at least ten [15 BOP] years, before the clinical onset of mesothelioma;inhaling asbestos fibres in an open environment for a cumulative period of at least 1 000 hours before the clinical onset of mesothelioma:(a) at the time material containing asbestos fibres was being applied, removed, dislodged, cut or drilled; and(b) the first inhalation of asbestos fibres occurred at least 15 years, before the clinical onset of mesothelioma;inhaling erionite fibres on more days than not for at least five [ten BOP] years before the clinical onset of mesothelioma, where the first inhalation of erionite fibres occurred at least ten [15 BOP] years before the clinical onset of mesothelioma; | Mesothelioma (104, 105/2015) |
| inhaling respirable asbestos fibres in an enclosed space, at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 1,000 hours before the clinical onset of malignant neoplasm of the kidney; and (b) where the first inhalation of asbestos fibres commenced at least five years before the clinical onset of malignant neoplasm of the kidney; Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an enclosed space may result in exposure to respirable asbestos fibres.inhaling respirable asbestos fibres in an open environment, at the time material containing asbestos was being applied, removed, cut, drilled, dislodged or disturbed: (a) for a cumulative period of at least 3,000 hours before the clinical onset of malignant neoplasm of the kidney; and (b) where the first inhalation of asbestos fibres commenced at least five years before the clinical onset of malignant neoplasm of the kidney; Note: Disturbance of debris or dust contaminated with asbestos fibres already present in an open environment may result in exposure to respirable asbestos fibres. | Malignant neoplasm of the kidney (41/2021) |
| inhaling respirable asbestos fibres in an enclosed space: (a) for a cumulative period of at least 1 000 hours before the clinical onset of malignant neoplasm of the liver; and (b) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (c) where the first inhalation of respirable asbestos fibres commenced at least five years before the clinical onset of malignant neoplasm of the liver; inhaling respirable asbestos fibres in an open environment: (a) for a cumulative period of at least 3 000 hours before the clinical onset of malignant neoplasm of the liver; and (b) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (c) where the first inhalation of respirable asbestos fibres commenced at least five years before the clinical onset of malignant neoplasm of the liver; | Malignant neoplasm of the liver(31/2020) |
| inhaling respirable asbestos fibres in an enclosed space:(a) for a cumulative period of at least 1 000 [2 000 BOP] hours before the clinical onset of malignant neoplasm of the ovary; and(b) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(c) where the first inhalation of respirable asbestos fibres occurred at least five [ten BOP] years before the clinical onset of malignant neoplasm of the ovary;inhaling respirable asbestos fibres in an open environment:(a) for a cumulative period of at least 3 000 [6 000 BOP] hours before the clinical onset of malignant neoplasm of the ovary; and(b) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and(c) where the first inhalation of respirable asbestos fibres occurred at least five [ten BOP] years before the clinical onset of malignant neoplasm of the ovary; | Malignant neoplasm of the ovary (9, 10/2018) |