Kerosene Engine Exhaust

Recommendations for the classification of mutagenicity and carcinogenicity and a health-based occupational exposure limit

To: the minister of Work and Participation
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Collaboration between the Dutch Expert Committee on Occupational Safety and the subcommittee on the Classification of Carcinogenic Substances, committees of the Health Council of the Netherlands, with the Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals

Health Council of the Netherlands

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Samenvatting

Kerosinemotoremissie (KME) is een complex mengsel van stoffen dat wordt uitgestoten bij de verbranding van kerosine door vliegtuigmotoren.
De samenstelling van de uitstoot wordt vooral beïnvloed door het type vliegtuigmotor, de brandstof en de omstandigheden (bijvoorbeeld het motorvermogen of stuwkracht van de vliegtuigmotoren en de weersomstandigheden). De gezondheidsrisico's van beroepsmatige blootstelling aan KME zijn beoordeeld door de commissie Gezondheid en beroepsmatige blootstelling aan stoffen (GBBS), de subcommissie voor de Classificatie van carcinogene stoffen van de Gezondheidsraad, en de Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals (NEG), hierna de commissies. De beoordelingen zijn uitgevoerd op verzoek van het Nederlandse ministerie van Sociale Zaken en Werkgelegenheid (SZW), en regelgevende overheden van Denemarken, Finland, Noorwegen en Zweden.

Classificatie en advieswaarde ter bescherming van werknemers
De commissies hebben als vaste taak om de gezondheidseffecten van blootstelling aan schadelijke stoffen en mengsels te beoordelen om werknemers te beschermen tegen nadelige gezondheidseffecten. Als er aanwijzingen zijn dat een stof of mengsel schadelijk is voor de gezondheid, doen de commissies een voorstel voor een classificatie in een gevarencategorie, of een aanbeveling voor een gezondheidskundige advieswaarde voor de stof of het mengsel. De overheden kunnen deze aanbevelingen voor een gezondheidskundige advieswaarde gebruiken als basis voor een wettelijke grenswaarde. Meer informatie over de commissies en hun taken is te vinden op gezondheidsraad.nl en nordicexpertgroup.org.

Dit adviesrapport bevat een voorstel voor de classificatie van KME, en een beoordeling van de toxiciteit en gezondheidsrisico's van KME als basis voor een gezondheidskundige advieswaarde.

KME-deeltjes kunnen na inademing diep in de longen terecht komen
KME bevat veel verschillende stoffen en stofgroepen, waaronder stofdeeltjes die bestaan uit elementair koolstof en organische koolstofverbindingen, polycyclische aromatische koolwaterstoffen (PAK's), metalen, zwaveloxiden en stikstofoxiden. KME wordt over het algemeen gekenmerkt door hoge concentraties zeer kleine deeltjes, die door verbranding worden gegenereerd. Deze ultrafijne deeltjes (ultrafijnstof) en de daaraan gebonden stoffen zoals PAK's kunnen na inademing diep in de longen terecht komen en de longblaasjes bereiken. Als de deeltjes in de longblaasjes komen, kunnen ze biologische membranen passeren, cellen binnendringen en vervolgens in de bloedsomloop komen.

Werknemers die werken in de directe omgeving van vliegtuigen kunnen worden blootgesteld aan KME, waaronder bagagemedewerkers, technici en personeel betrokken bij het slepen en terugduwen van taxiënde vliegtuigen. De hoogste concentraties KME-deeltjes in de lucht worden gemeten op het platform van vliegveld of luchthaven.

Blootstelling aan KME is in verband gebracht met verschillende gezondheidseffecten, zoals ontstekingen, vermindering van de longfunctie en verergering van longaandoeningen. Ook zijn verschillende componenten van KME geclassificeerd als bewezen, waarschijnlijke of mogelijke kankerverwekkende stoffen waarvoor soms ook wettelijke grenswaarden zijn vastgesteld. Dit geldt bijvoorbeeld voor sommige PAK's en metalen.

Analogiebenadering gebruikt voor classificatie

De commissies hebben de genotoxische en kankerverwekkende eigenschappen van KME beoordeeld op basis van de criteria van de CLP-regulering van de EU (*Classification, Labelling and Packaging of chemicals*). Op dit moment is er een beperkte hoeveelheid wetenschappelijke gegevens over (geno)toxische eigenschappen van KME. De beschikbare gegevens over KME zijn op zichzelf niet voldoende om als basis te dienen voor een classificatie. De commissies beschouwen dieselmotoremissie (DME) als een vergelijkbaar mengsel. Voor DME is wel een grote hoeveelheid wetenschappelijke gegevens beschikbaar. KME en DME zijn allebei complexe mengsels afkomstig van ruwe olie en de verbrandingsproducten bestaan uit vergelijkbare stoffen en stofgroepen, met vergelijkbare fysisch-chemische eigenschappen. Vanwege deze overeenkomsten zijn de commissies van oordeel dat de mengsels ook vergelijkbare toxicologische effecten hebben, waardoor een analogiebenadering gehanteerd kan worden. Hierbij wordt een classificatie voorgesteld op basis van gegevens over KME, aangevuld met gegevens over DME.

Classificeer KME voor mutageniteit in geslachtscellen

De commissies hebben beoordeeld of KME het genetisch materiaal in een cel kan beschadigen (genotoxiciteit). Er zijn geen gegevens over mutageniteit van KME in geslachtscellen (een maat voor genotoxiciteit) en onvoldoende gegevens over mutageniteit van DME in geslachtscellen. Er is beperkt bewijs voor genotoxiciteit van KME in somatisch weefsel op basis van literatuur over KME. Op basis van toxicologische onderzoeken naar DME, en de vergelijkbaarheid van DME met KME, kan worden geconcludeerd dat KME genotoxische effecten kan hebben in somatisch weefsel. De commissies adviseren om KME te classificeren in EU-gevarencategorie 2 voor mutageniteit (zie kader op pagina 7).

Classificeer KME voor carcinogeniteit

Er is beperkt bewijs voor de kankerverwekkende eigenschappen van KME op basis van onderzoek naar KME. Er is echter ruim voldoende bewijs uit epidemiologisch en toxicologisch onderzoek naar de kankerverwekkende eigenschappen van DME. Daarnaast bevatten zowel KME als DME

verschillende stoffen en stofgroepen waarvan bekend is dat ze kanker-verwekkende eigenschappen hebben. Ook is DME geclassificeerd als kankerverwekkend voor mensen (Groep 1) door de International Agency for Research on Cancer (IARC). Daarom adviseren de commissies om KME te classificeren in EU-gevarencategorie 1B voor kankerverwekkende stoffen (zie kader op pagina 7).

Gezondheidskundige advieswaarde kan niet worden afgeleid voor KME

Om blootstelling aan een mengsel zoals KME te meten, ten behoeve van het afleiden van een gezondheidskundige advieswaarde, is een specifieke en meetbare indicator van blootstelling nodig. Er is echter aanvullend onderzoek nodig om een geschikte indicator voor KME vast te stellen. Ook om een gezondheidskundige advieswaarde te kunnen afleiden is aanvullend wetenschappelijk bewijs nodig van de kwantitatieve relatie tussen blootstelling en gezondheidseffect(en); de blootstelling-respons-relatie. De commissies beschouwen de beperkte beschikbare gegevens als onvoldoende om een gezondheidskundige advieswaarde voor KME te kunnen afleiden. Een analogiebenadering op basis van DME kan hier niet worden toegepast, omdat niet duidelijk is of de kwantitatieve blootstelling-responsrelatie voor DME, die gebaseerd is op elementair koolstof, kan worden toegepast op KME. Hoewel er kwalitatief bewijs is voor de aan-wezigheid van elementair koolstof in KME-deeltjes, zijn de kwantitatieve hoeveelheden hiervan vooralsnog onvoldoende vastgesteld in relatie tot de mogelijke gezondheidseffecten.

Pas bestaande grenswaarden toe

Ondanks dat er geen gezondheidskundige advieswaarde kan worden afgeleid voor KME zijn er aanwijzingen dat beroepsmatige blootstelling aan KME kan leiden tot verschillende gezondheidseffecten. Daarnaast zijn er veel overeenkomsten tussen KME en DME. DME is een vergelijkbaar mengsel dat geclassificeerd is als kankerverwekkend voor mensen. Bovendien zijn verschillende stoffen en stofgroepen in KME bewezen, waarschijnlijke of mogelijke kankerverwekkende stoffen, en voor een aantal van deze stoffen en stofgroepen zijn ook wettelijke grenswaarden afgeleid. Dit geeft reden tot zorg voor beroepsmatige blootstelling aan KME. Daarom adviseren de commissies om in ieder geval bestaande grenswaarden zoals voor dieselmotoremissies, maar ook voor componen-ten van KME, zoals voor PAK's, metalen en andere stoffen, toe te passen om werknemers te beschermen. Het is echter onduidelijk of deze bestaan-de grenswaarden voldoende bescherming bieden tegen de mogelijk schadelijke effecten van beroepsmatige blootstelling aan het totale mengsel van KME-componenten. Daarom adviseren de commissies om aanvullend onderzoek te doen naar de blootstellingsresponsrelatie voor KME, zodat een gezondheidskundige advieswaarde voor KME kan worden afgeleid.

Meer onderzoek nodig

Om een gezondheidskundige advieswaarde af te leiden zijn goed opgezette en uitgevoerde epidemiologische onderzoeken nodig met

voldoende follow-up, gedegen karakterisering van blootstelling (waarvoor een geschikte indicator nodig is) en gezondheidseffecten, en relevante informatie over mogelijke factoren die de relatie tussen blootstelling en gezondheidseffect kunnen beïnvloeden (confounders). Ook adviseren de commissies onderzoek te doen waarin KME en DME vergeleken worden, waarbij onder andere het gehalte aan elementair koolstof wordt gekwantificeerd. De resultaten uit deze onderzoeken kunnen het afleiden van een gezondheidskundige advieswaarde mogelijk maken en/of de toepasbaarheid van de grenswaarde van DME op KME wetenschappelijk onderbouwen.

Classificatie mutagene en kankerverwekkende stoffen

In classificatievoorstellen gebruikt de Gezondheidsraad een indeling in gevarencategorieën. De categorieën zijn afgeleid van EU-verordening (EG) 1272/2008 en geven aan hoe sterk de bewijskracht is voor schadelijke effecten. Bij een gevarencategorie hoort ook een EU-gevarenaanduiding, die op verpakkingen kan worden gebruikt.

EU-gevarencategorieën voor mutageniteit in geslachtscellen

Categorie 1A	Stoffen waarvan bekend is dat ze erfelijke mutaties in de geslachtscellen van mensen veroorzaken (EU-gevarenaanduiding H340).
Categorie 1B	Stoffen waarvan verondersteld wordt dat ze erfelijke mutaties in de geslachtscellen van mensen veroorzaken (H340).
Categorie 2	Verdacht van het veroorzaken van erfelijke mutaties in de geslachtscellen van mensen (H341).

EU-gevarencategorieën voor kankerverwekkende stoffen

Categorie 1A	Stoffen waarvan bekend is dat ze kankerverwekkend zijn voor mensen (H350).
Categorie 1B	Stoffen waarvan verondersteld wordt dat ze kankerverwekkend zijn voor mensen (H350).
Categorie 2	Verdacht van het veroorzaken van kanker bij mensen (H351)

Betekenis voor de werkvloer

Werkgevers zijn op grond van de Nederlandse Arbowet wettelijk verplicht om gezondheids- en veiligheidsrisico's van het werken met stoffen zoveel mogelijk te voorkomen of te beperken. Op basis van de classificatievoorstellen van de Gezondheidsraad kan de staatssecretaris van SZW besluiten stoffen op te nemen in de officiële lijst van kankerverwekkende, mutagene en voor de voortplanting giftige stoffen (CMR-stoffen). Op die lijst staan kankerverwekkende en mutagene stoffen in categorie 1A en 1B en voor de voortplanting giftige stoffen in categorie 1A, 1B en 2. Afhankelijk van de classificatie vraagt de wetgever de werkgever aanvullende maatregelen te nemen om de werknemer te beschermen.

In Scandinavische landen is vergelijkbare wet- en regelgeving voor deze CMR-stoffen van kracht. Werkgevers zijn daardoor verplicht om gezondheids- en veiligheidsrisico's van werken met CMR-stoffen zoveel mogelijk te voorkomen (indien mogelijk moeten deze stoffen worden vervangen) of te beperken.

Executive summary

Kerosene engine exhaust (KEE) is a complex mixture that is emitted due to the combustion of kerosene jet fuel by aircraft engines. The composition and concentration of the combustion products of KEE are mainly influenced by the type of aircraft engine, the type of fuel and varying conditions, such as power settings and weather conditions. The health risks of occupational exposure to KEE were evaluated by the Dutch Expert Committee on Occupational Safety (DECOS), the Dutch subcommittee on the Classification of Carcinogenic Substances of the Health Council of the Netherlands, and the Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals (NEG). The hazard evaluation was carried out at the request of the Dutch Ministry of Social Affairs and Employment, and the regulatory authorities of Denmark, Finland, Norway and Sweden.

Evaluation of hazardous substances and mixtures to protect workers

The committees have the permanent task of carrying out hazard evaluations to protect workers against negative health effects due to their work. When there are indications that a substance or a mixture is a hazard, the committees propose a classification in an EU-hazard category and decide on a recommendation for a health-based occupational exposure limit (hb-OEL) for the substance or mixture. The national authorities can use these recommendations as a basis for setting legally binding occupational exposure limits (OELs). More information about the committees and their tasks can be found on healthcouncil.nl and nordicexpertgroup.org.

This advisory report contains an evaluation of the genotoxicity and carcinogenicity of KEE, and an evaluation of the toxicity and health effects as a basis for setting a hb-OEL for KEE.

KEE particles can penetrate deep into the lungs

KEE contains numerous components, such as carbonaceous particles (containing elemental carbon and organic carbon), polycyclic aromatic hydrocarbons (PAHs), metals, sulphur oxides and nitrogen oxides. KEE is generally characterised by high concentrations of very small (ultrafine) combustion-generated particles. These ultrafine particles (UFP), and particle-bound substances like PAHs, can penetrate deep into the lungs when inhaled, reaching the lung alveoli. If deposited, these UFP can pass biological membranes, enter cells and the systemic blood circulation.

Workers with activities related to operating kerosene-fuelled aircrafts are exposed to KEE, for example baggage handlers, flight officers, fuel operators, mechanics, engineers and personnel responsible for catering or towing/pushback. The highest concentrations of KEE particles in air have been reported at the apron of airfield and airport.

Exposure to KEE has been linked to several adverse health effects, such as inflammation, reduction of lung function, and worsening of respiratory diseases. In addition, several of the components of KEE are known, probable, or suspected carcinogens and/or have legally binding OELs. These components include some of the PAHs and metals.

Analogy approach used for classification

The committees evaluated the genotoxic and carcinogenic properties of KEE by using the criteria based on the CLP Regulation (Classification, Labelling and Packaging of chemicals). Currently, there is limited scientific data on KEE. The available data for KEE are (on itself) not sufficient to serve as a basis for a classification. However, the committees consider diesel engine exhaust (DEE) to be a substantially similar mixture to KEE. For DEE a large amount of data is available. Both KEE and DEE are complex mixtures derived from the combustion of crude oil, have a (relatively) similar composition of combustion products and similar physicochemical properties. Because of these similarities, the committees infer that they also have similar toxicological effects, which allows for an analogy approach. This means proposing a classification for KEE based on data on KEE supplemented with data on DEE.

Classify KEE for germ cell mutagenicity

The committees evaluated whether KEE can cause genotoxicity. There is no data on germ cell mutagenicity (a measure for genotoxicity) for KEE and insufficient data on germ cell mutagenicity for DEE. There is limited evidence for genotoxic properties of KEE in somatic tissues. Evidence from toxicological studies on DEE supports that KEE has genotoxic potential in somatic tissues. Because there are no strong indications for mutagenicity in germ cells, the committees recommend classification for germ cell mutagenicity of KEE in EU-hazard category 2 (see text box on page 11).

Classify KEE for carcinogenicity

The committees evaluated whether KEE can cause carcinogenicity. There is limited evidence for the carcinogenic properties of KEE, but there is ample evidence from epidemiological and toxicological studies on DEE. In addition, KEE and DEE both contain several known carcinogenic components, and DEE has been classified as carcinogenic to humans (Group 1) by the International Agency for Research on Cancer (IARC). Based on the limited evidence for KEE and the analogy with DEE, the committees recommend a classification for carcinogenicity of KEE in EU-hazard category 1B, presumed (or probably) carcinogenic to humans (see text box on page 11).

Health-based OEL for KEE could not be derived

For the (quantitative) exposure assessment of KEE, for the benefit of establishing a hb-OEL, a selective and measurable indicator of exposure is needed. However, no suitable indicator for KEE could be identified. Additional research is needed to establish a suitable indicator for KEE.

To recommend a hb-OEL, the committees also require quantitative exposure response data. The committees consider the available human data as insufficient to derive a hb-OEL for KEE. In this case, an analogy with DEE was not deemed feasible, because it is uncertain whether the quantitative exposure-response relationship for DEE, which is based on elemental carbon, can be applied to KEE. Moreover, although there is evidence for the presence of elemental carbon in KEE particles, the concentrations of elemental carbon in KEE particles have not yet been established in relation to potential adverse health effects.

Apply existing OELs

Although a hb-OEL for KEE could not be derived, the available data for KEE indicate that occupational exposure can lead to several health effects. Furthermore, KEE shows similarities with DEE, which has been classified as carcinogenic to humans. Also, several components of KEE are known, probable or suspected carcinogens, and for some of these components of KEE, OELs have been established. This gives reason for concern. Therefore, the committees recommend that existing occupational exposure limits for DEE as well as for components of KEE, such as PAHs, metals and others, be applied for KEE to protect workers with activities related to operating kerosene-fuelled aircrafts. However, it is uncertain whether existing OELs are sufficient to protect these workers against potential health effects of the entire KEE mixture. Therefore, the committees also recommend further research to establish an exposure response relationship based on data for KEE, to allow for the derivation of a hb-OEL for KEE.

More research needed

To establish a hb-OEL, high quality epidemiological studies, such as occupational cohort or case-control studies, are needed with sufficient follow-up time, thorough exposure (which requires a suitable indicator for KEE) and health assessment and relevant information on potential confounders. Such further research should also include studies comparing DEE and KEE, including studies particularly focussing on particles and (quantitative) elemental carbon content. Together, these findings could support the derivation of a hb-OEL for KEE and/or scientifically demonstrate the applicability of the OEL for DEE to KEE.

Classification for mutagenicity and carcinogenicity

The Health Council performs classification and labelling of substances according to the guidelines of the European Union (Regulation (EC) 1272/2008). The hazard categories described below indicate the strength of the evidence for hazardous properties of the substance. The substance is labelled using an EU Hazard statement code that can be used on packaging.

EU hazard categories for mutagenicity in germ cells

Category 1A	Known to induce heritable mutations in human germ cells (EU Hazard statement H340)
Category 1B	Presumed to induce heritable mutations in human germ cells (H340)
Category 2	Suspected to induce heritable mutations in human germ cells (H341)

EU hazard categories for carcinogenicity

Category 1A	Known to be carcinogenic to humans (H350)
Category 1B	Presumed to be carcinogenic to humans (H350)
Category 2	Suspected to be carcinogenic to humans (H351)

Implications for the workplace

According to the Dutch Working Conditions Act, employers are legally required to prevent or minimize the health and safety risks of working with hazardous substances as much as possible. Based on the Health Council's recommendations for classification, the State Secretary of Social Affairs and Employment can decide to add substances to the official list of substances that are carcinogenic, mutagenic or toxic to reproduction (CMR-substances). This list includes carcinogenic and mutagenic substances in categories 1A and 1B, and substances toxic to reproduction in categories 1A, 1B and 2. Depending on the classification, the government asks the employer to take additional measures to protect employees.

Nordic countries have similar regulations for CMR-substances, employers are required to prevent (by replacing CMR-substances) or to minimize the health and safety risks of working with hazardous substances as much as possible.

1 Scope

1.1 Background

The current evaluation of kerosene engine exhaust (KEE) is a collaboration between the Dutch Expert Committee on Occupational Safety (DECOS), the Dutch subcommittee on the Classification of Carcinogenic Substances, and the Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals (NEG). In the Netherlands a policy is in force with respect to occupational use and exposure to carcinogenic substances. Regarding this policy, the Minister of Social Affairs and Employment has asked the sub-committee on the Classification of Carcinogenic Substances, a permanent committee of the Health Council of the Netherlands, to evaluate the carcinogenic and genotoxic properties of substances (e.g., individual substances, mixtures or emissions), their carcinogenic mechanism and to propose a classification for germ cell mutagenicity and carcinogenicity. In addition, DECOS, also a permanent committee of the Health Council of the Netherlands, has been asked to perform overall scientific evaluations on the toxicity of chemical substances that are used in the workplace. Similarly, NEG performs evaluations at the request of the regulatory authorities of Denmark, Finland, Norway and Sweden.

The purpose of these scientific evaluations is to derive a health-based occupational exposure limit (hb-OEL) for the concentration of a substance in the air, provided that the available data allow the derivation of such a value. These recommendations serve as a basis in setting final, legally binding OELs by the Dutch and Nordic authorities.

This advisory report contains both an evaluation of the genotoxicity and carcinogenicity of kerosene-fuelled aircraft engine exhaust, in this report referred to as KEE, as well as an evaluation of the toxicity and health hazards as basis for a hb-OEL. Aircraft-related kerosene engine exhaust is a complex mixture of substances, including soot, polycyclic aromatic hydrocarbons (PAHs), metals, sulphur oxides, and nitrogen oxides.[1-3] Several of these substances have been classified as mutagenic and/or carcinogenic to humans by CLP, or carcinogenic to humans by IARC (Group 1), and/or have a legally binding OEL, see also Annex C of this advisory report.

1.2 Scope and definition

This advisory report focuses on occupational exposure to KEE from aircraft specifically. This report addresses the occupational exposure of airport and (military) airfield workers (e.g., baggage handlers, flight officers, fuel operators, mechanics, engineers, and personnel working in catering, towing/pushback) with activities related to operating kerosene-fuelled aircraft (including military aircraft, and helicopters operating on kerosene), usually on the apron of airports and (military) airfields. The definition of KEE

includes emissions from the combustion of kerosene jet fuel and lubricating oil used in aircraft engines (all types of aircraft engines operating on kerosene), and the kerosene-fuelled on-board auxiliary power units (APUs). Currently, approximately 99% of the global jet fuel use consists of kerosene jet fuel.[4] As a result, aircraft operating on aviation gasoline (AvGas) or other forms of jet fuel which do not predominantly contain kerosene (e.g., 100% bio-jet fuels) do not fall under the scope of this advisory report. Occupational exposures to cabin air (e.g., pilots and cabin crew during aircraft flights) are also excluded, because cabin air also contains substances not related to combustion of kerosene by aircraft engines. Furthermore, emissions from ground support equipment (GSE), which generally operate on diesel fuel, and emissions from oil leaks, tire, brake wear and runway surface wear, are outside of the scope of this advisory report.

Thus, the committees define KEE as a mixture of compounds resulting from the combustion of kerosene jet fuel by the main aircraft engine and the on-board APUs.

Kerosene jet fuel is derived from crude oil and distils between diesel and gasoline fractions. This increases the likelihood that the combustion products of diesel and gasoline consist of a similar complex mixture of substances which have similar physicochemical and structural properties. This observation may be relevant for the classification of carcinogenicity for KEE, because diesel engine exhaust (DEE) is classified as carcinogenic to humans by IARC (Group 1), and gasoline engine exhaust is classified as possibly carcinogenic to humans (Group 2B),[5] whereas KEE is not (yet) classified for carcinogenicity. In addition, DECOS has derived cancer risk values for DEE (based on respirable elemental carbon (REC)), which formed the basis for the current Dutch OEL.[6, 7] The Nordic countries have established legally binding OELs for DEE (based on elemental carbon (EC)) as well.[8-11]

1.3 Committees and procedures

This evaluation is a collaboration of DECOS, the Dutch subcommittee on the Classification of Carcinogenic Substances and NEG, hereafter referred to as the committees. There is an agreement between NEG and DECOS to collaborate in the evaluation and recommendation of hb-OELs if a chemical substance is on the work programme of both committees. The committees have the permanent task of giving scientific advice to help protect workers against the potentially harmful effects of chemical substances they may encounter in the workplace. The committees assess the toxic properties and health effects of these substances. The Dutch and Nordic regulatory authorities can use the joint advisory reports as a basis for setting policy for hazard classifications and legally binding OELs. Additional information on the tasks of the committees can be found at www.healthcouncil.nl and www.nordicexpertgroup.org. A list of the members of the committees can be found on the last pages of this advisory report.

This advisory report aims to recommend a classification for germ cell mutagenicity and carcinogenicity as well as a hb-OEL. This means that the usual outline of this advisory report is adapted and includes chapters and data that specifically addresses the classification for germ cell mutagenicity and carcinogenicity and the scientific evaluation of health hazards as basis for a hb-OEL for KEE.

For the classification, all the available individual studies on genotoxicity and carcinogenicity of KEE have been evaluated. The committees' considerations for determining the quality and contribution to the evidence base of a study can be found in the *Guideline for the classification of carcinogenic substances* (2023).[12] The criteria for the classification in EU-categories are based on the Globally Harmonized System (GHS), which has been incorporated into the system and guideline used by the European Union (Regulation (European Committee) No 1272/2008) for the classification, labelling, and packaging of substances and mixtures (the CLP regulation).[13,14]

For the scientific evaluation of the toxicity and health hazards, the committees refer to the *Guidance for recommending classifications and health-based occupational exposure limits* (2025)[15] for information on the general principles and advisory process.

The committees aim to evaluate and classify KEE as a mixture of numerous substances. In their hazard evaluation, the committees also consider the presence of well-known carcinogenic components in KEE and the potential similarities in composition between KEE, DEE and gasoline engine exhaust (e.g., analogy approach).

In January 2026, the chair of the Health Council released a draft of this advisory report for public review. The committees have taken the comments into account in deciding on the final version of this advisory report. The comments and replies can be found on healthcouncil.nl.

1.4 Data and literature

The evaluation and recommendations are based on scientific data, which are publicly available. The starting point of the classification reports are, if possible, the IARC Monographs. This means that the original sources of the studies, which are mentioned in the IARC Monograph, are evaluated only if these are considered most relevant in assessing the carcinogenicity and genotoxicity of the substance in question. In the case of KEE, such an IARC Monograph is *not* available. However, an IARC Monograph on diesel and gasoline engine exhaust is available,[5,16,17] which provides relevant information on corresponding combustion products of KEE.

In addition, a literature search of scientific papers was performed using the online databases PubMed, SCOPUS and EMBASE, with variations of the

following key words: kerosene exhaust emissions, aircraft exhaust, jet exhaust, jet fuel, kerosene, toxicity, occupational exposure, adverse health effects, dose-response relationship, hazard assessment, risk assessment, acute toxicity, chronic toxicity, pulmonary effects, cardiovascular disease, immune effects, haematological effects, genotoxicity, mutagenicity, carcinogenicity, tumorigenesis, cytotoxicity, DNA damage, chromosomal aberration(s), sister chromatid exchange, cell transformation, cancer mortality. Individual studies on mutagenicity and carcinogenicity are summarised in tables in Annex D of this advisory report. The last update of the literature search was performed in November 2025. The literature database for KEE has been extended with relevant publications cited by other selected relevant publications which were not identified during the initial literature search.

In addition, other sources of literature (grey literature) were used for the evaluation, for instance reports by the National Institute for Public Health and the Environment (RIVM), the Netherlands Organisation for Applied Scientific Research (TNO), the Agency for Toxic Substances and Disease Registry (ATSDR), the World Health Organization (WHO), the Federal Aviation Agency (FAA), the Intergovernmental Panel on Climate Change (IPCC) and several others. For more details on the literature search, see Annex B.

2 Characteristics

Emissions from the combustion of kerosene jet fuel by aircraft engines are generally recognised as a major source of occupational exposure and local air pollution at airports and (military) airfields. The composition of KEE is mainly determined by the type of aircraft engine (including operating conditions) and the composition of kerosene jet fuel. The focus in this chapter will be on these two factors and on the combustion products. Also, the composition of closely related petroleum-fuelled exhaust emissions is explored for similarities. Similarities in physicochemical and structural properties may provide additional or substitutional information for KEE.

2.1 Aircraft engines

The operating conditions (e.g., power or thrust settings) of aircraft engines form one of the main determinants of the composition of KEE. It should be noted that aircraft engines (particularly for passenger and cargo flights) are specifically designed for high performance while cruising at high altitudes. As a result, some aircraft operations within airports require that engines operate outside of their optimal regimes, for example maximum thrust during take-off or low power settings during operations on the ground. This may result in potentially higher emissions on the ground than during cruising, especially for those pollutants mainly emitted at lower power settings, such as carbon monoxide (CO) and hydrocarbons (HC).[1,18]

In general, five types of aircraft engines can be distinguished: turbojet, turbofan, turboprop, turboshaft and piston engines. A turbojet is composed of an inlet compressor, a combustion chamber adding and igniting fuel, and one or more turbines extracting energy from the exhaust gas in expansion and driving of the compressor. Turbofan engines use a turbojet as a core to produce energy for thrust and for driving a large fan placed in front of the compressor. Some small and regional airliners use aircraft with a turboprop engine, which use a turbine engine core fitted with a reduction gear to power propellers. Most modern helicopters are equipped with a turboshaft engine, which operates like a turbojet engine, but is optimised to generate shaft power instead of jet thrust.[1]

Piston engines are predominantly fitted in small-sized aircraft typically related to private use, flying clubs, flight training, crop spraying and tourism. The internal piston engine runs under the same basic principles as spark ignition engines for gasoline-fuelled cars but generally requires a higher performance. Aircraft with a piston engine generally operate on AvGas (tetraethyl lead may be added as antiknock additive), which is distilled separately from the most common motor gasoline.[1]

Emission regulation

For turbofan and turbojet engines with a maximum rated thrust at sea level greater than 26.7 kiloNewton (kN), typically used in commercial passenger and cargo aircraft, the International Civil Aviation Organization (ICAO)

regulates aircraft emissions by application of regulation standards. These standards intend to limit aircraft engine exhaust emissions (e.g., to reduce their general impact on the environment) during landing-and-take-off (LTO) operations. There are emission standards for non-volatile particulate matter (nvPM; i.e. particles directly emitted by aircraft engines, which are stable at temperatures and pressures reached in the exhaust plume), mass and number (since January 2023), unburnt hydrocarbons (UHC), CO and nitrogen oxides (NO_x) for new and in-production turbojet and turbofan aircraft engines. The certification process involves running the engine on a test bed at each thrust setting defined by the standard ICAO LTO cycle (100% thrust for take-off, 85% thrust for climb, 30% thrust for approach, and 7% thrust for taxi/ground idle (the lowest engine speed setting on the ground)). Emissions during the flight itself (cruise conditions), are therefore not regulated. This certification process results in an emission index (EI) for NO_x, UHC and CO (mass of emissions per kg fuel), the measured nvPM mass concentration, nvPM mass and nvPM number (total mass per kN and total nvPM number per kN). These values allow for the calculation of emission data for each of these pollutants.[19-22]

The ICAO LTO cycle and the emission indices ensuing from that are approximations and do not always represent the actual operations and emissions at airports. Also, other aircraft engine types such as turboprop, turboshaft, small turbofan engines with rated thrust below 26.7 kN (often used as smaller business jets or private jets), military turbofan engines, and APUs are not regulated by ICAO, and the publicly available emission data for these engine types is more limited. However, it should be noted that emissions from non-regulated engine types are also significantly lower than from regulated engine types.[1,19]

For the occupational exposure of airport workers, the emissions during taxi/ground idle are most relevant.

2.2 Fossil fuels

Kerosene jet fuels are extracted from the middle distillates of crude oil (kerosene fraction), which distils between the gasoline (including AvGas) and the diesel fractions (see Figure 1).[1,2,23]

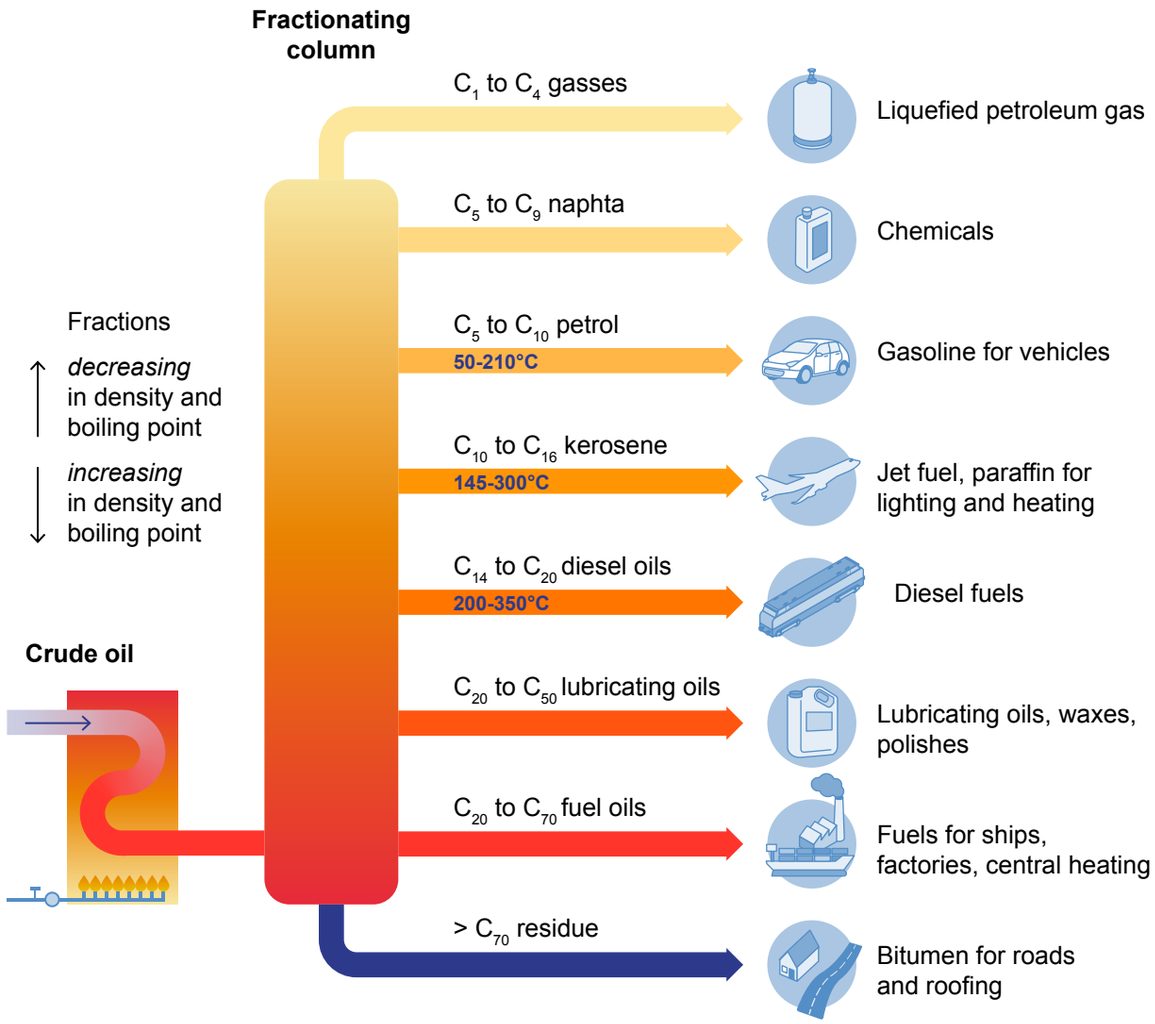


Figure 1. Schematic overview of refinery processes of jet fuels and other crude oil distillates. Adapted from ATSDR (2017)[23]

2.2.1 Kerosene jet fuels

In 2023, ~99% of global jet fuel use consisted of kerosene jet fuels, and ~0.2% consisted of sustainable aviation fuel (SAF) (see Chapter 2.4).

In 2025, the International Air Transport Association (IATA) expects ~0.7% of global jet fuel use to consist of SAF.[4,22]

The exact composition of kerosene jet fuel depends on the crude oil from which it is derived and on the refinery process used for its production.

Kerosene jet fuels consist of C9 to C16 hydrocarbons that boil in the range of 145-300°C.[23]

Kerosene jet fuels consist predominantly of branched and linear alkanes (paraffins) and naphthenes (cycloalkanes), which usually account for 70 to 80% of the components by volume. Aromatic compounds, such as alkylbenzenes and naphthalenes, account for 20 to 25% of the total composition. Alkenes (olefins) represent a minor fraction (<1%) of the total composition of kerosene jet fuels (see Figure 2).[23,24] The sulphur (S) content in kerosene jet fuels is limited to 0.3%. However, most kerosene jet fuels currently have a sulphur content well below this limit, averaging around 0.05% globally.[1,25,26] Trace amounts (up to 0.002%) of nitrogen may be present in kerosene jet fuel.[1] The differences between various kerosene jet fuels are related to physical properties and the inclusion of additives to enhance performance.[23,24]

For civil aviation 2 main types of kerosene jet fuels can be distinguished, Jet A and Jet A-1 (see Table 1). Jet A is predominantly used in continental

flights in the United States (US) while Jet A-1 is used throughout the rest of the world. These fuels are nearly identical, although Jet A-1 is refined to have a lower freezing point (-47°C) compared to Jet A (-40°C). The lower freezing point makes Jet A-1 a better choice for international flights, especially on polar routes during the winter season. In addition, Jet A typically does not contain a static dissipator additive that is required for Jet A-1 fuels.[1,23,24,27] In very cold climates (e.g., northern Canada), Jet B is used, which is a wide-cut type jet fuel, covering both naphtha and kerosene fractions of crude oil. Due to the thermodynamic characteristics of Jet B (e.g., lower freeze point and higher volatility), it is very suitable for handling and cold starting in these very cold climates. In Russia and former USSR states, TS-1 is mainly used for civil aviation, a kerosene jet fuel with slightly higher volatility and lower freeze point compared to Jet A and Jet A-1 fuels. In China, RP-3 is the main kerosene jet fuel for both civil and military aviation. RP-3 is similar to Jet A-1, whereas Chinese RP-4 is comparable to Jet B and Chinese RP-1 and RP-2 are comparable to Russian TS-1.[1,27]

Aviation fuels are subject to strict compositional requirements beyond those required for road transport fuels. Jet fuels must reach performance benchmarks that meet the operational and safety requirements of existing jet engines. Jet fuel specifications and test methods for certification of jet fuels is governed by the ASTM International (formerly known as American Society for Testing and Materials) and the British Ministry of Defence (UK-MoD).[1,23,24,28] Kerosene jet fuels for military purposes are formulated for higher performances and are regulated separately by several governments. The US military and North Atlantic Treaty Organization (NATO) use two types of kerosene jet fuels, Jet Propellent-5 (JP-5) and Jet-Propellent-8 (JP-8) (see also Table 1). JP-8 is the military equivalent of Jet A-1, although it contains a corrosion inhibitor and anti-icing additive that is not required in the ASTM specification of Jet A-1.[1,23,24,27] JP-8 may also contain thermal stability additives, since military aircraft engines generally have a greater need for thermal stability compared to commercial aircraft engines.[23,24] The primary difference between the two types of military jet fuels is that the flash point temperature for JP-5 is higher (60°C) as compared to JP-8 (38°C). The higher flash point for JP-5 is more suitable for safe handling and fuelling practices aboard aircraft carriers; therefore, is the primary jet fuel used by the US Navy.[23]

Table 1 General characteristics of commonly used jet fuels

Characteristic	Jet A / Jet A-1	JP-5	JP-8	Remarks
Freezing point (maximum)	-40°C (Jet A) -47°C (Jet A-1)	-46°C	-52°C	
Boiling range	145-300°C	150-290°C	150-290°C	
Density at 15°C	0.775-0.840 kg/L	0.788-0.845 kg/L	0.775-0.840 kg/L	
Solubility in water at 20°C	10.4 mg/L	-	12.44 mg/L (unspecified temperature)	ATSDR also reports ~5 mg/L for Jet A/ Jet A-1, JP-5 and JP-8
Solubility in organic solvents	Miscible with other petroleum solvents	Miscible with other petroleum solvents	Miscible with other petroleum solvents	
Vapour pressure at 21°C	>7.5 mm Hg at 37.8°C	2.25-25.1 mm Hg	2.25-25.1 mm Hg	
Flashpoint (minimum)	38°C	60°C	38°C	
Additives*	Antioxidant (hydro-processed Jet A-1); static dissipator	Antioxidant (hydroprocessed JP-5); corrosion inhibitor/ lubricity improver; fuel system icing inhibitor	Antioxidant (hydroprocessed JP-8); static dissipator; corrosion inhibitor/ lubricity improver; fuel system icing inhibitor	Only those additives specifically approved (along with allowed concentrations) may be added to jet fuel
Comparable to	RP-3 (China), TS-1 (Russia)			TS-1 has a slightly lower freezing point (-50°C) and higher volatility

* Only required additives are mentioned in this table
Abbreviation: ATSDR, Agency for Toxic Substances and Disease Registry.
Sources: ATSDR (2017)[23]; Chevron (2007)[24]; Masiol & Harrison (2014)[1]

2.2.2 Comparison with diesel and gasoline fuels

Diesel fuel generally consists of C12 to C20 hydrocarbons that boil in the range of 200-350°C.[1,23,29,30] Diesel fuel is a complex mixture of branched and normal cyclic alkanes (paraffins), which range from 65% to 85% of the components by volume; the remaining 5% to 30% consists mainly of aromatic compounds (especially alkylbenzenes), and small amounts of alkenes (which account for <10% of the total components by volume) (see also Figure 2). Benzene, toluene, ethylbenzene, and xylenes (generally referred to as BTEX) and PAHs, especially naphthalene, may be present at levels of parts per million in diesel fuels. The sulphur content of diesel fuels may depend on the source of crude oil and the refinery process, but it is currently on average 0.001% (regulated according to EU Directive 2023/2413). The sulphur content of diesel fuels used to be higher as it was regulated at 0.2% sulphur in the first emission regulation (EN590:1993).[5,25,26,31] The composition of diesel fuel influences the emissions of pollutants from diesel engines considerably. Kerosene jet fuels generally have a qualitatively similar composition compared with 'older' diesel fuels, although with different additives (see also Table 2).[16,30]

Gasoline fuel generally consists of C4 to C12 hydrocarbons that boil in the range of 50-210°C. Gasoline fuel consists mainly of alkanes (paraffins) ~49%, aromatic compounds (particularly alkylbenzenes) ~47.5%, and smaller amounts of alkenes (olefins) ~3.5% (see also Figure 2). Gasoline may contain butane, pentane, isopentane, benzene, toluene, ethylbenzene, and xylene.[16,32]

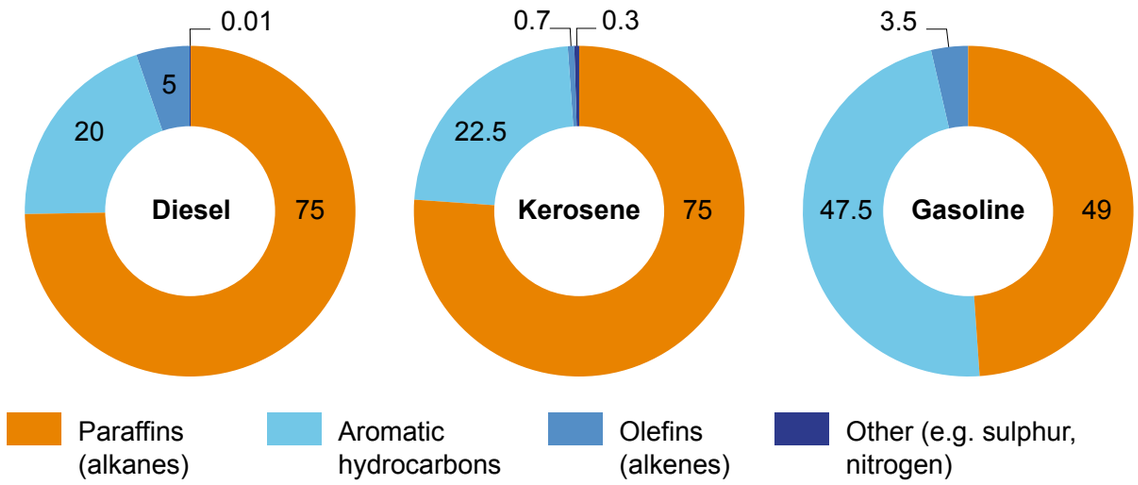


Figure 2. Overview of average composition of kerosene, 'older' diesel and gasoline fuels

Sources: ATSDR (2017)[23]; Chevron (2007, 2009)[24,32] IARC (1989, 2014)[5,16]; WHO (1996)[30]

2.2.3 Conclusion on fossil fuels

As is shown in Figure 2, kerosene jet fuel and diesel fuel (data on composition of 'older' diesel fuel (before introduction of EU emission regulations in 1993) used for comparison in this advisory report unless specified otherwise) show strong similarities in composition. As kerosene and diesel fuel are both derived from crude oil and have on average a comparable chemical composition, it is likely that their combustion products are also similar. Gasoline fuel contains generally similar compounds, however, the relative amounts in which these compounds occur differ from the composition of kerosene and diesel fuel. Gasoline contains relatively more (volatile) aromatic compounds and far less alkanes.

The combustion products of gasoline engine exhaust also contain remarkedly less particles and high amounts of carbon monoxide compared to diesel engine exhaust. Gasoline fuel and gasoline engine exhaust will therefore not be further considered in this advisory report.

Some of the chemical and physical properties of kerosene and diesel fuels are listed in Table 2. There is overlap in the number of carbon atoms, boiling range, density and solubility in water at room temperature between commonly used Jet A and Jet A-1 and the composition of 'older' diesel fuel. This is also shown in the average composition of the fuels, which is quite similar.

Additives are used in varying degrees in all petroleum derived fuels (e.g., kerosene jet fuels, diesel fuels), but only specifically approved additives may be added to kerosene jet fuels. In section 2.2.1 it was noted that the main difference in composition between various kerosene jet fuels lies in their additive content. This is particularly true for military jet fuels, JP-5 and JP-8 (see Table 1). Jet A generally contains no additives, although antioxidants, metal deactivators and static dissipators are allowed. For Jet A-1 a static dissipator is required and antioxidants are required only if the fuel is hydroprocessed. Diesel fuels generally contain several additives (see also Table 2)[30] which are not as strictly regulated as kerosene jet fuels. Remains of additives in fossil fuels may end up in the exhaust, particularly remains of metals with high boiling points.

Table 2 Chemical and physical properties of commonly used kerosene and diesel fuels (older diesel fuels)

	Kerosene (Jet A / Jet A-1)	Diesel
Carbon atoms	C9-C16	C12-C20
Cetane number	Not specified, so may contain a larger proportion of hydrocarbons	Minimum 45-49, usually 49-53 (in Europe) High cetane number improves cold starting, engine durability, reduces noise, fuel consumption and exhaust emissions
Boiling range	145-300°C	Generally 200-350°C, larger boiling ranges of 143-384 °C have been reported
Density at 15°C	0.775-0.840 kg/L	0.820-0.845 kg/L
Solubility in water at 20°C	10.4 mg/L ~5 mg/L (other data source)	~5 mg/L
Chemical composition fuel		
	Kerosene (Jet A / Jet A-1)	Diesel
Alkanes (paraffins)	Branched and linear 70-80%	Branched and cyclic 65-85%
Aromatic compounds	20-25% BTEX: trace amounts PAH content consists mostly of naphthalene	5-30% BTEX: trace amounts PAH content varies widely, total PAH <5% by volume, consists mostly of naphthalene
Alkenes (olefins)	<1%	Maximum 10%, probably much lower
Sulphur content	Sulphur: 0.05%-0.3% (currently regulated at 0.3%)	Sulphur: 0.05%-0.5% (currently regulated at 0.001%, in 1993 regulated at 0.2%)
Additives	Antioxidant (required for hydroprocessed Jet-A1), static dissipator (Jet-A1).	Cetane number improvers, cold-flow improvers, detergents, antioxidants, lubricity improvers, corrosion inhibitors, anti-foam agents, anti-emulsion agents, biocides.

Sources: ATSDR (2017)[23]; Chevron (2007)[24]; IARC (1989 & 2014)[5,33]; IPCC (1999)[25]; Li et al. (2009)[34]; Masiol & Harrison (2014)[1]; WHO (1996)[30]

2.3 Combustion products

The combustion of fossil fuels results in a complex mixture of numerous organic and inorganic substances distributed over gas-phase and particle-phase.

2.3.1 Composition of combustion products

Combustion of fossil fuels, such as kerosene jet fuel and diesel fuel, mainly consists of nitrogen (N_2), oxygen (O_2), carbon dioxide (CO_2) and water vapour (presence of N_2 and O_2 results from intake air passing through the engine for cooling). The proportions of CO_2 and water vapour (H_2O) depend on the specific carbon-hydrogen ratio of the fuel.[1,25]

The composition of the combustion products is influenced by the type of engine, the type of fuel, the operating conditions (e.g., thrust levels, weather conditions), and the composition of lubricating oil. This section describes the average composition of the combustion products of kerosene jet fuel by aircraft and diesel fuel for road transportation., it should be noted that quantitative data on emissions of various engine types, both aircraft engines and diesel engines, is relatively sparse and largely based on older data (e.g., 1980s, 1990s), published prior to the introduction of after-treatment systems for diesel engines. The data on the composition of the combustion products for kerosene and diesel should therefore be considered as indicative.[1,16,25,30]

Composition of kerosene engine exhaust

KEE forms a complex mixture of numerous organic and inorganic substances in gaseous, condensed (liquid) or solid form (combustion-generated particles). At least 140 individual substances and 13 substance groups (e.g., PAHs, particles) have been identified in KEE.[3]

The combustion products (~8.5% of the total mass flow from the aircraft engine, see Figure 3) of KEE during cruise conditions (data on ground idle, cruise and climb see Table 3) consist predominantly of CO_2 (~72%) and H_2O (~27.6%), which are, together with sulphur oxides (SO_x), directly related to the combustion of kerosene jet fuel with minor variations due to the carbon-hydrogen ratio in the fuel and the various flight phases of the aircraft. The remaining part (~0.4%) consists largely of nitrogen oxides (NO_x) (~84%), CO (~11.8%), hydrocarbons (HC) (~4%), and small amounts of soot (with adsorbed PAHs, metals and other organic compounds) (see Figure 3 and Tables 3 and 4).[1,25]

Emissions of NO_x, CO, HC (consisting mainly of low-molecular-weight hydrocarbons and derivates (e.g., carbonyls, alkanes (such as methane), aromatics)), and soot are strongly influenced by a wide range of variables, particularly thrust settings (see Table 3) and ambient engine inlet conditions (including air concentrations of N_2 and O_2). CO and HC are typically products of incomplete combustion. They are highest at low thrust settings, such as during ground idle operations (most relevant for occupational exposure of platform workers), when the temperature of the air is relatively low and fuel atomisation and mixing processes least efficient. NO_x and soot emissions, on the other hand, are highest at high thrust settings (see Table 3).[1-3,25,35] This contributes to (local) air pollution and occupational exposure of workers at airports and (military) airfields. The oxidation of atmospheric N_2 at very high temperatures during combustion drives the formation of

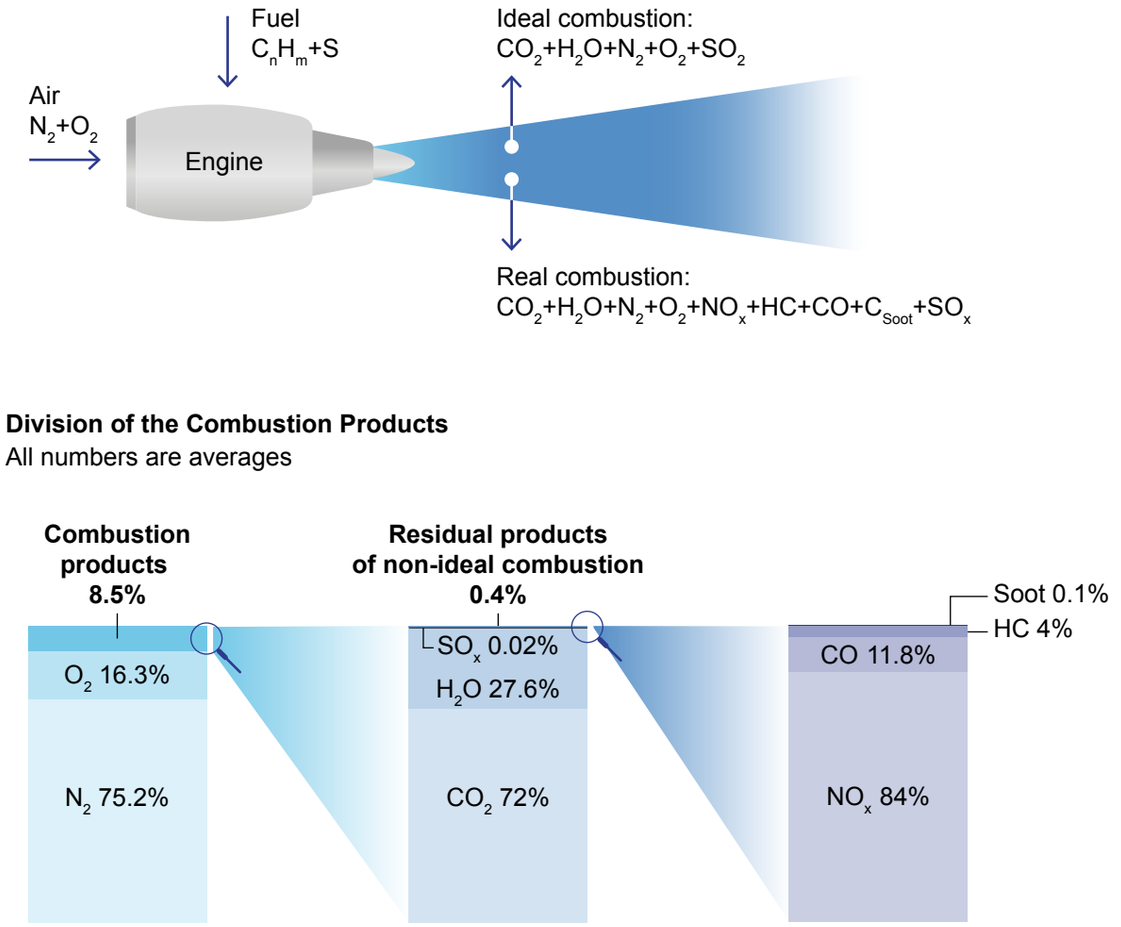


Figure 3 Average composition of combustion products of kerosene jet fuel during cruise conditions

Adopted from IPCC (1999)[25]

NO_x. In addition, the presence of trace amounts of S, N_2, and some metals in fuel (e.g., iron (Fe), copper (Cu), zinc (Zn)) may lead to the formation of SO_x, additional NO_x, HC and soot during incomplete combustion.[1]

Table 3 Emission index levels (in g/kg Jet A-1 fuel) for a turbofan engine for typical aircraft engine operating regimes

Substance	Ground idle (4% thrust)	Cruise (65% thrust)	Climb (85% thrust)
CO	66.8	0.8	0.9
Total hydrocarbons (as methane equivalents)	13.3	0.4	0.2
NO_x	4.5	15.5	20.9
NO	0.9	9.0	12.5
NO_2	3.1	1.6	1.7
Soot* (# particles / kg Jet A-1 fuel)**	$2.0x10^{15}$	$1.6x10^{15}$	$1.5x10^{15}$
Soot* (mg / kg Jet A-1 fuel)**	5.1	102.7	196.6

* Note: soot or carbonaceous particulate matter results from the incomplete combustion of fossil fuels or biomass. Soot forms a mixture of particles containing elemental and organic carbon (EC and OC).
** with particles loss corrections
Source: Heeb et al. (2024)[35]

Composition of diesel engine exhaust

Like KEE, DEE is a complex mixture of gaseous, condensed (or liquid), and solid combustion-generated particles produced during the combustion of petroleum-derived diesel fuel. The gaseous components of DEE consist primarily of N_2 (~75.2%), O_2 (~15%), CO_2 (~7.1%) and water vapour (~2.6%), like KEE (see Table 4). The remaining of the combustion products consists mainly of NO_x and small amounts of soot, sulphur dioxide (SO_2) and HC, such as low-molecular-weight hydrocarbons and their derivates (e.g., carbonyls, carboxylic acids, alkanes (paraffins), alkenes (olefins), aromatics).[6,16,29,30,36,37]

Table 4 Composition of kerosene engine exhaust (during cruise conditions) in % of total mass flow and diesel engine exhaust (automotive) in % by weight.

Substance	Kerosene engine exhaust	Diesel engine exhaust*
Nitrogen (N_2)	~75.2%	~75.2%
Oxygen (O_2)	~16.3%	~15%
Carbon dioxide (CO_2)	~6.1%	~7.1%
Water vapour (H_2O)	~2.3%	~2.6%
Sulphur oxides (SO_x)	~0.02%	~0.01% (currently only trace amounts due to regulations)
Nitrogen oxides (NO_x)	~0.03%	~0.03%
Hydrocarbons (HC)	~0.001%	~0.0007%
Carbon monoxide (CO)	~0.004%	~0.03%
Soot**	~0.00003%	~0.006%

* Note: original source for this information is a publication by Volkswagen (1989), so based on older data (older diesel engines without after-treatment systems (such as catalysators and particle filters) and old diesel fuel formulations).
** Note: soot or carbonaceous particulate matter results from the incomplete combustion of fossil fuels or biomass. Soot forms a mixture of particles containing elemental and organic carbon (EC and OC).
Sources: Hartikainen et al. (2024)[38]; IARC (2014)[5]; IPCC (1999)[25]; Liati et al. (2019)[39]; Masiol & Harrison (2014)[1]; Ris (2007)[37]; van Seters et al. (2024)[26]; WHO (1996)[30]

2.3.2 Toxicologically relevant components

This section identifies the toxicologically most relevant components of KEE and DEE with emphasis on their carcinogenic properties, i.e. combustion-generated particles, specifically soot, with adsorbed PAHs and metals. Because of the relatively high amount of sulphur compounds in KEE

(particularly compared to current DEE and current diesel fuel formulations), these compounds will be considered as well in this section.

Particulate matter

Particulate matter (PM), or combustion-generated particles (mainly soot), form a relatively small percentage of the total composition of KEE and DEE (both trace amounts). Combustion-generated particles are, however, considered main contributors to the toxicity of DEE and probably of KEE as well. IARC has classified DEE particles as carcinogenic to humans (Group 1).[5,40] In addition, PM in outdoor air is also classified as carcinogenic to humans (Group 1) by IARC.[3,40]

Several factors play an important role in the amount, size and composition of combustion-generated particles (such as KEE particles and DEE particles), such as engine type, engine power settings, chemical composition of fossil fuel and combustion temperature.[1,26]

Size distribution and particle structure

Several studies indicate that the combustion of kerosene jet fuel by aircraft generally leads to higher concentrations of smaller sized combustion-generated particles compared to combustion-generated particles from road transportation at freeways.[2,35,39,41-43]

KEE particles, as well as DEE particles, have a spherical shape.[1,43] They both have particle size distributions with a bimodal character, which corresponds to the formation of the particles. The two modes are commonly referred to as the nuclei (or nucleation) and accumulation mode.[44] The size boundaries and concentrations within the modes differ for KEE particles compared to those for DEE particles, but there is some overlap in the size distributions. It should be noted that varying particle size distributions may be reported in literature and that overlap between nucleation and accumulation mode particles is possible as well.

In general, KEE particles are small at low thrust levels (e.g., ground idle), approximately 3 to 40 nm in nucleation mode (nuclei), and tend to coagulate (clump together to form larger-sized particles) at higher power settings (e.g., climb and cruise conditions). The nuclei are unstable and tend to react by coagulation and aggregation (process forming large clusters of merged smaller-sized soot particles). As a result, the particles grow over time to a size of approximately 80 to 100 nm (accumulation mode).[1,2,38,39,43]

Approximately 80% of the soot agglomerates (large chain-like clusters of loosely clumped smaller-sized soot particles) have aerodynamic diameters ranging from 3 to 40 nm during ground idle conditions (of which ~50% is even <20 nm), higher power settings increased aerodynamic diameters of the particles with most particles (~40%) in the range of 40 to 80 nm.[1,2,39]

A recent study demonstrated that although particle size increases drastically upon chemical and physical processes, the size of KEE particles generally remained in the ultrafine particle (UFP) size range (<100 nm).[38]

Similar to KEE particles, DEE particles, consist of solid soot particles with adsorbed PAHs, nitro-PAHs and trace amounts of metals.[6,16,26,29,37,45] Approximately 1-20% of DEE particle mass consists of UFP-sized particles, with a mean particle aerodynamic diameter of 20 nm.[37]

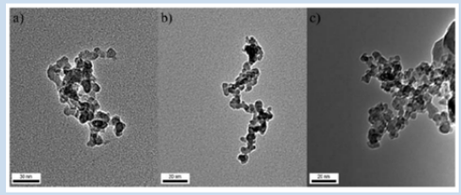
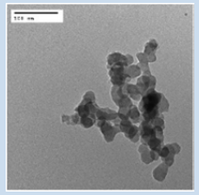
DEE particles in the nuclei mode (aerodynamic diameter ~3-30 nm) are formed through nucleation and condensation of SO_2 and hydrocarbons by homogeneous nucleation or by nucleation on solid core particles (see also Figure 4). The accumulation mode (aerodynamic diameter ~30-500 nm) contains agglomerates of soot formed in the engine cylinders. These soot particles are composed of elemental carbon (EC), organic carbon (OC) and other particle-adsorbed organic compounds. The accumulation mode contains most of the DEE particles.[29,44,46]

Carbonaceous particulate matter

Combustion-generated particles, like KEE particles and DEE particles, consist predominantly of solid carbonaceous PM that form a complex mixture of EC and OC, often referred to as soot.

Soot, or carbonaceous PM, is primarily generated by incomplete combustion processes through the pyrolysis of fossil fuels (e.g., kerosene jet fuel).[1,52] The formation of soot is driven by a high aromatic content in kerosene jet fuel,[24] which may lead to the formation of PAHs as a result of incomplete combustion processes in the aircraft engine. These PAHs may condense on the surface of pre-existing particles, better known as nuclei, formed during the combustion process and thus grow by further chemical reactions and accumulation of other molecules present in the fuel-rich environment, to form combustion-generated soot particles. The initial soot formation in the engine combustor occurs rapidly, often within a few seconds. Individual soot particles can also agglomerate to form larger soot particles or soot agglomerates (see also Figure 4).[38,44,53]

Table 5 Structural properties of kerosene engine exhaust particles (KEE particles) and diesel engine exhaust particles (DEE particles)

Structural property	KEE particles	DEE particles
TEM-image	 Note with figure above: a) and b) show KEE particles with aerodynamic diameters of 15 nm; and c) aggregate KEE particle with aero-dynamic diameter of 50 nm Source: Boies et al. (2015)[41]	 Note with figure above: DEE particle with aerodynamic diameter of 100 nm Source: Carlsten et al. (2016)[47]
Particle size distribution	The particle size distribution of KEE particles is bimodal: Nuclei mode of ~3 to 40 nm Accumulation mode of ~80 to 100 nm. (various size distributions reported)	The particle size distribution of DEE particles is bimodal: Nuclei mode of ~3 to 30 nm Accumulation mode of ~30 to 500 nm. (various size distributions reported)
Particle shape and structure	Spherical-like nature, forming fractal-like agglomerates.	Spherical-like nature, forming fractal-like agglomerates.
Ability to adsorb compounds	Large surface area which allows for adsorption of relatively large amounts of organic material.	Large surface area which allows for adsorption of relatively large amounts of organic material.

Abbreviations: TEM, transmission electron microscopy; nm, nanometre.
Sources: Abegglen et al. (2015)[48]; Boies et al. (2015)[41]; Carlsten et al. (2016)[47]; Gysel et al. (2003)[49]; Hartikainen et al. (2024)[38]; Heeb et al. (2024)[35]; Masiol & Harrison (2014)[1]; NTP (2021)[46]; Onasch et al. (2009)[18]; Park et al. (2004)[50]; Popovicheva et al. (2000)[51]; Ris (2007)[37]; Taxell & Santonen (2016)[29]

Compared to soot particles from other sources such as DEE or wildfires, aircraft-related soot contains the highest carbon content, the greatest oxygen content in the form of phenolic and carbonyl groups, and the widest range of elements, including S, Na, N, Zn and Ba.[1,54] Soot emissions in KEE are lower during idle but increase with increasing thrust settings and increasing combustion temperature. Soot composition changes with increasing thrust settings and shifts from OC-rich at idle to EC-rich with increasing thrust.[1,38,55-58]

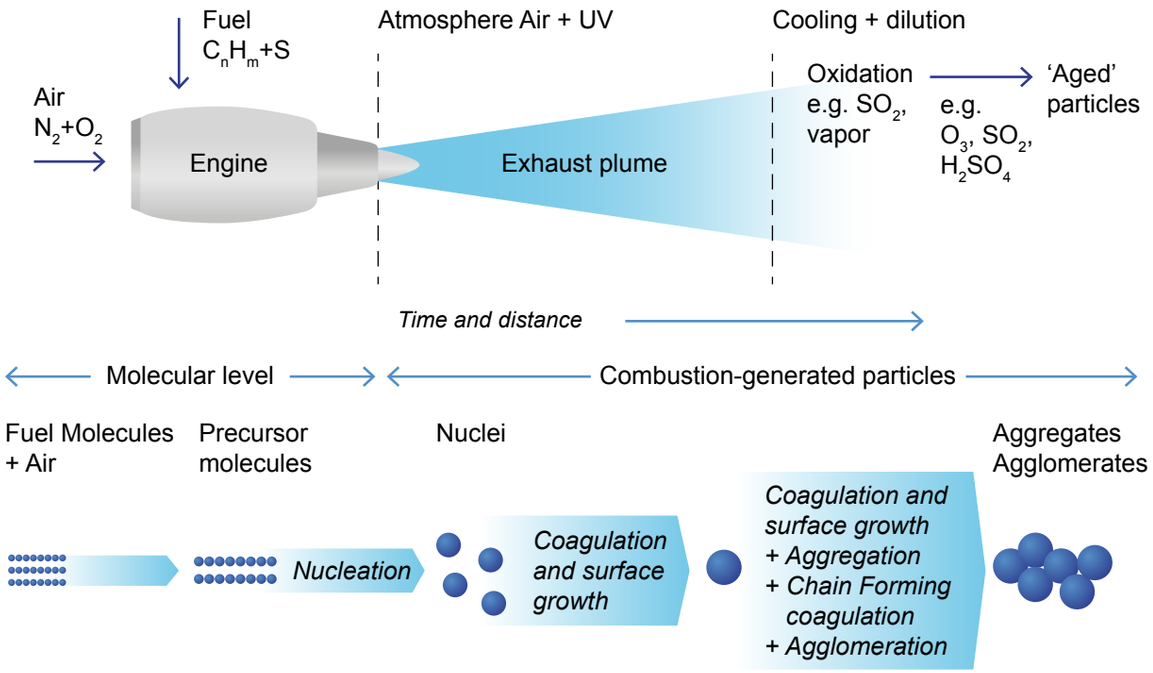


Figure 4 Process of combustion-generated particle formation

Aging and secondary aerosol formation

During atmospheric aging, volatile and semi-volatile organic compounds (SVOCs) become oxidized, leading to fragmentation or condensation and consequently to the formation of secondary inorganic and organic aerosols (SIAs and SOAs respectively). Since aircraft engines can emit significant quantities of VOCs, including known SOA precursors such as benzene and toluene, the formation of SOA may be substantial. However, data to underpin this is scarce due to the complexity of these processes.[1,44,56,59,60]

PAHs and other organic compounds

The main components adsorbed to both KEE particles and DEE particles are PAHs and other hydrocarbons. These PAHs deserve special attention, because most of the PAH-congeners are known, probable or possible human carcinogens.[1,61,62] PAHs are volatile and semi-volatile substances that are distributed between gaseous and particulate phases depending on the temperature in the exhaust plume. Almost all low molecular weight PAHs (≤4-rings) occur in the gaseous or vapour-phase, while almost all high molecular weight PAHs (>4-rings) occur almost completely particle-bound. Lighter congeners such as naphthalene and its 1-methyl and 2-methyl derivates contribute strongly to the total PAH mass in KEE at different thrust modes. Generally, approximately 60% of the total PAH emissions consist of naphthalene (2 rings), ~38% of PAHs with 3 ring-congeners and the remaining emissions (~2.5%) consist of high-molecular weight PAHs with 4 to 7 rings. The most abundant PAH in KEE are naphthalene,

acenaphthalene, phenanthrene, fluoranthene, fluorene and pyrene. Apart from naphthalene, which is most likely found in gas-phase, the other 3 to 4-ring PAHs can be found both particle-bound as well as gas-phase.[1-3,35,55] A more recent study by Heeb et al. (2024) reports the highest PAH (including carcinogenic 4 to 6 ring PAHs) emissions in KEE during ground idle operations, which is most relevant for occupational exposure. At higher thrust mode, most of the carcinogenic 4 to 6 ring PAHs were close to background levels.[35]

DEE also contains nitro-PAHs,[29] whereas there is no direct evidence that indicates whether KEE contains nitro-PAHs. However, nitro-PAH emissions, in the form of nitropyrenes, are reported after ignition of a kerosene heater.[63] Less than 1% of DEE-particle mass contains PAHs and its derivates (i.e., particle-bound PAHs).[29,37] Furthermore, naphthalene, phenanthrene and pyrene are also frequently found in DEE.[29]

Generally, the relative quantities of cyclic compounds in the fossil fuel, e.g., aromatics and naphthalene, determine how much soot is emitted during combustion. This increases with the amount of ring structures of the hydrocarbons.[24,26]

Benzene (classified as human carcinogen (Group 1) by IARC),[1,64,65] toluene, ethylbenzene and xylene (BTEX) are aromatic hydrocarbons principally emitted in exhaust plumes. BTEX compounds are highly reactive and play a key role in the formation of O_3 and SOAs. Emissions of benzene and toluene decrease rapidly with increasing aircraft engine power (higher power setting).[1]

A significant percentage (30-40%) of total hydrocarbon emissions in the exhaust plume at ground idle (low power settings) consists of aliphatic, cycloaliphatic and aromatic structures, predominantly ethylene, propylene, acetylene, 1-butene, methane and formaldehyde. Formaldehyde (a carbonyl classified as a human carcinogen (group 1 IARC)) was found to be the most predominant aldehyde in KEE. Carbonyl (e.g., formaldehyde, acetaldehyde, propionaldehyde, acrolein) emissions are generally higher during ground idle than at higher thrust levels or high-power settings. However, measurements of carbonyl emissions may show large variations due to changes in ambient temperature.[1,55]

Metals

Several metal compounds have been detected in trace amounts in the exhaust from aircraft engines. Some of these metal compounds have been classified as probable or possible human carcinogens, such as cobalt, nickel and lead.[3] These metal compounds, usually adsorbed to soot particles, include chromium (Cr), iron (Fe), molybdenum (Mo), sodium (Na), calcium (Ca), aluminium (Al), vanadium (V), barium (Ba), cobalt (Co), copper (Cu), nickel (Ni), lead (Pb), magnesium (Mg), manganese (Mn), silicon (Si), titanium (Ti), and zirconium (Zr).[1,3,66] The presence of zirconium and cobalt in the exhaust results from engine wear, since these compounds are not found in kerosene jet fuel or lubricating oil. Zirconium is known to be used as a thermal barrier coating in aircraft engine parts. Engine wear most likely also explains the presence of iron, copper, chromium, nickel, and

molybdenum in the exhaust, although smaller amounts of iron are also found in kerosene and lubricating oil. Chromium (metallic chromium can be a precursor of Cr(VI) and Cr(III); Cr(VI) is classified as carcinogenic to humans by IARC) is widely used in engine parts, such as turbine engine blades, and small amounts are also found in lubricating oil. For metals such as barium, vanadium, lead and titanium the main source is kerosene, although vanadium, lead and titanium are also found in lubricating oil. Calcium and sodium are found in both kerosene as well as lubricating oil. Barium may reduce soot emissions during combustion by acting as a nucleation core and was therefore used as an additive in kerosene. Compounds such as aluminium, silicon, magnesium, and manganese have no known main source[66] and are associated with kerosene, engine wear and/or lubricating oil.[1,3,66] DEE may contain some of the above-mentioned metals in trace amounts as well.

Sulphur oxides

Sulphur is a naturally occurring component of crude oil. Kerosene jet fuels averagely contain ~0.05% but is currently regulated at 0.3% sulphur content. While road transport fuels, currently sold in Europe, only contain ~0.001% sulphur content. 'Older' diesel fuel formulations, however, had a higher sulphur content, which was rather similar to the sulphur content in kerosene jet fuel.[1,25,26] Sulphur dioxide (SO_2) is the predominant sulphur-containing component in KEE and originates from the oxidation of sulphur in aircraft engines. The emission of SO_2 in the exhaust is therefore highly dependent on the fuel sulphur content.[1] During combustion, sulphuric oxides and sulphuric acids are formed. Sulphur trioxide (SO_3) can be formed by the oxidation of SO_2 and oxygen atoms, or by hydroxyl radicals present in the exhaust plume. The further reaction with water vapour converts SO_3 to sulphuric acid.[1,19,26,67,68] Sulphuric acid affects KEE particle formation by promoting the nucleation process and increasing the number of UFPs.[26,36]

2.3.3 Conclusion on combustion products

In Table 4, an overview of the composition of KEE and DEE is shown. KEE and DEE are very similar in composition regarding the relative amounts of nitrogen, oxygen, CO_2, NO_x, and hydrocarbons. There are, however, a few differences in the average composition of combustion products between KEE and DEE. First of all, that is that KEE contains relatively higher amounts of sulphur compounds compared to 'new' DEE emissions (new diesel fuel formulations and new diesel engines). However, when KEE is compared to 'older' data on DEE, the differences in sulphur content are small. Since the amount of sulphur compounds in the exhaust affects particle formation, these compounds may be relevant for the evaluation of the toxicity of KEE and will therefore be considered in the following chapters.

Secondly, KEE is generally characterised by high concentrations of smaller particles (mainly soot) compared to DEE. Most KEE particles have aero-dynamic diameters in the UFP size range. Table 5 shows that both KEE

particles and DEE particles have both bimodal size distributions with considerable overlap, morphology, fractal-like structures, and relatively large surface areas which allow for the adsorption of organic substances. Furthermore, KEE may contain lower concentrations of high-molecular weight PAHs (>4-ring PAHs) compared to 'older' DEE. These compounds generally have a higher carcinogenic potential, whereas the total PAH emitted in KEE generally consists of lower-molecular weight (2- and 3-ring) PAHs, such as naphthalene (2-ring PAHs).

2.4 Innovations in jet fuel and aircraft engine technology

Various ongoing innovations, such as sustainable aviation fuels (SAFs) and new engine technologies, may eventually affect the composition of KEE.[22,27,29,69] Some of these innovations are described below.

Sustainable aviation fuels

Several types of SAFs can be distinguished according to the feedstock and process technologies used.[22,25,27,28] Synthetic paraffinic kerosene (SPK) is derived from fossil feedstocks such as coal, natural gas and other hydrocarbons using a Fischer-Tropsch (FT) process (FT-SPK).[27,28,69]

FT jet fuels can also be derived from biomass (not 'synthetic').[27,69,70]

Bio-jet fuels (or renewable jet fuels (RJF)) can be produced from a wide range of biomass, including vegetable oils, plant materials and animal waste. These bio-jet fuels are often referred to as hydroprocessed renewable jet fuels (HRJ fuels) or hydroprocessed esters and fatty acids (HEFA) jet fuels.[27,69-71] Generally, bio-jet fuels have low energy density, poor high-temperature thermal stability and storage instability.[27,69,71]

SAFs must have adequate lubricity and be compatible with the existing aircraft fleet and all the materials (metallic and non-metallic) used in the aircraft engine fuel system to ensure that standards of safety-critical items (e.g., fuel pumps) are maintained.[22,25,28] This means that bio-jet fuels, used by the current aircraft fleet, are blended with common kerosene jet fuel (at least 50% kerosene).[27,28,70]

New aircraft technology

Apart from emission regulation (Chapter 2.1), new types of aircraft are being developed (e.g., hydrogen-powered aircraft) and/or in combination with new propulsion technologies that are compatible with either hydrogen (H_2), SAFs or electric energy. There are seven propulsion concepts: 1) disruptive gas turbine-based propulsion using drop-in SAF for combustion; 2) gas turbine-based propulsion using hydrogen for combustion; 3) battery electric propulsion; 4) fuel cell electric propulsion using hydrogen; 5) turbo-electric propulsion based on drop-in SAF; 6) other hybrid-electric propulsion using drop-in SAF; and 7) hybrid-electric propulsion using hydrogen as energy source. The application of these propulsion technologies has been limited to smaller aircraft with shorter ranges.[70,72]

As noted in 2.2.1, SAFs currently form less than 1% of globally used jet fuels. However, this will likely change in the future because the aviation industry

aspires, in line with the Paris Agreement and the European Green Deal, to reduce aircraft emissions to net zero by 2050.[4,73] By replacing kerosene jet fuel (Jet A-1) with SAFs, aircraft emissions (e.g., CO_2, sulphur oxides, soot) are likely to be reduced considerably.[19,27]

3 Workplace measurement methods

3.1 Workplace air monitoring

OELs are generally defined as a concentration of a substance in the air (i.e. the breathing zone of the worker) at the workplace. This means that for determination of occupational exposure the inhalable and respirable fractions which can penetrate the airways are most relevant. These particle size fractions, defined by standard CEN-EN-481 (1993),[74] are defined as follows:

- Inhalable fraction: Mass fraction of total airborne particles which is inhaled through the nose and mouth.
- Respirable fraction: Mass fraction of inhaled particles which penetrate the unciliated airways. This fraction consists of particles with a D_{50} (aerodynamic diameter) of 4 µm or smaller which can reach the alveolar region of the lungs.

Epidemiological studies have measured predominantly the following components as indicators for occupational exposure to KEE: particles, particularly UFP, PAH and metals (see also Chapter 4).[2,3]

For DEE respirable particles, EC, NO_x, CO and PAHs are generally applied as exposure indicators.[5,6,29] For the advisory report *Diesel engine exhaust (2019)*, DECOS focussed on respirable elemental carbon (REC) in DEE particles as indicator for DEE exposure.[6]

3.1.1 Particulate matter

The measurement of combustion-generated particles is heavily dependent on the adopted methodology. Since combustion-generated particles may undergo changes in time and space, the mass of sampled particles is determined by the sampling protocol, such as the distance from the engine exit, and other parameters that can influence the aging of plumes. In addition, the environmental conditions (e.g., temperature, humidity, sunlight, wind) can also affect particle mass, particularly through the potential for particle formation, coagulation, and growth (see Chapter 2.3).[1]

Gravimetric methods allow for determination of the relevant particle mass of a size selectively collected filter sample (e.g., MDHS 14/4) and is used for inhalable and respirable particle measurements (see Table 6 for sampling and analytical methods). However, the sensitivity of these methods to very small particle masses is low and it is not possible to distinguish KEE particles from other particles in the workplace air.[3] KEE particles consist mainly of carbonaceous particles which are generally identified as elemental carbon (EC) or black carbon (BC) depending on their corresponding measurement methods. The terms BC and EC are often

used interchangeably for the same carbonaceous fraction, but their definitions and measurement methods are different.[1,75] Methods for EC sampling and analyses are complex and typically concern a thermal optical analysis with a flame ionisation detector (FID) (NIOSH method 5040 for total carbon); filter-based sampling followed by analysis using gas chromatography with a flame ionisation detector (GC-FID) or gas chromatography with a mass spectrometer (GC-MS) (see also Table 7).[29] BC sampling is complicated by the lack of a simple widely accepted definition and absence of techniques that are uniquely sensitive to BC.[75-77] BC mass can be estimated in several ways, none of which fully represent BC. A commonly used method is the conversion of light absorption measured with an Aethalometer to give equivalent black carbon (see Chapter 4).[76,77]

Table 6 Overview of sampling and analytical methods for workplace exposure monitoring (in air) of inhalable and respirable dust

Method (reference)	Type of method	Substance(s)	Range	Limit of detection (LOD) or reporting limit (RL)	Remarks
CEN EN-482: 2021[78] EN-13205-1:2014[79] and EN-13205-2:2014[80]	Gravimetric analysis	Inhalable/ respirable dust	n.a.	n.a.	
HSE MDHS 14/4[81]	Gravimetric analysis	Inhalable/ respirable dust	n.a.	n.a.	For information on LOD is referred to ISO 15767:2009[82]
ISO 13137:2022[83]	Gravimetric analysis	Inhalable/ respirable dust	n.a.	0.05 µg (RL)	

Abbreviations: CEN, European Committee for Standardization; EN, European Standard; HSE, Health and Safety Executive; MDHS, Methods for the Determination of Hazardous Substances; n.a., not applicable; ISO, International Organisation for Standardization.

Source: RIVM (2024)[3]

Table 7 Overview of sampling and analytical methods for workplace exposure monitoring (in air) of total carbon and elemental carbon

Method (reference)	Type of method	Substance(s)	Range	Limit of detection (LOD) or reporting limit (RL)	Remarks
ISO 13137:2022[83]	Gravimetric analysis	EC	n.a.	0.1 µg (RL)	
NIOSH 5040[84]	Thermal-optical analysis; FID	Total carbon (OC and EC)	1-105 µg per filter portion	0.3 µg per filter portion	EC is recommended for workplace exposure.

Adapted from RIVM (2024)[3]

Abbreviations; n.a., not applicable; ISO, International Organisation for Standardization; FID, flame ionization detector; NIOSH, National Institute for Occupational Safety and Health.

Ultrafine particles

Since 2018 there has been a standard for assessment and measurement of workplace exposure to nano-objects, such as KEE particles in the UFP size range.[85,86] These particles have negligible mass but are the dominant contributor to the total number of combustion-generated particles in KEE emissions, which means that KEE particles are better quantified by number concentrations than by mass concentration.[36,87] According to EN 16966:2018 the main measurements used to assess workplace exposures are particle number concentration, particle surface area concentration and particle (volume) mass concentration.[85]

Sampling distance (e.g., breathing zone workers, stationary in the workplace) has a significant influence on measurement results. The measurement instruments used by studies for measuring UFP concentrations differ in operation principles and measurement ranges. The most used real-time and off-line instruments include direct-reading, handheld instruments, such as a condensation particle counter (CPC), a diffusion charger (DC; for instance, DiSCmini, NanoTracer, Naneos Partector) and an optical particle counter (OPC), which are used to detect releases of UFP. These are accompanied by sampling and subsequent chemical and electron microscopic analyses using scanning electron microscopy (SEM) or transmission electron microscopy (TEM) and/or x-ray fluorescence (XRF)/inductively coupled plasma-mass spectrometry (ICP-MS), which can be used for particle identification and elemental composition.[86,88]

The low-end of the measurement ranges for these instruments varies from 1 to 20 nanometer (nm). The upper-end of the measurement range is often not fixed.[67,87,88]

In addition, freshly emitted KEE particles may change rapidly in size and chemical composition, due to reaction processes of oxidation, condensation and coagulation with gaseous components. This can take minutes up to hours. Due to these processes, concentrations of KEE particles of UFP size particularly around 20 nm vary highly, more than concentrations of larger particles.[87]

3.1.2 PAHs and other organic compounds

For the sampling and analysis of PAHs (e.g., naphthalene) a high performance liquid chromatography with fluorescence detection (HPLC-Flu) is applied (e.g., NIOSH 5506[89]). A gas chromatography with a tandem mass spectrometry (GC-MS/MS) can be used for sampling and analysis of hydrocarbons and aromatic hydrocarbons (e.g., benzene, styrene) (NIOSH 1500[90] and 1501[91] respectively).

3.1.3 Metals

For the sampling and analysis of metals, such as chromium (Cr), nickel (Ni), vanadium (Va), zirconium (Zr), molybdenum (Mo), zinc (Zn), aluminium (Al), and iron (Fe) an ICP-MS can be used (e.g., ISO/DIS 30011[92]).[3]

3.1.4 Sulphur dioxide

KEE contains numerous gaseous components including sulphur dioxide (SO_2). For the sampling and determination of SO_2 extractive Fourier transform infrared (FTIR) spectrometry is usually used (e.g., NIOSH 3800).[93]

3.2 Biomonitoring of workplace exposures

Biomonitoring is used to assess exposure to hazardous substances by measuring the substance itself, its metabolites (breakdown products) or adducts, in biological materials such as urine, blood or exhaled breath condensate (EBC). An advantage of biomonitoring is that it integrates exposure from all exposure routes. In addition, biomonitoring provides information on the total body burden that is more directly related to the systemic effect.[94]

3.2.1 Ultrafine particles

In environmental health studies BC in blood, urine and other biological media (e.g., breast milk, brain tissue) has been used as biomarker of exposure.[95-97] BC content is expressed as number of particles per volume of sample. The measurement principle used is pulsed laser microscopy.[98]

3.2.2 Polycyclic aromatic hydrocarbons

Metabolites of PAHs and their conjugates are commonly analysed in urine, but determination in EBC has also been applied (see Chapter 4). Urinary monohydroxy polycyclic aromatic hydrocarbons (OH-PAHs) are a class of PAH metabolites commonly used as biomarker for assessing human exposure to PAHs. Due to the relatively short half-lives of urinary OH-PAHs (e.g., ranging 6-35 hours after inhalation of PAHs), these biomarkers only provide information on recent PAH exposures.[99] Occupational exposure to PAHs is commonly established using 1-hydroxypyrene (1-OH-P) in urine as a biomarker, because it correlates well with other PAH metabolites. Although 3-hydroxy benzo[a]pyrene (3-OH-BaP) may be toxicologically more relevant as a direct metabolite of benzo[a]pyrene, the analysis is more demanding with respect to laboratory equipment.[99,100]

4 Occupational exposure

As described in Chapter 2, KEE is a complex mixture of numerous substances. Because it is impossible and/or impractical to measure all these substances, most workplaces measure one or a few of these substances as indicators for exposure to KEE. Ideally, these indicators are specific for exposure to KEE, are measurable at relevant workplace concentrations, reflect the overall concentration of KEE and are directly linked to an OEL for KEE or, in the absence of this OEL, to its specific components.

Airport workers with activities related to operating aircraft can be exposed to a variety of combustion-generated particles from the exhaust directly or formed through various physical and chemical reactions and processes in the atmosphere.

Some important determinants for occupational exposure to KEE are type of job, proximity to operating aircraft and fluctuations in emissions due to various aircraft operating conditions.[2,3,101] Airport apron workers (e.g., baggage handlers, flight officers, catering drivers, fuel drivers, personnel working in pushback/towing) spend the highest proportion of their normal work time around the airport apron in the vicinity of operating aircraft. Baggage handlers at the apron spend approximately 76% of their total working time on the airport apron and are, consequently, considered to be highly exposed to KEE. Catering drivers, fuel drivers, inflight service drivers and other catering and inflight service personnel spend approximately 62% of their total working time on the airport apron in the proximity of aircraft. Airport workers such as airside security and firefighters spend less than 15% of the total working time on the airport apron. These percentages were estimated for an exposure assessment study at Copenhagen Airport. It is assumed that work time spent at the apron per type of job is similar for other (commercial) airports.[2,3,101]

4.1 Workplace monitoring data from personal samplers

Occupational exposure is determined for the individual worker during their whole work shift or during specific work tasks.[85]

4.1.1 Particulate matter

Several studies have measured inhalable, respirable and ultrafine particle fractions in air samples collected from airport workers. Some have also analysed the morphology and chemical composition. These studies are described in Table 8.

Table 8 Overview of occupational exposure levels to ultrafine particles (UFP) and particulate matter from personal monitoring

Reference / study location	Results personal exposure monitoring	Sampling method & measurement range	Remarks
Touri et al. (2025)[102] *Period*: June and July 2018 at Marseille-Provence airport; June and September 2019 at Paris-Roissey airport *Location*: Marseille-Provence airport and Paris-Roissey airport, France *Background aerosols or ambient air*: among 1 to 2 workers at Air France medical facilities (at the same airport, far from runways) *Study population:* Only 16 workers for personal aerosol measurements	Exposure to organic carbon (OC) was below the limit of detection in all samples irrespective of location. Quantifiable and similar concentrations of elemental carbon (EC) in personal and ambient air samples of mechanics and office workers from Paris-Roissey airport not in terminal or apron workers from Marseille-Provence airport. Mean EC concentrations (sd) in µg/m^3: • Mechanics: 10.1 (4.3) Background mechanics: 5.1 (0.7) • Office workers: 9.9 (3.9) Background office workers: 7.8 (1.3). Geographical location of the airport as well as seasonal influences may have played a role.	*Aerosol sampling:* • Sioutas: a personal cascade impactor, collecting airborne particles in 4 size fractions with 50% cut-points at 2.5, 1.0, 0.5, and 0.25 µm. Analysed for carbon content. Clipped onto clothing • Particlever: a size-selective impactor with a cut-off diameter of 4 µm. Analysed for elemental carbon content using thermo-optical analysis. Worn from strapnecklace. Other sampling and analysis included: lung function, exhaled CO, exhaled breath condensate (analysed for metal content (Cr, Cd, Al, and 8-isoprostane; particle size distribution), and urine analyses (analysed for metal content (Cr, Cd, and Al)	Marseille-Provence airport is located near the coast/sea (may influence results of UFP measurements). Follow-up of the study by Marie-Desvergne et al. (2016)[103] 471 participants (employees of Air France) from previous study, n=218 lost-to-follow-up, n=22 retired, n=13 no informed consent, n=3 no function data. A total of 215 participants in final study population. 4 work profiles: office workers (n=68), airport terminal workers (n=29), apron workers (n=35), and mechanics (n=83). All mechanics and apron workers are men. 23.72% of participants were current smokers Pregnant or lactating women or eligible subjects with contraindications for study procedures were excluded from the study.
Van der Meer et al. (2024)[104] *Period*: August-September 2023 (21 sampling days) *Location*: Schiphol Airport, Amsterdam, the Netherlands Background UFP: Osdorp (NL), continuous stationary UFP measurements using TSI particle counter	Background UFP concentrations measurement period (mean): 16,526 UFP/cm^3 *UFP (particles sizes <0.1 µm):* geometric mean, (GSD) 65,000 UFP/cm^3 (2,210 UFP/cm^3), n=114 measurements *Inhalable dust (particle sizes <100µm):* 0.19 mg/m^3 (1.82 mg/m^3), n=88 measurements *Respirable dust (particles sizes <10 µm):* not applicable, n=103 measurements 86% of the measurements was below detection limit. All respirable particles contained OC and 2/3 of the particles contained EC.	*UFP sampling*: Naneos Partector 2 based on diffusion charging, connected to Higgins-Dewell cyclone capturing larger sized particles Detection range: 10-300 nm Measuring range: till 1.10^6 particles/cm^3 *UFP measurements*: • Number of ultrafine particles/cm^3 • % time more than 100k ultrafine particles/cm^3 • Particle diameter GPS-loggers used for location *Inhalable dust sampling*: GilAir5 and gravimetric analysis. Concentrations of metals were determined using ICP-MS (ISO 30011 method). *Respirable dust sampling*: GilAir5 with Higgins-Dewell cyclone and gravimetric analysis (MDHS 14/4 method). Concentrations of EC determined using NIOSH 5040 method.	Airport is located near the coast/sea (may influence results of UFP measurements) Information on weather conditions, fly movements and participant activities (such as, smoking, driving vehicles (including type of fuel) and use of personal protection) was available. UFP measurements showed temporal and spatial variability, possibly due to location near coast/sea

Reference / study location	Results personal exposure monitoring	Sampling method & measurement range	Remarks
Esveld et al. (2024)[105] *Period*: May-June 2023 (7 sampling days for personal measurements) *Location*: Eindhoven Airport, Eindhoven, the Netherlands	*UFP measurements*: jobtitle, average mean • Flight officer: 77,000 UFP/cm^3 • Platform employee: 69,000 UFP/cm^3 • Airport operations manager: 8,700 UFP/cm^3 • Main door access worker: 37,000 UFP/cm^3 4 function groups selected based on: relative exposure, number of employees in group, and dispersion over airport and employers. 6 workers per function group measurements spread over multiple days, at least 4 days.	*UFP sampling*: 5x Naneos Partector 2 based on diffusion charging Detection range: 10-300 nm *UFP measurements*: • Number of ultrafine particles/cm^3 GPS-loggers used for location	Questionnaire filled in by one supervisor per function group. Worst-case weather conditions with respect to wind direction were selected for personal measurements. Data generated from Naneos Partectors have been compared with data from stationary TSI particle counter and variance between Partectors as well as between Partector and TSI particle counter was smaller than expected, 4-16% whereas 30% was expected.
Andersen et al. (2021)[106] *Period*: May-June 2018 *Location*: Military Air Force base, Denmark (location not further specified) JP-8 kerosene jet fuel used Study population: 79 self-reported healthy non-smoking employees	*UFP measurements*: job title (workplace; number of workers), mean (SD) • Crew chief (hangar; n=2/17)*: 13,800 (88,100) UFP/cm^3 • Aircraft engineer (workshop; n=1/14) 1,900 (1,700) UFP/cm^3 • Office workers (n=6/31): 2,400 (9,200) UFP/cm^3 Measurement data limited to 9 participants. * n=2/17 means 2 sampled out of 17 participants	*UFP sampling*: DiSCmini (4x) based on diffusion charging Detection range: 10-300 nm Sampling time: 4 hours 4 devices per sampling day, randomly distributed among participants willing to carry the devices *UFP measurements*: • Number of ultrafine particles/cm^3	In May 2017 same Air Force base was sampled: mean (SD) for the study by Bendtsen et al. (2019)[107] • Crew chief (n=1): 51,600 (330,500) UFP/cm^3. Measurement time 364 minutes, average particle size 61 nm. Difference in exposure levels probably due to monitoring time (2 hours longer), daily variations in weather and operational activities. Authors note that UFP measurements show erratic behaviour, characterised by short-time peak levels. Potentially exposed: aircraft engineers, crew chiefs, fuel operators** and munition specialists. Reference: avionics** and office workers. Only self-reported data on smoking, medication use, use of protective equipment available. ** Not included in personal UFP monitoring
Lecca et al. (2021)[108] *Period*: 16-18 March 2018 *Location*: airport in Italy (location not further specified) Cross-sectional study among 34 male operators	*UFP measurements*: mean (SD) for 33 workers • Particle number: 61,443 (351,475) particles/cm^3 • Particle size: 55.77 (25.63) nm • LDSA: 109.46 (506.38) m^2/cm^3	*UFP sampling:* DiSCmini based on diffusion charging Detection range: 10-300 nm *UFP measurements*: • Number of ultrafine particles/cm^3 • Particle diameter (mean) • Lung deposited surface area (LDSA)	*Study aim*: association between UFP and noise among airport ground staff Not mentioned in publication, but probably the same cohort as Marcias et al. (2019)[109]

Reference / study location	Results personal exposure monitoring	Sampling method & measurement range	Remarks
Bendtsen et al. (2019)[107] *Location*: commercial airport and military Air Base (same airfield as Andersen et al. (2021)), Denmark (locations not further specified)	Sampling stations were placed to measure the airborne particle concentrations (illustrations in supplementary material): Military airfield/non-commercial airfield • in the near field (stationary) ~1-2 meters from jetfighter • far field (stationary), NanoScan and OPC ~15 meters from jetfighter • in the breathing zone of flight personnel (personal monitoring), DiSCmini ~10-15 meters from jetfighter Commercial airfield • all devices placed close to the aircraft (according to illustration) Personal exposure levels were similar to stationary air concentrations, see also Table 9. crew chief n=1: 51,600 (sd=330,500) UFP/cm^3. See Andersen et al. (2021)[106] LDSA: 10.7% (take-off) to 11.5% (landing + refuelling) of the particle mass was predicted to deposit in the alveolar lung regions.	*UFP sampling military airfield/non-commercial airfield)*: Sampling in a jetfighter hangar with half elliptical roof (volume 4721 m^3) Real-time particle monitoring with an Electrical Low Pressure Impactor (ELPI; stationary device) placed in the near field, NanoScan and Optical Particle Counter (OPC) placed in the far field (at the front) and 4 DiSCmini's (portable device, based on diffusion charging) of which one device was placed in the breathing zone of flight personnel. Detection range DiSCmini's: 10-700 nm *UFP sampling commercial airport*: 4 DiSCmini's and NanoScan (portable devices, based on condensation particle counting) were used. *UFP measurements:* • Number of particles/cm^3 (PNC) • Particle diameter • Lung deposited surface area (LDSA)	*Study aim*: to assess pulmonary toxicity of aircraft emissions in mice and to compare the results with reference particles of known toxicity (carbon black and diesel engine particles) Only one of 4 DiSCmini's placed in breathing zone of the worker Andersen et al (2021)[106] refers to this study implicating that samples for this study were collected at a military airfield: *'We have previously measured UFP levels in connecting with personnel assisting the take-off and reception of aircrafts inside a hangar at a non-commercial airfield.'* Confirmed in personal communication with co-author
Marcias et al. (2019)[109] *Period*: 16-18 March 2018 *Location*: small provincial airport, Sardinia, Italy (location not further specified) Study population: 34 workers, study conducted within a cohort of airport workers.	*UFP measurements*: median (min-max) for 33 workers Number of particles (median): 2,440-13,000 particles/cm^3 Particle size range (median): 35-103 nm LDSA (median): 8.81-32.22 $\mu m^2/cm^3$ *UFP measurements per jobtitle*: (n) mean (SD) • All jobs: (n=33) 61,400 (351,400) UFP/cm^3 • Aircraft Ground Equipment personnel: (n=5) 44,500 (350,000) UFP/cm^3 • Firefighting officer: (n=9) 15,800 (54,300) UFP/cm^3 • Flight security agent: (n=7) 27,100 (88,100) UFP/cm^3 • Aviation fuel's administration staff: (n=12) 104,200 (475,600) UFP/cm^3 *LDSA:* mean (SD) in $\mu m^2/cm^3$: • *All jobs:* 109.46 (506.38) $\mu m^2/cm^3$ • *Aircraft Ground Equipment personnel:* 58.50 (307.01) $\mu m^2/cm^3$ • *Firefighting officer*: 33.33 (83.17) $\mu m^2/cm^3$ • *Flight security agent*: 94.49 (358.95) $\mu m^2/cm^3$ • *Aviation fuel's administration staff:* 174.27 (696.09) $\mu m^2/cm^3$	*UFP sampling*: DiSCmini based on diffusion charging Detection range: 10-300 nm Accuracy: ±30% Sampling time: 2.5 hours per sample, 2-3 samplings per sampling day *UFP measurements*: • Number of ultrafine particles/cm^3 • Particle diameter (mean) • Lung deposited surface area (LDSA) Sioutas Cascade impactor used for morphological and chemical analysis of particles, followed by electron microscopy: • Transmission electron microscopy (TEM) for morphology • Scanning electron microscopy (SEM) for chemical composition	Airport is probably located near the coast/sea (may influence results of UFP measurements). Activity logbook used One of the measurements failed due to malfunctioning equipment For the jobtitles of 'flight security officer' and 'aircraft ground equipment personnel' approximately 11 hours of sampling data available, for other jobtitles there was more data available

Reference / study location	Results personal exposure monitoring	Sampling method & measurement range	Remarks
Møller et al. (2014)[110] *Period*: October 2012 (8 sampling days divided over 2-week period) *Location*: Copenhagen Airport, Kastrup, Denmark	Data from 30 out of 40 workers available due to problems with measurements. *UFP measurements*: geometric mean (95% CI) • Baggage handlers (n=6): 37,000 (25,000-55,000) UFP/cm^3 • Landside security (n=2): 5,000 (2,000-11,000) UFP/cm^3 • Catering drivers (n=7): 20,000 (14,000-29,000) UFP/cm^3 • Cleaning staff (n=8): 12,000 (9,000-17,000) UFP/cm^3 • Airside security (n=7): 12,000 (8,000-18,000) UFP/cm^3	*UFP sampling*: NanoTracer based on diffusion charging Detection range: 10-300 nm Accuracy: ±30% GPS-loggers used for location.	Airport is located near the coast/sea (may influence results of UFP measurements). NanoTracer attached to belt on the hip. Catering drivers and cleaning staff possible co-exposure to diesel engine exhaust. Landside security workers were both females. Except for particles with diameters exceeding 125 nm, which appear to be overestimated by the NanoTracer there is good agreement of personal measurements with results from SMPS stationary sampler.
Buonanno et al. (2012)[111] *Period*: July-August 2011 *Location*: (military) aviation base, Italy (location not further specified) JP-8 kerosene jet fuel used	*UFP measurements*: median concentrations (min-max) • Crew chief: 25,000 (16,000-42,000) particles/cm^3 • Hangar operator: 17,000 (13,000-25,000) particles/cm^3 Particle number concentrations in the proximity of the airstrip show short term peaks during the working day mainly related to take-off, landing and pre-flight operations of jet engines.	*UFP sampling*: NanoTracer based on diffusion charging Detection range: 10-300 nm	Airport is probably located near the coast/sea (may influence results of UFP measurements). Results personal exposure measurements were higher than results from stationary monitoring, see also Table 9.

4.1.2 Polycyclic aromatic hydrocarbons and other aromatics

There is no available data on PAHs from personal monitoring in the context of aircraft emissions. See 4.2.2 and 4.3.2 for data on PAHs from stationary monitoring and biomonitoring, respectively.

Pitarque et al. (1999) investigated air concentrations of benzene, toluene and xylene in vapour samples collected using passive personal dosimeters from 39 workers at Barcelona airport. These workers assisted during charge and discharge of the aircraft, sometimes during refuelling. The authors reported 8-hour time weighted average (8h-TWA) air concentrations of 100 (sd=50), 130 (sd=10), and 130 (sd=20) µg/m^3 for respectively benzene, toluene and xylene.[112]

4.1.3 Metals

In the study by Van der Meer et al. (2024), metal concentrations of aluminium, chromium, iron, nickel, molybdenum, vanadium, zinc and zirconium were determined in inhalable particle fraction by using gravimetric analysis with ICP-MS. Most inhalable metal concentrations were below the limit of detection (i.e., 0.1 µg for chromium, nickel, vanadium, zirconium; 0.5 µg for molybdenum; 1.0 µg for zinc; 2.0 µg for aluminium; 3.0 µg for iron, see Chapter 3.1.3). In addition, only aluminium (14%), iron (15%), zinc (10%) and nickel (1%) were detected in measurements among workers with jobs in aircraft cargo transport or as external contractor (may be attributed to co-exposure).[104]

4.1.4 Sulphur dioxide

No personal monitoring or biomonitoring data are available for sulphur dioxide. There is very limited data available from stationary monitoring, see 4.2.4.

4.2 Workplace monitoring data from stationary samplers

In some situations, it is not possible to use personal samplers due to problems with accuracy, weight, size and/or power requirements of the sampler/monitor. In that case occupational exposure can also be determined by using stationary samplers, which have a fixed location in the workplace.[85]

4.2.1 Particulate matter

An overview of occupational exposure studies with stationary monitoring data of particles is shown in Table 9 and black carbon in Table 10. A few studies have also performed morphological and chemical analysis to determine the shape and composition of KEE particles.

Table 9 Overview of stationary monitoring data of particulate matter

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Ridolfo et al. (2024)[113] *Period*: 4 April-30 May 2023 *Location*: Barcelona-El Prat Airport, Catalonia, Spain	*UFP measurements*: Particle number concentrations all sizes (particle sizes 10-480 nm), averaged over 24h: • At the airport: 67,028 particles/cm^3 • Urban background: 8,406 particles/cm^3 Particle number concentrations in *nucleation mode* (particles sizes <25 nm): • At the airport: 55,718 particles/cm^3 • Urban background: 3,735 particles/cm^3 Particle number concentrations in *Aitken mode* (particles sizes 25-100 nm): • At the airport: 10,363 particles/cm^3 • Urban background: 3,831 particles/cm^3 Particle number concentrations in *accumulation mode* (particles sizes 100-480 nm): • At the airport: 948 particles/cm^3 • Urban background: 840 particles/cm^3	*UFP sampling*: Electrical Low-Pressure Impactor (ELPI+; range 6 nm-10 µm). Particle size and distribution: TSI Scanning Mobility Particle Sizer (SMPS) coupled to a TSI Condensation Particle Counter (CPC; range 10-480 nm) to measure number of particles. *Sampling site*: devices are placed in a laboratory van. The van was parked 80m north of taxiway and 250m from take-off runway. Devices inlet positioned on the roof of the van (~5m above ground).	Airport is located near the coast/sea (may influence results of UFP measurements).

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Esveld et al. (2024)[105] *Period*: May-June 2023 (7 sampling days for personal measurements) *Location*: Eindhoven Airport, Eindhoven, the Netherlands	*UFP measurements* in 2022: mean • Platform north side: 33,000 UFP/cm^3 • Platform south side: 76,000 UFP/cm^3 *UFP measurements* in 2023: mean • Platform average: 104,000 UFP/cm^3 • Aircraft stand: 110,000 UFP/cm^3 Average for total measurement period (May - June 2023): 49,000 UFP/cm^3 Maximum 8h-time weighted average (8h-TWA): 450,000 particles/cm^3	*UFP sampling*: TSI Environmental Particle Counter (detection range 7 nm-3 µm)	Same device and sampling site used during previous measurements. See table 8 for personal monitoring data
Pirhadi et al. (2020)[114] *Period*: May-October 2018 (32 sampling days with predominant wind from airport and nearby runways) *Location*: Schiphol Airport, Amsterdam, the Netherlands	*UFP measurements*: mean (standard error): Total PNC: 35,000 (615) particles/cm^3 Relative contributions to PNC: • Aircraft departures: 46.1% • Aircraft arrivals: 26.7% • Road traffic from nearby freeways: 18.0% • Ground support equipment (GSE) and local road traffic: 6.5% • Urban background: ~2.7% Contributions to mean particle sizes: • Aircraft departures and arrivals: <20 nm • Nearby freeways: 30-40 nm • GSE and local traffic: 60-80 nm. • Urban background: 150-225 nm	*UFP sampling:* particle number and size distributions using TSI Scanning Mobility Particle Sizer (SMPS) coupled to a TSI Condensation Particle Counter (CPC). *Sampling site*: 300m from 2 major runways, 2km northeast of the airport, and 500-1000m from A4 and A9 freeways.	Airport is located near the coast/sea (may influence results of UFP measurements). *Study aim*: contributions of airport activities to measured particle number concentration (PNC).
Bendtsen et al. (2019)[107] *Location*: commercial airport and military airfield/ non-commercial airfield, Denmark (location not further specified)	*UFP measurements*: average number of particles: • Full workflow: 1.220,000 UFP/cm^3 • Take-off: 7.700,000 UFP/cm^3 • Landing + refuelling: 2.670,000 UFP/cm^3 Mass concentration: average mean • Take-off: 1086 µg/m^3 • Landing + refuelling: 410 µg/m^3	*UFP sampling*: real-time particle monitoring with an Electrical Low Pressure Impactor (ELPI+) and 3 DiSCmini's placed at fixed locations. *Sampling time*: two full workflow cycles	Full workflow or whole flight cycle consists of: Plane Leaving (PL), Plane Arriving (PA) and refuelling by a Fuel Truck (FT). Personal exposure levels were similar (not reported). Military airfield or non-commercial airfield is the same location as study by Andersen et al. (2021)[106] (see also Tables 8 and 12)
Marcias et al. (2019)[109] *Period*: 16-18 March 2018 (2 sampling days) *Location*: Provincial airport, Sardinia, Italy (location not further specified)	*UFP measurements*: median (min-max) • Sampling day A: 10,200 (3,860-53,800) particles/cm^3 • Sampling day B: 13,300 (4,000-212,200) particles/cm^3 *UFP measurements*: mean (SD) • Sampling day A: 11,600 (5,360) particles/cm^3 • Sampling day B: 15,600 (14,900) particles/cm^3	*UFP sampling*: real-time detection of particle size and number using ELPI+: • Particle size (detection range: 6-10,000 nm) • Particle number (calculated) *Sampling time*: 5 hours per sample	Airport is located near the coast/sea (may influence results of UFP measurements). See table 8 for personal monitoring data.

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Marie-Desvergne et al. (2016)[103] *Period*: March-April 2012 *Location*: Marseille-Provence Airport, Marseille and Roissy-Charles de Gaulle Airport, Paris, France	*UFP measurements*: median (min-max) • Apron workers (n=248): 150,000 (10,000-21.000,000) particles/cm^3 • Administrative workers (n=210): 8,300 (617-23,000) particles/cm^3 Significant difference between particle concentrations on the apron and inside office buildings (p-value <0.001) Peak concentration levels correlated with aircraft activity on the apron Significantly smaller particle sizes found on the apron compared to office buildings. Geometric mean particle size 17.7 nm versus 23.7 nm (p-value <0.001)	*UFP sampling*: real-time particle collection using ELPI+ (sampling range 30 nm-10 µm). Number of particles using Condensation Particle Counter (CPC), range 5 nm-3 µm. Particle size using Fast Mobility Particle Sizer (FMPS) (range 30 nm-10 µm. *Sampling site:* For apron workers samples were taken 3-10m from aircraft parking. *Sampling times*: apron workers: 374 minutes administrative workers: 250 minutes	Marseille Provence airport is located near the coast/sea (may influence results of UFP measurements). UFP measurements were conducted once at 3 representative (for exposure groups) workplaces, either in Marseille or Paris. Particles were characterised using SEM-EDS, particles consisted of carbon, and some had trace amounts of sulphur.
Ellermann et al. (2012)[115] *Period*: 2009-2011, additional measurements Aug-Dec 2010 and Jan-June 2011 *Location*: Copenhagen Airport, Kastrup, Denmark	*UFP measurements*: period August-December 2010 Apron: • Total: 31900 particles/cm^3 • 6-40 nm: 27900 particles/cm^3 • 40-109 nm: 3100 particles/cm^3 • 109-700 nm: 900 particles/cm^3 Reference HCAB: • Total: 16100 particles/cm^3 • 6-40 nm: 9900 particles/cm^3 • 40-109 nm: 4700 particles/cm^3 • 109-700 nm: 1600 particles/cm^3 Period January-June 2011 Apron: • Total: 38600 particles/cm^3 • 6-40 nm: 32600 particles/cm^3 • 40-109 nm: 4600 particles/cm^3 • 109-700 nm: 1400 particles/cm^3 Reference HCAB: • Total: 13400 particles/cm^3 • 6-40 nm: 7800 particles/cm^3 • 40-109 nm: 4100 particles/cm^3 • 109-700 nm: 1400 particles/cm^3	*Sampling method for UFP not specified* Results from stationary monitoring data were compared to a reference, H.C. Andersens Boulevard (HCAB), which is a high-traffic street in Copenhagen	Airport is located near the coast/sea (may influence results of UFP measurements). The two most important sources to air pollution at the apron were handling vehicles, the airplanes main engine and Auxiliary Power Units (APU).

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Buonanno et al. (2012)[116] *Period*: July-August 2011 *Location*: (military) aviation base, Italy (location not further specified) JP-8 kerosene jet fuel was used	Number of particles: mean • Downwind side: 6,500 particles/cm^3 Mean particle size: 25 nm Total carbon content determined in inhalable particle fraction: 65%-80% carbon content. • 10-35% EC • 65-90% OC	*UFP sampling*: TSI condensation particle counter (CPC) used for number of particles. For particle concentrations and size distribution: TSI Fast Mobility Particle Sizer (FMPS), TSI SMPS, and TSI aerodynamic particle sizer (APS) *Sampling site*: 3 stationary measurement locations: • 500m downwind of the airstrip • Vicinity of the airstrip • Hangar near ground operations	Airport is located near the coast/sea (may influence results of UFP measurements). Results have been influenced by weather conditions and location (near the coast/sea). See table 8 for personal monitoring data.

Table 10 Overview of stationary monitoring data of black carbon (BC)*

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Ridolfo et al. (2024)[113] *Period*: 4 April-30 May 2023 *Location*: Barcelona-El Prat Airport, Catalonia, Spain	*Black carbon concentrations*: At the airport: 916 ng/m^3 Urban background: 366 ng/m^3	*Black carbon measurements*: Aethalometer. *Sampling site*: devices are placed in a laboratory van. Parked 80m north of taxiway and 250m from take-off runway. Devices inlet positioned on the roof of the van (~5m above ground).	Airport is located near the coast/sea. Harbour and industrial activities may have influenced the results
Pirhadi et al. (2020)[114] *Period*: May-October 2018 (32 sampling days) *Location*: Schiphol Airport, Amsterdam, the Netherlands	*Black carbon concentrations*: mean Sampling site: 0.6 µg/m^3 Range (min-max): 0.1-5.3 µg/m^3	*Black carbon measurements*: Aethalometer (portable). LOD (calculated): 0.095 µg/m^3 *Sampling site*: 2km northeast of the airport, 300m from 2 major runways and 500-1000m from A4 and A9 freeways.	Airport is located near the coast/sea Sampling days selected with predominant wind from airport and nearby runways Sampling site is impacted by both emissions from nearby airport activities as well as road traffic
Targino et al. (2017)[117] 12 airports in Europe and South America and 41 flights. • *Europe*: Amsterdam, the Netherlands; Paris, France; Milan, Italy; Florence, Italy. • *South America*: Montevideo, Uruguay; Porto Alegre, Brasil; Curitiba, Brasil; Londrina, Brasil; Sao Paulo (2x), Brasil; Rio de Janeiro (2x), Brasil.	*Black carbon concentrations*: mean During boarding/disembarking: 3.78 µg/m^3 Large variability in black carbon concentrations, which generally appeared to be highest at transit to/from aircraft.	*Black carbon measurements*: micro aethalometer used to measure black carbon concentrations. Device was placed in/on a *backpack* (to be held on passenger's lap during flights).	*Study aim:* to quantify the variability in black carbon concentrations at airport areas commonly visited by passengers. Only indirectly occupational exposure. Selected passengers did not smoke.

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Westerdahl et al. (2008)[118] *Period*: April 2003 (4 sampling days) *Location*: Los Angeles Airport, Los Angeles, USA	*Black carbon concentrations*: mean • At the taxiway: 1.8 µg/cm^3 • At the airport terminal: 3.8 µg/cm^3 • At nearby freeway: 22.7 µg/cm^3 Black carbon concentrations increased during take-off operations from 800 to 9550 ng/m^3	*Black carbon measurements*: Aethalometer. *Sampling site*: Measurements taken at various locations in the vicinity of the airport to determine the spread of airport emissions in up- and downwind.	Airport is located near the coast/sea. Measuring devices are placed on a motor vehicle, measurements were taken parked and driving.

* Black carbon (commonly used in aviation) is an assumed equivalent of elemental carbon (EC or soot). BC and EC have different measurement methods (see Chapter 3).

4.2.2 PAHs and other aromatics

Although no personal monitoring data is available for PAHs, there are several studies that measured vapour- or particle-bound PAH using stationary equipment. An overview of the measurement data is shown in Table 11.

Table 11 Overview of stationary monitoring data of PAHs

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Bendtsen et al. (2019)[107] *Location*: commercial and non-commercial airport, Denmark (location not further specified)	*PAH measurements:* mean Total for commercial airport: 0.081 mg/g Total for non-commercial airfield: 0.05 mg/g Higher concentrations of benzo[a]pyrene were found in particles from commercial airport (0.005 mg/g) and non-commercial airfield (0.009 mg/g) compared to standard reference material for DEE particles (0.0008 mg/g in NIST2975).	*PAH sampling:* 16 particle-bound PAHs extracted with cyclohexane and analysed by GC-MS	Findings were compared to standard reference material for DEE particles, NIST2975 (light duty vehicle) and NIST1650 (heavy duty vehicle). NIST standard reference materials are intended for evaluations of analytical methods
Westerdahl et al. (2008)[118] *Period*: April 2003 *Location*: Los Angeles Airport, Los Angeles, USA	*PAH measurements*: mean • At the taxiway: 50.1 ng/m^3 • At the nearby freeway: 47.0 ng/m^3 PAH mean concentrations increased during take-off operations from 37 to 124 ng/m^3	*PAH Sampling*: particle-bound PAHs measured using a photoelectric aerosol sensor. *Sampling time*: 4 sampling days *Sampling site*: devices are placed on a motor vehicle, measurements were taken parked and driving.	Airport is located near the coast/sea.

Reference / study characteristics	Stationary monitoring	Sampling method	Remarks
Iavicoli et al. (2006)[119] *Period*: January-February 2005 *Location*: Commercial airport, Italy (location not further specified)	*PAH measurements*: • Naphthalene: 130-13,050 ng/m^3 • 2-methylnaphthalene: 64-28,500 ng/m^3 • 1-methylnaphtalene: 24-35,300 ng/m^3 • Biphenyl: 24-1,610 ng/m^3 • Fluoranthene: 54.2 ng/m^3 • Benzo[a]pyrene: 8.6 ng/m^3 Results are difficult to interpret due to huge variations from day to day	*PAH sampling*: 25 vapour-phase and particle-bound PAHs including biphenyl *Sampling time*: 12 sampling days (12 air samples for 24h per sample) *Sampling site*: 3 different locations. Terminal C (baggage unloaded from transport vehicles onto conveyor belts) Sierra C (runway with heavy plane and motor vehicle traffic) Terminal C departure area	Measurements include both motor vehicle exhaust as well as aircraft exhaust. Highest levels measured for 24h Information on weather conditions available. Unclear how changes in weather conditions may have affected the results of PAH measurements
Cavallo et al. (2006)[120] *Period*: January-February 2005 *Location*: Leonardo Da Vinci airport, Rome, Italy	*PAH measurements*: mean • Apron: 27,703 ng/m^3 • Airport building: 17,275 ng/m^3 • Terminal departure area: 9,494 ng/m^3 Mean concentrations divided in 2-3 ringed PAH and 4-6 ringed PAH: *2-3 ringed PAH*: • Apron: 27,690 ng/m^3 • Airport building: 17,424 ng/m^3 • Terminal departure area: 9,481 ng/m^3 *4-6 ringed PAH:* • Apron: 0.013 ng/m^3 • Airport building: 0.033 ng/m^3 • Terminal departure area: 0.013 ng/m^3	*PAH sampling*: 23 vapour-phase PAHs and particle-bound PAHs. *Sampling time*: 5 working days	Measurements during 24h of 5 workdays at the airport apron, building and terminal/office area Possibly the same study location as study by Iavicoli et al. (2006)[119] Study included exposure measurements among airport personnel (n=41 exposed workers (n=24 high exposed and n=17 medium exposed) and n=31 non-exposed office workers). At the end of the work shift (8 h) of the working week urine samples were collected for OH-pyrene analysis. No significant differences between exposed and controls were found (p-value=0.978)
Childers et al. (2000)[121] *Period*: 4-6 May 1999 *Location*: Savannah Air National Guard base, Savannah, Georgia, USA	*PAH measurements*: mean total PAH concentrations in integrated air samples (vapour and particle-bound PAHs) • hangar background: 601.1 ng/m^3 • hangar taxiing: 1,025.4 ng/m^3 • engine test: 2,802.7 ng/m^3 • engine running on/off: 6,795.3 ng/m^3 • diesel-fuelled aerospace ground equipment: 9,811.1 ng/m^3	*PAH sampling*: real-time monitor for particle-bound PAHs based on photoelectric aerosol sensor (PAS) handheld device. For analysis of individual PAH integrated air samplers were used (not during flight-related exercises). *Sampling site*: real-time concentrations of particle-bound PAHs: • in a break room, • downwind from an aircraft during an engine run-up test, • in a maintenance hangar, • in an aircraft during cargo-drop training, • downwind from aerospace ground equipment, • in an aircraft during engine running on/off.	During flight-related exercises, PAH concentrations were 10-15 times higher than the ambient air. Real-time monitor responses generally followed integrated air sampler trends. Aircraft C-130H cargo bay was used for all aircraft related measurements and flight-related exercises

4.2.3 Metals

In the study by Ridolfo et al. (2024), metal concentrations were determined using ICP-MS and ICP-Atomic Emission Spectrometry (ICP-AES). Results show elevated concentrations of certain elements, including aluminium, iron, chromium, copper, molybdenum, manganese, lead, tin, and antimony, were observed at the airport, with aluminium exhibiting the finest size distribution and the most pronounced disparity compared to urban background levels. However, nearby metallurgical activities and shipping emissions may have significantly influenced the observed concentration of these elements.[113]

In the study by Bendtsen et al. (2019), metal concentrations were determined using ICP-MS. The general metal content in KEE particles from a commercial airport, non-commercial airfield and DEE particles (NIST2975) were similar. However, higher concentrations of magnesium, aluminium, copper, zinc, strontium and lead were found in particles from the commercial airport compared to particles from non-commercial airfield and DEE particles.[107]

In the study by Buonanno et al. (2012) concentrations of 40 elements including metals were determined in inhalable particles fraction. Chemical analysis showed that all the elements found can be attributed to long-range transport from the sea, which is in close vicinity of the airstrip.[116]

4.2.4 Sulphur dioxide

In the study by Ridolfo et al (2024), SO_2 has been measured using a fluorescence monitor. Concentrations for SO_2 were slightly higher inside the airport area (3.96 µg/m^3) compared to urban background (2.44 µg/m^3), possibly due to the proximity of the airport to the coast and shipping routes to and from the harbour.[113]

4.3 Biomonitoring of workplace exposures

The internal exposure to a substance can be assessed by measuring the substance itself, its metabolites (breakdown products) and conjugates in biological materials such as urine, blood or EBC.[94]

4.3.1 Particulate matter

A French study by Marie-Desvergne et al. (2016) used EBC to determine exposure to UFP (see also Table 9 for study description and stationary monitoring data). EBC was collected for 458 French airport workers working either on the apron (jobs not further specified) or in the offices. The apron workers were significantly higher exposed and to smaller sized particles, compared to the office workers. The EBC based particle size distribution was not influenced by gender, age, smoking status or exposure group. All participants had a main peak in particles of approximately 460 nm in size. A few participants also showed a second peak in the size distribution near 100 nm.[91]

4.3.2 PAHs and other aromatics

One study with biomonitoring data on PAHs is available, which is summarised in Table 12.

Overall, low PAH exposure levels were found for industrial hygiene measurements (e.g., silicone bands, skin wipes) as well as for biomonitoring (urinary excretion). Regarding the low levels of urinary OH-PAHs, the authors report that timing of sampling may be important as the window of excretion of OH-PAHs following exposure is quite narrow. Overall, the authors note that limitations of the study were the cross-sectional design, the small sample size, the complexity of the exposure and the fact that the reference group had had some background exposure.[106] See also Table 8 for results on personal UFP measurements.

Table 12 Industrial hygiene and biomonitoring data for PAHs

Reference / study characteristics	Biomonitoring data	Remarks
Andersen et al. (2021)[106] Period: May-June 2018 Location: Military Air Force base, Denmark (location not further specified) JP-8 kerosene jet fuel used Potentially exposed workers: aircraft engineers, crew chiefs, fuel operators, munition specialists. Reference: avionics, office workers.	Total urinary OH-PAHs (µmol/mol creatinine): average mean (SD). *Exposed workers*: 3.29 (1.7) µmol/mol creatinine *Reference*: 4.76 (3.9) µmol/mol creatinine Difference exposure groups *p-value* <0.004 Exposure levels of total-PAHs and OH-PAHs did not differ between exposure groups or job function. Uncertainties about results due to relatively large fraction of imputed data. For instance, only a few samples of naphthalene were above LOQ. Not clear if participants used personal protective equipment (PPE) during their work.	PAH measurements in silicon bands and skin wipes were also carried out. Total-PAH (ng/g) in *silicon band* per day: average mean (SD). *Exposed workers* (n=41): 479 (683) ng/g *Reference* (n=36): 473 (503) ng/g Difference in exposure groups *p-value* = 0.783 Total PAHs (ng/cm^2) per 1 hour using *skin wipes*: average mean (SD). *Exposed workers*: 2.05 (3.02) ng/cm^2 *Reference*: 2.10 (3.20) ng/cm^2 Difference exposure groups *p-value* = 0.718 There is no difference between exposure groups in total PAHs assessed through silicone bands and skin wipes. Except for fluorene in silicone bands driven by fuel operator's exposure (but then there is no association with urinary marker of fluorene).

4.3.3 Metals

In the French study by Marie-Desvergne et al. (2016), exposure to metals was investigated in EBC among airport workers (see also Table 9 and 4.3.1.). EBC was collected from 458 airport workers from 2 French airports, Marseille-Provence Airport and Roissy-Charles de Gaulle Airport in Paris, working directly on the apron (exposed, n=248) or in the offices (low or non-exposed, n=210). A multi-elemental analysis was used to measure Na, Al, cadmium (Cd), and Cr in EBC. Aluminium, cadmium, and chromium were detected in 19%, 22% and 79%, respectively, of all subjects' EBCs. No significant influence of gender, age or smoking status was found for these metals. A significantly higher concentration of cadmium was found among apron workers (mean=0.174 µg/l (sd=0.326)) in comparison with office workers (mean=0.108 µg/l (sd=0.106)).[103]

Touri et al. (2025)[102] conducted a prospective cohort study among the same group of airport workers as the study by Marie-Desvergne et al. (2016) (see also Table 8). Exposure to certain metals (chromium, cadmium and

aluminium) was determined in EBC as well as in urine samples. Most of the metals in EBC were below the levels of quantification (LOQ; only 1.9% to 4.8% above LOQ); there were no significant differences between the exposure groups. Also, in the urine samples, most metal concentrations for chromium and aluminium were below the limit of quantification. However, for cadmium significantly higher concentrations were found in urine samples of mechanics (p=0.0022) and apron workers (p=0.0074) compared to terminal workers (only graphically). The authors note that EBC may not consistently reflect exposure, and the lack of metal-containing particles suggests lung deposition of metals, rather than exhalation.[102]

4.3.4 Sulphur dioxide

No biomonitoring data are available for sulphur dioxide.

4.4 Summary on occupational exposure to kerosene engine exhaust

Several studies investigated occupational exposure to KEE particles.[104-106,109,110,113,114,116] However, in some of these studies the exposure estimates may have been influenced by the airport location (e.g., near industrial activities, near the coast) or the presence of other motor vehicles and road traffic at the airport or in the surroundings of the airport.[103,104,109,110,113,114,116]

In general, the highest particle number concentrations have been reported in stationary monitoring (mostly continuous measurements) data at the airfield and airport apron. Air concentrations of 110,000 particles/cm^3 at the aircraft stand,[105] and 150,000 particles/cm^3 at the apron[103] have been reported. The highest exposure estimates based on personal monitoring (mostly limited to a few hours) were seen for jobs such as flight officer of 77,000 particles/cm^3 (arithmetic mean), baggage handler of 37,000 particles/cm^3 (geometric mean), or crew chief of 25,000 particles/cm^3 (median).[105,110,116] These results should be carefully interpreted because of the different metrics in which the results are expressed. Furthermore, it should be noted that UFP measurements are particularly sensitive to varying conditions, such as high number of flight activities, weather conditions and seasonal influences. The relatively smaller (i.e. fewer flight operations) provincial airports and military airfields generally report lower particle number concentrations.[106,109]

Several studies with occupational monitoring data show that airport workers are particularly exposed to small sized particles compared to some other sources like road transportation.[103,109,113-116] The study by Ridolfo et al. (2024) showed that the highest particle concentrations were found at Barcelona-El Prat Airport (55,718 particles/cm^3 with particle sizes <25 nm (nucleation mode)) compared to urban background (3,735 particles/cm^3 for the same particle size). The mean particle number concentrations at the airport decreased with growing particle sizes to 948 particles/cm^3 for

particles in the range 100-480 nm size, almost as low as urban background concentrations (840 particles/cm^3).[113]

Furthermore, several studies have measured the amount of EC and OC in respirable or inhalable particle fractions. These studies indicate that most KEE particles contain OC and a substantial amount of these particles also contain EC.[104,116] There are also a few studies which measured BC concentrations using an aethalometer. The results indicate that BC air concentrations are higher at an airport and in the close vicinity of an aircraft,[113,117] but high concentrations are also found at nearby freeways, possibly caused by exhaust emissions from diesel cars.[118]

Based on stationary monitoring data for PAHs, the highest air concentrations for total PAHs were found on the airport apron.[118,120,121] In addition, in the study by Childers et al. (2000) authors reported that particle-bound PAH concentrations were 10-15 times higher during flight-related operations compared to ambient air concentrations.[121] Regarding the composition of particle samples collected at the apron, the most abundant species of vapour-phase PAHs in KEE were naphthalene and alkyl-substituted naphthalenes. Particle-bound PAHs, such as fluoranthene, pyrene, and benzo[a]pyrene were also found in some samples. The most dominant PAH in all exposure scenarios was naphthalene,[119-121] which is in accordance with the relative high levels of naphthalene in kerosene jet fuel (see Chapter 2) and consequently also in kerosene engine exhaust. In the study by Bendtsen et al. 2019 the authors reported higher concentrations of benzo[a]pyrene in particles collected at a commercial airport and non-commercial airfield in comparison with standard reference material representing DEE particles (NIST2975).[107]

In general, workplace monitoring data on metals and sulphur dioxide is limited and inconclusive. Also, biomonitoring data on workplace exposures to particles, PAHs and metals is limited and inconclusive.[102,103,106]

5 Mechanisms of toxicity

In this chapter, the toxicokinetics and underlying mechanisms of toxicity of KEE particles with adsorbed PAHs, metals, and sulphur compounds are described.

5.1 Toxicokinetics of KEE particles

How many KEE particles are inhaled into the human body depends on the properties of these particles, the source strengths of the emissions, the speed and direction of air movement near the body, breathing rate, and whether breathing is through nose or mouth. In addition, the site of deposition, or probability of exhalation, mainly depends on the properties of the particle and breathing pattern. It is generally considered that respirable particles (the mass fraction of inhaled particles that can reach the unciliated airways), like KEE particles, consist of particles with a D_{50} (aerodynamic diameter) of 4 µm or smaller which can reach the alveolar region of the lungs. Larger particles are less likely to reach the alveolar region and are more prone to be deposited higher up in the airways.[74] The smaller the size of the particles, the deeper they can penetrate into the respiratory system (see Figure 5). KEE particles are characterised by high concentrations of small sized particles (~20nm and smaller). Particles of

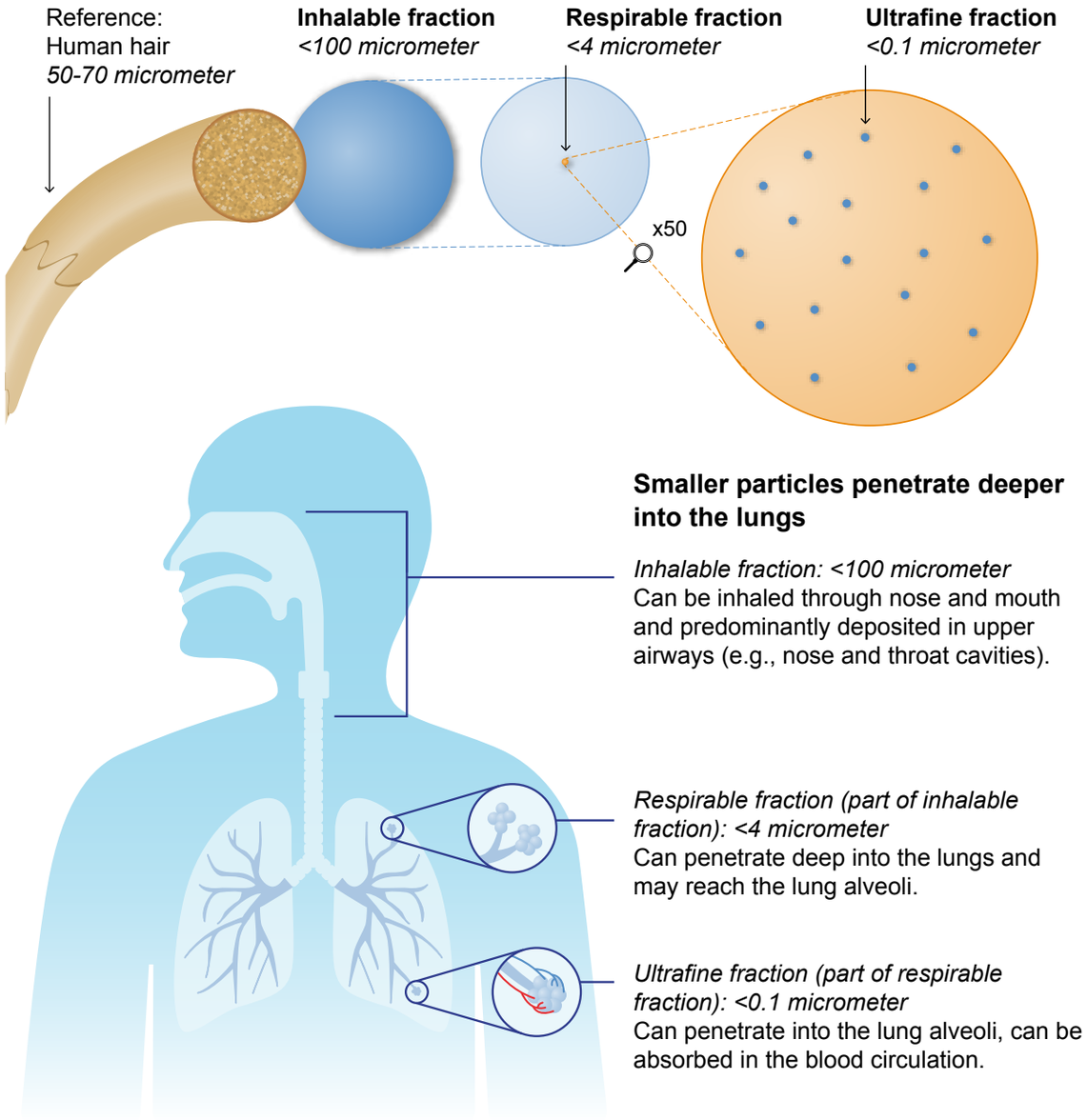


Figure 5 Particle sizes and their possibilities to reach specific lung regions

Adapted from Gezondheidsraad (2021)[87]

~20 nm in size have the highest deposition efficiency in the alveolar region (~50%); in tracheobronchial and nasopharyngeal regions the deposition efficiency is estimated at ~15%.[122]

Particle size is not static upon inhalation. Particle growth may also occur within the human respiratory system by both condensation and adsorption of water vapour and by particle agglomeration, until the particle reaches an equilibrium.[44,123] The overall toxicological consequence of particle growth and 'aging' of exhaust particles is unclear, since some components may be altered in more toxic forms and others in less toxic forms.[37]

In contrast to larger-sized particles, UFP, such as KEE particles, may translocate readily to extrapulmonary sites and reach other target organs through various transfer routes and mechanisms.[122] UFP can, upon inhalation, reach the alveoli and penetrate biological membranes, enabling them to pass into the systemic blood circulation and penetrate all organ systems including the brain, placenta and nervous system.[124-126] The presence of soluble compounds adsorbed to these particles may play an important role in the diffusion across membranes.[127]

From the tracheobronchial region, particles are cleared by mucociliary clearance and removed into the gastrointestinal system and excreted in faeces. From the alveolar region, particles are cleared by phagocytosis by alveolar macrophages and subsequently removed into the conducting airways followed by mucociliary clearance.[122]

5.2 Effect of particle-adsorbed substances on toxicity

Because they are small, KEE particles have a relatively large surface area (total surface area per unit of mass) and high surface reactivity, which enables them to adsorb greater quantities of hazardous metals, PAHs, and other organic compounds which can also generate oxidative stress.[26,36,122] For instance, particle-bound PAHs can generate the formation of reactive metabolites (e.g., epoxides), which may cause DNA adducts.[128]

It is even suggested that the organic compounds adsorbed to soot particles are partly responsible for the toxicity.[127] An in vitro study with human alveolar epithelial cells, using a novel thermophoresis-based air-liquid-interface (ALI) exposure system, investigated the toxicity of combustion-generated aerosols containing black carbon (soot particles) in various stages (e.g., fresh particles, aged particles) and with adsorbed organic material (e.g., sulfuric acid, hydrocarbons). The study demonstrated that adsorbed organic material attached to soot particles may increase its toxicity.[129] Another in vitro study with rat alveolar epithelial cells investigated the effect of metals, such as copper and iron (both are present in KEE), attached to soot particles. The results indicate a major contribution to the surface reactivity, increased cytotoxicity, increased release of pro-inflammatory cytokines, and increased release of macrophages, eventually leading to oxidative stress and DNA damage.[130] In conclusion, limited evidence suggests increased toxicity of soot particles with adsorbed substances. Further research is needed to confirm the effects of

particle-bound substances (both metals and organic compounds) on the toxicity.

The animal study by Bendtsen et al (2019)[107] in mice indicate that the toxicity of combustion-generated particles such as KEE particles is mainly driven by the presence of a carbon core (i.e., soot). Adsorbed organic substances, such as PAHs, may contribute to the toxicity of these particles.

5.3 Inflammation and oxidative stress

Deposited particles can induce health effects by causing oxidant-mediated cellular damage, resulting from reactive oxygen species (ROS) production and oxidative stress.[131,132]

Particles can induce these health effects via several modes of actions. Firstly, reactions on the particle surface can induce ROS formation resulting in intracellular oxidative stress, lipid peroxidation, and increased intracellular calcium.[122,123,132] Secondly, release of transition metals from particles or organic compounds can also cause ROS formation, (intracellular) oxidative stress, increased intracellular calcium, as well as the activation of cell surface receptors.[2,122,130-133] In vitro studies indicate that the underlying mechanisms, by which UFP induce effects, include oxidative stress-related changes of gene expression and cell signalling pathways resulting in inflammatory processes.[122,132] Lastly, the phagocytic capacity is insufficient to cope with high concentrations of particles. As a result, high concentrations of particles can damage airway epithelial cells and macrophages.[122,123,128,131,132]

Also, pulmonary exposure to combustion-generated particles may induce acute phase responses (i.e., local and systemic inflammatory and repair processes that accompany inflammation) and dose-dependent cytological changes in bronchoalveolar lavage (BAL) derived cell composition, which could indicate an exacerbated inflammatory response.[107]

5.4 Lung toxicity

Particles, such as KEE particles, can induce the formation of ROS, primarily superoxide (O_2-) and hydroxyl (OH-) radicals, and a subsequent inflammatory response in the lungs. In addition, adsorbed PAHs, other organic compounds and metals may produce ROS as well through redox chemistry both outside and inside the lung cells. ROS may also be produced by alveolar macrophages during particle phagocytosis.[128,131]

5.5 Cardiovascular toxicity

Several pathways have been suggested for the mechanisms of cardiovascular effects of combustion-generated particles. Firstly, inflammatory mediators (e.g., cytokines, acute phase proteins, activated inflammatory cells) released from the lungs may end up in the systemic circulation. These inflammatory mediators may affect the cardiovascular system directly, or indirectly by increasing the liver production of coagulation factors, or by affecting lipoprotein function. Secondly, particles or particle constituents may potentially translocate from the lungs into the systemic blood circulation and act directly on the vascular system with a

central role for ROS and oxidative stress in causing cardiovascular effects (e.g., by promoting systemic inflammation, stimulating vasoconstriction, promoting atherosclerotic plaque instability). Thirdly, particles may disturb the autonomic nervous system balance or heart rhythm by interaction with nerves or lung receptors.[128,134]

5.6 Genotoxicity

Particles, such as KEE particles, can induce genotoxicity either directly or through an inflammatory response in the lung followed by release of inflammatory mediators, apoptosis, generation of ROS and reactive nitrogen species (RNS), oxidative stress, ultimately resulting in DNA damage. These particles can easily adsorb metals, PAHs and other organic compounds, due to their large surface area. Some of these particle-bound PAHs and their derivatives or particle-bound metals can generate ROS directly, by auto-oxidation and redox-cycling, which may cause bulky DNA adducts (see Chapters 6 and 8).[26,128,135]

6 Mutagenicity

Available studies on genotoxicity and mutagenicity (an indicator of genotoxicity) of KEE are summarised below. Study summaries can also be found in Annex D of this advisory report.

6.1 Human data

Four epidemiological studies on genotoxicity (i.e., DNA damage) of KEE exposure in humans were included (see Table D1 in Annex D).
These studies investigated chromosomal and DNA damage using the micronucleus (MN) assay, sister chromatid exchange (SCE) and/or comet assay.

Andersen et al. (2021)[106] conducted a cross-sectional study among employees (n=79) of a military Air Force base (~700 employees in total) in Denmark, investigating lung function, inflammatory markers in plasma, and genetic damage in peripheral blood cells. Self-reported information on working history, personal protective equipment (PPE) use, health history, medication use, lifestyle and anthropometric data was available. Exclusion criteria were self-reported information on smoking, pregnancy and drug or alcohol misuse. Aircraft engineers (n=14), crew chiefs (n=17), fuel operators (n=6) and munition specialists (n=5) were considered potentially exposed (n=42) to fuel vapours, lubricants and jet exhaust. The reference group consisted of military staff working as office workers (n=31) or avionics (n=6) at the same base. See Chapter 4 for details on exposure estimates of UFP (Table 8) and PAHs (section 4.3.2). DNA and chromosome damage were assessed using a comet assay on peripheral blood mononuclear cells (DNA damage) and a MN assay on transferrin-positive peripheral blood reticulocytes (chromosome damage). No effects of exposure were found for biomarkers of systemic inflammation, genetic damage or lung function. No increases in micronuclei frequency or DNA strand breaks were found for exposed employees.[106] The committees noted that exposed and reference employees worked at the same military Air Force base, which means that the reference group may also have been exposed. The results for urinary PAH levels in Table 12 (Chapter 4.3.2 of this advisory report) indeed show higher levels for the reference group compared to the exposed group. Self-reported information on smoking, drug and alcohol misuse was used as exclusion criterion, which may have led to reporting bias. Furthermore, this study included small numbers of participants and exposure measurements, which may have resulted in imprecise estimates. Possible co-exposure to other carcinogenic substances could not be excluded.

A cross-sectional study by Cavallo et al. (2006)[120] investigated genotoxic effects among airport workers (n=41). Workers were categorised into three exposure groups, according to their working-time spent in the vicinity of in-service aircraft at the airport apron area: non-exposed n=31 (e.g., office

workers), medium-exposed n=17 (e.g., security staff, cleaning staff) and high-exposed n=24 (e.g., baggage handlers, aircraft towing). Information on clinical history, working history, and lifestyle (e.g., smoking, diet, alcohol use) was obtained through a medical-administered questionnaire. PAHs were sampled and analysed using air samples collected at three locations, and urinary OH-pyrene collected at the end of the work shift, used as marker of total PAHs adsorbed dose (see Chapter 4, Table 11 for exposure estimates). Results of urinary OH-pyrene analyses showed no difference between medium and high exposed and non-exposed airport workers (p-value=0.978). Genotoxic effects and early direct-oxidative DNA damage were evaluated by MN, by formamidopyrimidine DNA glycosylase-modified (Fpg-modified) comet assay on lymphocytes and exfoliated buccal cells (direct target tissue for inhalable substances), by chromosomal aberrations (CA), and by SCE analyses. For the comet assay, tail moment (the product of comet relative tail intensity and length) values from Fpg-enzyme treated cells (TM_{enz}) and from untreated cells (TM) were used as parameters of oxidative and direct DNA damage, respectively.

The MN assay did not show statistically significant differences between exposed and non-exposed workers in exfoliated buccal cells (p-value= 0.150), or in the lymphocytes (p-value=0.06), although a non-significant increase was found for high-exposed workers compared to the non-exposed group. The comet assay showed an increase in DNA strand breaks for exposed workers versus non-exposed workers of mean TM and TM_{enz} in both exfoliated buccal cells (TM 118.87 versus 68.20, *p-value* = 0.001; TM_{enz} 146.11 versus 78.32, *p* <0.001) and lymphocytes (TM 43.01 versus 36.01 *p* = 0.136; TM_{enz} 55.86 versus 43.98, *p* = 0.003). Exposed workers showed an increase of total structural CA (10.21% versus 7.73%, p = 0.014), as well as a higher mean value of SCE frequency compared to non-exposed workers (4.6 versus 3.8, p-value=< 0.001).[120] The committees note that the study included a small number of participants, resulting in low statistical power. Furthermore, this study lacks information on participation rate, and inclusion and exclusion criteria for selection of participants. The committees also note that exposure related differences in MN, strand breaks and CA may have been masked by age differences between the three groups.

Pitarque et al. (1999)[112] investigated genetic damage in peripheral blood lymphocytes among 39 male workers exposed to engine exhausts and petroleum derivatives at Barcelona airport. The reference group consisted of 11 non-exposed men working at the University Campus. Small differences were found in mean comet length between exposed (mean = 45.51, standard deviation (sd) = 1.55, p-value = <0.05) and non-exposed (39.25, sd=1.78). An increased genetic damage index was reported for exposed workers (1.11, sd=0.06) compared to non-exposed workers (0.77, sd=0.12). No statistically significant increases were found for SCE or MN. However, in exposed workers a significantly smaller amount of binucleated cells was shown with MN compared to the non-exposed workers.[112] The committees notes that there is no information on the timing of blood sampling versus

work shift or on the timing of exposure measurements. Information on confounders and potential exposure at the workplace was gathered through a questionnaire. However, there is no further reference of the type of information and how this information was retrieved (i.e., self-reported or otherwise). Furthermore, it should be noted that the non-exposed group consisted of 11 healthy men from the Barcelona Campus (different location) with no occupational exposure to petroleum derivatives or other potentially genotoxic agents. The non-exposed workers had a mean age of 34.8 years whereas the exposed workers had a mean age of 47.9 years. Also, 37.5% of the non-exposed workers were smokers compared to 56.4% of the exposed workers. Smoking is known to cause increased SCE and MN formation.

Lemasters et al. (1997)[136] investigated genotoxic effects related to exposure to fuel (mainly JP-4) and solvents among military personnel at a military base. The researchers conducted a prospective repeated measurement study in which each participant would serve as their own control (to reduce variability due to individual differences). Participants were eligible if they were 50 years of age or younger and had not worked with chemicals in the previous 12 months. The inclusion criterium selected 73 eligible participants of which 58 enrolled into the study. Information on work history, medical history, smoking, alcohol and caffein use were retrieved by questionnaires. There were 4 job-exposure groups selected: aircraft sheet metal workers (n=6); aircraft painters (n=6); jet fuelling operations (n=15); and flight line crew (n=23) which included ground crew and jet engine mechanics. The flight line crew group was the only exposure group with potential exposure to jet exhaust. Exposure measurements included full 8-hour work shift monitoring (5-7 compounds analysed) for 3 consecutive days, and exhaled breath sampling on the third day. Measurements were performed before the start of the exposed work activities and repeated at 15- and 30-week intervals during exposure. After 30 weeks of exposure, no increases in MN frequency were observed for the flight line crew. Also, no increases in SCE compared to prior exposure levels were observed for flight line crew. However, for sheet metal workers and aircraft painters SCE was increased.[136] The committees note that the study primarily aimed to investigate genotoxicity effects due to exposure to solvents and fuels rather than to engine exhaust. This resulted in only the flight line crew (n=23) being exposed to jet exhaust, but these workers were also exposed to fuel, solvents and possibly paint. This could have biased the results of this study in relation to effects of exposure to jet exhaust. Furthermore, due to the small number of participants, particularly considering only 23 workers were exposed to jet exhaust, the power of the study to detect genotoxic effects is limited.

6.2 Animal data

One in vivo study that investigated DNA damage of KEE was included (see Table D2 in Annex D).

Bendtsen et al. (2019)[107] investigated pulmonary toxicity of aircraft particle emissions by intratracheal instillation in the airways of mice (212 female C57BL / 6Tac mice) and compared the results with reference particles of known toxicity (e.g., diesel particles NIST2975 (diesel particles from a light duty vehicle), carbon black Printex90 nanoparticles, and published reference data on NIST1650 (diesel particles from a heavy-duty truck)). Particles were collected at 2 locations: a commercial airport (CAP) and at a military airfield or non-commercial airfield (NCA) (see Chapter 4 Table 8 and 9 for sampling and exposure estimates).

Acute phase response, inflammation and genotoxicity were assessed following pulmonary exposure to the 2 different aircraft particle samples at 3 different dose levels (single dose of either 6 µg, 18 µg, or 54 µg per mouse) by intratracheal instillation (6-8 mice per dose per particle sample) in 3 different exposure series. Histopathological analysis was performed on samples from mice that received 54 µg NCA, 54 µg CAP, and 162 µg NIST2975 on day 28 and day 90 after exposure. Genotoxicity was assessed by using the comet assay on BAL derived cells, lung cells and liver cells. Increased levels of DNA strand breaks in BAL cells were observed for NCA and NIST2975 (18 µg) on day 1 post-exposure. On day 28 post-exposure, CAP (6 µg) resulted in higher tail length and % tail DNA in liver cells (results only presented graphically). On day 90, no significant differences were found compared to vehicle controls. Inflammation was assessed by evaluating the total cell count and composition of inflammatory cell subsets in BAL fluid cellular content. Serum amyloid 3 (Saa3) mRNA levels in lung tissue, Saa1 mRNA levels in liver tissue, and SAA3m plasma proteins were used as biomarkers of pulmonary, hepatic, and systemic acute phase response, respectively. On day 1 post-exposure, exposure to NCA, CAP and NIST2975 led to an induced dose-dependent pulmonary acute phase response compared to the vehicle control. The acute phase response returned to baseline levels 28 days after exposure to NCA, CAP, and NIST2975.

The committees note that it is difficult to draw conclusions regarding genotoxicity in the lung due to the presence of inflammation. However, DNA damage was observed in the liver in the absence of inflammation. Besides the observed dose-dependent acute phase response for aircraft particles, standard diesel exhaust particles (NIST2975) and carbon black (Printext90) nanoparticles, which were considered as relevant controls in this analysis, induced inflammation at similar levels as the aircraft particles. Furthermore, physicochemical analysis of CAP and NCA showed that CAP contained various organic compounds including salt, pollen and soot. In addition, both aircraft particle samples contained metals and PAHs. The total PAH content in both aircraft particle samples was roughly comparable to the reported content of NIST2975 diesel particles. However, the aircraft particle samples contained higher concentrations of benzo[a]pyrene (NCA 0.009 mg/g and CAP 0.005 mg/g) compared to NIST2975 diesel particles (0.0008 mg/g). The metal content was higher in both aircraft particle samples compared to NIST2975 diesel particles.

It should also be noted that single intratracheal instillation is not considered

as a physiologically relevant method of application compared to inhalation exposure. Therefore, it cannot be used to derive a quantitative exposure-response relationship, but it could provide supportive data.

6.3 In vitro data

Two in vitro studies investigating DNA damage of KEE were included (see Table D3 in Annex D).

Melzi et al. (2024)[137] conducted an in vitro toxicological study in which human lung epithelial Calu-3 cells, in monoculture and co-culture with macrophages, were exposed to primary particulate matter (PM) extracts. The PM extracts generated from the combustion of 12 types of aviation fuel were collected on PTFE filters using a standardised unheated sampling system and subsequently extracted in methanol. Cells were exposed via ALI using a nebulization method that mimicked respiratory exposure of airway epithelium. Exposure to PM extracts from combustion aerosol standard (CAST) generated samples, particularly from high-aromatic fuels, resulted in a statistically significant increase in DNA damage as assessed by the comet assay. DNA damage was also evaluated with inclusion of enzymatic modification (Endonuclease III (ENDOIII), Fpg) which showed that the observed DNA damage was unlikely due to the oxidation of DNA bases. No cytotoxicity or significant changes in transepithelial electrical resistance (TEER) was observed at the tested doses (~450 ng/cm^2 particles).[137]

The committees note that the results on DNA damage are only presented graphically, which hinders quantitative estimation of the results.

McCartney et al. (1986)[138] carried out a bacterial reverse mutation assay with *Salmonella typhimurium* strains TA98, TA98NR and TA98/1,8-DNP6 exposed to the extracts derived from airplane particulates. Particles were collected for 6 hours at ~18 meters distance from an active runway, and from an operating aircraft in ground idle at 9 meters distance and 61 cm above the ground for 7 minutes. A control sample was collected at the same position and time period as the sample from aircraft in ground idle. Exposure of the *Salmonella typhimurium* strains to the extracts derived from runway particles (0; 0.10; 0.33 and 1.0 mg of particulate equivalent) and ground idle particles (0; 0.015; 0.050 and 0.150 mg of particulate equivalent) resulted in a dose-dependent increase in the number of mutations per plate, with the highest number of mutations per plate for the ground idle particles. It should be noted that this is the only study describing the presence of nitroarenes in aircraft particle emissions both during ground idling and during take-off/ landing operations. This conclusion is based on the low mutagenicity in the TA98NR (in comparison to the native TA98 strain as a positive control) which is a bacterial strain lacking nitro-reductase that activates nitro-PAHs by enzymatic conversion to amino-PAHs that are known mutagens.[138]
The committees noted that the control air sample taken at the site

contained no measurable particles and upon extraction no mutagenicity was observed. A similar result was found for the extracted unexposed filter.

6.4 Summary on mutagenicity

Regarding cytogenetic effects in humans, one study[120] reported an increase in chromosomal aberrations, while four studies showed no effect on micronuclei frequency.[106,112,120,136] Two studies[112,120] showed a positive result for sister chromatid exchange, but another study[136] did not. The results for DNA damage were inconclusive.[106,112] Overall, the results of epidemiological studies show indications of potential genotoxic effects. However, the epidemiological studies were limited due to the small numbers of participants, differences in exposure profiles and the limited quality of exposure assessment, which may have influenced the results.

An animal study[107] observed that pulmonary exposure to KEE related particles induced genotoxicity in liver tissue and cells obtained by broncho-alveolar lavages, which provides limited evidence for the genotoxic properties of KEE.

An in vitro study[138] showed a positive result for gene mutation after exposure of *Salmonella typhimurium* strains to extracts derived from runway and ground idle particles. Another in vitro study[137] showed an increase in DNA damage in lung cells upon exposure to particle extracts of jet engine exhaust. Considering both in vitro studies, the outcomes provide limited evidence for genotoxicity of KEE.

Overall, these findings suggest some evidence of genotoxic effects due to occupational exposure to aircraft-related engine exhaust. However, the results may have been biased by limitations in the studies, such as the small numbers of participants and the possibility of co-exposure(s) to other genotoxic substances at the workplace. Chapter 8 describes the hazard evaluation and recommendation on the classification of germ cell mutagenicity.

7 Carcinogenicity

Summaries on the available carcinogenicity studies on KEE are given below. Summaries of study descriptions of the individual studies are reported in annex D.

7.1 Human data

Six epidemiological studies (cohort and case-control studies) on carcinogenicity of KEE exposure in humans were included (see also Table D4 in Annex D). These studies investigated the incidence of testicular cancer, renal cell carcinomas and other cancer sites.

7.1.1 Cohort studies

Garland et al. (1998)[139] investigated testicular cancer incidence among active-duty US Navy personnel. Information on demographics, hospitalizations, occupational and service history was retrieved from registries of the Naval Health Research Centre for white men serving during 1974-1979 (2,275,829 person years). A total of 143 diagnosed testicular cancer (ICD-code 186 (8th revision)) cases were identified within that period. For 87% of the cases the pathology reports were available. The National Cancer Institute Surveillance and End Results (SEER) provided incidence and mortality data for 1973-1977. Standardised incidence ratios (SIR) were calculated for all naval occupations with diagnosed testicular cancer cases. Navy aviation support equipment technicians had a statistically significant increased SIR for testicular cancer (n=5; 9,951 person years) compared to both SEER population (SIR=6.2, p-value=<0.001, 95% CI 1.9-13.0) and total Navy personnel (6.9, p<0.001, 95% CI 2.1-14.4). There was no relationship with length of service. Co-exposures were possible, such as to lubricating oils, paints (including chromate-based paints), degreasing agents, other solvents, and internal combustion exhaust emissions from diesel and gasoline engines.[139]

The committees note that co-exposures to carcinogenic substances other than aircraft engine exhaust could not be excluded. Furthermore, the results were based on a small number of cases, which means that the results may be attributed to chance.

Foley et al. (1995)[140] investigated testicular cancer incidence among UK Royal Air Force (RAF) personnel. Information on demographics, occupational and service history, categorized by age group and occupation, was retrieved for serving personnel between 1984-1989. A total of 148 diagnosed testicular cancers (ICD-code 186) were identified. Incidence rates were compared with the general population (supplied by Office of Population Census and Surveys). Incidence rates were particularly high among RAF personnel working in close vicinity of aircrafts. The overall relative risk was 3.27 (148 observed cases versus 45.2 expected; 95% CI 2.43-4.31). RAF personnel who were less closely involved with aircrafts

(e.g., catering) had testicular cancer incidence rates closer to the national average.[140]

The committees note that the study does not include information on the number of cases per occupation. The wide confidence intervals for the different occupations suggest a small number of cases per specific occupational group. The reported relative risk of 3.27 concerns all diagnosed testicular cancer cases. Furthermore, co-exposure to other carcinogenic substances present in the workplace could not be excluded.

7.1.2 Case-control studies

Barul et al. (2025)[141] conducted a population-based case–control study investigating prostate cancer from 2005-2012 in Montreal, Canada. Individuals were eligible if they were men ≤75 years of age, residents of Greater Montreal and registered on the electoral list. Controls resided in the same area, randomly selected from the electoral list and frequency-matched to cases on age (± 5 years). A total of 1,924 cases (participation rate 79%) and 1,989 population-controls (participation rate 56%) enrolled in the study. Information on socio-demographics, anthropometrics, lifestyle factors, medical and occupational history were collected by in-person interviews. Prostate cancer cases were actively ascertained from 7 Montreal hospitals (80% coverage of prostate cancer cases). Pathology reports were extracted for identification of incident cases. Industrial hygienists evaluated the intensity, frequency and reliability of exposure to engine exhausts for each job held for ≥2 years. They used details on chemical agents and equipment used, tasks, protective measures and workplace characteristics. Exposure to jet fuel engine exhaust occurred among aircraft mechanics, aircraft repairmen and air transport operating support occupations. No association was found between exposure to jet fuel engine exhaust and prostate cancer. The committees note that the results are based on a small number of cases (n=15) and controls (n=50) with exposure to jet fuel engine exhaust. Furthermore, the participation rate among cases is higher compared to controls, which may have caused selection bias.

The population-based case-control study by Parent et al. (2000)[142] investigated renal cell cancer in Montreal, Canada. Individuals were eligible if they were men, aged 35-70 years and residents of the Montreal area. All large hospitals participated in the study, leading to an almost complete (97%) ascertainment of cases. A total of 142 histologically confirmed renal cell carcinoma cases (ICD-code 189.0 (9th revision)) and 533 population-controls, selected using random digit dialling, were enrolled in the study. Information on lifestyle factors and detailed work history were ascertained by in-person interviews between 1979 and 1985. Industrial hygienists and chemists evaluated potential exposures by intensity (low, medium, or high), frequency (how often during a working week) and reliability (likeliness of actual exposure). The study also included 1,900 cancer-controls, diagnosed with other types of cancer, and a pooled control group which included both cancer-controls as well as population-controls. Because the results with the

3 control groups were very similar, Parent et al. (2000) only presented results of the pooled control group. An excess risk was reported for aircraft mechanics (OR=2.8; 95% CI 1.0-8.4), based on a small number of cases (n=4). An further in-dept analysis showed a non-significant relationship between exposure to jet fuel engine emissions and renal cell carcinoma (OR=2.7; 95% CI 0.9-8.1; n=4), OR was adjusted for age, smoking and body mass index.[142] This study used the same data as Siemiatycki et al. (1988).[143] The committees note that the wide confidence intervals indicate low precision of the estimate, due to a small number of cases (n=4). In this study both population-controls and cancer-controls were used. Generally, the participation rate among population-controls is lower, which may induce selection bias. Using a pooled control group would minimize that effect. In addition, the inclusion of 533 population-based controls allows for comparison with the general population.

A case-control study by Ryder et al. (1997)[144] investigated testicular cancer among UK Royal Navy personnel serving between 1976 and June 1994. Cases were retrieved from 2 naval hospital registries and records at the Defence Analytical Services Agency (DASA). A total of 110 histopathological confirmed testicular cancer cases (ICD-code 186) were enrolled in the study. The study included 4 randomly selected controls from personnel records, matched by date of birth (± 2 years) and length of service (at least as long as the case until diagnosis of the case) to each case. Increased ORs were reported for members for the Fleet Air Arm relative to all other branches combined (OR=1.90, 95% CI 1.04-3.48, n=19 cases), Air Engineers relative to all other specialities combined (2.32, 95% CI 1.20-4.48, n=17) and Aircraft Handling sub-specialty relative to all other sub-specialities combined (7.31, 95% CI 1.81-29.53, n=6).[144]
The committees note that information about the working conditions or lifestyle factors is limited, which means that the effect of other contributors to the relationship than job title could not be investigated. Furthermore, the wide confidence intervals for the sub-analyses indicate low precision of the estimates, because of a small number of cases.

Siemiatycki et al. (1988)[143] conducted a population-based case-referent study in Montreal, Canada from 1979 until 1985. A total of 3,726 cancer cases (men, aged 35-70 years) were enrolled in the study. Cases were diagnosed in any of the 19 participating hospitals in the Montreal-area. Associations between 15 types of cancer and exposure to different types of exhaust and combustion products were investigated. Other cancer cases were used as reference in the analysis. Each type of cancer formed a case series, and for each case series, a reference group was selected from the other cancer cases. This study used the same data as Parent et al. (2000).[142] Potential exposure was assigned to each subject by a group of industrial hygienists and chemists based on information from work histories (see also Parent et al. (2000). No statistically significant associations with jet engine exhaust and any of the cancer sites were found.[143] The committees note that the statistical power is low because of a small

number of cases per cancer site and a small number of exposed participants. Furthermore, each cancer case also served as a control for other cancer cases This may have biased the results if the cancer of the cancer-control is related to the exposure of interest (here jet engine exhaust). In addition, the study did not include adjustments for co-exposures to other carcinogenic substances.

7.2 Animal data

No animal studies have been identified that investigated the carcinogenicity of KEE exposure.

7.3 Summary on carcinogenicity

There are indications[139,140,144] that KEE exposure may lead to increased testicular cancer rates. This is in line with other findings suggesting that testicular cancer may be linked to ambient air pollutants.[145] There is limited evidence for a relationship between exposure to aircraft engine exhaust and renal cell cancer.[142] One study investigated the relationship between KEE exposure and prostate cancer risk, but did not observe an association.[141] All these findings were hampered by small numbers of (exposed) cases, leading to imprecise outcomes. Furthermore, co-exposures to other carcinogenic substances in the workplace could not be excluded.

See Chapter 8 for the hazard evaluation and recommendation on the classification of carcinogenicity.

8 Evaluation and recommendation on the classification of mutagenicity and carcinogenicity

The committees prepare classification proposals for germ cell mutagens and carcinogens. The applied criteria and classification EU-categories are based on the Globally Harmonized System, which has been incorporated into the CLP regulation. The CLP regulation is used by the European Union for the classification, labelling, and packaging of substances and mixtures (Regulation EC 1272/2008: Section 3.5 for germ cell mutagenicity, and Section 3.6 for carcinogenicity).[13] Although the criteria mentioned in the EU Regulation are set for manufactured substances and mixtures that are evaluated according to the CLP regulation, the criteria are also considered useful, because the CLP regulation forms a widely accepted legal frame-work, in recommending classifications for both CLP regulated and not regulated individual substances, mixtures and emissions.[12,15]

8.1 Application of CLP criteria

Regarding the classification of mixtures for germ cell mutagenicity and carcinogenicity, there are several scenarios described in the CLP regulation (see also Annex E in this advisory report). Based on scenario 1 for germ cell mutagenicity and scenario 2 for carcinogenicity, the committees conclude that the available data on KEE as a mixture is insufficient to recommend a classification for germ cell mutagenicity and for carcinogenicity (see Annex E for detailed description). The CLP also describes possibilities for the application of an analogy approach for similar mixtures such as the bridging principles for ‘substantially similar mixtures’ (CLP, Annex I section 1.1.3.5.). However, the bridging principles as included in CLP are not designed to evaluate complex combustion-generated mixtures such as KEE. But article 9(3) and CLP Annex I, section 1.1.1 states that ‘where the criteria cannot be directly applied to the available data, expert judgement should be used for the evaluation of the available information in a weight of evidence determination’. Based on the information provided in Chapter 2, the committees performed a hazard evaluation to decide whether KEE and DEE can be considered as similar mixtures with similar hazardous properties. This means that if these mixtures can be considered as similar mixtures, data used to substantiate the classification of DEE may be used to predict the hazard properties of KEE where data is missing. Therefore, an analogy approach with DEE, in line with Article 9(3), is further explored below (see Chapter 8.2).

8.2 Analogy with diesel engine exhaust

In the following section the similarities between KEE and DEE are addressed in more detail regarding their genotoxic and carcinogenic properties. DEE has been classified as carcinogenic to humans (Group 1) by IARC[5] based on sufficient evidence from epidemiological and toxicological studies.

8.2.1 Exhaust composition

KEE and DEE are complex mixtures containing numerous substances, but their relative compositions are rather similar (see Table 4 in Chapter 2.3.1). Both KEE and DEE contain the same toxicologically relevant components such as combustion-generated particles (or soot) with adsorbed metals, PAHs and organic sulphur compounds. The differences between KEE and DEE mainly concern the concentrations in which some of these substances occur in the exhausts, which may influence the toxicity. These differences and their effect on the hazard properties of KEE are discussed in more detail below.

KEE particles versus DEE particles

Both KEE particles and DEE particles have an approximately spherical shape forming fractal agglomerates with a large surface area which gives these particles the ability to adsorb large amounts of organic (e.g., PAHs, sulphur compounds) and inorganic (e.g., metals) compounds. Although KEE predominantly consists of very small particles, there is overlap in the particle size distributions between KEE and DEE.[44] Once emitted, the particles may grow in size to form larger particles through various physical and chemical reactions[1,2,44] until they reach an equilibrium (see Chapter 2.3). This starts immediately after formation and continues during dilution and cooling of the exhaust in ambient air.[1]

The committees acknowledge that KEE contains higher concentrations of very small particles and that the smaller sized particles can penetrate deeply into the lungs and may, if deposited, cause toxicological effects. The very small particles (<100 nm) can easily pass through membranes and distribute to all tissues (see Chapter 5).[35,122,124] The committees also note that there is some overlap in the particle size distributions for KEE and DEE (DEE also contains very small particles) and that particles may expand to form larger particles even after inhalation.[44,123] Evidence for DEE shows that the toxicity is driven by the presence of carbonaceous particles. Like DEE particles, KEE particles predominantly consist of carbonaceous particles (soot).[107]

Sulphur compounds

KEE generally contains higher concentrations of organic and inorganic sulphur compounds compared to current DEE emissions (see Chapter 2.3). An in vitro study showed that combustion-generated particles with particle-adsorbed organic compounds, such as hydrocarbons and sulphuric acid, may increase genotoxicity and immunosuppression (see also Chapter 5).[129] The presence of higher amounts of organic and inorganic sulphur

compounds in KEE may increase its toxicity, but further research is needed. In addition, most of the toxicity studies, used by other scientific bodies to substantiate their hazard evaluations for DEE, were carried out using 'older' diesel fuels with higher sulphur content. As was noted in Chapter 2.2, current kerosene jet fuels and KEE are rather similar to 'older' diesel fuels and 'older' DEE. Hence, the classification of DEE is based on studies in which humans and animals were exposed to a wider range of sulphur concentrations than used in modern diesel fuels.

Low and high-molecular weight PAHs

Limited data on composition indicate that KEE has relatively lower concentrations of high-molecular weight PAHs and higher concentrations of low-molecular weight PAHs (see also Chapter 2) compared to 'older' DEE. A recent study by Heeb et al. (2024) showed that the highest PAHs (including genotoxic PAH) emissions in KEE were reported during ground idle and taxi operations, which are most relevant for occupational exposure. At ground idle the PAH emissions are dominated by naphthalene (IARC group 2B, possible carcinogenic) other PAH only contribute up to 19% to the genotoxic potential of KEE. At higher thrust benzo(a)pyrene and dibenzo(ah)anthracene (both IARC Group 1 carcinogens) contribute more to the genotoxic potential of KEE. PAH emissions for DEE (current diesel fuel and a particle filter) fell between low and high thrust patterns for PAH emissions of KEE. However, the overall genotoxic potential of KEE based on PAH emissions during ground idle was considered much higher (8 to 400 times) compared to DEE in this study.[35] A few studies, described in Chapter 4, have examined particle-bound PAH concentrations. Two studies (possibly the same study location) showed lower concentrations for high-molecular weight PAHs in KEE.[119,120] One other study which compared KEE particles with DEE particles found higher concentrations of benzo[a]pyrene in KEE particles. However, these findings are based on a small number of measurements.[107]

Conclusions on combustion products

Evidence for DEE shows that the toxicity of DEE is primarily related to the toxicity of DEE particles. DEE particles show strong similarities with KEE particles, except that KEE particles are generally smaller in size compared to DEE particles ('older' DEE particles). However, KEE particles (with PAHs or other compounds adsorbed to the particles) can penetrate deeply into the lungs due to their small size.[2] It has also been shown that toxicity increases with decreasing particle size. It is plausible that size has an effect on genotoxicity, because smaller particles have a greater surface area per unit mass to adsorb relatively large amounts of organic substances and metals.[129] Furthermore, particle-bound PAHs (predominantly high-molecular weight PAHs), even in small amounts, have been linked to immunosuppression, and exert their toxicity primarily through genotoxic pathways, such as DNA adduct formation and DNA strand breaks.[129] Additionally, there is some evidence that sulphur compounds adsorbed to KEE particles might also increase the toxicity of KEE.

Considering the similarities in characteristics between DEE particles and KEE particles and that smaller particles can penetrate deeper into the lungs, the committees expect that the toxicity of KEE is mainly driven by the toxicity of KEE particles. The mode of action related to particle toxicity is further described below.

8.2.2 Mechanism of action

The carcinogenic mechanism of KEE particles involves genotoxicity by inflammatory-induced oxidative reactions and bulky DNA adducts. PAHs adsorbed to KEE particles may increase the number of mutations and structural chromosome damage. The findings regarding oxidative damage, formation of ROS, and lung inflammation for particle-induced genotoxicity are provided below. Where relevant, information on DEE particles has been used to fill information gaps for KEE particles.

Lung inflammation

Particles, such as KEE particles, can induce inflammation and oxidative stress, leading to further inflammatory responses in the lungs (see also Chapter 5). This is supported by an animal study in mice, which showed a highly increased influx of inflammatory cells in BAL fluid on day 1 post-exposure after intratracheal exposure to KEE particles. This influx of inflammatory cells was similar or even higher for KEE particles compared to the same mass dose of DEE particles used as reference.[107] Similar findings regarding the influx of inflammatory cells were observed in mouse studies with DEE particles.[29,146,147]

Formation of reactive oxygen species

There is no specific data on KEE particles concerning ROS formation. Based on information on carbonaceous DEE particles (KEE also contains carbonaceous particles), ROS and RNS could be generated by inflammatory cells during particle-induced inflammation.[29] Oxidative stress may also arise from the direct generation of ROS at the particle surface. In addition, soluble compounds such as transition metals (iron and copper are well-known redox-active metals and present in KEE) or organic compounds (PAH-derived redox-cycling semiquinones contribute to ROS formation) attached to these particles can alter the function of mitochondria or NADPH-oxidase.[2,130,133]

Oxidative DNA damage

Increased levels of DNA strand breaks were observed in mice after intratracheal exposure to KEE particles with the Comet assay. Most DNA damage was observed in BAL cells for NCA and in liver cells for CAP. The observed levels of DNA damage were overall low, but similar levels were found for DEE particles in this study.[107]
Similar findings have been reported for intratracheal and inhalation exposure to DEE and DEE particles specifically in animal studies.[29]

Particle-adsorbed substances with genotoxic potential

Chemical analyses have shown that the particle fraction of KEE is enriched with PAHs and a variety of structurally related aromatic compounds (see Chapter 2.3). Many of these substances are established, probable and possible mutagens and carcinogens. Experimental evidence supports the concern for genotoxicity, because extracts of KEE particles were mutagenic in bacterial assays[138] and caused DNA damage in mammalian cells in vitro[137] (see Chapters 5, 6 and 7). Furthermore, a recent study reported that the genotoxic potential, expressed in ng-TEQ/kg fuel, (calculated as the sum of the amounts of eight priority carcinogenic PAHs multiplied by their respective toxicity equivalence factors) of jet engine exhaust with Jet A-1 fuel is 90 times higher during ground idle compared to a diesel vehicle with a particle filter. In addition, particle number concentrations were 400 times higher for an aircraft engine compared to a diesel vehicle.[35]

These findings suggest that the particulate phase may serve as an efficient carrier for genotoxic substances, facilitating their deposition in tissues and enhancing the likelihood of cellular exposure.

8.2.3 Inflammation, genotoxic effects and cancer

Only a limited number of studies have directly examined the genotoxicity of KEE, both in vitro and in vivo. However, the available evidence, together with the comparison to the more extensively studied DEE, indicate that KEE also has genotoxic potential.

The limited number of studies for KEE give evidence of oxidative stress, ROS formation and lung inflammation after exposure to KEE derived particles.[2,107,148-150] These findings are similar to the described effects for DEE. Epidemiological studies on KEE exposure have reported increased risk of testicular cancer and renal cell cancer. The findings on testicular cancers are not supported by evidence for DEE. DEE is particularly associated with lung and bladder cancer, but a relationship with kidney cancer has also been suggested.[151,152] The epidemiological studies for KEE had limitations due to the small number of (exposed) cases and possible co-exposure to other carcinogens present in the workplace. Nevertheless, the reported increased risk for testicular and renal cell cancer could also be attributed to exposure to particles in the ambient air.[145,153]

8.2.4 Conclusion

The committees conclude that there are substantial similarities in characteristics between KEE and DEE particles, which allow for an analogy with DEE. In particular, the presence of (ultra)fine particles of similar shape and composition together with PAHs in overlapping concentrations in both KEE as DEE, support the classification of KEE, as these components are likely responsible for the genotoxic and carcinogenic properties of these mixtures.

There are, however, some differences between KEE and DEE, but these differences are not expected to lead to a different outcome regarding genotoxicity and carcinogenicity due to exposure to KEE compared to DEE.

The sulphur content and smaller particle size may even be expected to increase rather than decrease the toxicity of KEE in comparison with DEE. The committees infer that the toxicity of KEE is primarily related to the presence of combustion-generated particles (with adsorbed metals, PAHs and other organic compounds) in the exhaust. This is supported by the ample available evidence for the toxicity of DEE particles. In addition, IARC has classified DEE particles as carcinogenic to humans (Group 1).[5,40]

8.3 Recommendation on the classification of germ cell mutagenicity and carcinogenicity

Considering the available data for KEE substantiated with information from the analogy with DEE, the committees have concluded on the following recommendations for germ cell mutagenicity and carcinogenicity.

Recommendation on the classification of germ cell mutagenicity

There is no data on germ cell mutagenicity for KEE and the information on DEE is too limited to be used as basis for a conclusion on KEE. There is a limited amount of evidence for genotoxicity of KEE in somatic tissues, consisting of some inconclusive epidemiological data, one positive in vivo study and two positive in vitro assays. Evidence from toxicological studies on DEE support that KEE has genotoxic potential in somatic tissues. Therefore, the committees recommend a classification for germ cell mutagenicity in category 2 'suspected to induce heritable mutations in the germ cells of humans' for KEE (see text box).

Recommendation on the classification of carcinogenicity

There is limited evidence for the carcinogenic potential of KEE. There is sufficient evidence from epidemiological and toxicological studies for DEE. In addition, KEE contains several known carcinogenic components. The committees recommend a classification in category 1B 'presumed to be carcinogenic to humans' for KEE (see text box).

Classification categories for mutagenicity and carcinogenicity based on CLP criteria[12]

Classification for germ cell mutagenicity

Category 1A	Known to induce heritable mutations in the germ cells of humans (H340)
Category 1B	Presumed to induce heritable mutations in the germ cells of humans (H340)
Category 2	Suspected to induce heritable mutations in the germ cells of humans (H341)

EU Hazard statement codes for germ cell mutagenicity

H340	May cause genetic effects
H341	Suspected of causing genetic effects

Classification for carcinogenicity

Category 1A	Known to be carcinogenic to humans (H350)
Category 1B	Presumed to be carcinogenic to humans (H350)
Category 2	Suspected to be carcinogenic to humans (H351)

EU Hazard statement codes for carcinogenicity

H350	May cause cancer
H351	Suspected of causing cancer

9 Other health effects

In this chapter, health effects other than genotoxic and carcinogenic effects associated with (occupational) exposure to KEE are described. Both short and long-term health effects are described, in accordance with the reported literature.

Toxicological studies suggest that UFP generally elicit a greater toxicity (based on mass concentrations) and a higher likelihood to induce systemic effects compared to larger-sized particles of the same composition. This is due to their small diameter by which these particles can reach the alveoli, high surface area-to-mass ratio (can adsorb large amounts of organic and inorganic compounds) and high number concentrations.[2,124,154] For more details on mechanisms of toxicity see Chapter 5. Several in vitro and in vivo studies have shown that combustion-generated particles or KEE particles can cause inflammation, formation of ROS and DNA damage.[2,148-150]

9.1 Short-term health effects

The association between exposure to UFP and effect on human urinary metabolome (a measure to characterise changes in cellular pathway activity) was investigated among 21 healthy non-smoking participants. Participants were university students residing in Amsterdam >2 kilometres from the airport and not within 300 meters from a highway or busy road (>10,000 vehicles per day). Participants were repeatedly (2-5 visits) exposed to ambient air at Schiphol Airport for 5 hours, while performing intermittent moderate exercise. Exposure changes in urinary metabolome were measured before and after exposure (morning of the next day, on average 18 hours after exposure). The total particle number concentration was on average 53,500 particles/cm^3 (range 10,500-173,200). There were significant reductions reported in urinary taurine (an indirect antioxidant; reduction may reflect mitochondrial dysregulation, inflammatory cell recruitment and cytokine secretion), dimethylamine (reflects enhanced dimethylnitrosamine synthesis, which exerts genotoxicity in mammalian cell lines) and pyroglutamate (precursor to glutathione) which were associated with UFP because of landing and take-off operations of aircraft in the vicinity of the study location. This study location was ~300 m away from two runways, ~500 m away from two highways and ~10 kilometres away from Amsterdam.[155]

9.1.1 Pulmonary effects

A study by Janssen et al. (2019) investigated short-term health effects of exposure to UFP among neighbouring residents of Schiphol Airport and reported reductions in lung function and temporarily exacerbated existing respiratory diseases. Temporary reductions in heart function were also measured in otherwise healthy adults. Overall, these changes do not necessarily have to result in immediate health problems.[26,156]

In a randomized crossover study, asthma patients experienced increased acute systemic inflammation and oxidative stress after exposure to UFP (personal sampling using DiSCmini diffusion charger). In this study spirometry, multiple flow exhaled nitric oxide and circulating inflammatory cytokines were measured before and after exposure, in 22 non-smoking participants with asthma. Participants performed scripted mild walking activity either in the proximity of Los Angeles Airport (mean particle number concentrations of 53,342 particles/cm^3) or further away from the airport (mean 19,557 particles/cm^3).[26,157] In a controlled human exposure study 21 healthy non-smoking participants were exposed to UFP while performing intermittent moderate exercise in the proximity of Schiphol Airport (laboratory located northwest of the airport, ~300 m from two runways and ~500 m from two highways). Short-term exposure to UFP (~125,400 particles/cm^3, especially to small particles <20 nm), was associated with a decreased lung function.[158]

9.1.2 Cardiovascular effects

Lecca et al. (2021) studied the association between occupational exposure to aircraft-related UFP and heart rate variability (HRV) in a cross-sectional study among 33 airport ground workers. The total lung deposited surface area (LDSA) (mean LDSA=109.46 m^2/cm^3) was significantly associated with a lower HRV total power (ß=-0.038, p-value=0.016), an early indicator of cardiovascular autonomic response.[108]

9.2 Long-term health effects

9.2.1 Pulmonary effects

A recent study by Touri et al. (2025) investigated lung function among airport workers of Air France. A change in lung function over a period of approximately 6.6 years was investigated in a small cohort of airport workers located at Paris-Roissy (Charles de Gaulle) Airport or Marseille Airport in France. A total of 215 workers were investigated: 68 office workers (location Paris-Roissy (PR)), 83 mechanics (PR), 29 airport terminal workers (location Marseille (M)), and 35 apron workers (M). Personal measurements of carbonaceous particles were performed among 16 workers. Only mechanics (10.1 µg/m^3 EC, sd=4.3) and office workers (9.9 µg/m^3 EC, sd=3.9) from Paris-Roissy Airport had measurable EC levels. Metal concentrations (n=206) were measured in EBC, most samples had estimates below levels of quantification and no significant differences between the groups were found (see also Chapter 4 Table 8 and 4.3.3). An overall decline in lung function was found for 24.75% of all airport workers in the cohort. The decline in lung function occurred more often for terminal workers (44.83%) as compared to mechanics (14.47%, p-value = 0.0056), and for apron workers (35.29%) as compared to mechanics (p-value = 0.0785). It should be noted that, considering individual participants, lung function decline was found in all employment-type groups (including office workers), especially in terminal and apron workers at Marseille Airport.[102]

A cross-sectional study by Tunnicliffe et al (1999) among airport workers found significant associations for runny nose (OR=2.9) and cough with phlegm (OR=3.5) for high exposed workers (adjusted for age, smoking and seasonal rhinitis, $p<0.05$). Furthermore, this study showed that aircraft-related exposure is associated with an excess of some respiratory symptoms but yielded no evidence for an association with occupational asthma.[159] A cross-sectional study conducted by Yang et al. (2003) among 106 airport workers (exposed group) and 305 terminal or office workers (non-exposed group) found significantly increased prevalence (adjusted for age, smoking, education, previous occupational dust and fumes exposures) for chronic respiratory symptoms such as cough and dyspnoea.[154,160]

9.2.2 Cardiovascular effects

A Danish occupational cohort study by Møller et al. (2020) among 6,515 male airport workers (reference group of 61,617 men from greater Copenhagen area in unskilled jobs) did not find an association between cumulative apron-years and incidence of ischemic heart disease or cerebrovascular disease during a mean follow-up of 14.4 years. However, the median age for the reference group and the exposed group (see Chapter 4 Table 8 for exposure estimates) was 31 and 28 years, respectively, and therefore the study population might have been too young to observe enough events to detect cardiovascular effects.[2,154,161]

A study by Janssen et al. (2022) studying long-term health effects as a result of UFP exposure among neighbouring residents of an airport reported possible associations with cardiovascular effects based on more use of cardiovascular medication.[26,162]

9.2.3 Reproductive and developmental effects

A study by Wing et al (2020) report that exposure to UFP is associated with increased pre-term birth rates near the LAX airport in Los Angeles. Pre-term birth may increase the infant's risk for developing complications, such as respiratory problems, infections, developmental delays, and vision or hearing impairments.[163]

9.3 Conclusions on other health effects

A few studies have investigated potential health effects due to (occupational) exposure to UFP.[102,108,155-163] Five of these studies were conducted in an occupational setting or among airport workers,[102,108,159-161] and three 'controlled' human exposure studies.[155,157,158] In two of the 'controlled' human exposure studies participants were exposed during specific exercise in the laboratory located in the vicinity of an airport (~300 m from two runways and ~500 m from two highways). Participants were exposed to aircraft-related UFP as well as ambient UFP.[155,158] Exposure to UFP was associated with effects on urinary metabolome, reductions in lung function, worsening of existing respiratory diseases, chronic respiratory symptoms and reduction in heart rate variability. No

associations were reported for occupational asthma, ischaemic heart disease or cerebrovascular disease. However, most of the studies had some limitations, such as a lack of quantitative exposure assessment,[159,160] limited number of participants or workers,[108,155,157,158] or some results may have been biased due to limitations in exposure or health assessment[108,155,158-160] or confounding by co-exposures to traffic-related UFP.[102,156,162]

Thus far there are no occupational cohort studies for KEE exposure with long-term follow-up, thorough exposure and health assessment and relevant information on potential confounding factors.

10 Evaluation and recommendation on an occupational exposure limit

10.1 Existing occupational exposure limits

There is, to our knowledge, no occupational exposure limit for KEE.[3,7] KEE is a complex mixture of numerous inorganic and organic substances and substance groups. This advisory report focuses on the toxicologically most relevant components of this mixture: particles with adsorbed PAHs, metals, and sulphur compounds (mainly sulphur dioxide). For several of these components OELs have been established,[3,7] see Table 13. Unlike for KEE, there are OELs for DEE, based on respirable elemental carbon (REC) or EC as indicator, see Table 13. It is emphasized that EC represents a surrogate or indicator of exposure to carcinogenic components in DEE particles.[6] EC is considered as a suitable indicator of particulate diesel exhaust, because EC in DEE particles is primarily derived from the combustion of fossil fuels. In addition, other sources of fossil combustion, such as gasoline engines, emit far less EC than diesel engines.[164] For KEE, only a few studies have investigated EC content in KEE particles and reported varying relative amounts of EC in KEE particles. The quantitative concentrations of EC in KEE have not (yet) been established (see Chapter 4).[104,116]

10.2 Identification of an exposure indicator for KEE

KEE is a highly complex mixture of numerous gas-phase and particle-phase substances, for which it is impractical to measure all these substances. Therefore, (occupational) exposure to mixtures is typically established by measuring one or a few markers or indicator substances present in the mixture. Such indicator(s) should be measurable, appropriate and preferably specific for the mixture, in this case KEE. KEE is generally characterised by large numbers of small particles, mainly in the UFP range. These UFP have negligible mass but are the dominant contributor to the total number of particles in the mixture. UFP may be better quantified by number concentrations or surface area rather than commonly applied mass concentration measurements.[36,44,87] Measuring UFP number and/or surface area concentrations may be a suitable exposure indicator. However, other sources of UFP are present at the apron or in the proximity of the airport or (military) airfield (e.g., road traffic), which means that UFP measurements are not unique for exposure to KEE. Other candidate indicators are EC, BC, OC, and other combustion-generated components such as PAHs and sulphur dioxide (SO_2).[164,167] Considering that an indicator must be selective, SO_2 may be appropriate since exhaust emissions from road traffic, such as

Table 13 Some existing OELs (as inhalable fraction unless otherwise indicated) of toxicological relevant components of KEE

Substance	Denmark (OEL in mg/m^3)	Finland (OEL in mg/m^3)	The Netherlands (OEL in mg/m^3)	Norway (OEL in mg/m^3)	Sweden (OEL in mg/m^3)	EU (OEL in mg/m^3)	Remarks
Reference	9	11	7	10	8	[7]	
Respirable or ultrafine particles	5* (respirable dust)	n.a.	n.a.	5* (respirable dust)	2.5* (respirable inorganic dust)	n.a.	No OELs for UFP. OEL for respirable dust (5 mg/m^3) is withdrawn in the Netherlands
PAH	0.2*	0.01*	0.0055*	0.04*	0.002* 0.02**	n.a.	EU Commission is currently (June 2026) discussing a BOEL proposal.[165]
SO_2	1.3* 2.7**	1.3* 2.7**	0.7* 0.7**	1.3* 2.7**	1.3* 2.7**	1.3* 2.7** (IOELs)	DECOS derived OEL for short-term exposure only.[166]
NO_2	0.96* 1.9**	0.96* 1.9**	0.96* 1.91**	0.96* 1.91**	0.96* 1.9**	0.96* 1.91** (IOELs)	
Metallic components							
Aluminium	2-5*#	0.1-1.0*#	n.a.	2-10*#	2* (respirable) 5* (total dust)	n.a.	
Barium	0.5*	0.5*	0.5*	0.5*	0.5* (total dust)	0.5(IOEL)	
Chromium (metallic)	0.5*	0.5*	0.5*	0.5*	0.5* (total dust)	2* (IOEL)	
Cobalt	0.01*	0.02*	0.02*	0.02*	0.02*	n.a.	EU Commission is currently (June 026) discussing a BOEL proposal.[165]
Copper	n.a.	0.02* (respirable)	0.1*	1* (copper dust) 0.1* (copper smoke)	0.01* (respirable)		
Iron	1*	1-5*#	n.a.	1*	n.a.	n.a.	
Molybdenum	5-10*#	0.5*	n.a.	5* (soluble) 10* (insoluble)	5* (respirable) 10* (total dust)	n.a.	
Nickel	0.05* 0.01* (respirable)	0.05* 0.01* (respirable)	n.a.	0.05* 0.01* (respirable)	0.05* 0.01* (respirable)	0.05* 0.01* (respirable) (BOELs)	
Vanadium	0.03*	0.02*	n.a.	0.05*	0.2* (total dust) 0.05** (respirable)	n.a.	
Zirconium	5*	1*	n.a.	5*	n.a.	n.a.	
Similar mixture							
Diesel engine exhaust	0.005* (as EC)	0.05* (as REC)	0.01* (as REC)	0.05* (as EC)	0.05* (as EC)	0.05* (as EC) (BOEL)	DECOS established a prohibition risk level of 0.001 mg REC/m^3 and a target risk level of 0.00001 mg REC/m^3.[6]

* Time-weighted average 8 hours (TWA-8h); ** Short-Term Exposure Limit (STEL); # depending on chemical compound.

Abbreviations: n.a., not available; UFP, ultrafine particles; PAH, polycyclic aromatic hydrocarbons; BaP, benzo(a)pyrene; IOEL, Indicative Occupational Exposure Limit; SO_2, sulphur dioxide; DECOS, Dutch Expert Committee on Occupational Safety; NO_2, nitrogen dioxide; ACGIH, American Conference of Governmental Industrial Hygienist; EC, elemental carbon; REC, respirable elemental carbon; BOEL, binding occupational exposure limit.

Sources: Danish Working Environment Authority (DWEA)[9]; Social- och hälsovårdsministeriet (SHM)[11]; Social and Economic Council of the Netherlands (SER)[7]; Norwegian Labour Inspection Authority (NLIA)[10]; Swedish Work Environment Authority (SWEA).[8] All sources accessed November 2025.

DEE, currently contains only trace amounts compared to KEE. Although Yu et al. (2004) suggested that SO_2 can be used as indicator of aircraft emissions if used within the contours of the airport, there are globally major other sources of SO_2 emissions, particularly from shipping (marine diesel oil may contain 0.5% of sulphur).[1,168] In addition, SO_2 may be difficult to measure using air sampling; because of its high reactivity, sulphur has many chemical occurrences in the atmosphere. EC, BC, and OC reflect the presence of carbonaceous particles in KEE. There is, however, only limited data on EC, BC and OC content in KEE particles (see also Chapter 4). In addition, other carbonaceous sources are present at the airport apron and in the surroundings of airports. Furthermore, EC is used as indicator for DEE exposure.[164,167]

PAHs, like UFP, have multiple sources present at the apron and in the vicinity of airports and (military) airfields. Further research is needed to establish a suitable indicator for occupational exposure to KEE. This includes research on how these indicators relate to other components in the exhaust plume of KEE and how the size, composition and transformational behaviour of particles, and the speed of these processes, affect those relations. The mechanisms around the formation of carbonaceous particles, the effect of adsorbed inorganic and organic substances, and the effect of aging have not been fully understood yet.[67]

10.3 Considerations for an OEL for KEE

Health-based OELs are obtained by a quantitative risk analysis, for which the committees need reliable quantitative exposure-response data. A thorough exposure and health assessment is necessary to establish a quantitative exposure-response relationship. This is different than for a hazard classification, i.e. for mutagenicity and carcinogenicity, which are based on an qualitative hazard evaluation on the potential for inducing mutagenic and carcinogenic effects.[15]

For KEE, a limited number of human studies are available in which the relationship between exposure to components of KEE, predominantly UFP, and adverse health effects are investigated. From the available epidemiological studies discussed in Chapters 7 (carcinogenicity) and 9 (other health effects) only observational cohort or case-control studies are relevant for establishing a quantitative exposure-response relationship. Cross-sectional studies generally lack a clear timeline from exposure to effect, because measurements and observations of effect are performed at one time point which are not suitable for establishing long-term effects such as cancer or some respiratory diseases.

For carcinogenicity a few case-control and cohort studies are available, but these studies provide limited evidence because of small number of (exposed) cases and limitations in the exposure assessment (effect of co-exposure(s) cannot be excluded), which may have affected the outcomes.[139,140,142,144]

For other health effects there is even less evidence, with two cohort studies which either have too limited follow-up to detect health effects[161] or limitations in the exposure assessment.[102] In addition, the committees have not identified any suitable animal studies (e.g., inhalation studies) that may support derivation of a hb-OEL. The only animal study by Bendtsen et al. (2019)[107] investigated pulmonary toxicity in mice using intratracheal instillation. This method is suitable for hazard comparison and the study suggests that KEE and DEE have very similar toxicity. However, intra-tracheal instillation is not considered a physiologically relevant method of application for establishing a quantitative exposure-response relationship.
In conclusion, the committees do not have sufficient quantitative exposure-response data available for the derivation of a hb-OEL for KEE.
In this case, an analogy with DEE was not deemed feasible, because it is uncertain whether the quantitative exposure-response relationship for DEE, which is based on EC, is applicable to KEE. Moreover, although there is evidence for the presence of EC in KEE particles, the concentrations of EC have not yet been established in relation to potential adverse health effects.

10.4 Evaluation and recommendation

The committees consider the available human data as insufficient to derive a hb-OEL for KEE. There is, however, cause for concern because of the similarities with DEE, which is classified as carcinogenic to humans (Group 1) by IARC. In addition, there are existing OELs for DEE (see Table 13).
Furthermore, the available data for KEE indicate that occupational exposure to KEE can lead to several health effects, including inflammation, reductions in lung function, worsening of existing respiratory disease and chronic respiratory symptoms. Also, several components of KEE are known, probable, or suspected mutagens, carcinogens and/or OELs have been established (see also Table 13). Finally, the committees consider a classification in Category 1B for carcinogenicity and Category 2 for germ cell mutagenicity warranted, based on the limited evidence from a limited number of epidemiological and toxicological studies with exposure to KEE and sufficient evidence for DEE using an analogy approach.
The committees consider that there are ample indications to stress that exposure to KEE should be limited, even in the absence of a hb-OEL for KEE. Therefore, the committees recommend that existing OELs for DEE and components of KEE, such as PAHs, metals, SO_2, NO_2, should be applied for KEE to reduce occupational exposure of airport workers. Because it is uncertain if these OELs are sufficiently protecting workers, the committees recommend further research to establish exposure-response data for KEE that would allow for the derivation of a hb-OEL.

11 Research needs

Previous chapters have indicated several data gaps related to KEE exposure assessment. Therefore, the committees make several suggestions for further research.

Exposure assessment

To improve exposure measurements and allow for comparison of measurement data between studies, a standardised UFP measurement method including low-end and upper-end definition would have to be developed. Furthermore, improvement of our knowledge of mechanisms of particle formation, growth and the effect of aging is needed, particularly on how the numerous components in the aircraft exhaust plume are related. In line with this, research activities should focus on establishing a sensitive and measurable indicator for KEE, to be able to disentangle exposure to KEE emissions specifically from numerous other sources of exposure to the same substances present at the apron. A combination of two or more indicators among UFP, EC, SO_2, and PAH might be a way forward, as none of these are unique to KEE, but their relative abundance may help to distinguish exposure to KEE from DEE and other pollutants.

Effect assessment

For the hazard evaluation and classification for germ cell mutagenicity and carcinogenicity specifically for KEE, epidemiological and controlled in vitro and in vivo (e.g., inhalation studies) experimental studies are needed with relevant and validated outcomes for genotoxicity and carcinogenicity. In vitro studies should include relevant exposures such as continuous flow exposure in an Air Liquid Interface (ALI) set-up. Epidemiological studies, such as cohort, case-control and cross-sectional studies should include thorough exposure and health assessment and for cohort studies a sufficient follow-up time to detect effects. Cross-sectional studies using relevant biomarkers can help substantiate the findings.
Further investigation is also needed to determine the elemental carbon content of KEE particles, and how particle-adsorbed metals and organic compounds, such as PAHs and organic sulphur compounds, may affect the toxicity.

Exposure-response relationship

As already mentioned, a suitable indicator for KEE is needed, and will be important for deriving exposure-response data for KEE.
For a hb-OEL the committees require high-quality (both setup and execution) observational studies, such as occupational cohort or case-control studies. Occupational cohort studies should include a long-term follow-up and a thorough exposure (which requires a suitable indicator) and health assessment, including complete work histories and relevant

information on potential confounders. Such additional research can also include studies comparing DEE and KEE, including studies particularly focussing on particles and elemental carbon content. Together, these findings would support the derivation of a hb-OEL for KEE and/or scientifically demonstrate the applicability of a health-based OEL for DEE to KEE.

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Annex A

Abbreviations and acronyms

ALI	air liquid interface
APU	auxiliary power units
ATSDR	Agency for Toxic Substances and Disease Registry (USA)
AvGas	aviation gasoline
BAL	bronchoalveolar lavage
BC	black carbon
BTEX	benzene, toluene, ethylbenzene, xylene
CA	chromosomal aberrations
CI	confidence interval
CLP	Classification, Labelling and Packaging
CO	carbon monoxide
CO_2	carbon dioxide
DECOS	Dutch Expert Committee on Occupational Safety
DEE	diesel engine exhaust
EBC	exhaled breath condensate
ECHA	European Chemicals Agency
EC	elemental carbon
EI	emission index
EPA	Environmental Protection Agency (USA)
EU	European Union
FID	flame ionisation detector
FPG	formamidopyrimidine-DNA-glycosilase
GC-FID	gas chromatography with flame ionization detection
GR	Gezondheidsraad (Health Council of the Netherlands)
GSE	ground support equipment
H_2O	water
hb-OEL	health-based occupational exposure limit
HC	hydrocarbons
HPLC	high performance liquid chromatography
IARC	International Agency for Research on Cancer
IATA	International Air Transport Association
ICAO	International Civil Aviation Organization
ICD	International Classification of Diseases
ICP-MS	inductively coupled plasma mass spectrometry
IPCC	Intergovernmental Panel on Climate Change
KEE	kerosene engine exhaust
kN	kiloNewton
LDSA	lung deposited surface area
LTO	landing and take-off
MN	micronucleus
NEG	Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals
NO_x	nitrogen oxides
NO_2	nitrogen dioxide

nvPM	non-volatile particulate matter
OC	organic carbon
OEL	occupational exposure limit
OH-PAHs	urinary hydroxylated polycyclic aromatic hydrocarbons
OR	odds ratio
PAH	polycyclic aromatic hydrocarbon
PM	particulate matter
PNC	particle number concentration
ppm	parts per million
REC	respirable elemental carbon
RIVM	National Institute for Public Health and the Environment
RNS	reactive nitrogen species
ROS	reactive oxygen species
SAF	sustainable aviation fuel
SCE	Sister chromatid exchange
SCOEL	Scientific Committee on Occupational Exposure Limits
SIA	secondary inorganic aerosol
SIR	standardised incidence rate
SO_2	sulphur dioxide
SOA	secondary organic aerosol
SO_x	sulphur oxides
TEM	transmission electron microscopy
TNO	Netherlands Organisation for Applied Scientific Research
UFP	ultrafine particles
UHC	unburned hydrocarbons
WHO	World Health Organization

Annex B

Literature search

Scientific literature databases PubMed, Scopus and EMBASE were searched for relevant publications on the composition of the KEE and predominantly on the health endpoints studied in human (worker) populations or *in vitro* or *in vivo* animal experiments. The search covered all years up to November 2025. Duplicates were removed and records were screened and included if considered relevant. In addition, these literature databases, grey literature (e.g., publications, reports, evaluations) from relevant agencies were consulted. This included publications or reports from RIVM, TNO, ATSDR, WHO, IARC, SCOEL, ECHA, IPCC, NEG-DECOS, United States Environmental Protection Agency (EPA), Health Effects Institute (HEI), Chevron, ICAO, FAA, European Union Aviation Safety Agency (EASA).

Additionally, relevant publications cited in selected publications or grey literature, but not identified with the primary search, were included. A substantial number of publications with relevant information concerning the composition and characteristics of KEE or its components have been retrieved this way since PubMed, Scopus and EMBASE mainly contain health-related scientific journals and publications. Retrieving publications by citations in other scientific publications or reports is a continuous process, not bound by the overall literature search.

The following keywords and combinations of keywords were used:
Keywords substance: jet engine exhaust, aircraft exhaust, jet fuel exhaust, jet A-1, jet A, kerosene exhaust, kerosene exhaust emissions, jet emissions, aircraft emissions, jet fuel emissions, aircraft combustion.

Keywords occupational exposure/workplace: airport, aviation, aeroengine, airplane, air traffic, engine take-off, landing, take off, LTO, ground idle, ground support, ground personnel, ground crew/staff, apron workers, baggage handlers, aircraft workers, airport staff, marshalls, engineers, technicians, fitters, flight line, job occupation*, work, job, occupational exposure.

Keywords health effects:
General toxicity: toxicity, toxicogenetic*.
Genotoxicity: genotoxicity, mutagenicity, mutations, DNA damage, micronuclei, mutagen, comet, transgenic, epigenetic, air liquid interface, cellular mechanisms
Carcinogenicity: cancer, carcinogenicity, tumorigenesis, cancer mortality, adenoma, carcinoma, occupational carcinogenesis

Keywords type of studies: cohort(s), case-cohort, epidemiological study, epidemiolog*, hospital-based, industrial-based, clinical, meta-analysis, meta-analyses, cross-sectional, case-control, nested case-control, pooled-analysis, pooled-analyses, carcinogenicity study, animal study, animal experiment, in vitro, in vivo, cell study.

Annex C

Overview of existing classifications for similar mixtures and components of kerosene engine exhaust

Table C1 Overview of similar mixtures and their classifications for carcinogenicity and mutagenicity

KEE component	EC-number	CAS-number	Harmonized classification according to Annex VI[1,2]	Notified classification[3]	IARC classification[4]	Remarks
Diesel engine exhaust	n.a.	n.a.			Group 1	Dutch classification[5] Group 1B (1995)
Diesel exhaust particles	n.a.	n.a.			Group 1	IARC considers the particulate component of diesel engine exhaust also as carcinogenic to humans
Gasoline engine exhaust	n.a.	n.a.			Group 2B	Gasoline itself is classified as mutagenic and carcinogenic, mainly due to the presence of benzene.

Abbreviations: n.a., not available.

[1] Harmonised classification - Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation).

[2] Harmonised classification and labelling (CLH) Hazard classes: Carc. 1A - *Known to have carcinogenic potential for humans, the placing of a substance is largely based on human evidence*. Carc. 1B - *Presumed to have carcinogenic potential for humans, the placing of a substance is largely ba sed on animal evidence*. Carc. 2 - *Suspected human carcinogen*. Category statement codes: H350 - *May cause cancer*; H351 - *Suspected of causing cancer*. Muta. 1A - *Substances known to induce heritable mutations in the germ cells of humans*. Muta. 1B - *Substances which should be regarded as if they induce heritable mutations in the germ cells of humans*. Muta. 2 - *Substances which cause concern for humans owing to the possibility that they may induce heritable mutations in the germ cells of humans*. Category statement codes: H340 - *May cause genetic defects*; H341: *Suspected of causing genetic defects* (CLP-regulation l272/2008).

[3] Notified classification and labelling according to CLP criteria based on ECHA's C&L inventory (ECHA, 2023). Only presented in absence of a harmonized classification for carcinogenicity and/or mutagenicity.

[4] IARC classification. Group 1: *Carcinogenic to humans*; Group 2A: *Probably carcinogenic to humans*; Group 2B: *Possibly carcinogenic to humans*; Group 3: *Not classifiable as to its carcinogenicity to humans*.

[5] GR classification. Group 1A*: Known to have carcinogenic potential for humans, classification is largely based on human evidence*; Group 1B: *Presumed to have carcinogenic potential for humans, classification is largely based on animal evidence*; Group 2: *Suspected human carcinogen*; Group 3: *The available data are insufficient to evaluate the carcinogenic properties of the substance*; Group 4: *The substance is probably not carcinogenic to man*.

Table C2 Overview of components of kerosene engine exhaust (KEE) and classifications for carcinogenicity and mutagenicity

KEE component	EC-number	CAS-number	Harmonized classification according to Annex VI[1,2]	Notified classification[3]	IARC classification[4]	Remarks
1,2,4-Trimethylbenzene	202-436-9	95-63-6				
1,2-Dimethylnaphtalene	209-364-7	573-98-8				
1,3,5-Trimethylbenzene	203-604-4	108-67-8				
1,3-Butadiene	203-450-8	106-99-0	Carc. 1A / H350 Muta. 1B / H340		Group 1	
1,4-Dimethylnaphthalene	209-335-9	571-58-4				
1,7-Dimethyl naphthalene	209-382-5	575-37-1				
1-Butene	203-449-2	106-98-9				
1-Methyl naphthalene	201-966-8	90-12-0				
1-Methylanthracene	210-224-2	610-48-0				
1-Methylphenanthrene	212-622-1	832-69-9		Carc. 2 / H351	Group 3	
2,6-dimethyl naphthalene	209-464-0	581-42-0				
2-Methylanthracene	210-329-3	613-12-7				
2-Methylnaphthalene	202-078-3	91-57-6				
2-Methylpentane	203-523-4	107-83-5				
2-Methylphenanthrene	219-791-0	2531-84-2				
3-Methylpentane	202-481-4	96-14-0				
5-Methylchrysene	681-936-2	3697-24-3		Carc. 1B/ H350 Carc. 2 / H351	Group 2B	Dutch classification[5] Group 1B
9,10-Anthracenedione	201-549-0	84-65-1	Carc. 1B / H350		Group 2B	
9-Fluorenone	237-116-8	13629-22-6				
9-Methylanthracene	212-299-7	779-02-2				
Acenaphthene	201-469-6	83-32-9			Group 3	
Acenaphthylene	205-917-1	208-96-8				
Acetaldehyde	200-836-8	75-07-0	Carc. 1B / H350 Muta. 2 / H341		Group 2B	Dutch classification[5] Group 1B
Acrolein	203-453-4	107-02-8		Carc. 2 / H351	Group 2A	
Aluminium	231-072-3	7429-90-5				
Anthanthrene	205-884-3	191-26-4		Carc. 1B/ H350	Group 3	
Anthracene	204-371-1	120-12-7		Carc. 1B / H350 Carc. 2 / H351	Group 2B	

KEE component	EC-number	CAS-number	Harmonized classification according to Annex VI[1,2]	Notified classification[3]	IARC classification[4]	Remarks
Arsenic	231-148-6	7440-38-2		Carc. 1A / H350 Muta. 2 / H341	Group 1	
Barium	231-149-1	7440-39-3				
Benz[a]anthracene	200-280-6	56-55-3	Carc. 1B / H350		Group 2B	
Benzene	200-753-7	71-43-2	Carc. 1A / H350 Muta. 1B / H340		Group 1	
Benzo(a)pyrene	200-028-5	50-32-8	Carc. 1B / H350 Muta. 1B / H340		Group 1	
Benzo(b)fluoranthene	205-911-9	205-99-2	Carc. 1B / H350		Group 2B	
Benzo(k)fluoranthene	205-916-6	207-08-9	Carc. 1B / H350		Group 2B	
Benzo[b]naph[2,l- d]thiophene	205-948-0	239-35-0			Group 3	
Benzo[c]phenanthrene	205-896-9	195-19-7		Carc. 2 / H351 Muta. 2 / H341	Group 2B	
Benzo[e]pyrene	205-892-7	192-97-2	Carc. 1B / H350		Group 3	
Benzo[ghi]perylene	205-883-8	191-24-2			Group 3	
Benzo[j]fluoranthene	205-910-3	205-82-3	Carc. 1B / H350		Group 2B	
Benzo[k]fluoranthene	205-916-6	207-08-9	Carc. 1B / H350		Group 2B	
Biphenyl	202-163-5	92-52-4				
Black carbon	n.a.	n.a.			Group 1	PM in outdoor air (including its components) is classified as carcinogenic by IARC.
Cadmium	231-152-8	7440-43-9	Carc. 1B / H350 Muta. 2 / H341		Group 1	
Calcium	231-179-5	7440-70-2				
Carbon dioxide	204-696-9	124-38-9				
Carbon disulphide	200-843-6	75-15-0				
Carbon monoxide	211-128-3	630-08-0				
Chloromethane	200-817-4	74-87-3	Carc. 2 / H351		Group 3	
Cholanthrene	207-528-2	479-23-2				
Chromium (metallic)	231-157-5	7440-47-3	Carc. 1B / H350 Cr(VI)		Group 1 Cr(VI)	Metallic chromium can be a precursor of Cr(VI) or Cr(III) Metallic chromium is classified as Group 3 in the Netherlands[5]
Chrysene	205-923-4	218-01-9	Carc. 1B / H350 Muta. 2 / H341		Group 2B	
cis-2-Butene	209-673-7	590-18-1				
Cobalt	231-158-0	7440-48-4	Carc. 1B / H350 Muta. 2 / H341		Group 2A	
Copper	231-159-6	7440-50-8				
Coronene	205-881-7	191-07-1			Group 3	

KEE component	EC-number	CAS-number	Harmonized classification according to Annex VI[1,2]	Notified classification[3]	IARC classification[4]	Remarks
Cyclopenta[c,d]pyrene	690-388-3	27208-37-3			Group 2A	
Dibenz[ac]anthracene	205-920-8	215-58-7		Carc. 1A / H350 Muta. 2 / H341	Group 3	
Dibenz[ah]anthracene	200-181-8	53-70-3	Carc. 1B / H350		Group 2A	
Dibenzo[ae]pyrene	205-891-1	192-65-4		Carc. 1B / H350 Carc. 2 / H351 Muta. 2 / H341	Group 3	Dutch classification[5] Group 2
Dibenzo[ah]pyrene	205-878-0	189-64-0	Carc. 1B / H350 Muta. 2 / H341		Group 2B	Dutch classification[5] Group 1B
Dibenzo[ai]pyrene	205-877-5	189-55-9	Carc. 1B / H350 Muta. 2 / H341		Group 2B	Dutch classification[5] Group 1B
Dibenzo[al]pyrene	205-886-4	191-30-0	Carc. 1B / H350 Muta. 2 / H341		Group 2A	Dutch classification[5] Group 2
Dimethyl sulphide	200-846-2	75-18-3				
Docosane	211-121-5	629-97-0				
Dodecane	203-967-9	112-40-3				
Dotriacontane	208-881-5	544-85-4				
Eicosane	204-018-1	112-95-8				
Elemental carbon	n.a.	n.a.			Group 1	Both diesel exhaust particles as PM in outdoor air are classified as carcinogenic to humans
Ethane	200-814-8	74-84-0				
Ethine (ethyne, acetylene)	200-816-9	74-86-2				
Ethyl nitrate	210-903-3	625-58-1				
Ethylbenzene	202-849-4	100-41-4		Carc. 1A / H350 Carc. 2 / H351 Muta. 1B	Group 2B	
Ethylene	200-815-3	74-85-1				Dutch classification[5] Group 3
Fluoranthene	205-912-4	206-44-0			Group 3	
Fluorene	201-695-5	86-73-7			Group 3	
Formaldehyde	200-001-8	50-00-0	Carc. 1B / H350 Muta. 2 / H341		Group 1	
Heneicosane	211-118-9	629-94-7				
Hentriacontane	685-676-0	630-04-6				
Heptacosane	209-792-4	593-49-7				
Heptadecane	211-108-4	629-78-7				
Hexacosane	211-124-1	630-01-3				

KEE component	EC-number	CAS-number	Harmonized classification according to Annex VI[1,2]	Notified classification[3]	IARC classification[4]	Remarks
Hexadecane	208-878-9	544-76-3				
Indeno[l,2,3-cd]pyrene	205-893-2	193-39-5		Carc. 2 / H351	Group 2B	Dutch classification[5] Group 1B
i-Pentane (Isopentane)	201-142-8	78-78-4				
Iron (metallic)	640-395-2	7439-89-6				Process of iron and steel founding is classified as carcinogenic in the Netherlands[5]
Isobutane	200-857-2	75-28-5				
Isoprene	201-143-3	78-79-5	Carc. 1B / H350 Muta. 2 / H341		Group 2B	
Lead	231-100-4	7439-92-1		Carc. 1A / H350 Carc. 2 / H351 Muta. 2 / H341	Group 2B	
Magnesium	231-104-6	7439-95-4				
Manganese	231-105-1	7439-96-5				
Mercury	231-106-7	7439-97-6		Muta. 2 / H341	Group 3	
Methane	200-812-7	74-82-8				
Methyl nitrate	209-941-3	598-58-3				
Molybdenum	231-107-2	7439-98-7				
m-Xylene	203-576-3	108-38-3			Group 3	
Naphthalene	202-049-5	91-20-3	Carc. 2 / H351		Group 2B	Dutch classification[5] Group 3
n-Butane	203-448-7	106-97-8				
n-Decyl cyclohexane	217-272-3	1795-16-0				
n-Heptane	205-563-8	142-82-5		Carc. 1B / H350 Muta. 1B / H340		
n-Heptyl cyclohexane	227-041-9	5617-41-4				
n-Hexane	203-777-6	110-54-3				
Nickel	231-111-4	7440-02-0	Carc. 2 / H351		Group 2B	
Nitrogen oxides (NO_x)	n.a.	n.a.				Comprises primarily NO and NO_2
n-nonyl cyclohexane	220-739-4	2883-02-5				
n-octyl cyclohexane	217-271-8	1795-15-9				
Nonacosane	211-126-2	630-03-5				
Nonadecane	211-116-8	629-92-5				
non-volatile particulate matter (nvPM)	n.a.	n.a.			Group 1	PM in outdoor air is classified as carcinogenic by IARC
Norhopane	n.a.	n.a.				Not further specified

KEE component	EC-number	CAS-number	Harmonized classification according to Annex VI[1,2]	Notified classification[3]	IARC classification[4]	Remarks
n-Pentane	203-692-4	109-66-0				
n-Undecyl cyclohexane	258-976-0	54105-66-7				
Octacosane	211-125-7	630-02-4				
Octadecane	209-790-3	593-45-3				
o-Xylene	202-422-2	95-47-6		Carc. 1B / H350	Group 3	m-, o-, p- xylenes have a Group 3 IARC rating; only o- xylene has a notified carcinogenic classification as well
PAHs	n.a.	n.a.			Group 1 (particle-bound PAH)	PM in outdoor air are classified as carcinogenic by IARC PAH emissions from processes with soot, tar or coal are classified as carcinogenic in the Netherlands[5]
Pentacosane	211-123-6	629-99-2				
Pentadecane	211-098-1	629-62-9				
Perylene	205-900-9	198-55-0			Group 3	
Phenanthrene	201-581-5	85-01-8		Carc. 2 / H351	Group 3	
Phytane	211-332-2	638-36-8				
Pristane	217-650-8	1921-70-6				
Propane	200-827-9	74-98-6		Carc. 1A / H350 Muta 1B / H340		
Propene (propylene)	204-062-1	115-07-1			Group 3	
Propionaldehye	204-623-0	123-38-6				
p-Xylene	203-396-5	106-42-3			Group 3	
Pyrene	204-927-3	129-00-0			Group 3	
Respirable particulate matter	n.a.	n.a.			Group 1	PM in outdoor air is classified as carcinogenic by IARC
Retene	207-597-9	483-65-8				
Silicon	231-130-8	7440-21-3				
Sodium	231-132-9	7440-23-5				
Soot (carbonaceous PM)	n.a.	n.a.			Group 1	PM in outdoor air is classified as carcinogenic by IARC
Styrene	202-851-5	100-42-5		Carc. 2 / H351 Muta. 2 / H341	Group 2A	Dutch classification[5] Group 1B
Sulphur oxides (SO_x)	na	na				primarily SO_2 and SO_3
Tetracosane	211-474-5	646-31-1				
Tetradecane	211-096-0	629-59-4				
Tetratriacontane	238-013-0	14167-59-0				
Titanium	231-142-3	7440-32-6				
Toluene	203-625-9	108-88-3		Carc. 1A / H350 Muta 1B / H340	Group 3	

KEE component	EC-number	CAS-number	Harmonized classification according to Annex VI[1,2]	Notified classification[3]	IARC classification[4]	Remarks
Total alkanes	n.a.	n.a.				Not further specified
Total alkenes	n.a.	n.a.				Not further specified
Total alkynes	n.a.	n.a.				Not further specified
Total aromatics	n.a.	n.a.				Not further specified
trans-2-Butene	210-855-3	624-64-6				
Triacontane	211-349-5	638-68-6				
Tricosane	211-347-4	638-67-5				
Tridecane	211-093-4	629-50-5				
Tritriacontane	686-232-9	630-05-7				
Ultrafine particulate matter	n.a.	n.a.			Group 1	PM in outdoor air is classified as carcinogenic by IARC
Unburned hydrocarbons	n.a.	n.a.			Group 1	e.g., benzene, hexane. Benzene is classified as carcinogenic by IARC.
Vanadium	231-171-1	7440-62-2				
Xylene	215-535-7	1330-20-7			Group 3	
Zinc	231-175-3	7440-66-6				
Zirconium	231-176-9	7440-67-7				

Sources: RIVM (2024)[3], Gezondheidsraad (2023)[169], SER (accessed September 2024)[7]

Abbreviations: IARC, International Agency for Research on Cancer; GR, Gezondheidsraad (Health Council of the Netherlands); NL-OEL, Dutch occupational exposure limit; EU-BOEL, European Union binding occupational exposure limit; EU-IOEL European Union indicative occupational exposure limit; TWA-8h, time weighted average over 8 hours; TWA-15min, time weighted average over 15 minutes; ppm, particles per million; n.a., not available.

1 Harmonised classification - Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation).

2 Harmonised classification and labelling (CLH) Hazard classes: Carc. 1A - *Known to have carcinogenic potential for humans, the placing of a substance is largely based on human evidence*. Carc. 1B - *Presumed to have carcinogenic potential for humans, the placing of a substance is largely based on animal evidence*. Carc. 2 - *Suspected human carcinogen*. Category statement codes: H350 - *May cause cancer*; H351 - *Suspected of causing cancer*. Muta. 1A - *Substances known to induce heritable mutations in the germ cells of humans*. Muta. 1B - *Substances which should be regarded as if they induce heritable mutations in the germ cells of humans*. Muta. 2 - *Substances which cause concern for humans owing to the possibility that they may induce heritable mutations in the germ cells of humans*. Category statement codes: H340 - *May cause genetic defects*; H341: *Suspected of causing genetic defects* (CLP-regulation l272/2008).

3 Notified classification and labelling according to CLP criteria based on ECHA's C&L inventory (ECHA, 2023). Only presented in absence of a harmonized classification for carcinogenicity and/or mutagenicity.

4 IARC classification. Group 1: *Carcinogenic to humans*; Group 2A: *Probably carcinogenic to humans*; Group 2B: *Possibly carcinogenic to humans*; Group 3: *Not classifiable as to its carcinogenicity to humans*.

5 GR classification. Group 1A*: Known to have carcinogenic potential for humans, classification is largely based on human evidence*; Group 1B: *Presumed to have carcinogenic potential for humans, classification is largely based on animal evidence*; Group 2: *Suspected human carcinogen*; Group 3: *The available data are insufficient to evaluate the carcinogenic properties of the substance*; Group 4: *The substance is probably not carcinogenic to man*.

Annex D

Study summaries on mutagenicity and carcinogenicity

Table D1 Summary table of mutagenicity in humans

Study design and population	Exposure assessment	Health assessment	Results	Analysis	Remarks
Andersen *et al.* (2021)[106] Cross-sectional study Military Air Force base, Denmark *Study population:* 79 employees (87% males): 42 exposed (crew chiefs (n=17), aircraft engineers (n=14), fuel operators (n=6), munition specialists (n=5)), 37 non-exposed (office workers (n=31), avionics (n=6), from the same military base) Employees were 25-61 years old, mean age: 46.9 years	*Exposure measurements:* • PAH • organophosphate esters (OPEs) • UFP See Chapter 4 for details	Self-reported information: working history, PPE use, medical history, lifestyle factors. *Outcome(s): g*enetic damage levels, lung function, acute phase inflammatory markers *Assessment:* Genotoxicity: • Micronucleus (MN) assay for chromosome damage in transferrin- positive peripheral blood reticulocytes. • DNA damage by comet assay on peripheral blood mononuclear cells. Inflammation markers: • ELISA assay used to determine serum amyloid A and C- reactive protein in plasma. Lung function: • Spirometery: forced vital capacity (FVC), forced expiratory volume in one second (FEV1), peak expiratory flow (PEF). Settings adjusted for ethnicity.	No evidence for genetic damage. DNA strand breaks (number of lesions/10^6 basepairs) • Exposed, n=42: mean 0.09, sd=0.04 • Non-exposed, n=35: mean 0.10, sd=0.04 p-value Welch one-way test: 0.202 Micronucleated reticulocytes (‰): • Exposed, n=37: 3.01, sd=1.2 • Non-exposed, n=33: 3.04, sd=1.4 p-value Welch one-way test: 0.990 No evidence for effects on biomarkers of systemic inflammation, or lung function.	*Statistical analyses:* Welch one-way test (group means), Wilcoxon rank sum test (OPEs), Hotelling T-squared test (means outcomes). *Sensitivity analysis:* excluding female participants; excluding participants from not exposed group. Linear model adjusted for age, sex, BMI, relevant health history, lung function and smoking history.	Small numbers of participants and exposure measurements. Co-exposure to other carcinogenic substances possible. Non-exposed (reference) group may have been exposed as well (see Chapter 4). Self-reported information on smoking, drug or alcohol use was used as exclusion criterium Possible reporting bias. No specific evidence for mutagenic or carcinogenic effects.

Study design and population	Exposure assessment	Health assessment	Results	Analysis	Remarks
Cavallo *et al.* (2006)[120] Cross-sectional study Leonardo da Vinci Airport, Rome, Italy Study population: Airport personnel: • High exposed n=24 (e.g., baggage handlers). • Medium exposed n=17 (e.g., security staff, cleaning staff). • Non-exposed group n=31 (e.g., airport office workers).	*Exposure measurements:* • 23 PAHs (gas-phase and particle-bound PAHs) See Chapter 4 for details	*Outcome:* genotoxic effects and direct-oxidative DNA damage. *Assessment: in blood* • Micronucleus (MN) assay to identify DNA damage in lymphocytes and exfoliated buccal cells. • Fpg-modified comet assay used to evaluate oxidative DNA damage in lymphocytes. • Sister chromatid exchange • (SCE) in lymphocytes (SCE / cell per subject) blood samples collected on day 3 of workweek. • Analysis of chromosomal aberrations (CA).	*SCE frequency (mean (sd)):* Exposed: 4.61 (0.80) Non-exposed: 3.84 (0.58) M-W-test: p-value <0.001 High exposed: 4.61 (0.87) Non-exposed: 3.84 (0.58) K-W: p<0.001 Medium exposed: 4.59 (0.71) Non-exposed: 3.84 (0.58) K-W: p<0.001 Exposed showed higher mean SCE frequencies compared to non-exposed. *Fpg-modified comet assay (tail moment).* • Fpg- treated cells: Exposed: 55.86 Non-exposed: 43.98 • Untreated cells: Exposed: 43.01 Non-exposed: 36.01 Exposed showed increased levels of CA and increased tail moment in lymphocytes compared to non-exposed. *MN assay in exfoliated buccal cells and lymphocytes.* • Buccal cells (mean (sd)): Exposed: 0.64 (0.98 Non-exposed: 0.64 (0.54) M-W: p <0.251 • Lymphocytes (mean (sd)): Exposed: 8.15 (3.70) Non-exposed: 7.10 (4.21) M-W: p <0.129 There was no difference in MN between exposed and non-exposed for exfoliated buccal cells and lymphocytes.	*Statistical analyses:* • Student's t-test, Mann-Whitney U- test (M-W), ANOVA, Kruskal-Wallis (K-W) used to test for significant differences in biological parameters. • Z normal test to evaluate statistically significant differences in CA	Small number of participants. No information on participation Rate, inclusion and exclusion criteria. Age difference between high and medium exposed. Co-exposure to other carcinogenic substances (e.g., diesel engine exhaust, solvents and fuels) was possible.

Study design and population	Exposure assessment	Health assessment	Results	Analysis	Remarks
Pitarque *et al.* (1999)[112] Cross-sectional study Barcelona Airport, Spain *Study population* Exposed n=39 workers assisting during charge and discharge of aircraft. Non-exposed n=11 workers from the University Campus, Barcelona.	*Exposure substances:* petroleum derivatives, engine exhausts *Exposure assessment:* Benzene, toluene and xylene. See Chapter 4 for details *Questionnaire*: information on confounders and potential exposure at the workplace.	*Outcome:* genetic damage in peripheral blood lymphocytes. *Assessment: in blood* • SCE analysis • Micronuclei (MN) assay: total number of MN and the frequency of binucleated cells with MN (BNMN) were scored • Comet assay Test for cytokinesis block proliferation index (CBPI) calculated based on MN.	*SCE frequency* (mean (sd)): • Exposed n=39: 8.07 (0.24) • Non-exposed n=11: 7.30 (0.28) Analysis was also performed for smokers and non-smokers separately. *SCE in smokers* (mean (sd)): • Exposed n=22: 8.70 (0.31) • Non-exposed n=3: 7.39 (0.23) p-value t-test: <0.01 *total MN assay* (mean (sd)): • Exposed n=39: 7.62 (0.49) • Non-exposed n=11: 12.55 (1.65) p-value t-test: <0.001 *Comet length* (mean (sd)): • Exposed n=37: 45.51 (1.55) • Non-exposed n=11: 39.25 (1.78) p-value t-test: <0.05 Slight but significant differences in the mean comet length and genetic damage index (GDI, not reported) were observed.	*Statistical analysis:* t-tests: SCE, total MN, BNMN, percentage of binucleated cells (%BN), High Frequency Cells (HFC), mean comet length, and p21 values (pooled two-sample one-sided t-test) Chi^2 test for Proliferation Rate Index (PRI), and distribution of DNA damage.	No information on timing of blood samples vs. work shift or on timing of exposure measurements. No detailed information on sort of information collected through questionnaire or how the questionnaire was administered Workers exposed to petroleum derivatives and engine exhausts, co-exposure to other carcinogenic substances could not be excluded Considerable age difference between exposed and non-exposed workers. Also % smokers among the exposed was considerably higher than among the controls.

Study design and population	Exposure assessment	Health assessment	Results	Analysis	Remarks
Lemasters et al. (1997)[136] Prospective repeated measurement study *Study population:* N=58 military personnel: aircraft sheet metal workers (n=6), aircraft painters (n=6), jet fuelling operation workers (n=15), flight line crew (n=23; ground crew, jet engine mechanics). Age 18-50 years. Each participant served as his own control. Response rate: 79%	Fuel and solvents, including jet engine exhaust. *Exposure assessment:* 3 consecutive days of full-shift air monitoring with breath sampling on day 3. Only flight line crew (n=23) were exposed to jet engine exhaust *Questionnaire*: Work history, medical history, smoking, alcohol and caffein use. Flight line crew showed statistically significant differences for fuel and benzene exposure in personal air samples between 15- and 30-week sampling period.	*Outcome: g*enotoxic effects, SCE and MN frequency. *Assessment:* Pre- and post (after 15 or 30 weeks) assessment of SCE and MN frequency using blood lymphocyte MN and SCE analysis. Cytogeneticist was blind to exposure status.	No difference between baseline measurements for SCE and MN in non-exposed and exposed subjects with relevant exposure.	*Statistical analysis:* Paired t-tests for significant changes in the outcome variable within groups from baseline to 15 or 30 weeks after exposure	Study investigated occupational exposure to fuels, solvents and jet engine exhaust. Jet engine exhaust was not the aim of the study. Co-exposure to other carcinogenic substances could not be excluded Small number of participants with relevant exposure, n=23 flight line crew. Flight line crew were also exposed to fuel, solvents and possibly paints.

Table D2 Summary table of mutagenicity in animals

Species	Experimental period and design	Concentration/Dose	Observations and results	Remarks
Bendtsen *et al.* (2019)[107] Female C57BL / 6Tac mice, 8 weeks old at time of exposure 212 mice, 6-8 mice per exposure group	Single intratracheal instillation. Mice sacrificed after 1, 28 or 90 (high dose only) days. *Effects studied:* Genotoxicity: DNA strand breaks (Comet Assay) on lung and liver tissue and cells from the BAL fluid. Inflammation: Lung pathology, cellular content of Broncho-alveolar lavage (BAL) fluid. Acute phase response: Serum amyloid A (Saa) levels in lung (mRNA), liver (mRNA) and blood (protein). *Exposure agents*: Particles collected at a commercial airport (CAP) and a non-commercial airfield (NCA) (see Chapter 4 Table 8 and 9 for exposure estimates). Positive controls: carbon black (CB) (Printex 90) and diesel exhaust particles (NIST2975 and NIST1650). Negative control: Nanopure water (vehicle).	CAP/NCA: 6, 18 and 54 µg per mouse Positive control CB: 54 µg per mouse, NIST2975: 18, 54, 162 µg per mouse	Increased levels of DNA strand breaks in BAL cells were observed for NCA and NIST2975 (18 µg), day 1 post-exposure. Day 28 post-exposure CAP (6 µg) resulted in higher tail length and % tail DNA in liver cells. No dose-response was observed. Inflammation and acute phase response: NCA, CAP, NIST2975 and CB exposure resulted in dose-dependent increases in total number of cells, neutrophils, and eosinophils after 1 day in BAL, as well as increased levels of Saa3 mRNA in lung tissue and Saa3 protein in plasma. Day 28 post-exposure, lymphocytes were increased for NCA, CAP, NIST2975 and CB, as well as neutrophils for NCA and CB. *Statistics*: One-way ANOVA with Dunnett's or Sidak's multiple comparison test. Nonparametric data with Kruskal-Wallis with Dunn's multiple comparisons test. Dose-response effects were tested for linear trend.	Difficult to draw conclusions for the lung due to inflammation. However, an effect was observed in the liver (in the absence of inflammation), with a positive result for DNA damage. The controls included in this analysis are considered relevant, and induced inflammation at similar levels as aircraft particles. CAP contained organic compounds including salt, pollen and soot. CAP and NCA contained metals and PAHs, of which the total PAH content was similar to that of NIST2975. The metal content was higher in NCA and CAP than NIST2975. Intratracheal instillation is not considered as a physiological relevant exposure route compared to inhalation exposure.

Table D3 Summary table of mutagenicity studies in vitro

Tissue/cell line	Experimental period and design	Concentration/Dose	Observations and results	Remarks
Melzi et al. (2024)[137] Lung epithelial cells Blank filter: negative control NIST2975 (DEE particles): positive control	*Effects studied:* DNA strand breaks and oxidative DNA damage (comet assay) *Exposure agent:* Primary particulate matter (PM) collected from the combustion of twelve aviation fuels. *Experiment:* • PM collected on filters, from 1) Rich-Quench-Lean (RQL) combustion rig, 2) liquid-fuelled Combustion Aerosol Standard (CAST) Generator. • PM samples extracted in methanol and re-suspended in water. • Nebulized onto human lung epithelial cells (Calu-3) using an Air-Liquid Interface (ALI) exposure system. • Cells collected 24h after exposure.	Expected deposition: 450 ng/cm^2 Deposited dose in ng/cm^2 (mean (SEM)): • negative control: 0 (0) ng/cm^2 • positive control: 456 (32) ng/cm^2	*Statistics:* One- and two-way ANOVA, Dunnett's test No observed cytotoxicity or significant changes in transepithelial electrical resistance (TEER). *DNA damage:* Statistically significant increase in DNA damage after exposure to PM extracts from combustion aerosol standard (CAST) generated samples, both in monoculture and co-culture. No significant difference in DNA damage in samples treated with diesel soot compared to the control.	Results on DNA damage are only presented graphically, which limits the quantitative estimation of the results.
McCartney et al. (1986)[138] Salmonella typhimurium strains TA98, TA98NR and TA98/1,8-DNP$_6$	*Effects studied:* Bacterial reverse mutation assay (Ames test); number of mutant colonies per plate. Experiment in triplicate. *Particle sampling:* • Near the runway (sampling time 6 hours at 18 m from runway) • Directly behind idling aircraft engine (sampling time 7 min at 9 m from the engine, 61 cm above the ground). • Control sample was taken at the site in absence of idling planes (sampling time 7 min). *Sampler:* High-volume filter collector with a glass filter. After solvent extraction and evapouration, the residues were dissolved in DMSO. *Experiment:* Exposure via suspension on petri-plates. Control sample (each assay) obtained from unexposed filter. Positive controls: 2-nitrofluorene (33 µg/plate), 1-nitropyrene (0.1 µg/plate) and 1,8-dinitropyrene (0.008 µg/plate).	Amount of particulate equivalent applied: Runway: 0, 0.10, 0.33 and 1.0 mg Idling: 0, 0.015, 0.050 and 0.150 mg Control: amount not given as no particles were detected in this sample	*Statistics:* not reported. Dose-dependent increase in the number of mutant (revertant) colonies per plate: Runway particles: TA98: 19, 32, 30, 132, TA98NR: 15, 31, 31, 44, TA98/1,8-DNP$_6$: 5, 6, 11, 20 Idling particles: TA98: 17, 40, 67, 156, TA98NR: 12, 19, 53, 76, TA98/1,8-DNP$_6$: 8, 9, 13, 20	Distinction made between runway and idling. The air control sample taken at the site contained no measurable particles and upon extraction no mutagenicity was shown, i.e. identical to the extracted unexposed filter.

Table D4 Summary table of carcinogenicity in humans

Study design and population	Exposure assessment	Health assessment	Results	Remarks
Cohort studies				
Garland *et al.* (1998)[139] Cohort study *Study population:* white US naval men diagnosed with testicular cancer (n=143) *Study period:* 1974-1979 *Reference population:* US SEER* population and the total Navy personnel.	Information on demographics, hospitalizations, work and service history available. Job titles used as proxy for occupational exposure. Aviation support technicians whose activities may occur on flight decks of aircraft carriers, at aircraft repair facilities, and at airports.	*Health outcome:* testicular cancer (n=143), ICD-code 186 (8th revision.) Cases identified through medical files from Naval Health Research Centre. For 87% pathology reports were available. *Outcomes:* standardised incidence ratios (SIR), 95% confidence intervals (95% CI), p-value	2,275,829 person-years in the study Aviation Support Equipment Technician (n=5, 9,951 person-years): • SEER population: SIR=6.2, 95% CI 1.9-13.0, p<0.001 • total Navy: SIR=6.9, 95% CI 2.1-14.4, p<0.001	Small number of cases, leading to wider confidence intervals. Co-exposures to other carcinogenic substances could not be excluded Only data for active-duty naval personnel. No follow-up after discharge. *US SEER = Surveillance, Epidemiology and End Results (SEER) of National Cancer Institute
Foley *et al.* (1995)[140] *Study population:* Royal Air Force (RAF) UK personnel *Study period*: 1984-1989. *Reference population:* General UK population (expected number of cases)	Information on demographics, work and service history categorized by age group and occupation available. RAF occupation used as proxy for exposure.	*Health outcome:* testicular cancer (n=148), ICD-code 186 (8th revision). Histologically confirmed. *Outcome(s): a*bsolute number of cases and incidence per 100,000. Relative risks (RR), 95% confidence intervals (95% CI), p-value	Incidence rates of testicular cancer were particularly high for RAF personnel working closely in the vicinity of aircraft. Total RR testicular cancer: UK population: (O/E=148/45.2), RR=3.27, 95% CI 2.43-4.31, P<0.001.	Small number of cases, leading to wider confidence intervals in stratified analyses. Co-exposures to other carcinogenic substances could not be excluded Only data for active-duty naval personnel. No follow-up after discharge.
Case-control studies				
Barul et al. (2025)[141] Population-based case-control study Montreal, Canada *Study period*: 2005-2012 *Study population:* *Eligible*: men ≤75 years of age, residents of Greater Montreal, registered on the electoral list *Cases*: 1924 cases *Controls*: 1989 population-controls	*In-person interviews*: socio-demographics, anthropometrics, lifestyle factors, medical and occupational history. *Exposure assessment*: • Evaluation by industrial hygienists. • Intensity, frequency and reliability of exposure to engine exhausts for each job held for ≥2 years. • Details on chemical agents and equipment used, tasks, protective measures and workplace characteristics.	*Health outcomes:* prostate cancer (n=1924) *Outcome assessment:* cases actively ascertained (80% coverage of prostate cancer cases). 7 Montreal hospitals Pathology reports extracted to identify incident cases	No associations between exposure to jet fuel engine exhaust and prostate cancer was found.	Small number of cases (n=15) and controls (n=50) exposed to jet fuel engine exhaust Participation rate among cases (79%) was higher compared to controls (56%).

Study design and population	Exposure assessment	Health assessment	Results	Remarks
Parent *et al.* (2000)[142] *(see Siemiatycki et al. 1988)* Population-based case-control study Montreal, Canada *Study population:* men 35-70 years of age, derived from 19 Montreal hospitals *Cases*: 142 renal cell carcinomas *Controls*: 1900 cancer-controls, 533 population-controls, 2433 pooled-controls.	*Exposure agent(s):* see Siemiatycki et al. (1988) Interviews 1979-1985 *Statistical analysis* Unconditional logistic regression, adjusted for: 1) non-occupational factors, and 2) both non-occupational and occupational.	*Health outcome:* renal cell carcinoma (n=142), ICD-code 189.0 (9th revision) Ascertainment of cases 97%. Histologically confirmed.	*Outcomes*: adjusted OR, 95% CI using the pooled-control group. Adjusted for age, smoking and BMI. Exposure to jet fuel engine emissions (n=4): OR=2.7, 95% CI 0.9-8.1 Aircraft mechanics (n=4): OR=2.8, 95% CI 1.0-8.4	Small numbers of exposed cases (n=4) leading to imprecise estimates. Pooled-controls as reference may reduce possible selection bias.
Ryder et al. (1997)[144] Matched case-control study *Study period*: 1976-June 1994 *Study population*: serving naval personnel, 15-59 years of age *Cases*: 110 testicular cancer cases *Controls*: 4 controls per case, matched on date of birth (± 2 years) and length of service (at least as long as the case until diagnosis of the case).	*Assessment:* Job title used as proxy for exposure	*Health outcome:* testicular cancer (n=110), ICD code 186. *Assessment:* record linkage with 2 naval hospitals and the Defense Analytical Services Agency (DASA). Histopathological confirmed.	*Outcomes*: OR, 95% CI Relative to all other branches combined, Fleet Air Arm (n=19): OR=1.90, 95% CI 1.04-3.48 Relative to all other specilities combined, Air Engineers (n=17): OR=2.32, 95% CI 1.20-4.48 Sub-group analysis relative to all other sub-specialities combined, Aircraft Handling sub-speciality (n=6): OR=7.31, 95% CI 1.81-29.53	Small number of cases in sub-analyses leading to imprecise estimates and wide confidence intervals Limited information about the working conditions or lifestyle factors, no detailed information (e.g., tasks, working conditions) was considered.
Siemiatycki (1988)[143] *(see Parent et al. 2000)* Population-based case-referent study Montreal, Canada *Study population:* male cancer patients, 35-70 years of age *Cases*: 3726 cancer cases *References*: for each case-series a referent was selected from the other cancer cases	*Exposure:* 10 types of exhaust and combustion products, including jet engine exhaust *Assessment:* • In-depth interviews 1979-1985 with detailed information on work history and lifestyle • Evaluation by industrial hygienists and chemists of intensity (low, medium, high), frequency and reliability of exposure to engine exhausts based on work history.	*Outcome:* cancer at several sites (esophageal, stomach, colon, rectosigmoid, rectal, pancreatic, lung, prostate, bladder, kidney, melanoma of the skin, non-Hodgkin's lymphoma. *Assessment:* cases ascertained if diagnosed in any of 19 participating hospitals. Histologically confirmed.	*Statistics*: logistic regression analysis Criteria for assigning covariate as a confounder was if it changed the estimate of the disease-exposure ratio by >10% No statistically significant associations between jet engine exhaust and any of the other cancer sites were found.	Cancer cases were also references for other cancer cases, assuming that the other cancers are not related to the exposure of interest. Small number of cases for outcomes, leading to low statistical power to detect effects.

Annex E

Application of CLP criteria

The sections below describe the application of the CLP criteria to the classification of KEE for germ cell mutagenicity (also referred to as mutagenicity) and carcinogenicity.

Germ cell mutagenicity

Regarding the classification of mixtures for germ cell mutagenicity, there are two possible scenarios described in the CLP regulation. Scenario 1 describes 2 routes. The first route is applicable when data are available for the individual ingredients of the complete mixture (route 1, CLP Annex I: *3.5.3.2.1*). The second route is applicable on a case-by-case basis. Test data on mixtures may be used for classification when demonstrating effects that have not been established from the evaluation based on the individual ingredients (route 2, CLP Annex I: *3.5.3.2.1*). Scenario 2 can be applied when test results of the complete mixture are not conclusive *(*CLP Annex I: 3.5.3.3.1).

Scenario 1 – Annex I: 3.5.3.2.1 of the CLP regulation

Route 1 describes the following '*classification of mixtures will be based on the available data for the individual ingredients of the mixture using concentration limits for the ingredients classified as germ cell mutagens*.'

However, in the case of KEE, the mixture does not contain one or more components which are individually classified as a Category 1 or 2 mutagen at concentrations of 0.1% or higher. Chapter 2.3.1 describes that soot (~0.1% of ~0.4% of the residual products of incomplete combustion) and hydrocarbons (~4.0% of ~0.4% of the residual products of incomplete combustion) form a very small fraction of the total composition of KEE (see also Figure 3 in Chapter 2). Therefore, based on the concentrations of the individual components KEE cannot be classified for germ cell mutagenicity (see also Figure E1).

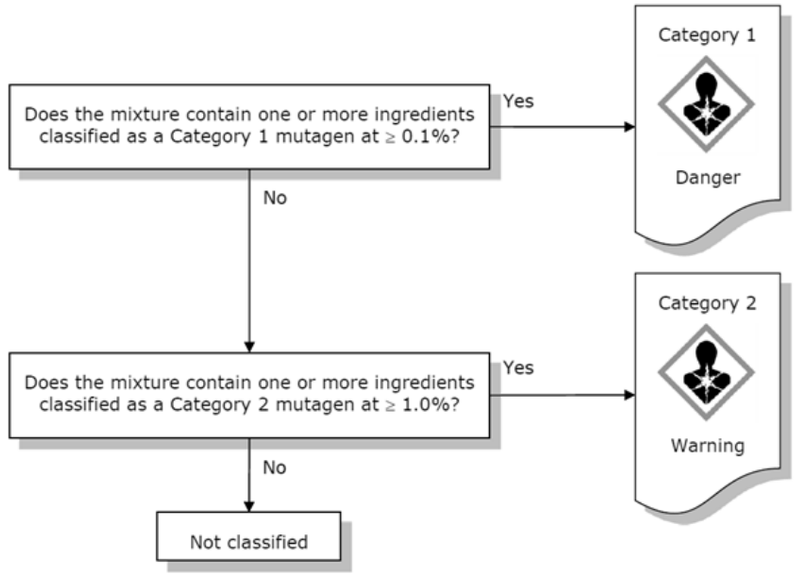

Figure E1 Schematic decision tree for classification for germ cell mutagenicity, based on individual components of the mixture.

Note with figure E1: The choices made in the decision tree are marked with grey squares. Source: CLP Regulation [14]

Route 2 describes the following: '*on a case-by-case basis, test data on mixtures may be used for classification when demonstrating effects that have not been established from the evaluation based on the individual ingredients*.'

There is limited data for the mutagenicity of KEE. The available epidemiological studies indicate potential genotoxic effects. However, these studies also have some limitations. Therefore, no firm conclusions can be drawn for germ cell mutagenicity (see also Chapter 6). In conclusion, based on the limited data available on the mixture itself, KEE cannot be classified for germ cell mutagenicity.

Scenario 2 – Annex I: 3.5.3.3.1 of the CLP regulation

In case the test results on the mixture are not conclusive, Annex I reports that '*in case there are sufficient data on similar tested mixtures to adequately characterise the hazards of the mixture, these data shall be used in accordance with the applicable bridging rules set out in section 1.1.3.'*.

KEE as a mixture shows similarities with DEE, a mixture of compounds derived from the same source (i.e., crude oil), with a similar relatively composition of substances (see Table 4), and for which sufficient data (i.e., epidemiological and toxicological studies) is available. However, the bridging principles as included in the CLP are not designed to evaluate complex combustion-generated mixtures with varying composition such as KEE. But article 9(3) and CLP Annex I, section 1.1.1 states that 'where the criteria cannot be directly applied to the available data, expert judgement should be used for the evaluation of the available information in a weight of evidence determination'. Based on the information provided in Chapter 2, the committees performed a hazard evaluation to decide whether KEE and DEE can be considered as similar mixtures with similar hazardous properties. This means that if these mixtures can be considered as similar mixtures, data used to substantiate the classification of DEE may be used to predict the hazard properties of KEE where data is missing. The committees concluded that KEE and DEE can be considered as substantially similar mixtures with similar toxicity and an analogy with DEE is made.

Carcinogenicity

Regarding the classification of mixtures for carcinogenicity, there are three possible scenarios described in the CLP regulation. Scenario 1 is applicable when data are available for all ingredients or only for some ingredients. Scenario 2 is applicable when data are available for the complete mixture. Scenario 3 can be applied when test results of the complete mixture are not conclusive.

Scenario 1 - Annex I: 3.6.3.1.1

Annex I of the CLP describes that '*the mixture will be classified as a carcinogen when at least one ingredient has been classified as a Category 1A, Category 1B or Category 2 carcinogen and is present at or above the*

appropriate generic concentration limit of 0.1%' as shown in Table 3.6.2 of the CLP regulation.

However, in the case of KEE, the mixture does not contain one or more components which are individually classified as a Category 1 or 2 carcinogens at 0.1% or higher, or above a specific concentration limit (SCL) set for the specific components (see also Chapter 2 and Annex C). In conclusion, based on the concentrations of the individual components of KEE, KEE cannot be classified for carcinogenicity (see also Figure E2).

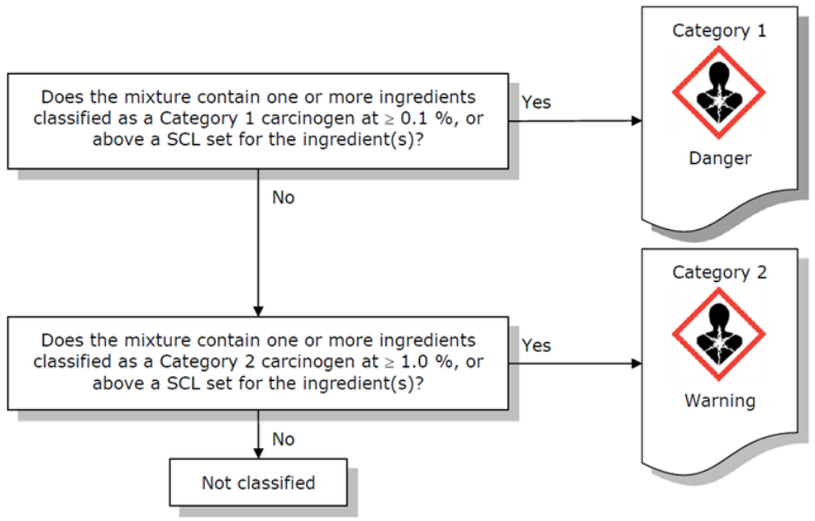

Figure E2 Schematic decision tree for classification for carcinogenicity, based on individual components of the mixture.

Note with figure E2: The choices made in the decision tree are marked with grey squares. Source: CLP Regulation [14]

Scenario 2 - Annex I: 3.6.3.2.1

Annex I of the CLP describes that '*when data are available for the complete mixture, the classification of mixtures will be based on the available test data for the individual ingredients of the mixture using concentration limits for the ingredients classified as carcinogens.*'

There is limited evidence for the carcinogenic effects of KEE. However, the available epidemiological studies also have some limitations (see also Chapter 7).

In conclusion, based on the limited data available on the mixture itself, KEE cannot be classified for carcinogenicity.

Scenario 3 - Annex I: 3.6.3.3.1

Annex I of the CLP describes that '*when data are not conclusive for the complete mixture, that where the mixture itself has not been tested to determine its carcinogenic hazard, but there are sufficient data on the individual ingredients and similar tested mixtures (subject to the provisions of paragraph 3.6.3.2.1) to adequately characterize the hazards of the mixture, these data shall be used in accordance with the applicable bridging rules set out in section 1.1.3.*'

KEE as a mixture shows similarities with DEE, a mixture of compounds derived from the same source (i.e., crude oil), with a similar relatively composition of substances (see Table 4), and for which sufficient data (i.e., epidemiological and toxicological studies) is available. However, the bridging principles as included in the CLP are not designed to evaluate

complex combustion-generated mixtures with varying composition such as KEE. But article 9(3) and CLP Annex I, section 1.1.1 states that ‘where the criteria cannot be directly applied to the available data, expert judgement should be used for the evaluation of the available information in a weight of evidence determination’. Based on the information provided in Chapter 2, the committees performed a hazard evaluation to decide whether KEE and DEE can be considered as similar mixtures with similar hazardous properties. This means that if these mixtures can be considered as similar mixtures, data used to substantiate the classification of DEE may be used to predict the hazard properties of KEE where data is missing. The committees concluded that KEE and DEE can be considered as substantially similar mixtures with similar toxicity and an analogy with DEE is made.

Committees and consulted expert[a]

Members of the Dutch Expert Committee on Occupational Safety (DECOS) for the advisory report *Kerosene Engine Exhaust*

- Prof. A.P. van Wezel, Professor of Environmental Quality and Health and dean of the Faculty of Geosciences, Utrecht University, Utrecht, *chair*
- Prof. H. Bouwmeester, Professor of Toxicology, Wageningen University and Research Centre, Wageningen
- Prof. M.B.M. van Duursen, Professor Environmental Health and Toxicology, VU Amsterdam, Amsterdam
- Dr W. Fransman, Senior Scientist, TNO, Zeist
- Prof. I.A. Kreis, retired Physician-Epidemiologist, Royal College of Surgeons of England, London
- Dr E.D. Kroese, retired Toxicologist, TNO, Zeist
- Dr A.L. Menke, Toxicological Pathologist, TNO Leiden Metabolic Health Research
- Dr S. Peters, Associate Professor, Institute for Risk Assessment Sciences (IRAS), Utrecht University, Utrecht
- Prof. G.B.G.J. van Rooy, Professor of Early Detection and Prevention of Occupational Diseases, Institute for Risk Assessment Sciences (IRAS) of the faculties Veterinary Sciences and Medical Sciences, Utrecht University, Utrecht
- Dr P.T.J. Scheepers, Associate Professor Molecular Epidemiology and Risk Assessment, Radboud University, Nijmegen
- Dr P.F.R. Schins, Associate Professor, Department of Pharmacology and Toxicology, Maastricht University, Maastricht
- Dr B.J.W. Scholten, toxicologist, Risk Analysis for Prevention, Innovation & Development (RAPID), Netherlands Organisation for Applied Scientific Research (TNO), Utrecht
- Prof. L.A. Smit, Professor One Health and Environmental Epidemiology, Institute for Risk Assessment Sciences (IRAS), Utrecht University, Utrecht

Observers[a]

- N.L. Luijckx, Social and Economic Council, The Hague
- Dr R. Renirie, Ministry of Social Affairs and Employment, The Hague

Members of the subcommittee on the Classification of Carcinogenic Substances for the advisory report *Kerosene Engine Exhaust*

- Dr R.W.L. Godschalk, Genetic Toxicology and Molecular Epidemiology, Maastricht University, Maastricht, *chair*
- Dr F.A.A. van Acker, PreClinical Safety Leader & Screening Toxicology Expert, Galapagos BV (PreClinical Development Department), Leiden
- Prof. M.L. de Bruin, Professor Drug Regulatory Science, Department Farmaceutical Sciences, Utrecht University, Utrecht
- Dr E. de Rijk, Toxicological Pathologist, Charles River Laboratories, ‘s Hertogenbosch
- Dr P.T.J. Scheepers, Associate Professor Molecular Epidemiology and Risk Assessment, Radboud University, Nijmegen
- Dr J.J. Vlaanderen, epidemiologist, coordinating researcher, National Institute for Public Health and the Environment, Bilthoven, *structurally consulted expert*[a]

[a] Observers and consulted experts are entitled to speak during the meeting. They do not have any voting rights and do not bear any responsibility for the content of the Committee’s advisory report.

Observer[a]

- M. Woutersen, Bureau REACH, National Institute for Public Health and the Environment, Bilthoven

Scientific secretariat

- Dr D. Boers, Health Council of the Netherlands, The Hague
- L. Souhoka, MSc, Health Council of the Netherlands, The Hague

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Members of the committee of the Nordic Expert Group (NEG) for the advisory report *Kerosene Engine Exhaust*

- Prof. G. Johanson, Professor of Occupational Toxicology and Risk Assessment, Institute of Environmental Medicine, Karolinska Institutet, Stockholm, *chair*
- Dr M.D. Bugge, Lead Head Physician, National Institute of Occupational Health, Oslo
- Dr L. Schenk, Associate Professor of Toxicological Risk Management, Institute of Environmental Medicine, Karolinska Institutet, Stockholm
- Dr P. Taxell, Senior Specialist, Finnish Institute of Occupational Health, Helsinki
- Dr A.T. Saber, Senior Researcher in Toxicology, National Research Centre for the Working Environment, Copenhagen
- Dr S. Wolf, Researcher, National Institute of Occupational Health, Oslo
- Dr M. Öberg, Associate Professor of Toxicology, Institute of Environmental Medicine, Karolinska Institutet, Stockholm

Scientific secretariat

Dr A.K. Alexandrie, Swedish Work Environment Authority, Stockholm

The Nordic Expert Group – appointment and interests procedures

Members of the Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals (NEG) are appointed by the Director General of the Swedish Work Environment Authority (SWEA) following nominations from the Danish, Finnish, Norwegian and Swedish occupational health institutes. They are appointed in a personal capacity because of their special expertise in relevant areas. NEG does not follow a formal procedure regarding conflict of interest, however, being employed by state institutes, the members are obliged to report any potential conflict of interest. See also nordicexpertgroup.org for more information about the procedures of NEG.

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