17. Health Impact

17.1 Introduction

17.1.1.1 This section presents an assessment of potential impacts on human health in relation to (i) air pollutants from aircraft emissions and associated activities, as well as (ii) aircraft noise arising from operation of the project, which has been conducted in accordance with the requirements given in Clause 3.4.14 together with section II of Appendix A and section II of Appendix C of the EIA Study Brief (ESB-250/2012).

17.2 Health Impact Assessment of Air Pollutants

17.2.1 Technical Requirements

17.2.1.1 This section presents a Health Impact Assessment (HIA) for emissions of toxic air pollutants associated with the operation of the 3RS. Toxic air pollutants (TAP), also known as hazardous air pollutants, refer to those air pollutants that are known or suspected to cause cancer or other serious health effects or adverse environmental effects. Although criteria pollutants are not normally considered as TAP, the current HIA also covers an evaluation of potential public health risk from exposure to criteria pollutants.

17.2.1.2 In accordance with 3.4.14.2 of the EIA Study Brief, the HIA shall be based on established practices in countries around the world. A literature search shall be carried out to determine the best approach and methodology for the HIA, including any codes of practices, guidelines, etc. applied locally in Hong Kong and elsewhere in the world. The approach and methodology to be adopted shall be agreed by the Director prior to the commencement of assessment.

17.2.1.3 Besides, in accordance with the technical requirements set out in section II of Appendix A of the EIA Study Brief, the HIA of TAP shall include the following key steps:

- Identification of key components of TAP from the aircraft emissions and associated activities during the operation of the project for health impact assessment;
- Assessment of the likelihood and consequences of exposure to the identified key components of TAP emissions;
- Identification of means by which the health impact could be further reduced;
- Recommendations of reasonably practical measures, if any, to reduce the health impact during the operation of the project.

17.2.2 Literature Review

17.2.2.1 A literature search for determining the best approach and methodology for the HIA was carried out in accordance with the EIA Study Brief requirements described above. The following sources were covered in the literature review:

- World Health Organization (WHO) publications (e.g. WHO guidelines for indoor air quality: selected pollutants, 2010; Air Quality Guidelines Global Updates, 2005; Air Pollution and

Cancer. IARC Scientific Publication no. 161., 2013; Concise International Chemical Assessment Document 43 - Acrolein, 2002);

- United States Environmental Protection Agency (USEPA) publications (e.g. Toxicological Review, 2012; Health Assessment Document For Diesel Engine Exhaust, 2002);
- International Air Transport Association (IATA), Federal Aviation Administration (FAA) and International Civil Aviation Organization (ICAO) publications (e.g. ICAO Airport Air Quality Manual, 2011); and
- Public domain websites (e.g. USEPA IRIS: http://www.epa.gov/IRIS/; USEPA SPECIATE Data Browser http://cfpub.epa.gov/si/speciate/ehpa_speciate_browse.cfm; OEHHA – Hot Spots Guidelines http://oehha.ca.gov/air/hot_spots/index.html).

17.2.2.2 Relevant literatures published in the past twenty years (i.e., from years 1993 to 2013) were selected for the review. **Tables 17.2.1** to **17.2.3** present a summary of the review results with respect to available international HIA methodology guidelines, HIA methodologies adopted in other airport-related studies, and HIA methodologies adopted in other EIA studies and feasibility studies.

Table 17.2.1: Summary of desktop research on international HIA methodology guidelines

Item	Literature	Organisation	Year	Methodology
1	Evaluation and use of epidemiological evidence for environmental health risk assessment	WHO	2000	• The document specifies the key HIA steps as follows: – Specify the purpose and framework of the impact assessment – Specify the method(s) used to quantify uncertainty – Specify the measure(s) of exposure – Specify the range of exposure to be considered – Derive the population exposure distribution – Specify the time window between exposure and effect – Select appropriate health outcome(s) – Estimate the exposure-response relationship in the population of interest – Derive population baseline frequency measures for the relevant health outcomes – Calculate the number of attributable cases
2	WHO Air Quality Guidelines - Global Update 2005	WHO	2005	• The HIA assessment due to outdoor air pollution is based on four components as follows: – Identify pre- and post-air-pollution concentrations and exposure assessment; – Determine size and composition of population groups exposed to current levels of air pollution – Establish concentration response (CR) functions for background incidence of mortality and morbidity – Estimate the impact functions
3	APHEIS: Health Impact Assessment of Air Pollution and Communication Strategy	Air Pollution and Health : A European Information	2005	• Focus on Black Smoke and PM • The key HIA steps shall include: – Specification of exposure – Defining the appropriate health outcomes – Specification of the exposure-response relationship

Item	Literature	Organisation	Year	Methodology
		System, EU		– Derivation population baseline frequency measures for the health outcomes – Calculation of the number of cases • Analysis of the acute effects of PM_{10} and Black Smoke on premature mortality and hospital admissions • Estimation of the impact on premature mortality of long-term exposure to PM_{10} and $PM_{2.5}$
4	CAFÉ Program Methodology for the Cost-Benefit analysis for CAFE: Volume 2: Health Impact Assessment	AEA Technology Environment / European Commission DG Environment	2005	The systematic approach for HIA shall include: • Identification of sources and quantification of pollutant emissions • Calculation of dispersion • Incorporation of exposure response functions to estimate yield loss • Valuation of yield loss using world market prices
5	ACRP Report 7 - Aircraft and Airport-Related Hazardous Air Pollutants: Research Needs and Analysis	Airport Cooperative Research Program Transportation Research Board	2008	• Emissions of TAP were considered • Integration of Emission Rates with Toxicology for Prioritization of Airport Hazardous Air Pollutants • Risk-based concentrations (RBCs) for cancer and noncancerous health effects were proposed • Acute exposure guidelines (AEGLs), acute minimal risk levels (MRLs), and acute inhalation reference exposure levels (RELs) were proposed
6	A Method to Estimate the Chronic Health Impact of Air Pollutants in .US. Residences	Ernest Orlando Lawrence Berkeley National Laboratory	2011	• Disability-Adjusted Life Years (DALY) Method adopted • For criteria pollutants (ozone, NO_2, $PM_{2.5}$, SO_2, and CO), an Intake-Incidence-DALY (IND) method that uses epidemiology based concentration-response functions shall be adopted to quantify disease incidence rates. These are combined with estimates of DALYs per disease incidence reported in the literature • For TAP, an Intake-DALY (ID) approach which used the work of Huijbregts *et al.* (2005) shall be adopted to calculate the health impact associated with intake of non-criteria pollutants based on animal toxicity literature

Table 17.2.2: Summary of desktop research on HIA methodologies adopted in other airport related studies

Item	Literature	Organisation	Year	Methodology
7	Health Impact Assessment Schiphol Airport	National Institute of Public Health and Environmental Protection (RIVM)	1994	• Both criteria pollutants and TAP were considered • Inhalation exposure pathway was considered • Incremental risk was considered • Increase in respiratory symptoms was considered • Increase in cancer risk was considered
8	Human health risk assessment for LAX Master Plan EIS / EIR	Los Angeles World Airport	2001	• Only TAP were considered. • Inhalation and ingestion pathway were considered • Toxicity-weighted emissions were established for TAP screening • Acute and chronic risk were calculated
9	EIS for Chicago O'Hare International Airport	Chicago O'Hare International Airport	2005	• Only TAP were considered • Inhalation exposure pathway was considered • No quantification of TAP concentrations • Comparison of toxicity-weighted emissions between with and without-project scenarios

Item	Literature	Organisation	Year	Methodology
10	HIA - Finningley Airport	Doncaster Health Authority	2000	• Both criteria pollutants and TAP were considered • Inhalation exposure pathway was considered • Comparison with Air Quality Criteria and WHO guidelines
11	Health Impact Assessment for Brisbane Airport	Brisbane Airport Corporation Pty Limited	2007	• Both criteria pollutants and TAP were considered • Inhalation exposure pathway was considered • Incremental risk was considered • The acute health effects examined include: – Mortality and hospital admission (for criteria pollutants) – Lung function, symptoms and GP visits (for criteria pollutants) • The long term effects considered include: – Mortality (for criteria pollutants) – Cancer incidence (for TAP) – Lung function growth in children (for criteria pollutants)
12	EIS for Fort Lauderdale-Hollywood International Airport	Landrum & Brown, Incorporated	2007	• Only TAP were considered • Comparison of TAP emission inventories only • No quantification of TAP concentrations
13	Health Impact Assessment of Second Runway for Stansted Airport - Generation 2 Development	Stansted Airport	2008	• Incremental risks due to PM and NO_2 were considered • Inhalation exposure pathway was considered • Years of life lost were considered • Respiratory hospital admissions were considered • Cardiovascular hospital admissions were considered • GP consultations for asthma were considered • Chronic Bronchitis was considered • Restricted activity days were considered • Lower respiratory symptoms were considered • Qualitatively addressed the ingestion exposure path due to polycyclic aromatic hydrocarbons (PAHs)
14	High-Priority Compounds Associated with Aircraft Emissions	U.S. Federal Aviation Administration Office of Environment and Energy (Funder)	2008	• The risk-based prioritisation included three components: – Emissions determination – Identification of the emission-to-exposure relationship (including pollutant fate and transport and population patterns) – Determination of the toxicity of compounds • Primarily focused on total population health risks, rather than considering the maximum individual health risks found within the population • Optimal spatial domain and resolution for atmospheric dispersion modelling may differ across pollutants as well as across airports • Ranking differences across airports
15	Santa Monica Airport Health Impact Assessment (HIA)	UCLA Department of Paediatrics	2010	• Consideration of both criteria pollutants and TAP Adopt rapid non-participatory health impact assessment • Inhalation exposure path was considered • Conducting empirical and scientific literature reviews
16	General Aviation Airport Air Monitoring Study	USEPA	2010	• Long-term monitoring of TAP concentrations within and in the vicinity of the airports

Table 17.2.3: Summary of desktop research on HIA methodologies adopted in other EIA studies and feasibility studies

Item	Literature	Organisation	Year	Methodology
17	Assessment of Toxic Air Pollutant Measurements in Hong Kong	HKEPD	2003	• Both criteria pollutants and TAP were considered • Inhalation exposure path was considered • Non-cancer risk and cancer risk was presented • Dietary intake for dioxin was considered
18	EIA for Sludge Treatment Facilities	HKEPD	2008	• Both criteria pollutants and TAP were considered • Inhalation exposure path was considered • Acute and chronic non-cancer risk, and cancer risk were presented • Risk due to criteria pollutant (except Pb) was checked against the Air Quality Objectives (AQO)
19	Review of Air Quality Objectives and Development of a Long Term Air Quality Strategy for Hong Kong – Feasibility Study	Arup / HKEPD	2009	• Criteria pollutants were considered • Inhalation exposure was considered • Incremental risk was considered • Increase in hospital admission, mortality, etc were considered • Health cost was quantified
20	EIA for Development of the Integrated Waste Management Facilities Phase 1	HKEPD	2011	• Both criteria pollutants and TAP were considered • Inhalation, ingestion exposure (including consumption of drinking water and fish, consumption of animal products, consumption of aboveground produce) were considered • Incremental cancer risk was considered • Risk due to criteria pollutant (except Pb) was checked against the AQO • Project contribution concentration to determine the acute and other non-cancer risk

17.2.2.3 It is noted from the literature review that inhalation pathway is the major exposure pathway evaluated for airport-related emissions of TAP and criteria pollutants. The approaches to address the potential health impact from exposure to TAP and criteria pollutants identified from the review are summarized in **Table 17.2.4**. It is noted that the methods of comparing toxicity-weighted emissions, comparison with air quality criteria, or conducting empirical and scientific literature review, which have been identified to be the approaches adopted in some of the above-listed studies cannot be used for determining the consequence of exposure. On the other hand, it is noted that while calculated Disability-Adjusted Life Years (DALY) may be used as an indicator of health that integrates both mortality and morbidity data, the methodology of calculating DALY is not standardized and results would depend very much on the assumptions adopted. The limitation of the above-mentioned risk assessment approaches are summarized in **Table 17.2.5**.

Table 17.2.4: Summary of approaches in determination of health risk identified from the literature review

Approaches	Item	Pollutants Covered
Comparison of toxicity-weighted emissions	Items 9 and 12	TAP
Comparison with air quality criteria	Item 10	Criteria Pollutants
Conducting empirical and scientific literature reviews	Item 15	TAP and Criteria Pollutants
Disability-Adjusted Life Years (DALY) Method	Item 6	Criteria Pollutants
Evaluation of acute and chronic non-cancer health risks as well as cancer risk	Items 1-5, 7, 8, 11, 13, 14, 17 – 20	TAP and Criteria Pollutants

17.2.2.4 The limitation of different risk assessment approaches are summarized in **Table 17.2.5**.

Table 17.2.5: The limitation of different risk assessment approaches

Approaches	Limitation
Comparison of toxicity-weighted emissions	Not able to determine the consequences of exposure as specified in the EIA Study Brief
Comparison with air quality criteria	Applicable to criteria pollutants only; not able to determine the likelihood and consequences of exposure as specified in the EIA Study Brief for TAP.
Conducting empirical and scientific literature reviews	Not able to determine the likelihood and consequences of exposure as specified in the EIA Study Brief
Disability-Adjusted Life Years (DALY) Method	Not standardised method and results depend very much on the assumptions used.

17.2.2.5 Therefore, the human health risk assessment approach, which involves an evaluation of acute and chronic non-cancer health risks and cancer risk and forms the basis of many of the above-mentioned studies, is adopted as the methodology for the HIA of air pollutants. The typical approach to a human health risk assessment involves (i) hazard identification, (ii) exposure assessment, (iii) dose-response assessment, and (iv) risk characterization and the key tasks involved under each step are described below:

Hazard Identification

Task 1: Identification of the key air pollutants of interest, including both criteria pollutants and TAP;

Exposure Assessment

Task 2: Determination of modelling scenarios;

Task 3: Identification of exposure pathways;

Task 4: Assessment of the likelihood of exposure to the key air pollutants;

Dose-Response Assessment

Task 5: Determination of the relationship between dose and toxic effect;

Risk Characterisation

Task 6: Integration of the information from the proceeding steps of the risk assessment to synthesise an overall conclusion about risk; and

Task 7: Identification of reasonably practicable measures to reduce the health impact.

17.2.2.6 For TAP, the acute risk, chronic non-carcinogenic risk and incremental carcinogenic risk associated with the future operation of the 3RS were evaluated. For criteria pollutants, the increased risk of hospital admission and risk of mortality were determined.

17.2.3 Hazard Identification

17.2.3.1 The criteria air pollutants specified under the AQOs in Hong Kong include respirable suspended and fine particulates (RSP and FSP, also known as PM10 and PM2.5), sulphur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), carbon monoxide (CO) and lead (Pb). The effects of these air pollutants on health have been thoroughly researched internationally and are well-known.

17.2.3.2 Unlike other criteria pollutants such as NO_2, ozone (O_3) is not a pollutant directly emitted from man-made sources but formed by photochemical reactions of primary pollutants such as nitrogen oxides (NO_x) and volatile organic compounds (VOCs) under sunlight. As the photochemical reactions take place in minutes in the presence of solar radiation and could accumulate over several hours, ozone recorded in one place could be attributed to VOC and NO_x emissions from places afar.

17.2.3.3 A hypothetical sensitivity test was conducted using the PATH model to compare the simulated ozone concentrations in downwind areas for the "with-airport" (under 3RS scenario) and the "without-airport" scenarios. **Tables 17.2.6 to 17.2.8** summarise the results under different wind directions.

Table 17.2.6: Ozone concentrations for with and without airport scenarios under northern wind direction

Area	Ozone under the with airport case (3RS), µg / m^3	Ozone under the without airport case, µg / m^3	Difference (with airport – without airport), µg / m^3
Lung Kwu Chau PATH grid (8,30)	361	361	0
PH1(Airport North Station) PATH grid (12,28)	316	325	- 9
PH5 (Airport South Station) PATH grid (11,26)	287	321	- 34
Tung Chung Air Quality Monitoring Station PATH grid (12,25)	277	302	- 25
Lantau Central PATH grid (12,23)	269	272	-4
Lantau South PATH grid (12,21)	244	244	0

Table 17.2.7: Ozone concentrations for with and without airport scenarios under southern wind direction

Area	Ozone under the with airport case (3RS), µg / m^3	Ozone under the without-airport case, µg / m^3	Difference (with airport – without airport), µg / m^3
Lantau Central PATH grid (12,23)	128	128	0
Tung Chung Air Quality Monitoring Station PATH grid (12,25)	121	122	-1
PH5 (Airport South Station) PATH grid (11,26)	106	111	-5
PH1(Airport North Station)	75	79	-4

Area	Ozone under the with airport case (3RS), µg / m^3	Ozone under the without-airport case, µg / m^3	Difference (with airport – without airport), µg / m^3
PATH grid (12,28)			
Lung Kwu Chau PATH grid (8,30)	93	103	-10
Yuen Long Air Quality Monitoring Station (18,38)	133	133	0

Table 17.2.8: Ozone concentrations for with and without airport scenarios under western wind direction

Area	Ozone under the with airport case (3RS), µg / m^3	Ozone under the without-airport case, µg / m^3	Difference (with airport – without airport), µg / m^3
Lung Kwu Chau PATH grid (8,30)	162	162	0
PH1 (Airport North Station) PATH grid (12,28)	115	225	-110
Central Western Air Quality Monitoring Station PATH grid (27, 25)	146	174	-28

17.2.3.4 There are no potential human receptors to the west of the airport. Hence, the ozone concentration under the eastern wind direction were not considered.

17.2.3.5 On comparing the ozone concentrations under the downwind direction, the ozone concentration under the 3RS scenario are in general lower than that of the without-airport scenario. The ozone will start to restore at more than 5 km which is near the boundary of assessment area. This suggests that ozone is consumed by the presence of airport-related operational activities. Nitric oxide emitted from airport-related operational activities readily reacts with ozone to form NO_2 thereby removing ozone. Hence, ozone is not considered as a key air pollutant of interest in evaluating the potential air quality and health impact from the operation of the project.

17.2.3.6 Apart from the criteria pollutants, a number of TAP would be emitted from operation of the project. As pointed out in **Section 17.2.2.3**, the key exposure pathway is expected to be from inhalation based on the literature review. In Hong Kong, a designated list of TAP is not present. A desktop review has been conducted to identify the key components of TAP that may arise from aircraft emissions and associated activities during the operation of the project. **Table 17.2.9** summarises the TAP as listed in relevant guidelines. **Table 17.2.10** summarises the air pollutants, including TAP, that have been considered in various airport-related HIA studies. As described further below, this initial long list of TAP generated from a review of relevant guidelines and airport-related HIA studies has been reviewed and evaluated quantitatively for identification of the key components of TAP in the HIA study.

Table 17.2.9: TAP considered in various international guidelines

TAP	ICAO [1]	USEPA [2]	USEPA [3]	FAA [4]	ACRP [5]
1,3-butadiene	✓	✓	✓	✓	✓
Acetaldehyde	✓	✓	✓	✓	✓
Acrolein	✓	✓	✓	✓	✓

TAP	ICAO [1]	USEPA [2]	USEPA [3]	FAA [4]	ACRP [5]
Benzene	✓	✓	✓	✓	✓
Diesel Particulate Matters	✓				
Ethylbenzene		✓	✓		✓
Formaldehyde	✓	✓	✓	✓	✓
Isopropylbenzene (cumene)			✓		
Lead	✓	✓		✓	
m-Xylene + P-xylene +o-Xylene	✓	✓	✓	✓	
Naphthalene	✓		✓	✓	✓
n-Hexane		✓			
PAH / POM		✓			
Phenol (carbolic acid)			✓		
Propionaldehyde	✓	✓	✓	✓	
Styrene		✓			
Toluene	✓	✓	✓	✓	

Notes:

[1] ICAO, Air Quality Manual, 2011.

[2] USEPA, Source Identification and Base Year 1990 Emission Inventory Guidance for Mobile Sources HAPs on the OAQPS List of 40 Priority HAPs, 1997.

[3] USEPA, Recommended Best Practice for Quantifying Speciated Organic Gas Emissions from Aircraft Equipped with Turbofan, Turbojet, and Turboprop Engines, 2009.

[4] FAA, Select resource materials and annotated bibliography on the topic of hazardous air pollutants (HAPs) associated with aircraft, airports and aviation, 2003.

[5] ACRP, Aircraft and Airport-Related Hazardous Air Pollutants: Research Needs and Analysis, 2008.

Table 17.2.10: TAP considered in various airport-related health impact assessments / monitoring

TAP	Brisbane Airport	Finningley Airport	Chicago O'Hare International Airport	Fort Lauderdale-Hollywood International Airport	Los Angeles International Airport	Santa Monica Airport	Schipol Airport	General Aviation Airport Air Monitoring Study	London Stansted Airport	High-Priority Compounds Associated with Aircraft Emissions
1,3-butadiene			✓	✓	✓			✓		✓
Acetaldehyde			✓	✓	✓			✓		✓
Acrolein			✓	✓	✓					✓
Benzene	✓	✓	✓	✓	✓		✓	✓		✓
Diesel Particulate Matter			✓	✓	✓					
Formaldehyde	✓		✓	✓	✓			✓		✓
Lead		✓		✓				✓		
Naphthalene			✓	✓	✓					✓
Propionaldehyde				✓						✓
Toluene	✓		✓	✓	✓			✓		✓
Xylene	✓			✓	✓					✓

TAP	Brisbane Airport	Finningley Airport	Chicago O'Hare International Airport	Fort Lauderdale-Hollywood International Airport	Los Angeles International Airport	Santa Monica Airport	Schipol Airport	General Aviation Airport Air Monitoring Study	London Stansted Airport	High-Priority Compounds Associated with Aircraft Emissions
Ethylbenzene				✓				✓		✓
n-Hexane				✓						
Styrene				✓						✓
PAH / POM		✓		✓	✓	✓	✓			✓
Arsenic			✓	✓	✓					
Chromium VI			✓	✓	✓					
Nickel			✓	✓	✓					
2,2,4-Trimethylpentane				✓						
TCDD					✓					
Beryllium					✓					
Cadmium					✓					
Copper					✓					
Manganese					✓					
Zinc					✓					
SO_2		✓					✓			✓
NO_2	✓	✓					✓		✓	✓
CO	✓	✓					✓	✓		✓
RSP / FSP / UFP	✓	✓				✓		✓	✓	✓
Black Smoke						✓	✓			
Ozone		✓								✓

Note:

[1] **Table 17.2.2** shall be referred to for a summary of the approaches adopted in the various HIA studies.

17.2.3.7 A three-tiered approach has been adopted to short-list the key TAP. The first tier involves a quantitative screening that considers the emission quantities and toxicity levels of the TAP. Tier 2 is used for further selection of carcinogenic chemicals and Tier 3 is used to retain airport-specific chemicals:

- Tier 1: Screening based on calculation of emission-toxicity values;
- Tier 2: Reference to IARC Group 1 (Carcinogenic to human) Chemicals; and
- Tier 3: Reference to TAP identified in other airport-related studies.

Tier 1: Screening based on calculation of emission-toxicity values

17.2.3.8 The speciation profiles of TAP from aircraft or airport operation-related emissions are listed in **Appendix 17.2.1**. To identify the key components of TAP emissions in accordance with the EIA Study Brief requirements, the standard approach of a quantitative screening analysis as part of the human health risk assessment was conducted to determine a subset of chemicals from the initial list of TAP to be considered as chemical of potential concern (COPC) for subsequent assessment. In order to identify the COPC of interest, a standardised screening process was carried out taking into account the estimated emission rates of each chemical and toxicity values. More than 120 species of TAP were screened for health impacts, as shown in **Appendix 17.2.2**. A relative potency expressed in terms of toxicity-weighted emissions, or referred to as "emission-toxicity values" here, was calculated for each chemical in the initial long-list of TAP by multiplying the total emission level of each TAP by an applicable carcinogenic and/or non-carcinogenic toxicity weight, based on the methodology developed by USEPA as detailed in its publication "Air Toxics Risk Assessment Reference Library – Volume 2 Facility-Specific Assessment (EPA-453-K-04-001B) 2004". The chemicals that had the highest relative potency were retained as COPCs in the current human health risk assessment. To summarise, the screening procedures are listed as follows:

- Identify all the inhalation unit risks (IURs) and reference concentrations (RfCs) for the TAP of interest;
- Determine the emission rate (e.g. tons/year) of each TAP;
- Multiply the emission rate of each TAP by its IUR where applicable to obtain an emission-toxicity value for each TAP of interest with respect to carcinogenic effect;
- Similarly, divide the emission rate of each TAP by its RfC to obtain an emission-toxicity value for each TAP with respect to non-carcinogenic effect;
- Rank the emission-toxicity values calculated and sum all emission-toxicity values for carcinogenic and non-carcinogenic effects respectively; and
- Starting with the highest emission-toxicity values calculated, proceed down the list until the cumulative sum of the emission-toxicity values reaches a large proportion (i.e. 99.9%) of the total for all the TAP.

17.2.3.9 USEPA's Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (December 1989) suggests a value of 1 percent for toxicity screening. With reference to the Human Health Risk Assessment Technical Report prepared as part of the Los Angeles International Airport (LAX) Master Plan EIS / EIR study, it is noted that one-tenth of this value was used in their screening analysis to ensure that the analysis would be protective for all TAP that might be released. Moreover, in the Human Health Risk Assessment of Air Emissions from Proposed Biosolids Incinerator, Revised Final Report, 2010, it is noted that the same "cut-off" value of total toxicity-weighted emissions (i.e. 99.9%) was adopted. Hence, the cut-off value of 99.9% was adopted for screening of TAP in the current study in accordance with the international practice.

17.2.3.10 A review of TAP toxicity due to aircraft emissions and associated activities has been performed. Data on unit risks of TAP for potential or confirmed carcinogens, and reference concentrations may be derived from 'no observed adverse effect levels' (NOAEL) or 'lowest observed adverse

effect levels' (LOAEL). Reference concentrations indicate the concentrations to which long-term exposure has no appreciable risk to health. It is noted that different cities / countries might have adopted different acceptable toxicity values taking into account relevant factors including local conditions. Hence, in determination of the generally accepted toxicity values for the assessment, the following hierarchy was adopted:

1. Worldwide level (such as WHO);

2. Country level (e.g. countries with well-established environmental regulations, such as USEPA – IRIS, USEPA – ASTDR); and

3. Local level (i.e. state / city of countries with well-established environmental regulations, such as California EPA – OEHHA).

17.2.3.11 **Appendix 17.2.2** shows the screening results of the Tier 1 assessment. Based on the screening results, the key TAP which attribute to 99.9% of emission-toxicity values include:

- 1,3-Butadiene
- Acetaldehyde
- Acrolein
- Arsenic
- Benzene
- Cadmium
- Chromium VI
- Copper
- Diesel Particulate Matter
- Formaldehyde
- Lead
- Manganese
- Naphthalene
- Nickel
- Propionaldehyde
- Xylene

Tier 2: Reference to IARC Group 1 Chemicals

17.2.3.12 Apart from the shortlisted TAP identified from the quantitative screening presented under the Tier 1 analysis above, all those IARC Group 1 chemicals that are related to emissions from aircraft and associated activities arising from the operation of the project have been selected. These chemicals include:

- Benzo(a)pyrene
- Beryllium
- TCDD

Tier 3: Reference to TAP identified in other airport-related studies

17.2.3.13 In addition to the above key TAP, other TAP considered in the international guidelines and airport-related studies (listed in **Tables 17.2.9** and **17.2.10**) have also been selected. These TAP include:

- 2,2,4-Trimethylpentane
- Benzo(a)anthracene
- Benzo(bk)fluoranthene
- Chrysene
- Dibenz(a,h)anthracene
- Ethylbenzene
- Indeno(1,2,3-cd)pyrene
- Isopropylbenzene (cumene)
- Phenol (carbolic acid)
- Methanol
- n-Hexane
- Styrene
- Toluene

17.2.3.14 There is no published toxicity value for 2,2,4-trimethylpentane available from WHO, IRIS, ATSDR and OEHHA and therefore no further evaluation is possible.The TAP covered in this human health risk assessment based on the three-tier screening procedure are summarised in **Table 17.2.11**.

Table 17.2.11: Summary of short-listed TAP

Type	TAP
VOC	1,3-Butadiene, Acetaldehyde, Acrolein, Benzene, Formaldehyde, Propionaldehyde, Xylene, Isopropylbenzene (cumene), Methanol, Phenol (carbolic acid), n-Hexane, Ethylbenzene, Styrene, Toluene
PM	Diesel Particulate Matter
Heavy Metals	Arsenic, Beryllium, Cadmium, Chromium VI, Copper, Manganese, Nickel, Lead
PAH/Dioxin	Naphthalene, Benzo(a)pyrene, Benzo(a)anthracene, Benzo(bk)fluoranthene, Chrysene, Dibenz(a,h)anthracene, Indeno(1,2,3-cd)pyrene, TCDD equivalent

Note: Lead is evaluated as both a TAP and a criteria pollutant.

17.2.4 Exposure Assessment

Determination of Modelling Scenarios

17.2.4.1 For the related operational air quality impact assessment presented in **Chapter 5**, according to the requirement sets out in Clause 5(iv) of Appendix A of the EIA Study Brief (ESB-250/2012), the air pollution impacts of the future air traffic shall be calculated based on the highest aircraft emissions, due specifically to aircraft landing and take-off (LTO) cycles, within the period when the project commences operation to the year the project reaches and operates at full capacity.

17.2.4.2 As described in **Section 5.3.4**, the selected worst assessment year has been identified to be year 2031. Hence, year 2031 has also been adopted as the assessment year for the HIA. All activities arising from the operation of the project in accordance with Clause 3.4.14.1(i) of the EIA Study Brief, such as aircraft LTOs, marine traffic, road traffic, fuel tank, catering, aircraft maintenance, etc. have been taken into account in the HIA.

17.2.4.3 The potential increase in human health risks has been established by a comparison of the "with-project" and "without-project" scenarios (i.e., based on a business as usual (BAU) scenario under the existing two-runway system (2RS)) for the same worst assessment year identified for the three-runway system. **Table 17.2.12** summaries the scenarios to be conducted under the HIA.

Table 17.2.12: Modelling scenarios to be assessed

Scenario	Description	Assessment Year
1	Highest aircraft emission scenario	Year 2031 According to the EIA Study Brief, the selected year of assessment represents the highest aircraft emission scenario, taking into consideration the number of landing take-off cycles and the corresponding aircraft engine emission factors for the selected year. Moreover, the highest incremental changes (3RS – 2RS) of aircraft emissions of RSP, NO_2, SO_2, CO and VOC are predicted to occur in Yr 2031.
2	Without project scenario	Same year as Scenario 1, but based on a two- runway system under the business as usual case. The purpose of this scenario is to establish the baseline scenario of the additional health impact due to the increase, if any, of the air pollutants arising from the project.

Human Receptors Identification

17.2.4.4 For the related operational air quality impact assessment that is focusing on assessing human inhalation exposure levels to criteria pollutants based on the established Air Quality Objectives (AQO), the Air Sensitive Receivers (ASRs) of interest are located within 5 km from the project boundary as per the requirements set out in the EIA Study Brief. Specifically, Clause 4(i) in Appendix A of the EIA Study Brief requires the expected air pollutant concentrations at the identified ASRs within 5 km from the project boundary to be quantified based on the highest aircraft emissions scenario under normal operating conditions with the project.

17.2.4.5 The identified ASRs in the vicinity of the project site have been identified as potential human receptors and a similar study area of 5 km from the project boundary has been adopted for the HIA. **Drawing No. MCL/P132/EIA/5-3-001** illustrates the extent of the study area. The study area generally covers the entire areas of Tung Chung, San Tau, Sha Lo Wan, San Shek Wan, Siu Ho Wan, Sham Wat Wan in Lantau North, Tap Shek Kok and areas adjacent to Butterfly Beach in Tuen Mun.

17.2.4.6 Within the project site (i.e., the airport island), there are other potential human receptors involving general public. These include visitors to hotels, AsiaWorld-Expo and the planned North Commercial District (NCD), though any potential exposures to air pollutants are expected to be transient in nature for visitors.

17.2.4.7 Within the airport island there are also commercial and industrial workers at various existing and planned facilities and there would also be workers involved in construction/ maintenance projects. However, for on-airport workers, exposures are occupational and these are not the focus of the current HIA. It shall also be noted that the Factories and Industrial Undertakings Ordinance (Cap. 59) (FIUO), which applies to industrial undertakings (i.e. factories, construction sites, catering establishments, cargo and container handling undertakings, repair workshops and other industrial workplaces), is applicable to work activities within the airport island. The definition of industrial

undertakings in the FIUO also covers specifically the loading, unloading, or handling of goods or cargo at the airport.

17.2.4.8 In relation to control of air pollution in industrial undertakings, the Labour Department has published the "Code of Practice on Control of Air Impurities (Chemical Substance) in the Workplace" under Section 7A(1) of the FIUO, to provide practical guidance for proprietors to take adequate measures for safeguarding workers against air impurities i.e., airborne chemical substances in form of dust, fumes or gases that are emitted into the workplace environment as a result of work activities. Hence, workers on the airport island are not selected as potential human receptors in the current HIA. It is also noted that this approach is in line with that adopted in other local and international HIA studies and a few examples are summarized in **Table 17.2.13**.

Table 17.2.13: Human receptor locations considered in local and international HIA studies

HIA Studies	Country / Cities	Year	Identification of Human Receptor Locations
The Stansted Generation 2 project – Health Impact Assessment	UK	2008	The area within the airport boundary was excluded from the calculation of health effects
EIS – Los Angeles International Airport	US	2009	Off-site sensitive receptors (i.e. residential and schools) were identified and evaluated against the acute and chronic non - carcinogenic risk, and carcinogenic risk. Workers inside airport were identified and evaluated against the occupational standards (OSHA).
EIS – New Parallel Runway for Brisbane Airport	Australia	2007	No sensitive receptor was selected inside airport boundary
EIA – Sludge Treatment Facilities (EIA 155/2008)	HK	2008	No on-site sensitive receptor was selected
EIA – IWMF (EIA 201/2011)	HK	2011	No on-site sensitive receptor was selected

17.2.4.9 In relation to planned land uses in the vicinity of the project boundary, it is understood that a Planning and Engineering Study on the remaining development in Tung Chung is being undertaken by the Civil Engineering and Development Department (CEDD). The objective of the Planning and Engineering Study is to assess the feasibility of the remaining development in the east and west of Tung Chung. Since the Recommended Outline Development Plan from CEDD is not available, the representative planned ASRs have been identified and included as potential human receptors in the current HIA study.

17.2.4.10 Locations of the representative human receptors (HSR) selected for the HIA are illustrated in **Drawing No MCL/P132/EIA/17-3-002** to **MCL/P132/EIA/17-3-006** and are summarised in **Table 17.2.14**. These include the representative ASRs in the vicinity of the airport and also representative locations selected within the airport island that represent incidental exposures of visitors to air pollutants. For evaluating chronic effects from long-term exposure to air pollutants, the exposure period is usually from at least 7 years to lifetime according to “Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A), USEPA”. Given that visitors to the hotels, AsiaWorld-Expo and the planned NCD will not be subject to long-term exposure to air pollutants from the airport operation, it is considered appropriate to exclude these potential human receptors in the evaluation of chronic health effects.

Table 17.2.14: Representative existing and planned human receptors

HSR ID	Location	Landuse [1]	Acute Risk	Chronic Risk
Airport Island (Drawing No. MCL/P132/EIA/17-3-006)				
AI-C1	Regal Airport Hotel	C	√	-
AI-C2	AsiaWorld-Expo	C	√	-
AI-C3	AsiaWorld-Expo	C	√	-
AI-C4	Hong Kong SkyCity Marriot Hotel	C	√	-
AI-C5	Planned Northern Commercial District	C	√	-
AI-C6	Planned Northern Commercial District	C	√	-
Hong Kong Boundary Crossing Facilities (HKBCF)(Drawing No MCL/P132/EIA/17-3-006)				
BCF-1	Planned Passenger Building	GIC	√	-
Tung Chung (Drawing No MCL/P132/EIA/17-3-003)				
TC-1	Caribbean Coast Block 1	R	√	√
TC-2	Caribbean Coast Block 6	R	√	√
TC-3	Caribbean Coast Block 11	R	√	√
TC-4	Caribbean Coast Block 16	R	√	√
TC-5	Ho Yu College	E	√	√
TC-6	Ho Yu Primary School	E	√	√
TC-7	Coastal Skyline Block 1	R	√	√
TC-8	Coastal Skyline Block 5	R	√	√
TC-9	La Rossa Block B	R	√	√
TC-10	Le Bleu Deux Block 1	R	√	√
TC-11	Le Bleu Deux Block 3	R	√	√
TC-12	Le Bleu Deux Block 7	R	√	√
TC-13	Seaview Crescent Block 1	R	√	√
TC-14	Seaview Crescent Block 3	R	√	√
TC-15	Seaview Crescent Block 5	R	√	√
TC-16	Ling Liang Church E Wun Secondary School	E	√	√
TC-17	Ling Liang Church Sau Tak Primary School	E	√	√
TC-18	Tung Chung Public Library	GIC	√	-
TC-19	Tung Chung North Park	P	√	-
TC-20	Novotel Citygate Hong Kong	C	√	-
TC-21	One Citygate	C	√	-
TC-22	One Citygate Bridge	C	√	-
TC-23	Fu Tung Shopping Centre	C	√	-

HSR ID	Location	Landuse [1]	Acute Risk	Chronic Risk
TC-24	Tung Chung Health Centre and Air Quality Monitoring Station	GIC	√	-
TC-25	Ching Chung Hau Po Woon Primary School	E	√	√
TC-26	Po On Commercial Association Wan Ho Kan Primary School	E	√	√
TC-27	Po Leung Kuk Mrs. Ma Kam Min Cheung Fook Sien College	E	√	√
TC-28	Wong Cho Bau Secondary School	E	√	√
TC-29	Yu Tung Court - Hei Tung House	R	√	√
TC-30	Yu Tung Court - Hor Tung House	R	√	√
TC-31	Fu Tung Estate - Tung Ma House	R	√	√
TC-32	Fu Tung Estate - Tung Shing House	R	√	√
TC-33	Tung Chung Crescent Block 1	R	√	√
TC-34	Tung Chung Crescent Block 3	R	√	√
TC-35	Tung Chung Crescent Block 5	R	√	√
TC-36	Tung Chung Crescent Block 7	R	√	√
TC-37	Tung Chung Crescent Block 9	R	√	√
TC-38	Yat Tung Estate - Shun Yat House	R	√	√
TC-39	Yat Tung Estate - Mei Yat House	R	√	√
TC-40	Yat Tung Estate - Hong Yat House	R	√	√
TC-41	Yat Tung Estate - Ping Yat House	R	√	√
TC-42	Yat Tung Estate - Fuk Yat House	R	√	√
TC-43	Yat Tung Estate - Ying Yat House	R	√	√
TC-44	Yat Tung Estate - Sui Yat House	R	√	√
TC-45	Village house at Ma Wan Chung	R	√	√
TC-46	Ma Wan New Village	R	√	√
TC-47	Tung Chung Our Lady Kindergarden	E	√	√
TC-48	Sheung Ling Pei	R	√	√
TC-49	Tung Chung Public School	E	√	√
TC-50	Ha Ling Pei	R	√	√
TC-51	Lung Tseung Tau	R	√	√
TC-52	YMCA of Hong Kong Christian College	E	√	√
TC-53	Hau Wong Temple	W	√	√
TC-54	Sha Tsui Tau	R	√	√
TC-55	Ngan Au	R	√	√

HSR ID	Location	Landuse [1]	Acute Risk	Chronic Risk
TC-56	Shek Lau Po	R	√	√
TC-57	Mo Ka	R	√	√
TC-58	Shek Mun Kap	R	√	√
TC-59	Shek Mun Kap Lo Hon Monastery	W	√	√
TC-P1	Planned North Lantau Hospital	H	√	√
TC-P2	Planned Park near One Citygate	P	√	-
TC-P5	Tung Chung West Development	N/A	√	√
TC-P6	Tung Chung West Development	N/A	√	√
TC-P7	Tung Chung West Development	N/A	√	√
TC-P8	Tung Chung East Development	N/A	√	√
TC-P9	Tung Chung East Development	N/A	√	√
TC-P10	Tung Chung East Development	N/A	√	√
TC-P11	Tung Chung East Development	N/A	√	√
TC-P12	Tung Chung Area 53a - Planned Hotel	C	√	-
TC-P13	Tung Chung Area 54 - Planned Residential Development	R	√	√
TC-P14	Tung Chung Area 55a - Planned Residential Development	R	√	√
TC-P15	Tung Chung Area 89 - Planned Primary / Secondary School	E	√	√
TC-P16	Tung Chung Area 90 - Planned Special School	E	√	√
TC-P17	Tung Chung Area 39	N/A	√	√
San Tau (Drawing No MCL/P132/EIA/17-3-002)				
ST-1	Village house at Tin Sum	R	√	√
ST-2	Village house at Kau Liu	R	√	√
ST-3	Village house at San Tau	R	√	√
Sha Lo Wan (Drawing No MCL/P132/EIA/17-3-002)				
SLW-1	Sha Lo Wan House No.1	R	√	√
SLW-2	Sha Lo Wan House No.5	R	√	√
SLW-3	Sha Lo Wan House No.9	R	√	√
SLW-4	Tin Hau Temple at Sha Lo Wan	W	√	√
San Shek Wan (Drawing No MCL/P132/EIA/17-3-002)				
SSW-1	San Shek Wan	R	√	√
Sham Wat (Drawing No MCL/P132/EIA/17-3-002)				
SW-1	Sham Wat House No. 39	R	√	√
SW-2	Sham Wat House No. 30	R	√	√
Siu Ho Wan (Drawing No MCL/P132/EIA/17-3-004)				

HSR ID	Location	Landuse [1]	Acute Risk	Chronic Risk
SHW-1	Village house at Pak Mong	R	√	√
SHW-2	Village house at Ngau Kwu Long	R	√	√
SHW-3	Village house at Tai Ho San Tsuen	R	√	√
SHW-4	Siu Ho Wan MTRC Depot	I	√	-
SHW-5	Tin Liu Village	R	√	√
Proposed Lantau Logistic Park (Drawing No MCL/P132/EIA/17-3-004)				
LLP-P1	Proposed Lantau Logistics Park - 1	N/A	√	-
LLP-P2	Proposed Lantau Logistics Park - 2	N/A	√	-
LLP-P3	Proposed Lantau Logistics Park - 3	N/A	√	-
LLP-P4	Proposed Lantau Logistics Park - 4	N/A	√	-
Tuen Mun (Drawing No MCL/P132/EIA/17-3-005)				
TM-7	Tuen Mun Fireboat Station	GIC	√	-
TM-8	DSD Pillar Point Preliminary Treatment Works	GIC	√	-
TM-9	EMSD Tuen Mun Vehicle Service Station	GIC	√	-
TM-10	Pillar Point Fire Station	GIC	√	-
TM-11	Butterfly Beach Laundry	I	√	-
TM-12	River Trade Terminal	I	√	-
TM-13	Planned GIC use opposite to TM Fill Bank	GIC	√	-
TM-14	EcoPark Administration Building	C	√	-
TM-15	Castle Peak Power Plant Administration Building	C	√	-
TM-16	Customs and Excise Department Harbour River Trade Division	I	√	-
TM-17	Saw Mil Number 61-69	I	√	-
TM-18	Saw Mil Number 35-49	I	√	-
TM-19	Ho Yeung Street Number 22	I	√	-

Notes:

[1] R– residential; C – Commercial; E – educational; I – Industrial; H – clinic/ home for the aged/hospital; W – worship; GIC – government, institution and community; P – Recreational/Park; OS – Open Space; N/A – Not Available.

[2] The exposure time of students in school is around 8 hours per day and 5 days per week. It is similar to the working times of the working population. In addition, most students are likely to come from adjacent residential areas at which the potential health risk has already been evaluated as part of the HIA by selection of representative HSR. Therefore, they may not need to be considered in the chronic health risk. Nevertheless, as a conservative approach, schools have also been included for evaluation of potential chronic health risk.

[3] Except for Lantau Logistics Park, for those land use with "N/A", both acute and chronic health risk were considered from a conservative point of view.

Identification of Exposure Pathways and Assessment of the Likelihood of Exposure to the Identified TAP

17.2.4.11 Toxic substances can enter the human body through different routes. The major pathways include inhalation, ingestion through food and water, and skin absorption. For chemicals in form of solid or liquid that contaminate food and water, ingestion is a major pathway. For gases and volatile chemicals, inhalation is the most important route. For toxic substances in liquid form that are lipid soluble, such as benzene, toluene, xylene, n-hexane and other organic solvents, skin absorption through contact with the liquid can be an important route of entry into the human body.

17.2.4.12 TAP from aircraft emissions and associated activities are either gases or suspended particulates (PM_{10}, with about 70% made up of $PM_{2.5}$). Metal are usually deposit on the surface of PM_{10} or $PM_{2.5}$. These pollutants will remain airborne and dispersed by air movements. They will not easily settle on to the soil and water by gravity. Even if these air pollutants have been brought down to the land by rainfall, they will be rapidly diluted to very low concentrations and will unlikely contribute to the pollution of the soil and water. Hence, the chance of dermal exposure through contacts with contaminated water and soil is very low. For workers, there is a potential of exposure through contact with aircraft fuel onsite. Again, as site workers would be protected by enforcing relevant labour safety regulations and provision of suitable personal protective equipment (e.g., gloves and aprons), it is considered that the risk from skin absorption of chemicals can be adequately controlled for site workers. In addition, occupational risks are beyond the scope of a public health impact assessment.

17.2.4.13 A literature search was carried out to determine the key exposure pathways that should be considered in the HIA of human exposure to the TAP. The relevant guidelines and reports identified include the following:

- Environmental health criteria for human exposure assessment, WHO (2000);
- Principles for evaluating health risks in children associated with exposure to chemicals, WHO (2006);
- Guidelines for exposure assessment, USEPA (1992);
- Human health risk assessment protocol for hazardous waste combustion facilities, USEPA (2005); and
- USEPA's Air Toxics Risk Assessment Reference Library - Volume 2 Facility-Specific Assessment (EPA-453-K-04-001B) (2004).

17.2.4.14 With reference to the representative sensitive receivers presented in **Chapter 5**, it can be noted that the key human receptors of interest are mainly residential developments. **Table 17.2.15** summarises the potential exposure pathways that will be further evaluated for residents as well as for transient population (e.g. visitors) in the evaluation of potential exposure to TAP.

Table 17.2.15: Potential exposure pathways for different population

Potential Affected Population	Risk	Exposure Pathways
Residents in Tung Chung, Sha Lo Wan, San Tau, Siu Ho Wan, etc Transient Population (e.g. visitors in hotel)	• Acute	**Inhalation** • Inhalation of vapours and particulates
Residents in Tung Chung, Sha Lo	• Chronic	**Ingestion**

Potential Affected Population	Risk	Exposure Pathways
Wan, San Tau, Siu Ho Wan, etc		• Ingestion of potable water • Incidental ingestion of soil • Ingestion of home-grown product • Ingestion of contaminated food **Inhalation** • Inhalation of vapours and particulates

17.2.4.15 Taking into account the nature of the key components of TAP identified, the findings of the literature search, which covered scientific papers, Government studies and relevant websites, the chance of exposure to TAP from the ingestion pathway has been evaluated. Examples of the relevant guidelines, reports and websites reviewed include the following:

- Oral reference dose and cancer slope factors of metallic contaminants under the "Integrated Risk Information System" developed by U.S. Environmental Protection Agency (EPA);
- Agriculture, Fisheries and Conservation Department (AFCD) website (Reference to http://www.afcd.gov.hk/english/fisheries/fish_aqu/ fish_aqu.html);
- Food Adulteration (Metal Contamination) Regulations (Chapter 132V); and

  Food Safety Report under the Food Surveillance Programme conducted by Centre of Food Safety (CFS) (Reference to http://www.cfs.gov.hk/english/programme/programme_fs/programme_fs.html).

Ingestion of potable water

17.2.4.16 Potable water for Hong Kong is derived mainly from the 'Dongjiang' river in China, and the likelihood of the river being contaminated by TAP from aircraft emissions and associated activities arising from the operation of the project is considered very low given the separation distance.

Incidental ingestion of soil

17.2.4.17 Young children may ingest soil accidentally by transferring soil present in their hands, food or toys (that have contacted the soil) to their mouths. However, this activity is unlikely to result in ingestion of TAP generated from the operation of the project because the emissions generated from aircraft emissions and associated activities of the project are either gases or suspended particulates (PM_{10}, with about 70% made up of $PM_{2.5}$). These pollutants will remain airborne and dispersed by air movements. Even when these TAP have been brought down to the land by rainfall, they will be rapidly diluted to very low concentrations. Besides, for most representative human receptors in the study area, the chance of direct contact with soil resulting in accidental ingestion of soil is considered very low.

Ingestion of home-grown product

17.2.4.18 Most agricultural products consumed in Hong Kong is imported from neighbouring mainland China. Locally raised pigs and chickens could be exposed to emissions of TAP through ingestion of locally raised grain and silage or through grazing on locally impacted lands. However, given the small percentage of land that is used for farming in Hong Kong, and the low probability of

significant soil contamination by airborne pollutants, it is highly unlikely that home-grown vegetables and local livestock fed on locally-produced silage or grazed on local pasture land would constitute a significant source of ingestion risk arising from emission of TAP from the project. Hence, the risk due to ingestion of home-grown produce for exposure to TAP from the project is considered low.

17.2.4.19 According to AFCD's information, there are no fish ponds within 5 km of the project site (http://www.afcd.gov.hk/english/ fisheries/fish_aqu/fish_aqu_mpo/fish_aqu_mpo.html). Hence, ingestion of TAP due to the consumption of fishes from fish ponds contaminated by TAP from the project is considered unlikely.

Ingestion of contaminated seafood

17.2.4.20 According to the 2006 Port Survey of AFCD, the waters to the north and north-west of HKIA have fisheries production value. However, most identified TAP will be transformed to other chemicals instead of accumulating in the environment. Therefore, when the TAP resulting from the operation of the project enter the sea, they will be quickly diluted. In the natural environment, they will then be degraded, usually into less toxic or non-toxic products. Hence, emissions of most TAP from the project should not lead to bioaccumulation inside fish.

Inhalation Exposure

17.2.4.21 Most TAP emitted from aircraft and associated activities during the operation of the project will be in gaseous form, vapour form or particulates suspended in the air. These include, for example, benzene, toluene, xylene, acrolein, acetaldehyde, proprionaldeyde. These toxic chemicals are highly volatile. As they are released at high temperature, they tend to remain in gaseous phase. Even as they cool down, they would exist as vapours instead of liquids. Since it is released at high temperature, it tends to remain in gaseous phase. Most TAP will exist in gaseous or vapour state instead of solid state.

17.2.4.22 In vapour state, inhalation of TAP is the predominant route of entry into the human body because a large volume of air is breathed into the human body every minute. The inhalation exposure route is therefore the prominent pathway considered in the quantitative health impact assessment.

Determination of Exposure to TAP

TAP Speciation Profile

17.2.4.23 The quantification of criteria pollutants emission inventory and their dispersion have been documented in **Chapter 5** of this EIA report. This section summarises the quantification of TAP emission inventories and their dispersion.

17.2.4.24 To estimate the quantity of an individual TAP, speciation factors have been used. These factors estimate the quantity of an individual TAP with consideration of emission levels of volatile organic compounds and particulate matter. As there is no locally available speciation factor, the speciation profiles or factors used in this study has been based on the Emissions and Dispersion Modeling System (EDMS) default value and/or available research findings / data from USEPA, FAA, etc. which represent the best available information. Details of the speciation factors

are presented in **Appendix 17.2.1**. The following subsections summarise the methodology for determining the speciation profiles of the emission sources associated with airport-related activities, proximity infrastructure, and background contributions. Those TAP not related to the airport operation were not considered.

Airport Related activities TAP Emissions

17.2.4.25 **Table 17.2.16** summarises the methodology to determine the speciated VOC, diesel particulate matter (DPM), heavy metal, PAH and dioxin speciation factors for aircraft emissions and associated activities arising from the operation of the project.

Table 17.2.16: Methodology for determination of TAP speciation profile for airport activities

Emission Sources	TAP	Speciation Method
Aircraft and business jet	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	N/A
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• Naphthalene: EDMS V5.1.4.1 built-in TAP speciation profile • Other PAH: Select Resource Materials and Annotated Bibliography on the Topic of Hazardous Air Pollutants (HAPs) Associated with Aircraft, Airports, and Aviation (FAA, 2003)
	TCDD	N/A
APU	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	N/A
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• Naphthalene: EDMS V5.1.4.1 built-in TAP speciation profile • Other PAH: Select Resource Materials and Annotated Bibliography on the Topic of Hazardous Air Pollutants (HAPs) Associated with Aircraft, Airports, and Aviation (FAA, 2003)
	TCDD	N/A
GSE	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	• All PM from diesel engine are assumed as DPM.
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012
	TCDD	• MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012
Helicopter	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	N/A
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• Naphthalene: EDMS V5.1.4.1 built-in TAP speciation profile • Other PAH: Select Resource Materials and Annotated Bibliography on the Topic of Hazardous Air Pollutants (HAPs) Associated with Aircraft, Airports, and Aviation (FAA, 2003)
	TCDD	N/A
Aviation Fuel Farm	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	N/A
	Heavy metal	N/A
	PAH	N/A
	TCDD	N/A

Emission Sources	TAP	Speciation Method
Fire Training Activities	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	N/A
	Heavy metal	N/A
	PAH	N/A
	TCDD	N/A
Engine Maintenance Center	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	N/A
	Heavy metal	N/A
	PAH	N/A
	TCDD	N/A
Engine Testing Facilities	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	N/A
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• Naphthalene: EDMS V5.1.4.1 built-in TAP speciation profile • Other PAH: Select Resource Materials and Annotated Bibliography on the Topic of Hazardous Air Pollutants (HAPs) Associated with Aircraft, Airports, and Aviation (FAA, 2003)
	TCDD	N/A
Catering	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	• All PM from diesel engine are assumed as DPM.
	Heavy metal	• U.S. EPA AP-42
	PAH	• U.S. EPA AP-42
	TCDD	• U.S. EPA AP-42
Airport Ferries	VOC(speciated)	• USEPA, 2009. Documentation for Commercial Marine Vessel of the National Emission Inventory Methodology
	DPM	• All PM from diesel engine are assumed as DPM.
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• Methodology for calculating emissions from ships: 1. Update of emission factors, Swedish Environmental Protection Agency, 2004
	TCDD	• Methodology for calculating emissions from ships: 1. Update of emission factors, Swedish Environmental Protection Agency, 2004
Car Parks	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	• All PM from diesel engine were assumed as DPM.
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012
	TCDD	• MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012
Motor Vehicles	VOC(speciated)	• EDMS V5.1.4.1 built-in TAP speciation profile
	DPM	• All PM from diesel engine were assumed as DPM.
	Heavy metal	• USEPA SPECIATE 4.3 database
	PAH	• MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012
	TCDD	• MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012

Note: N/A means not applicable.

Proximity Infrastructure TAP Emission

17.2.4.26 **Tables 17.2.17** and **17.2.18** summarise the methodology in determining the TAP speciation factors for different proximity infrastructure emissions in Lantau area and Tuen Mun area respectively.

Table 17.2.17: Methodology for determination of TAP speciation profile for proximity infrastructure emission in Lantau area

Project / Sources	Emission Type	TAP Speciation
Hong Kong Boundary Crossing Facilities (HKBCF)	Vehicular emissions	• VOC (speciated) : EDMS V5.1.4.1 built-in TAP speciation profile • DPM: All PM from diesel engine were assumed as DPM. • Heavy Metals: USEPA SPECIATE 4.3 database • PAH: MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012 • Dioxin: MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012
Hong Kong Link Road (HKLR)	Vehicular emissions	
Tuen Mun – Chek Lap Kok Link (TM-CLKL) (Lantau section)	Vehicular emissions	
North Lantau Highway (NLH) and other roads in Tung Chung	Vehicular emissions	
Tung Chung New Town Extension Study	Vehicular emissions	
Organic Wastes Treatment Facilities (OWTF) Phase 1	Chimney emissions	• VOC (speciated): USEPA SPECIATE 4.3 database • No DPM, heavy metals, PAH, and dioxin were identified in the approved EIA of "Organic Waste Treatment Facilities, Phase I"

Table 17.2.18: Methodology for determination of TAP speciation profile for proximity infrastructure emission in Tuen Mun area

Project / Sources	Emission Type	TAP Speciation
Tuen Mun Western Bypass (TMWB)	Vehicular emissions	• VOC (speciated): EDMS V5.1.4.1 built-in TAP speciation profile • DPM: All PM from diesel engine were assumed as DPM. • Heavy Metals: USEPA SPECIATE 4.3 database • PAH: MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012 • Dioxin: MOVES2010b - Additional Toxics Added to MOVES, USEPA, 2012
TM-CLKL (Tuen Mun section)	Vehicular emissions	
Other roads in Tuen Mun	Vehicular emissions	
Shiu Wing Steel Mill	Chimney emissions	• VOC (speciated): USEPA AP-42 • Heavy metal: USEPA AP-42 • PAH: USEPA AP-42 • Dioxin: USEPA AP-42
Green Island Cement	Chimney emissions	• VOC (speciated): USEPA AP-42 • Heavy metals: USEPA AP-42 • PAH: USEPA AP-42 • Dioxin: USEPA AP-42
Castle Peak Power Plant (CPPP)	Chimney emissions	• Given that the plume from the chimney will be dispersed at a height higher than 200m and has less influence on the administrative building inside the CLPP site, the effect of chimney is thus not taking into account for Tap Shek Kok Receivers.
EcoPark in Tuen Mun Area 38	Chimney emissions	• VOC (speciated): USEPA AP-42 • Heavy metals: USEPA AP-42 • PAH: USEPA AP-42 • Dioxin: USEPA AP-42
Butterfly Beach Laundry	Chimney emissions	• VOC (speciated): USEPA AP-42

Project / Sources	Emission Type	TAP Speciation
		• Heavy metals: USEPA AP-42 • PAH: USEPA AP-42 • Dioxin: USEPA AP-42
Flare at Pillar Point Valley Landfill (PPVL)	Chimney emissions	• VOC (speciated): EDMS V5.1.4.1 built-in TAP speciation profile • Follow approved EIA of "Organic Waste Treatment Facilities, Phase I" - No DPM, heavy metals, PAH, and dioxin were identified
Permanent Aviation Fuel Facility (PAFF)	Emissions from fuel tanks	• VOC (speciated) from fuel tank : EDMS V5.1.4.1 built-in TAP speciation profile • VOC (speciated) from chimney: USEPA AP-42 • Heavy metals from chimney : USEPA AP-42 • PAH from Chimney: USEPA AP-42 • Dioxin from chimney: USEPA AP-42
Marine	Exhaust Emissions	• VOC (speciated): USEPA, 2009. Documentation for Commercial Marine Vessel of the National Emission Inventory Methodology • DPM: All PM from diesel engine were assumed as DPM. • Heavy Metals: USEPA SPECIATE 4.3 database • PAH: Methodology for calculating emissions from ships: 1. Update of emission factors, Swedish Environmental Protection Agency, 2004 • Dioxin: Methodology for calculating emissions from ships: 1. Update of emission factors, Swedish Environmental Protection Agency, 2004

17.2.4.27 **Appendix 17.2.2** summarises the speciated TAP emissions estimated for different activities in 2031.

Background Contributions

17.2.4.28 EPD has conducted VOC and carbonyl compounds measurements at the air quality monitoring stations (AQMS) in Yuen Long, Tung Chung, Tsuen Wan and Central Western. The Yuen Long AQMS and Tsuen Wan AQMS were characterised by the industrial activities in the vicinity. Therefore, the Tung Chung AQMS, the station closest to the airport, was selected for available information on background TAP concentrations. The Central Western AQMS, which is less influenced by industrial and marine emission sources than the other air quality monitoring stations, has also been considered for information on available background TAP concentrations in cases relevant data were not available from the Tung Chung AQMS.

17.2.4.29 According to EPD, 30 VOC species were measured at the Tung Chung AQMS, while 143 VOC species and 16 carbonyl species were measured at the Central Western AQMS. The latest available measurement data at Tung Chung and Central Western AQMS in Year 2011 have formed the basis in determining the best available information on ambient TAP concentrations. **Appendix 17.2.3** lists the detailed TAP measurements in Tung Chung AQMS and Central Western AQMS in Year 2011.

17.2.4.30 The ambient DPM is derived from EC based on the following equation:

Diesel Particulate Matter (DPM) = 1.04 x Elemental Carbon (EC) concentration (Wong *et al*. 2002a).

Air Quality Modelling for TAP

17.2.4.31 TAP levels have been estimated from the VOC and PM concentrations based on the TAP speciation factors. **Table 17.2.19** summarises the modelling methodologies adopted for representative human receptors in North Lantau and Tuen Mun, repspectively.

Table 17.2.19: Modelling Methodology for different type of receivers

Area	Airport related sources	Proximity infrastructure sources in Lantau	Proximity infrastructure sources in Tuen Mun	Ambient Concentrations of TAP
North Lantau	AERMOD / CALINE	AERMOD / CALINE	-	EPD Tung Chung AQMS and Central Western AQMS
Tuen Mun	AERMOD / CALINE	-	AERMOD / CALINE	EPD Tung Chung AQMS and Central Western AQMS

Impact from Airport Related Sources

17.2.4.32 AERMOD model has been adopted as the air quality impact model for VOC / PM modelling from major airport related activities, except for roads on the airport island which were modelled by the EMFAC-HK v2.6 and CALINE4 model. The AERMOD model allows three types of sources: Point, Area and Volume. Hence, the emission sources inside the HKIA were modelled as one of the three sources according to their sources emission characteristics. Hourly meteorological data including wind speed, wind direction, air temperature, and upper air data (such as cloud coverage, mixing height, etc.) from the Fifth-Generation Penn State / NCAR Mesoscale Model (MM5) were adopted.

17.2.4.33 Details of the air quality modelling methodology shall refer to **Chapter 5**. The concentrations of TAP of interest due to airport related activities have been determined by applying the derived speciation factors as discussed in **Table 17.2.16** on the modelled VOC / PM concentrations.

Impact from Proximity Infrastructure Sources

17.2.4.34 CALINE-4 model has been used to predict the VOC / PM at representative human receptors near open roadways by taking into account the composite emission factors generated from EMFAC-HK v2.6 model. Roadways are divided into a series of segments from which incremental concentrations are computed and then summed to form a total concentration estimate at the representative human receptors and simulated grid points. Hourly meteorological data including wind speed, wind direction, air temperature, mixing height from MM5 have been adopted. Pasquill stability class has been determined using the USEPA PCRAMMET programme.

17.2.4.35 Potential emission sources in the vicinity, including Green Island Cement Plant, Shiu Wing Steel Mill, river trade terminal, etc., have been included in proximity infrastructure emissions (i.e. modelled by near-field dispersion model). The power plants of CLPP were modeled in PATH model. The emission characteristics were based on the relevant information/data including approved EIA reports or EPD's modelling guideline. The AERMOD model has been adopted to predict concentrations of VOC and PM at the representative human receptors. Hourly

meteorological data including wind speed, wind direction, air temperature, and mixing height data from MM5 have been adopted.

17.2.4.36 Details of the air quality modelling methodology shall refer to **Chapter 5**. The concentrations of TAP of interest due to the various proximity infrastructure and different industrial activities emission sources have been determined by applying the derived speciation factors as discussed in **Tables 17.2.17** and **17.2.18** on the modelled VOC / PM concentrations.

Impact from Ambient Sources of Air pollutants

17.2.4.37 With respect to the next phase of the emissions reduction plan, the two Governments (Hong Kong and Guangdong) endorsed the emission reduction targets for 2015, and agreed to set emission reduction for 2020. As compared with the emission levels in 2010, the emission targets of the four major air pollutants in HKSAR and in Pearl River Delta Economic Zone (PRDEZ) for 2015 and 2020 are shown in **Tables 17.2.20** and **17.2.21**.

Table 17.2.20: Summary of emission targets in PRDEZ

Year	Pollutants *(Thousand Tonnes)*				References
	SO_2	NO_x	PM_{10}	VOC	
2010	507	889	637	903	The Hong Kong-Guangdong Joint Working Group on Sustainable Development and Environmental Protection (JWGSDEP) 12th meeting, 2012
2015	426	729	573	813	
2020	406	711	541	768	

Table 17.2.21: Summary of emission targets in HKSAR

Year	Pollutants *(Thousand Tonnes)*				References
	SO_2	NO_x	PM_{10}	VOC	
2010	35.5	108.6	0.63	33.7	The Hong Kong-Guangdong Joint Working Group on Sustainable Development and Environmental Protection (JWGSDEP) 12th meeting, 2012
2015	26.6	97.7	0.57	32	
2020	23.1	86.9	0.54	28.6	

17.2.4.38 As shown in **Tables 17.2.20** and **17.2.21**, the VOC and PM_{10} emission inventory shows a decreasing trend from years 2010 to 2020. As a conservative approach, the VOC and EC monitoring data at EPD's monitoring stations in Year 2011 have been adopted to represent the background concentrations for the future years.

Cumulative Impact

17.2.4.39 The estimated concentrations of the key components of TAP from AERMOD and CALINE4 models have been combined hour by hour to determine the acute and chronic risks.

17.2.5 Dose-Response Assessment

17.2.5.1 Human health risk assessment is a combination of procedures, models and tools by which a proposed development may be judged as to its potential effects on the health of the nearby representative human receptors. The dose-response assessment step involves an evaluation of the relationship between exposures and responses in human with respect carcinogenic health effects as well as acute and chronic non-carcinogenic health effects. Acute effects are obvious

and usually occur soon after exposure. Some may be reversible when exposure to the pollutant ends and some are often irreversible, even after exposure to the pollutant come to an end.

Consequence of Exposure

17.2.5.2 The consequences of exposure to the key TAP identified for airport related sources are summarised in **Table 17.2.22**.

Table 17.2.22: Consequences of exposure to the key TAP for airport related sources

Pollutant	Characteristics	Consequence
1,3-butadiene	1,3-butadiene is a colorless gas. At room temperature, the gas has a gasoline-like odour. This pollutant is a byproduct of petroleum processing and is used in the production of synthetic rubber and plastics. It is also found in automobile exhaust, gasoline vapor, fossil fuel incineration products, and cigarette smoke.	The majority of 1,3-butadiene is released into the air and humans are typically exposed to the pollutant via inhalation. Breathing very high levels of 1,3-butadiene for a short time may cause central nervous system damage, blurred vision, nausea, fatigue, headache, decreased blood pressure and pulse rate, and unconsciousness. Breathing lower levels of this pollutant may cause irritation of the eyes, nose, and throat. The IARC has classified 1,3-butadiene as a “group 1 known carcinogen".
Acetaldehyde	Acetaldehyde is a colorless, volatile liquid with a characteristic pungent, fruity odour. Acetaldehyde is used primarily as a chemical intermediate in the production of acetic acid, as well as a synthetic flavouring agent. Acetaldehyde is released to the environment in vehicle exhaust and as a product of open burning of gas, fuel oil, and coal.	Acute exposure to acetaldehyde can cause eye, nose, and throat irritation and subsequent inflammation of the eyes and coughing. This pollutant can also cause central nervous system depression, delayed pulmonary edema, and moderate unconsciousness. Chronic inhalation exposure at high concentrations causes adverse respiratory tract effects in animals. Carcinogenicity studies in rats have shown that acetaldehyde causes respiratory tract tumors. The IARC has classified acetaldehyde as a “group 2B possibly carcinogenic to humans".
Acrolein	Acrolein is a clear or yellow liquid with a disagreeable odour. Acrolein is used as an intermediate in the production of acrylic acid, as well as a pesticide to control algae, weeds, bacteria, and mollusks. Small amounts of acrolein can be formed and emitted into the air when trees, tobacco, other plants, gasoline, and oil are burned. Acrolein may also be released in to the environment in emissions and effluents from its manufacturing and use facilities and in emissions from combustion processes.	Exposure to high concentrations of acrolein may damage the lungs and could cause death. Breathing lower amounts may cause eye watering and burning of the nose and throat and a decreased breathing rate. The USEPA has classified acrolein as “not classifiable” as to human carcinogenicity.
Arsenic	Arsenic is a naturally occurring element. It is released into the air by volcanoes, the weathering of arsenic-containing minerals and ores, and by commercial or industrial processes.	Acute high-level inhalation exposure to arsenic dust or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain); central and peripheral nervous system disorders. Chronic inhalation exposure to inorganic arsenic of humans leads to lung cancer, irritation of the skin and mucous membranes and effects in the brain and nervous system. IARC classifies inorganic arsenic as "group 1 human carcinogen".

Pollutant	Characteristics	Consequence
Benz(a) anthracene	Benz(a) anthracene is present as a major component of the total content of polynuclear aromatic hydrocarbons in the environment. Human exposure to benz(a)anthracene occurs primarily through smoking of tobacco, inhalation of exhaust emissions from gasoline engines and incomplete combustion of fossil fuels.	No acute health effect of Benz (a) anthracene is known this time. There is evidence that it causes cancer in humans and it has been shown to cause liver and lung cancer in animals. IARC classifies it as "group 2B probable human carcinogen".
Benzene	Benzene is a volatile, colorless, flammable liquid that has a sweet odour. It is a chemical intermediate in the synthesis of compounds such as plastics, resins, nylon, synthetic fibers, synthetic rubbers, lubricants, dyes, detergents, drugs, and pesticides. Major sources of atmospheric releases include vehicle exhaust emissions, evaporative gasoline fumes, emissions from vehicle service stations, and industrial emissions. Other sources of atmospheric benzene include cigarette smoke and landfill emissions.	Acute inhalation exposure to benzene can result in death, while high levels can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death. Chronic exposure to benzene causes leukemia and aplastic anemia. The IARC has classified benzene as a “group 1 known carcinogen".
Benzo(a)pyrene	Benzo(a)pyrene is present as a major component of the total content of polynuclear aromatic hydrocarbons in the environment. Major sources of PAHs in ambient air (both outdoors and indoors) include residential and commercial heating with wood, coal or other biomasses (oil and gas heating produce much lower quantities of PAH), other indoor sources such as cooking and tobacco smoke, and outdoor sources like motor-vehicle exhaust (especially from diesel engines), industrial emissions and forest fires.	In humans, BaP has been associated with chromosomal replication errors and altered DNA in gametes (sperm and eggs). In adults, BaP exposure was associated with altered sperm morphology and decreased sperm numbers, and decreased egg numbers. At high levels of acute exposure in adults, BaP has been reported to be associated with red blood cell damage, which can lead to anemia. The IARC has classified benzo(a)pyrene as a “group 1 known carcinogen"
Benzo(bk)fluoranthene	Benzo(bk)fluoranthene is PAH. Sources of Benzo(bk)fluoranthene in ambient air (both outdoors and indoors) include forest fires, industrial emissions, residential and commercial heating with wood, coal, or other biomass fuels (oil and gas heating produce much lower quantities of PAHs), motor vehicle exhaust (especially diesel), and other indoor sources such as cooking and tobacco smoke.	Carcinogenicity studies in animals show that health concerns of Benzo(bk)fluoranthene are associated with tumors in in the nasal cavity, larynx, trachea, pharynx, lung and oesophagus. Benzo(bk)fluoranthene is classified as "group 2B probable human carcinogen' based on sufficient data from animal bioassays by IARC.
Beryllium	Beryllium (Be) is a dark gray metal of the alkaline earth family and is moderately rare in its natural form. Beryllium is used industrially to harden copper, for the manufacture of nonsparking alloys for tools, in the manufacture of lightweight alloys and ceramics, and in the construction of nuclear reactors. However, most beryllium in the environment is released through coal	Data on human toxicity from beryllium are only available following inhalation exposures. The lung is the major target organ following inhalation of beryllium in a variety of forms. High levels of beryllium in air can cause an acute pneumonitis (acute beryllium disease) characterized by edema and inflammation. Extreme cases can be fatal. Chronic exposure to low levels of beryllium in air may lead to chronic beryllium disease (berylliosis). The IARC has classified beryllium as

Pollutant	Characteristics	Consequence
	burning operations.	a “group 1 known carcinogen".
Cadmium	Cadmium is an element of the transitional metal series that occurs widely in nature, usually in sulfide or zinc ores. Natural weathering of minerals releases small amounts of cadmium to the environment, but human activities are responsible for the majority of cadmium releases. Anthropogenic sources of cadmium include releases from mining and smelting, fuel combustion, manufacture and use of phosphate fertilizer, application of sewage sludges, waste incineration, and primary and secondary metal production.	Absorption of cadmium following inhalation exposure varies depending on particle size. Large particles (>10 microns in diameter) tend to be deposited in the upper airway, while smaller particles (about 0.1 microns) tend to penetrate into the alveoli. Cadmium bioaccumulates in mammals, particularly in the kidney and liver. Epidemiological studies have revealed an association between nonmalignant pulmonary diseases and inhalation of cadmium. It is also suspected that chronic exposure to cadmium produces anemia, sensory loss (particularly smell), and immunosuppression in humans. The IARC has classified cadmium as a “group 1 known carcinogen".
Chromium VI	Chromium is a naturally occurring metal present in low concentrations in the earth's crust. Chromium (VI) is the second most stable chromium compound, after chromium (III). Natural occurrence of hexavalent chromium (chromium [VI]) is infrequent; it occurs in nature in the rare mineral crocoite (PbCrO4). It is primarily produced from anthropogenic sources. Chromium (VI) is used extensively in industry, mainly for plating metals such as stainless and alloy steels and aluminum. It is also used as an additive in cleansing agents, paints, catalysts, fungicides, and wood preservatives.	Hexavalent chromium compounds are strong oxidizing agents and are severely irritating and corrosive. Acute inhalation exposure to chromium (VI) may cause asthma attacks in sensitive individuals; concentrations at which these effects occur were not described. Acute inhalation exposure to chromium fumes may also cause fever, chills, and muscle aches. Chronic inhalation of dust containing chromium (VI) concentrations may cause respiratory irritation, emphysema, chronic bronchitis, and other respiratory conditions. USEPA has classified inhaled chromium (VI) as Group A - Human Carcinogen. The IARC has classified chromium(VI) as a “group 1 known carcinogen".
Chrysene	Chrysene is present as a major component of the total content of polynuclear aromatic compounds in the environment. Human exposure to chrysene occurs primarily through the smoking of tobacco, inhalation of polluted air.	Inhalation of Chrysene may irritate the nose and throat causing coughing and wheezing as acute effects. Chrysene is classified as "group 2B probable human carcinogen" as it has shown to cause liver and lung cancer in animals. The IARC has classified chrysene as a “group 2B Possibly carcinogenic to humans".
Copper	Copper is a reddish metal that occurs naturally in rock, soil, water, sediment, and, at low levels, air. Copper can enter the environment through releases from the mining of copper, and from factories that make or use copper metal or copper compounds. Copper can also enter the environment through waste dumps, domestic waste water, combustion of fossil fuels and wastes, wood production, phosphate fertilizer production, and natural sources.	In humans, copper is a respiratory irritant. Workers exposed to copper dust report a number of symptoms that are suggestive of respiratory irritation, including coughing, sneezing, thoracic pain, and runny nose. Copper is also considered the etiologic agent in the occupational disease referred to as “vineyard sprayer’s lung”. USEPA has not yet classified copper as a human carcinogen.

Pollutant	Characteristics	Consequence
Dibenz(a,h)anthracene	Dibenz(a,h)anthracene is a specie of Polycyclic aromatic hydrocarbons (PAHs). PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and charbroiled meat. PAHs usually occur naturally, but they can be manufactured as individual compounds for research purposes.	Studies of people show that individuals exposed by breathing or skin contact for long periods to mixtures that contain PAHs and other compounds can develop cancer. United States Department of Health and Human Services (USHHS) has determined that Dibenz(a,h)anthracene is known animal carcinogens. The IARC has classified dibenz(a,h)anthracene as a "Group 2A Probably carcinogenic to humans".
Diesel Particulate Matters	Diesel PM is formed primarily through the incomplete combustion of diesel fuel. PM in diesel exhaust can be emitted from on- and off-road vehicles, stationary area sources, and stationary point sources. Typical diesel exhaust particles have diameters ranging from 0.1 to 0.25 micrometers (µm). The particles are mainly aggregates of spherical elemental carbon particles coated with organic and inorganic substances.	The primary route by which humans are exposed to diesel exhaust PM is via inhalation. Numerous epidemiological and clinical studies have conclusively shown that exposure to PM in diesel emissions is associated with increases in respiratory illnesses such as bronchitis, emphysema and asthma, as well as premature deaths from cardio-pulmonary disorders. Diesel exhaust is classified as Group 1 “Carcinogenic to human” as to its carcinogenicity to humans" by IARC.
Ethylbenzene	Ethylbenzene is a colourless liquid with an aromatic odour. It is used primarily in the production of styrene. It is also used as a solvent, as a constituent of asphalt and naphtha, and in fuels. It may occur naturally, as it has been found in orange peel, parsley leaves, dried legumes and other foodstuffs.	Short-term inhalation exposure of people to Ethylbenzene can cause respiratory effects, such as throat irritation and chest constriction, irritation of the eyes, and neurological effects such as dizziness. Long-term inhalation exposure of people to Ethylbenzene may results effects on the blood. IARC has classified ethylbenzene as a "group 2B Possibly carcinogenic to humans".
Formaldehyde	At room temperature, formaldehyde is a colorless, flammable gas that has a distinct, pungent smell. Formaldehyde is a product of incomplete combustion and is emitted into the air by burning wood, coal, kerosene, and natural gas, by automobiles, and by cigarettes; it is also a naturally occurring substance. Formaldehyde can be released to soil, water, and air by industrial sources and can off-gas from materials made with it. Humans can be exposed to formaldehyde through inhalation of contaminated air and smog.	Low levels of formaldehyde can cause irritation of the eyes, nose, throat, and skin. Chronic exposure leads to cancer of the nasopharynx and leukemia. The IARC classifies formaldehyde as a “group 1 carcinogen”.
Indeno(1,2,3-cd)pyrene	Indeno(1,2,3-cd)pyrene belongs to aromatic hydrocarbon compounds (PAHs), which are formed primarily from combustion and are present in the atmosphere in particulate form. Sources of air emissions are diverse and include cigarette smoke, vehicle exhaust, home heating, laying tar, and grilling meat.	No reports of effects to humans following acute exposure to polycyclic organic matter (POM) are available. Epidemiologic studies have reported an increase in lung cancer in humans exposed to coke oven emission, roofing tar emissions, and cigarette smoke. Each of these mixtures contains a number of POM compounds. USEPA has classified Indeno(1,2,3-cd)pyreneas "group B2, probable human carcinogens".

Pollutant	Characteristics	Consequence
Isopropylbenzene (cumene)	Cumene is a water-insoluble petrochemical used in the manufacture of several chemicals, including phenol and acetone. It readily volatilizes into the atmosphere from water and dry soil/sediments and to undergo biodegradation in water and soil.	Short-term inhalation exposure to cumene may cause headaches, dizziness, drowsiness, slight incoordination, and unconsciousness in humans. Cumene has a potent central nervous system (CNS) depressant action characterized by a slow induction period and long duration of narcotic effects in animals. Cumene is a skin and eye irritant. No information is available on the chronic, reproductive, developmental, or carcinogenic effects of cumene in humans. USEPA has classified cumene as a Group D, not classifiable as to human carcinogenicity.
Lead	Lead is a naturally occurring, soft, bluish-gray heavy metal. Due to its abundance, low cost and physical properties (low melting point, corrosion resistance, waterproof nature and malleability) lead and lead compounds have been utilized in a variety of products including cable covers, petrol (gasoline), paint, plastics, pesticides, solder, etc. This widespread use of lead has caused extensive environmental contamination and health problems in many parts of the world.	Short-term inhalation exposure of people to high levels of lead can cause gastrointestinal disturbances (anorexia, nausea, vomiting, abdominal pain), hepatic and renal damage, hypertension and neurological effects (malaise, drowsiness, encephalopathy) that may lead to convulsions and death. Long-term inhalation exposure of lead commonly causes haematological effects, such as anaemia, or neurological disturbances. There is some evidence that long-term occupational exposure to lead may contribute to the development of cancer. IARC has classified inorganic lead compounds as a "Group 2A Probably carcinogenic to humans".
Manganese	Manganese is naturally ubiquitous in the environment. Manganese is essential for normal physiologic functioning in humans and animals, and exposure to low levels of manganese in the diet is considered to be nutritionally essential in humans. Metallic manganese is used primarily in steel production to improve hardness, stiffness, and strength. Manganese compounds have a variety of uses. Manganese dioxide is used in the production of dry-cell batteries, matches, fireworks, etc.	Long-term inhalation exposure of people to manganese results primarily in effects on the nervous system. Long-term inhalation exposure of people to high levels may result in a syndrome called manganism and typically begins with feelings of weakness and lethargy and progresses to other symptoms such as gait disturbances, clumsiness, and psychological disturbances. USEPA has classified manganese as a Group D, not classifiable as to carcinogenicity in humans.
Methyl alcohol (Methanol)	Methanol is a colorless liquid that may explode when exposed to an open flame. It is primarily used as an industrial solvent for inks, resins, adhesives, and dyes. It is also used as a solvent in the manufacture, antifreeze for automotive radiators, ingredient of gasoline, etc. Natural emission sources of methanol include volcanic gases, vegetation, microbes, and insects; methanol is also formed during biological decomposition of biological wastes, sewage, and sludge.	Short-term inhalation exposure of people to high levels of methanol may result in visual disturbances, such as blurred or dimness of vision, leading to blindness. Long-term inhalation exposure to methanol may result in headache, dizziness blurred vision, and blindness in humans. Neurological damage, specifically permanent motor dysfunction, may also result. No information is available on the carcinogenic effects of methanol in humans or animals. USEPA has not classified methanol with respect to carcinogenicity.
Xylene	Xylene is a colorless, sweet-smelling liquid that catches on fire easily. It occurs naturally in petroleum and coal tar. Chemical industries produce xylene from petroleum. Xylene is used as a solvent and in the printing, rubber, and leather	Short-term exposure of people to high levels of xylene can cause irritation of the skin, eyes, nose, and throat; difficulty in breathing; impaired function of the lungs; delayed response to a visual stimulus; impaired memory; stomach discomfort; and possible changes in the liver and

Pollutant	Characteristics	Consequence
	industries. It is also used as a cleaning agent, a thinner for paint, and in paints and varnishes. It is found in small amounts in airplane fuel and gasoline.	kidneys. Both short- and long-term exposure to high concentrations of xylene can also cause a number of effects on the nervous system, such as headaches, lack of muscle coordination, dizziness, confusion, and changes in one's sense of balance. Some people exposed to very high levels of xylene for a short period of time have died. USEPA has classified mixed xylenes as a Group D, not classifiable as to human carcinogenicity.
Naphthalene	Naphthalene is a white solid with the odour of mothballs or tar, and is found naturally in fuels when they are burned. Burning tobacco or wood also produces naphthalene. The major commercial use of naphthalene is in the manufacture of polyvinyl chloride (PVC) plastics. Naphthalene is released into the air through the burning of tobacco, wood, oil and coal.	Exposure to large amounts of naphthalene may damage or destroy some red blood cells. This condition is called hemolytic anemia, with symptoms including fatigue, lack of appetite, restlessness, and pale skin. Exposure to large amounts of naphthalene may also cause nausea, vomiting, diarrhea, blood in the urine, and a yellow color to the skin. The IARC has classified naphthalene as a "group 2B Possibly carcinogenic to humans".
n-Hexane	n-Hexane is a chemical made from crude oil. It is a colorless liquid with a slightly unpleasant odor. It evaporates very easily into the air and dissolves only slightly in water. Pure n-hexane is used in laboratories. Most of the n-hexane used in industry is mixed with similar chemicals in products known as solvents. The major use for solvents containing n-hexane is to extract vegetable oils from crops such as soybeans. They are also used as cleaning agents in the printing, textile, furniture, and shoemaking industries.	Long-term inhalation exposure of humans to n-hexane results primarily in effects on the nervous system. Feeling of numbness in feet and hands, followed by muscle weakness in the feet and lower legs were reported in several studies of workers occupationally exposed air containing high concentrations of n-hexane. USEPA has classified hexane as a Group D, not classifiable as to human carcinogenicity.
Nickel	Nickel is a hard, silvery-white metal. It is a natural element of the earth's crust, therefore, small amounts are found in food, water, soil, and air. Nickel can be combined with other metals, such as iron, copper, chromium, and zinc, to form alloys. These alloys are used to make coins, jewelry, and items such as valves and heat exchangers. Most nickel is used to make stainless steel.	Short-term inhalation exposure of people to an extremely high level of nickel suffered severe damage to the lungs and kidneys. Long-term inhalation exposure of people to nickel results in respiratory effects, including a type of asthma specific to nickel, decreased lung function, and bronchitis. The IARC has classified nickel compounds as "Group 1 Carcinogenic to humans".
Phenol (carbolic acid)	Phenol has a wide range of uses, including in the preparation of phenolic and epoxy resins, nylon-6, selective solvents for refining lubricating oils, adipic acid, phenolphthalein, etc.	Short-term inhalation exposure of people to high level of Phenol can cause irregular breathing, muscle weakness and respiratory. Anorexia, progressive weight loss and diarrhea have been reported in chronically exposed humans. Gastrointestinal irritation and blood and liver effects have also been reported. In one study, muscle pain, weakness, enlarged liver and elevated levels of liver enzymes were found in an individual after long-term inhalation and dermal exposure to phenol and a few other chemicals. USEPA has classified phenol as a Group D, not classifiable as to human carcinogenicity, based on a lack of data concerning carcinogenic effects in humans and animals.

Pollutant	Characteristics	Consequence
Propionaldehyde	Propionaldehyde is a colorless, flammable liquid with a suffocating fruity odour. Propionaldehyde is released to the atmosphere via the combustion of wood, gasoline, diesel fuel, and polyethylene. Municipal waste incinerators can release it to ambient air.	The vapor may cause respiratory irritation but is not a strong enough irritant of eyes or respiratory tract to be considered significant factor in smog. USEPA has not classified propionaldehyde for carcinogenicity. IARC has classified it as "group 3 Not classifiable as to its carcinogenicity to humans".
Styrene	Styrene is a colourless, viscous liquid with a pungent odour. It is used predominately in the production of polystyrene plastics and resins. It is also used as an intermediate in the synthesis of materials used for ion exchange resins and to produce copolymers. Indoor air is the principal route of styrene exposure for the general population, occupational exposure to styrene occurs in the reinforced plastics industry and polystyrene factories.	Short-term exposure to styrene in humans results in respiratory effects, such as mucous membrane irritation, eye irritation, and gastrointestinal effects. Long-term exposure to styrene in humans results in effects on the CNS, with symptoms such as headache, fatigue, weakness, depression, CNS dysfunction and on the blood. IARC classified styrene as "Group 2B Possibly carcinogenic to humans".
TCDD	TCDD is not intentionally produced by industry. It can be inadvertently produced in very small amounts as an impurity during the incineration of municipal and industrial wastes and during the manufacture of certain chemicals. It may be formed during the chlorine bleaching process used by pulp and paper mills, and as a by-product from the manufacture of certain chlorinated organic chemicals, such as chlorinated phenols. It is primarily released to the environment during the combustion of fossil fuels (including motor vehicles) and wood, and during incineration processes.	Short-term inhalation exposure of people to TCDD can cause chloracne, and a severe acne-like condition that can develop within months of first exposure. Chronic effects (non-cancer) from TCDD of inhalation in humans have not been reported in the literature. Human studies, primarily of workers occupationally exposed to 2,3,7,8-TCDD by inhalation, have found an association between 2,3,7,8-TCDD and lung cancer, soft-tissue sarcomas, lymphomas, and stomach carcinomas. IARC has classified it as "Group 1 Carcinogenic to humans".
Toluene	Toluene is a clear, colorless, inflammable liquid with benzene-like odour. It is used as a high-octane blending stock in gasoline; as a solvent for paints and coatings, gums, resins, oils, rubber and adhesives; and as an intermediate in the preparation of many chemicals, dyes, pharmaceuticals, detergents and explosives. It is released into the atmosphere principally from the volatilization of petroleum fuels and toluene-based solvents and thinners and in motor vehicle exhaust. It is also present in emissions from volcanoes, forest fires and crude oil.	The central nervous system (CNS) is the primary target organ for toluene toxicity in both humans and animals for acute and chronic exposures. CNS dysfunction and narcosis have been frequently observed in humans acutely exposed to low or moderate levels of toluene by inhalation; symptoms include fatigue, sleepiness, headaches, and nausea. CNS depression and death have occurred at higher levels of exposure. Long-term inhalation exposure of humans to toluene causes irritation of the upper respiratory tract and eyes. Under the Guidelines for Carcinogen Risk Assessments (USEPA, 2005), the USEPA considers that there is inadequate information to assess the carcinogenic potential of toluene.

Non-carcinogenic Health Risk of TAP

17.2.5.3 For acute and chronic non-carcinogenic risks, a threshold level of exposure (a reference concentration) can usually be identified, below which it is generally considered that significant health effects will not occur on acute or, for chronic risk, continuous long-term exposure. The

literature from WHO, USEPA (i.e. IRIS), ATSDR and CalEPA (i.e. OEHHA) have been reviewed to establish the threshold levels of exposure for the TAP. **Table 17.2.23** summarises the basis of risk values from the different guidelines.

Table 17.2.23: Basis of risk values in different guidelines

Risk value	Description
WHO non-carcinogenic chemicals guideline (WHO)	The air quality guidelines for non-carcinogenic pollutants can only be applied if the averaging times are specified. The averaging time associated with a guideline value depends on the type of effects that are caused by short-term exposure producing acute effects, or long term exposure producing chronic effects. Typical averaging times are 24 hours for acute exposure and one year for chronic health effects.
USEPA non-cancer risk value (USEPA-IRIS; Scorecard)	The non-cancer risk values from the USEPA are reference doses or concentrations and are estimates of the daily exposure to the human population (including subgroups) that is likely without an appreciable risk of deleterious effects over a lifetime.
Minimum risk level (US – ATSDR)	ATSDR developed minimum risk level. It is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciating risk of adverse, non-cancer health effect over a specified duration of period. According to its definition, acute-duration means exposure less than 14 days. Chronic duration means exposure longer than 1 year.
Acute reference exposure levels (CEPA-OEHHA)	OEHHA developed acute REL for assessing potential non-cancer health impacts for short-term, generally one-hour peak exposures to facility emissions. By definition, an acute REL is an exposure that is unlikely to cause adverse health effects in a human population, including sensitive subgroups, exposed to that concentration for the specified exposure duration on an intermittent basis.
Chronic reference exposure levels (CEPA – OEHHA)	OEHHA developed chronic RfC for assessing non-cancer health impact from long term exposure. A chronic RfC is a concentration level at or below which no adverse health effects are anticipated due to long term exposure. Long term exposure for these purposes has been defined as 12% of a lifetime, or 8.4 years for humans.

17.2.5.4 For non-carcinogenic health risk from chronic inhalation exposure, the potential health risk can be evaluated by comparing the chemical-specific annual average concentration (EC_c) of the TAP with the relevant Reference Concentrations (RfC):

If $EC_c \leq RfC$, adverse chronic non-cancer health effects are not anticipated.

17.2.5.5 The acute inhalation health impact can also be evaluated by comparing the chemical-specific short-term maximum concentration (EC_{ST}) of the TAP with acute reference dose-response value (AV):

If $EC_{ST} \leq AV$, adverse acute health effects are not anticipated.

17.2.5.6 The incremental carcinogenic risk due to the increase of TAP concentrations arising from the project has also been evaluated.

Carcinogenic Health Risk of TAP

17.2.5.7 The carcinogenic health risk is measured as the increase in the number of cases of cancer per million population that is attributable to a TAP. The inhalation dose-response estimate is usually assumed to be linear with no threshold, and the risk is expressed as a "unit risk", defined as the risk of developing cancer if a person is continuously exposed to a unit concentration (usually presented as one $\mu g/m^3$) for a life time of 70 years. The international unit risk factors are usually derived for 70 years. Hence, a lifetime of 70 years is the basis for the assessment of potential long term effect from exposure to TAP on health for carcinogenic health risk.

17.2.5.8 The following literature have been reviewed to establish the carcinogenic classification and unit risk for cancer:

Table 17.2.24: Summary of key literature to establish the carcinogenic classification and unit risk for cancer

Purpose	Key Literature
Carcinogenic classification	• WHO • USEPA – IRIS • CEPA –OEHHA • California Air Resources (CARB) - California Air Toxic Programme • IARC
Unit risk for cancer	• WHO – Air Quality Guideline • USEPA – IRIS • CEPA – OEHHA

Hence,

$$\text{Cancer Risk}_i = \Delta EC_L \times IUR$$

Where:

ΔEC_L = estimate of incremental long-term inhalation exposure concentration (i.e. concentrations due to the Project less concentrations due to the "business as usual" scenario under the existing 2RS) for a specific TAP;

IUR = the corresponding inhalation unit risk estimate for that TAP

Characterization of Cancer Risk from Exposure to Multiple Pollutants is summarised as follows:

Total Incremental Cancer $Risk_T$ = Incremental Cancer $Risk_1$ + Incremental Cancer $Risk_2$ + ... + Incremental Cancer Risk.

17.2.5.9 The USEPA has established relevant criteria for human health impact assessment and for cancer risk, the USEPA focuses on evaluation of incremental cancer risk for an individual potentially exposed to one or more TAP. As explained in Section 27.4 of its publication "Air Toxics Risk Assessment Reference Library – Volume 1 – Technical Resources Manual (EPA-453-K-04-001A) 2004", in protecting public health with an ample margin of safety, the USEPA strives to provide maximum feasible protection against risks to human from TAP by (1) protecting the greatest number of persons possible to an individual lifetime risk level of no higher than 1×10^{-6} and (2) limiting to no higher than approximately 1×10^{-4} the estimated risk that a person living near a source would have if exposed to the maximum pollutant concentrations for 70 years.

17.2.5.10 According to Technical Manual 1003 - Guidance on Risk Assessment for Air Contaminant Emissions (New Jersey Department of Environmental Protection, 2009), the focus of required human health risk assessment shall be the incremental inhalation risk from exposure to air toxics. Existing risks of cancer associated with smoking, occupational or domestic exposures, dietary habits, inherited traits, or other factors that may contribute to cancer are not required to be evaluated; nor does it requires consideration of risks from other nearby air toxics sources or existing levels of toxics in the ambient air. **Table 17.2.25** summarises the cancer risk guidelines.

Table17.2.25: Cancer risk guidelines

Risk value	Description
Cancer risks less than or equal to one in a million ($1x10^{-6}$)	Negligible
Cancer risks that fall in-between 1×10^{-4} and 1×10^{-6}	Considered by the DAQ Risk Management Committee on a case-by-case basis. Sources with risk falling within this range must take steps to minimize the projected risk before a Pre-Construction Permit can be issued.
Cancer risks greater than or equal to one in ten thousand ($1x10^{-4}$)	Unacceptable

17.2.5.11 For the purpose of the current HIA which involves calculation of incremental cancer risks that represent upper-bound predictions of exposure at representative human receptors, the criterion of limiting to no higher than approximately $1x10^{-4}$ the estimated risk for an individual living near a source adopted by the USEPA is considered relevant and has been adopted as the benchmark for evaluating carcinogenic health risk from TAP associated with the operation of the project.

17.2.5.12 **Table 17.2.26** lists the toxicity criteria of these TAP evaluated in the human health risk assessment.

Table 17.2.26: Toxicity criteria of the acute, carcinogenic and chronic non-carcinogenic risks of the identified TAP

TAP	IUR		RfC		AV	
	(per µg/m^3)	Source [1]	(µg/m^3)	Source [1]	(µg/m^3)	Source [1]
1,3-butadiene	3.00E-05	IRIS	2	IRIS	660 (1-hr)	OEHHA
Acetaldehyde	2.20E-06	IRIS	9	IRIS	470 (1-hr)	OEHHA
Acrolein			0.35[2]	WHO	7 (daily)	ATSDR
Arsenic	1.50E-03	WHO	0.015	OEHHA	0.2 (1-hr)	OEHHA
Benz(a)anthracene	1.10E-04	OEHHA				
Benzene	6.00E-06	WHO	30	IRIS	29 (daily)	ATSDR
Benzo(a)pyrene	8.70E-02	WHO				
Benzo(bk)fluoranthene	1.10E-04	OEHHA				
Beryllium	2.40E-03	IRIS	0.02	WHO		
Cadmium	1.80E-03	IRIS	0.01	ATSDR	0.03 (daily)	ATSDR
Chromium VI	4.00E-02	WHO	0.1	IRIS	0.3 (daily)[6]	ATSDR
Chrysene	1.10E-05	OEHHA				
Copper			2.4	OEHHA	100 (1-hr)	OEHHA
Dibenz(a,h)anthracene	1.20E-03	OEHHA				
Diesel Particulate Matter	3.00E-04	OEHHA	5	IRIS [3]		

TAP	IUR		RfC		AV	
	(per µg/m^3)	Source [1]	(µg/m^3)	Source [1]	(µg/m^3)	Source [1]
Ethylbenzene	2.50E-06	OEHHA	1,000	IRIS	21,700 (daily)	ATSDR
Formaldehyde	1.30E-05	IRIS	100 [4]	WHO	100 (30-min)	WHO
Indeno(1,2,3-cd)pyrene	1.10E-04	OEHHA				
Isopropylbenzene (cumene)			400	IRIS		
Lead	1.20E-05	OEHHA	0.5	WHO		
Manganese			0.150	WHO		
Methyl alcohol (Methanol)			20,000	IRIS	2,8000 (1-hr)	OEHHA
Xylene			100	IRIS	8,820 (daily)	ATSDR
Naphthalene	3.40E-05	OEHAA	10	WHO		
n-Hexane			700	IRIS		
Nickel	3.80E-04	WHO	0.09	ATSDR	0.2 (daily)	ATSDR
Phenol (carbolic acid)			200	OEHHA	5,800 (1-hr)	OEHHA
Propionaldehyde			8	IRIS		
Styrene			1,000	IRIS	21,630 [5] (daily)	ATSDR
TCDD	3.80E+01	OEHHA				
Toluene			5,000	IRIS	3,750 (daily)	ASTDR

Notes:
[1] The hierarchy in selecting information source is WHO > USEPA – IRIS > US – ATSDR > CARB-OEHAA.
[2] Concise International Chemical Assessment Document 43, WHO 2002. This value was further supported by the updated RfC values (as of Oct 2013) from OEHHA (http://oehha.org/air/allrels.html).
[3] Diesel Particulate Matter (DPM) is the particulate component of diesel exhaust.
[4] According to WHO guidelines for indoor air quality: Selected pollutants, 2010, this short-term guideline would also prevent effects on lung function as well as long-term health effects, including nasopharyngeal cancer and myeloid leukaemia. Hence, it was adopted as reference concentration.
[5] According to WHO Air Quality Guidelines for Europe, 2000, the air quality guideline for styrene was based on odour detection threshold level of 70 µg/m^3 (30-minute average). Hence, it is not selected as acute risk level.
[6] Based on intermediate inhalation minimal risk level on particulates phase.

Health Risk of Criteria Pollutants

Short-Term Health Effect

17.2.5.13 For the short-term health effects of CO, SO_2 and NO_2, the highest 1-hr CO, highest 10-min SO_2 and 19th highest 1-hr NO_2 have been compared with the respective AQO, which were derived from the WHO Air Quality Guidelines (AQG).

17.2.5.14 The short-term mortalities and morbidities associated with the exposure to SO_2, NO_2 and PM_{10} have been determined. Data from local and international epidemiological studies on short-term

relative risks of cardiovascular and respiratory diseases have been collected through the literature review (**Appendix 17.2.4**). It is considered that whenever local data are available, their use would be preferred for the assessment of health effects. Hence, available data from a recent comprehensive study on short-term health effects of air pollutants (Wong, CM *et al.*, 2010, the PAPA Study) was adopted in the health risk assessment. **Table 17.2.27** summarises the excessive risk of mortalities and morbidities attributable to a 10 $\mu g/m^3$ increase in air pollutant concentration in Hong Kong locally. Available data on NO_2, PM_{10} and SO_2 were adopted for short-term mortalities and morbidities calculations.

Table 17.2.27: Percentage of excess risk (95% of confidence interval) of short-term mortalities and morbidities attributable to a 10 $\mu g/m^3$ increase in air pollutant concentrations (for all ages)

Air Pollutant	All-cause Mortality	Cardio-vascular Mortality	Respiratory Mortality	Cardio-vascular Diseases	Respiratory Diseases
Hong Kong [1]					
NO_2	1.03 (0.69-1.37)	1.38 (0.75-2.01)	1.41 (0.67-2.15)	1.00 (0.73-1.26)	0.75 (0.50 - 1.00)
PM_{10}	0.51 (0.23-0.80)	0.63 (0.11-1.16)	0.69 (0.08-1.31)	0.58 (0.36-0.80)	0.60 (0.40-0.80)
SO_2	0.91 (0.40-1.42)	1.23 (0.27-2.21)	1.31 (0.21-2.43)	0.98 (0.53-1.39)	0.13 (-0.24-0.50)

Note: [1] Wong, C.M., *et al.*, 2010.

Long-Term Health Effect

17.2.5.15 To assess the long-term health risk from exposure to SO_2, NO_2 and PM_{10} / $PM_{2.5}$, data from local and international epidemiological studies on long-term relative risks of cardiovascular and respiratory diseases have been collected through the literature review (**Appendix 17.2.4**). The RR for long-term mortality (i.e. $PM_{2.5}$) conducted by Pope et al. (2002) are commonly adopted by other countries (and WHO in deriving the AQG) and are therefore proposed for use as the best available data for the heath risk assessment. **Table 17.2.28** summarises the excessive risk of long-term mortalities attributable to a 10 $\mu g/m^3$ increase in air pollutant concentrations.

Table 17.2.28: Percentage of excess risk (95% of confidence interval) of long-term mortalities attributable to air pollutants

Air Pollutant	All-cause Mortality	Cardiopulmonary Mortality	Lung Cancer Mortality
NO_2	Effects cannot be separated from PM_{10} or $PM_{2.5}$ effects [2]		
PM_{10} [3] [4]	5 (Not Statistically Significant)	16.3 (Not Statistically Significant)	28.5 (Not Statistically Significant)
$PM_{2.5}$ [5]	4 (1-8)[6]	6 (2-10)[6]	8 (1-16)[6]
SO_2	WHO recommends a 24 hr AQG of 20 $\mu g/m^3$. No annual AQG is recommended.		

Notes:

[1] % excess risks are expressed as per 10 $\mu g/m^3$ increase in air pollutant concentrations.

[2] It is difficult to separate the long-term effects of NO_2 from PM and other traffic generated fumes. WHO maintains a long-term Air Quality Guideline of 40 $\mu g/m^3$.

[3] McDonnell WF et al. Relationships of mortality with the fine and coarse fractions of long-term ambient PM_{10} concentrations in nonsmokers. Journal of Exposure Analysis and Environmental Epidemiology 2000;10:427-436;

[4] Evidence for a separate RR of mortality for long-term exposure to PM_{10} is insufficient, but RRs for short-term exposure are well-documented.

[5] ACS study by Pope, A.C., et al., Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution, Journal of the American Medical Association, 2002.

[6] Mean value was adopted as the excess risk factor for health impact assessment according to international practices.

17.2.5.16 For lead (Pb) which has also been identified as a TAP, the approach in assessing potential health effects from chronic exposure was similar to that adopted for the other identified TAP, i.e., the chronic carcinogenic and/or non-carcinogenic health risks were evaluated by using available IUR and RfC that are presented in **Table 17.2.28**. It can be noted that the annual concentration limit adopted for lead in the AQO is the same as the adopted RfC, which is based on the reference value from WHO.

17.2.5.17 Projected incremental changes in the concentrations of these key criteria pollutants were estimated from a comparison of the "with-project" and "without-project" scenarios. Population data within 5 km assessment area in Year 2031 was based on the Territorial Population and Employment Data Matrices (TPEDM) 2009. The affected population in Tung Chung East and West Development was based on Theme 1 in the Stage 2 Public consultation digest released in May 2013. Together with the projected changes in air pollutant concentrations in the neighbouring districts, the 'population attributable risk' with respect to the increases in mortality and morbidity for cardiovascular and respiratory diseases among the populations in the nearby human receptors were estimated. These estimates reflect the potential health impact on human associated with the future operation of the project on the nearby human receptors.

17.2.5.18 The methods for assessing the increased premature deaths and illnesses attributed to air pollution, which have been similarly adopted by the European Community in their health impact assessment studies, are summarised below:

- Attributable proportion (AP) = [(RRc – 1) x Pc] / [RRc x Pc]

  Where RRc = relative risk in category c of exposure;

  Pc = percentage of population in category c of exposure;

- I_e (Incidence or number of cases attributable to exposure per 10 µg/m^3 increase in pollutants) = I x AP;

  Where I = frequency of outcome in the current HK population (e.g. Numbers of in-patient discharges and deaths in hospitals for cardiovascular or respiratory disease)

- Incidence or mortality rate attributable to air pollution = I_e / current HK population

- No. of hospital admissions or premature deaths attributed to air pollution in representative human receptors in the future = Future population in representative human receptors x I_e / current HK population x 0.1 x incremental change in the concentration of criteria pollutants (i.e. the difference between the concentration of air pollutants in 3RS and 2RS)

17.2.5.19 Summary tables of hospital illnesses and mortality health outcomes are provided in **Tables 17.2.29** and **17.2.30** respectively.

Table 17.2.29: Summary of parameters for hospital illnesses health outcome

Health Outcome		RR (per 10µg/m^3)	AP (per 10µg/m^3)	I [5]	Ie [1] (per 10µg/m^3)
Short-term hospital illnesses effects of NO_2	Cardiovascular [2] & [3]	1.0100	0.00990	155,299	1,537.6
	Respiratory [2] & [4]	1.0075	0.00744	169,071	1,258.6
Short-term hospital illnesses effects of RSP	Cardiovascular [2] & [3]	1.0058	0.00577	155,299	895.5
	Respiratory [2] & [4]	1.0060	0.00596	169,071	1,008.4
Short-term hospital illnesses effects of SO_2	Cardiovascular [2] & [3]	1.0098	0.00970	155,299	1,507.2
	Respiratory [2] & [4]	1.0013	0.00130	169,071	219.5

Notes:

[1] Ie = Total population in HK in mid-2012 = 7,154,600 (Census and Statistics Department).

[2] Wong C.M. et al, 2010.

[3] In HK in 2012, numbers of in-patient discharges of cardiovascular diseases in hospital (ICD10: I00-I99) = 155299 (Department of Health: http://www.dh.gov.hk/english/pub_rec/pub_rec_ar/pdf/1213/supplementary_table2012.pdf).

[4] In HK in 2012, numbers of in-patient discharges of respiratory diseases in hospital (ICD10: J00-J99) = 169071 (Department of Health: http://www.dh.gov.hk/english/pub_rec/pub_rec_ar/pdf/1213/supplementary_table2012.pdf).

[5] Numbers of in-patient discharges in hospitals for cardiovascular or respiratory disease.

Table 17.2.30: Summary of parameters for premature death mortality health outcome

Health Outcome		RR (per 10µg/m^3)	AP (per 10µg/m^3)	I[7]	Ie [1] (per 10µg/m^3)
Long-term mortality effects of FSP	All-causes mortality [2] & [3]	1.0400	0.03846	42,017	1,616.0
	Cardiopulmonary [2] & [4]	1.0600	0.05660	19,952	1,129.4
	Malignant Neoplasm of Trachea, Bronchus and Lung [2] & [5]	1.0800	0.07407	3,893	288.4
Short-term mortality effects of NO_2	All-causes mortality [3] & [4]	1.0103	0.01019	42,017	428.4
	Cardiovascular [4] & [6]	1.0138	0.01361	10,320	140.5
	Respiratory [4] & [6]	1.0141	0.01390	9,632	133.9
Short-term mortality effects of RSP	All-causes mortality [3] & [4]	1.0051	0.00507	42,017	213.2
	Cardiovascular [4] & [6]	1.0063	0.00626	10,320	64.6
	Respiratory [4] & [6]	1.0069	0.00685	9,632	66.0
Short-term mortality effects of SO_2	All-causes mortality [3] & [4]	1.0091	0.00902	42,017	378.9
	Cardiovascular [4] & [6]	1.0123	0.01215	10,320	125.4
	Respiratory [4] & [6]	1.0131	0.01293	9632	124.5

Notes:

[1] Total population in HK in 2012 = 7,154,600 (Census and Statistics Department).

[2] ACS study by Pope et al, 2002.

[3] In 2012, Numbers of deaths in hospital for all-causes in HK = 43672; numbers of deaths in hospital from external causes of morbidity and mortality = 1655. Hence, total numbers of natural deaths in hospital = 43672 - 1655 = 42017. (Department of Health: http://www.dh.gov.hk/english/pub_rec/pub_rec_ar/pdf/1213/supplementary_table2012.pdf)

[4] In 2012, numbers of deaths in hospital from cardiovascular diseases (ICD10: I00-I99) in HK = 10320; numbers of deaths in hospital from respiratory diseases (ICD10: J00-J99) = 9632. Total cardiopulmonary deaths in HK in 2012= 10320 + 9632 = 19952.

[5] Numbers of deaths in hospital from malignant neoplasm of trachea, bronchus and lung in HK in 2012 (ICD10: C33-C34) = 3893 (Department of Health: http://www.dh.gov.hk/english/pub_rec/pub_rec_ar/pdf/1213/supplementary_table2012.pdf)

[6] Wong C.M. et al, 2010.

[7] Numbers of deaths in hospitals for cardiovascular or respiratory disease.

17.2.6 Risk Characterisation

17.2.6.1 The risk characterisation step combines the results of both the exposure assessment and the dose-response assessment to estimate the potential health risks.

Toxic Air Pollutants

Chronic Non-carcinogenic Health Risk

17.2.6.2 The annual average TAP concentrations for 3RS and 2RS, and their incremental change in concentrations arising from the aircraft and related activities plus the background contribution at different human receptors, are listed in **Appendix 17.2.5**. The maximum predicted annual average air concentrations for 3RS in different areas are extracted and summarised in **Table 17.2.31.** The maximum incremental change (3RS – 2RS) of annual average air concentrations in different areas are summarised in **Table 17.2.32**.

17.2.6.3 Based on the annual-average TAP concentrations for 3RS presented in **Table 17.2.31**, it can be noted that the predicted TAP concentrations at all human receptors would comply with all chronic non-carcinogenic criteria. Therefore, the exposure of human receptors to the TAP is not anticipated to cause an adverse chronic non-carcinogenic health effects.

Table 17.2.31: Maximum predicted cumulative annual average TAP concentrations for 3RS (μg/m^3)

Major Area	Criteria	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung
1,3-butadiene	2	3.35E-01 (Yes)	4.06E-01 (Yes)	3.54E-01 (Yes)	3.70E-01 (Yes)	3.35E-01 (Yes)	3.41E-01 (Yes)
Acetaldehyde	9	1.52E+00 (Yes)	1.72E+00 (Yes)	1.58E+00 (Yes)	1.62E+00 (Yes)	1.52E+00 (Yes)	1.54E+00 (Yes)
Acrolein	0.35	2.37E-02 (Yes)	1.14E-01 (Yes)	4.79E-02 (Yes)	6.56E-02 (Yes)	2.39E-02 (Yes)	2.95E-02 (Yes)
Benzene	30	1.51E+00 (Yes)	1.51E+00 (Yes)	1.44E+00 (Yes)	1.47E+00 (Yes)	1.41E+00 (Yes)	1.50E+00 (Yes)
Ethylbenzene	1000	1.70E+00 (Yes)	1.71E+00 (Yes)	1.71E+00 (Yes)	1.71E+00 (Yes)	1.71E+00 (Yes)	1.71E+00 (Yes)
Formaldehyde	100	3.69E+00 (Yes)	4.26E+00 (Yes)	3.85E+00 (Yes)	3.98E+00 (Yes)	3.70E+00 (Yes)	3.74E+00 (Yes)
Isopropylbenzene (cumene)	400	1.18E-01 (Yes)	1.19E-01 (Yes)	1.18E-01 (Yes)	1.18E-01 (Yes)	1.18E-01 (Yes)	1.18E-01 (Yes)
Methyl alcohol (Methanol) [1]	20000	9.44E-03 (Yes)	7.09E-02 (Yes)	2.58E-02 (Yes)	3.74E-02 (Yes)	9.43E-03 (Yes)	1.30E-02 (Yes)
Xylene (Total)	100	4.98E+00 (Yes)	5.00E+00 (Yes)	4.99E+00 (Yes)	4.99E+00 (Yes)	4.98E+00 (Yes)	4.98E+00 (Yes)
n-Hexane	700	1.37E+00 (Yes)	1.39E+00 (Yes)	1.37E+00 (Yes)	1.42E+00 (Yes)	1.37E+00 (Yes)	1.40E+00 (Yes)
Naphthalene	10	7.65E-01 (Yes)	7.99E-01 (Yes)	7.74E-01 (Yes)	7.86E-01 (Yes)	7.65E-01 (Yes)	7.85E-01 (Yes)
Phenol (carbolic acid) [1]	200	3.80E-03 (Yes)	2.85E-02 (Yes)	1.04E-02 (Yes)	1.51E-02 (Yes)	3.79E-03 (Yes)	5.24E-03 (Yes)
Propionaldehyde	8	1.79E-01 (Yes)	2.06E-01 (Yes)	1.86E-01 (Yes)	1.92E-01 (Yes)	1.79E-01 (Yes)	1.81E-01 (Yes)
Styrene	1000	2.13E-01	2.23E-01	2.16E-01	2.17E-01	2.13E-01	2.13E-01

Major Area	Criteria	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung
		(Yes)	(Yes)	(Yes)	(Yes)	(Yes)	(Yes)
Toluene	5000	6.64E+00 (Yes)	6.70E+00 (Yes)	6.66E+00 (Yes)	6.67E+00 (Yes)	6.64E+00 (Yes)	6.65E+00 (Yes)
Arsenic	0.015	5.00E-03 (Yes)	5.02E-03 (Yes)	5.01E-03 (Yes)	5.01E-03 (Yes)	5.00E-03 (Yes)	5.00E-03 (Yes)
Beryllium	0.02	4.02E-05 (Yes)	4.12E-05 (Yes)	4.04E-05 (Yes)	4.12E-05 (Yes)	4.02E-05 (Yes)	4.05E-05 (Yes)
Cadmium	0.01	1.32E-03 (Yes)	1.32E-03 (Yes)	1.32E-03 (Yes)	1.32E-03 (Yes)	1.32E-03 (Yes)	1.32E-03 (Yes)
Chromium VI	0.1	2.61E-03 (Yes)	2.74E-03 (Yes)	2.64E-03 (Yes)	2.65E-03 (Yes)	2.61E-03 (Yes)	2.61E-03 (Yes)
Copper	2.4	6.07E-02 (Yes)	6.23E-02 (Yes)	6.11E-02 (Yes)	6.12E-02 (Yes)	6.07E-02 (Yes)	6.07E-02 (Yes)
Diesel Particulate Matters	5	2.66E+00 (Yes)	3.17E+00 (Yes)	2.74E+00 (Yes)	2.93E+00 (Yes)	2.62E+00 (Yes)	3.03E+00 (Yes)
Lead	0.5	5.00E-02 (Yes)	5.02E-02 (Yes)	5.01E-02 (Yes)	5.01E-02 (Yes)	5.00E-02 (Yes)	5.00E-02 (Yes)
Manganese	0.15	2.06E-02 (Yes)	2.09E-02 (Yes)	2.07E-02 (Yes)	2.07E-02 (Yes)	2.06E-02 (Yes)	2.06E-02 (Yes)
Nickel	0.09	4.76E-03 (Yes)	4.92E-03 (Yes)	4.80E-03 (Yes)	4.82E-03 (Yes)	4.76E-03 (Yes)	4.77E-03 (Yes)

Notes:

[1] Background concentration in EPD's Tung Chung Air Quality Monitoring Station and Central Western Air Quality Monitoring Station is not available.

[2] For predicted annual average TAP concentration, "Yes" refers to its compliance with criteria and "No" refers to its non-compliance with criteria.

[3] Compliance against the criteria is shown in the ( ).

Table 17.2.32: Maximum incremental annual average TAP concentrations ($\mu g/m^3$)

Major Area	Criteria	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung
1,3-butadiene	2	3.27E-03 (1.0%)	1.22E-02 (3.1%)	6.91E-03 (2.0%)	4.98E-03 (1.4%)	3.14E-03 (0.9%)	3.49E-03 (1.0%)
Acetaldehyde	9	8.77E-03 (0.6%)	3.17E-02 (1.9%)	1.83E-02 (1.2%)	1.28E-02 (0.8%)	8.34E-03 (0.6%)	9.39E-03 (0.6%)
Acrolein	0.35	4.45E-03 (23.2%)	1.73E-02 (18.0%)	9.52E-03 (24.8%)	6.95E-03 (11.8%)	4.35E-03 (22.3%)	4.65E-03 (18.8%)
Benzene	30	3.68E-03 (0.3%)	1.33E-02 (0.9%)	7.70E-03 (0.5%)	5.69E-03 (0.4%)	3.37E-03 (0.2%)	4.80E-03 (0.3%)
Ethylbenzene	1000	3.59E-04 (0.0%)	1.49E-03 (0.1%)	8.45E-04 (0.0%)	6.07E-04 (0.0%)	3.85E-04 (0.0%)	3.76E-04 (0.0%)
Formaldehyde	100	2.52E-02 (0.7%)	9.13E-02 (2.2%)	5.27E-02 (1.4%)	3.70E-02 (0.9%)	2.40E-02 (0.7%)	2.69E-02 (0.7%)
Isopropylbenzene (cumene)	400	7.37E-06 (0.0%)	3.16E-05 (0.0%)	1.86E-05 (0.0%)	1.30E-05 (0.0%)	8.42E-06 (0.0%)	7.80E-06 (0.0%)
Methyl alcohol (Methanol)	20000	3.16E-03 (50.4%)	1.26E-02 (21.7%)	6.84E-03 (36.0%)	5.04E-03 (15.6%)	3.13E-03 (49.7%)	3.28E-03 (33.7%)
Xylene (Total)	100	1.04E-03 (0.0%)	4.41E-03 (0.1%)	2.54E-03 (0.1%)	1.81E-03 (0.0%)	1.16E-03 (0.0%)	1.10E-03 (0.0%)
n-Hexane	700	7.18E-04	3.72E-03	1.19E-03	7.59E-03	4.13E-04	5.00E-03

Major Area	Criteria	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung
		(0.1%)	(0.3%)	(0.1%)	(0.5%)	(0.0%)	(0.4%)
Naphthalene	10	1.21E-03 (0. 2%)	4.50E-03 (0.6%)	2.52E-03 (0.3%)	1.99E-03 (0.3%)	1.14E-03 (0.1%)	2.46E-03 (0. 3%)
Phenol (carbolic acid)	200	1.27E-03 (50.4%)	5.08E-03 (21.7%)	2.75E-03 (36.0%)	2.03E-03 (15.6%)	1.26E-03 (49.7%)	1.32E-03 (33.7%)
Propionaldehyde	8	1.34E-03 (0.8%)	5.25E-03 (2.6%)	2.89E-03 (1.6%)	2.02E-03 (1.1%)	1.34E-03 (0.8%)	1.37E-03 (0.8%)
Styrene	1000	5.23E-04 (0.2%)	2.09E-03 (0.9%)	1.13E-03 (0.5%)	8.33E-04 (0.4%)	5.18E-04 (0.2%)	5.43E-04 (0.3%)
Toluene	5000	2.46E-03 (0.0%)	1.11E-02 (0.2%)	6.46E-03 (0.1%)	6.38E-03 (0.1%)	2.88E-03 (0.0%)	4.03E-03 (0.1%)
Arsenic	0.015	7.03E-07 (0.0%)	5.05E-07 (0.0%)	1.13E-06 (0.0%)	7.52E-07 (0.0%)	7.50E-07 (0.0%)	7.02E-07 (0.0%)
Beryllium	0.02	6.07E-08 (0.2%)	1.88E-07 (0.5%)	1.23E-07 (0.3%)	2.38E-07 (0.6%)	5.37E-08 (0.1%)	8.53E-08 (0.2%)
Cadmium	0.01	6.07E-08 (0.0%)	1.88E-07 (0.0%)	1.23E-07 (0.0%)	2.38E-07 (0.0%)	5.37E-08 (0.0%)	8.53E-08 (0.0%)
Chromium VI	0.1	2.17E-06 (0.1%)	2.73E-05 (1.0%)	2.90E-06 (0.1%)	8.00E-06 (0.3%)	2.11E-06 (0.1%)	4.38E-06 (0.2%)
Copper	2.4	2.79E-05 (0.0%)	3.42E-04 (0.6%)	3.70E-05 (0.1%)	9.98E-05 (0.2%)	2.73E-05 (0.0%)	5.53E-05 (0.1%)
Diesel Particulate Matters	5	7.36E-03 (0.3%)	2.86E-02 (1.0%)	1.63E-02 (0.6%)	2.95E-02 (1.0%)	5.99E-03 (0.2%)	2.32E-02 (0.8%)
Lead	0.5	3.07E-06 (0.0%)	3.61E-05 (0.1%)	4.19E-06 (0.0%)	1.09E-05 (0.0%)	2.98E-06 (0.0%)	5.93E-06 (0.0%)
Manganese	0.15	4.86E-06 (0.0%)	5.51E-05 (0.3%)	6.58E-06 (0.0%)	1.66E-05 (0.1%)	4.78E-06 (0.0%)	9.15E-06 (0.0%)
Nickel	0.09	2.70E-06 (0.1%)	3.27E-05 (0.7%)	3.61E-06 (0.1%)	9.65E-06 (0.2%)	2.63E-06 (0.1%)	5.31E-06 (0.1%)

Note:

[1] Incremental percentage changes are listed in the ( ).

Acute Health Risk

17.2.6.4 In addition to the potential long-term risk to human health due to TAP emitted from the aircraft and related activities, short-term or acute risk has been evaluated for direct inhalation of TAP. Acute exposure has been estimated, based on maximum 1-hr / 24-hr average air concentrations predicted from the atmospheric dispersion modelling. To determine the likelihood of adverse acute effects, the maximum predicted 1-hr / 24-hr average air concentrations are compared with the criteria for short-term inhalation exposures.

17.2.6.5 The maximum 1-hr / 24-hr average TAP concentrations for 3RS and 2RS, and their incremental change in concentrations arising from the aircraft and related activities plus the background contribution at different human receptors, are listed in **Appendix 17.2.5**. The maximum predicted 1-hr/ 24-hr average TAP concentrations for 3RS in different areas are extracted and summarised in **Table 17.2.33.** The maximum incremental change of 1-hr/ 24-hr average TAP concentrations in different areas are extracted and summarised in **Table 17.2.34.**

17.2.6.6 Based on the maximum predicted 1-hr / 24-hr average air concentrations for 3RS presented in **Table 17.2.33**, it can be noted that the predicted maximum TAP concentrations at all HSRs would comply with all acute criteria.

Table 17.2.33: Maximum predicted cumulative 1-hr / 24-hr average TAP concentrations for 3RS (µg/m^3)

Major Area	Criteria	Airport Island	BCF	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung	Tuen Mun
1,3-butadiene	660[1]	4.03E+00 (Yes)	2.81E+00 (Yes)	2.23E+00 (Yes)	3.71E+00 (Yes)	2.90E+00 (Yes)	3.40E+00 (Yes)	2.53E+00 (Yes)	3.19E+00 (Yes)	2.32E+00 (Yes)
Acetaldehyde	470[1]	1.18E+01 (Yes)	8.63E+00 (Yes)	6.84E+00 (Yes)	1.07E+01 (Yes)	8.56E+00 (Yes)	9.97E+00 (Yes)	7.59E+00 (Yes)	9.33E+00 (Yes)	7.04E+00 (Yes)
Acrolein	7[2]	1.10E+00 (Yes)	8.86E-01 (Yes)	2.47E-01 (Yes)	8.04E-01 (Yes)	4.31E-01 (Yes)	8.05E-01 (Yes)	2.81E-01 (Yes)	4.04E-01 (Yes)	3.78E-01 (Yes)
Benzene	29[2]	3.14E+00 (Yes)	3.35E+00 (Yes)	4.40E+00 (Yes)	2.92E+00 (Yes)	2.59E+00 (Yes)	2.93E+00 (Yes)	2.47E+00 (Yes)	2.93E+00 (Yes)	2.52E+00 (Yes)
Ethylbenzene	21700[2]	4.64E+00 (Yes)	4.63E+00 (Yes)	4.59E+00 (Yes)	4.63E+00 (Yes)	4.61E+00 (Yes)	4.62E+00 (Yes)	4.59E+00 (Yes)	4.60E+00 (Yes)	4.60E+00 (Yes)
Formaldehyde	100[3] & [6]	4.51E+01 (Yes)	3.17E+01 (Yes)	2.48E+01 (Yes)	4.05E+01 (Yes)	3.18E+01 (Yes)	3.75E+01 (Yes)	2.79E+01 (Yes)	3.50E+01 (Yes)	2.57E+01 (Yes)
Methyl alcohol (Methanol)	28000[1] & [4]	3.22E+00 (Yes)	1.87E+00 (Yes)	1.42E+00 (Yes)	2.97E+00 (Yes)	2.13E+00 (Yes)	2.62E+00 (Yes)	1.75E+00 (Yes)	2.43E+00 (Yes)	1.54E+00 (Yes)
Xylene (Total)	8820[2]	1.31E+01 (Yes)	1.31E+01 (Yes)	1.30E+01 (Yes)	1.31E+01 (Yes)	1.31E+01 (Yes)	1.31E+01 (Yes)	1.30E+01 (Yes)	1.30E+01 (Yes)	1.30E+01 (Yes)
Phenol (carbolic acid)	5800[1] & [4]	1.30E+00 (Yes)	7.54E-01 (Yes)	5.70E-01 (Yes)	1.19E+00 (Yes)	8.58E-01 (Yes)	1.05E+00 (Yes)	7.03E-01 (Yes)	9.78E-01 (Yes)	6.19E-01 (Yes)
Styrene	21630[2]	1.43E+00 (Yes)	1.40E+00 (Yes)	1.33E+00 (Yes)	1.39E+00 (Yes)	1.35E+00 (Yes)	1.39E+00 (Yes)	1.33E+00 (Yes)	1.35E+00 (Yes)	1.34E+00 (Yes)
Toluene	3750[2]	1.49E+01 (Yes)	1.49E+01 (Yes)	1.47E+01 (Yes)	1.53E+01 (Yes)	1.49E+01 (Yes)	1.48E+01 (Yes)	1.48E+01 (Yes)	1.48E+01 (Yes)	1.65E+01 (Yes)
Arsenic	0.2[1]	2.21E-02 (Yes)	2.15E-02 (Yes)	2.14E-02 (Yes)	2.19E-02 (Yes)	2.16E-02 (Yes)	2.16E-02 (Yes)	2.15E-02 (Yes)	2.16E-02 (Yes)	8.58E-02 (Yes)
Cadmium	0.03[2]	6.21E-03 (Yes)	6.20E-03 (Yes)	6.21E-03 (Yes)	6.21E-03 (Yes)	6.20E-03 (Yes)	6.21E-03 (Yes)	6.20E-03 (Yes)	6.22E-03 (Yes)	1.87E-02 (Yes)
Chromium VI	0.3[2]	8.55E-03 (Yes)	8.72E-03 (Yes)	8.21E-03 (Yes)	9.13E-03 (Yes)	8.49E-03 (Yes)	8.82E-03 (Yes)	8.19E-03 (Yes)	8.30E-03 (Yes)	2.03E-02 (Yes)
Copper	100[1]	2.62E-01 (Yes)	2.06E-01 (Yes)	1.64E-01 (Yes)	2.17E-01 (Yes)	1.73E-01 (Yes)	1.84E-01 (Yes)	1.65E-01 (Yes)	1.90E-01 (Yes)	2.17E-01 (Yes)
Nickel	0.2[2]	1.49E-02 (Yes)	1.51E-02 (Yes)	1.45E-02 (Yes)	1.56E-02 (Yes)	1.48E-02 (Yes)	1.53E-02 (Yes)	1.45E-02 (Yes)	1.46E-02 (Yes)	2.65E-02 (Yes)

Notes:

[1] Based on hourly average concentration.

[2] Based on daily average concentration.

[3] Based on 30-min average concentration

[4] Background concentration in EPD's Tung Chung Air Quality Monitoring Station and Central Western Air Quality Monitoring Station is not available.

[5] For predicted annual average TAP concentration, "Yes" refers to its compliance with criteria and "No" refers to its non-compliance with criteria.

[6] 1-hour concentration was converted to 30-minute concentration by multiplying a factor of 1.41, based on Air Dispersion Modeling Guideline for Ontario by Toronto Ministry of the Environment.

Table17.2.34: Maximum incremental 1-hr / 24-hr average TAP concentrations (µg/m^3)

Major Area	Criteria	Airport Island	BCF	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung	Tuen Mun
1,3-butadiene	660[1]	4.60E-01 (15.0%)	1.98E-01 (7.6%)	4.58E-01 (27.5%)	1.12E+00 (49.2%)	1.04E+00 (56.2%)	8.25E-01 (32.1%)	8.77E-01 (53.2%)	1.16E+00 (56.9%)	6.92E-01 (50.7%)
Acetaldehyde	470[1]	1.11E+00 (10.4%)	4.90E-01 (6.0%)	1.20E+00 (22.4%)	2.95E+00 (42.4%)	2.71E+00 (46.2%)	2.17E+00 (27.8%)	2.33E+00 (44.2%)	3.01E+00 (47.6%)	1.85E+00 (40.6%)
Acrolein	7[2]	2.79E-01 (34.0%)	2.28E-01 (34.6%)	8.37E-02 (59.1%)	1.97E-01 (50.5%)	1.56E-01 (56.5%)	7.57E-02 (10.4%)	1.28E-01 (83.3%)	9.87E-02 (37.0%)	1.79E-01 (105.8%)
Benzene	29[2]	2.22E-01	2.11E-02	1.05E-02	1.38E-01	9.41E-02	5.09E-02	8.14E-02	4.80E-02	1.43E-01

Major Area	Criteria	Airport Island	BCF	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung	Tuen Mun
		(7.6%)	(0.6%)	(0.4%)	(5.3%)	(3.8%)	(1.8%)	(3.4%)	(1.7%)	(6.1%)
Ethylbenzene	21700[2]	2.08E-02 (0.4%)	1.78E-02 (0.4%)	6.57E-03 (0.1%)	1.52E-02 (0.3%)	1.38E-02 (0.3%)	5.36E-03 (0.1%)	9.90E-03 (0.2%)	7.38E-03 (0.2%)	1.46E-02 (0.3%)
Formaldehyde	100[3] & [6]	4.48E+00 (11.0%)	1.97E+00 (6.6%)	4.81E+00 (25.6%)	1.20E+01 (47.3%)	1.10E+01 (52.8%)	8.86E+00 (30.9%)	9.43E+00 (51.1%)	1.22E+01 (53.8%)	7.45E+00 (47.9%)
Methyl alcohol (Methanol)	28000[1]	5.59E-01 (25.5%)	1.41E-01 (8.1%)	4.34E-01 (48.9%)	1.13E+00 (76.2%)	1.05E+00 (97.0%)	8.86E-01 (51.2%)	8.78E-01 (101.0%)	1.18E+00 (94.5%)	6.70E-01 (102.0%)
Xylene (Total)	8820[2]	5.82E-02 (0.4%)	5.10E-02 (0.4%)	1.89E-02 (0.1%)	4.59E-02 (0.4%)	4.11E-02 (0.3%)	1.43E-02 (0.1%)	2.80E-02 (0.2%)	2.09E-02 (0.2%)	4.22E-02 (0.3%)
Phenol (carbolic acid)	5800[1]	2.25E-01 (25.5%)	5.66E-02 (8.1%)	1.75E-01 (48.9%)	4.55E-01 (76.2%)	4.23E-01 (97.0%)	3.56E-01 (51.2%)	3.53E-01 (101.0%)	4.75E-01 (94.5%)	2.70E-01 (102.0%)
Styrene	21630[2]	3.28E-02 (2.4%)	2.67E-02 (1.9%)	9.83E-03 (0.7%)	2.37E-02 (1.8%)	1.91E-02 (1.4%)	9.29E-03 (0.7%)	1.54E-02 (1.2%)	1.19E-02 (0.9%)	2.14E-02 (1.6%)
Toluene	3750[2]	1.04E-01 (0.7%)	1.05E-01 (0.7%)	3.64E-02 (0.2%)	1.14E-01 (0.8%)	8.94E-02 (0.6%)	2.37E-02 (0.2%)	5.45E-02 (0.4%)	5.93E-02 (0.4%)	7.94E-02 (0.5%)
Arsenic	0.2[1]	4.84E-04 (2.2%)	4.89E-05 (0.2%)	5.66E-05 (0.3%)	2.91E-04 (1.4%)	2.13E-04 (1.0%)	9.22E-05 (0.4%)	2.02E-04 (1.0%)	2.26E-04 (1.1%)	2.20E-06 (0.0%)
Cadmium	0.03[2]	5.63E-06 (0.1%)	2.11E-06 (0.0%)	9.33E-07 (0.0%)	6.20E-08 (0.0%)	4.69E-07 (0.0%)	7.27E-08 (0.0%)	2.35E-07 (0.0%)	2.66E-06 (0.0%)	1.03E-06 (0.0%)
Chromium VI	0.3[2]	-1.76E-04 (-2.0%)	1.63E-05 (0.2%)	5.45E-05 (0.7%)	4.00E-04 (4.6%)	3.78E-05 (0.4%)	1.62E-04 (1.9%)	1.20E-05 (0.1%)	1.72E-04 (2.1%)	1.37E-04 (1.6%)
Copper	100[1]	-3.48E-03 (-1.3%)	-3.35E-02 (-14.0%)	4.96E-03 (3.3%)	2.31E-02 (12.2%)	-2.19E-03 (-1.3%)	1.36E-03 (0.7%)	-8.77E-03 (-5.0%)	1.78E-02 (10.9%)	5.98E-03 (3.7%)
Nickel	0.2[2]	-2.04E-04 (-1.3%)	3.72E-05 (0.2%)	6.74E-05 (0.5%)	4.82E-04 (3.2%)	4.97E-05 (0.3%)	1.94E-04 (1.3%)	1.81E-05 (0.1%)	2.12E-04 (1.5%)	1.66E-04 (1.1%)

Notes:

[1] Based on hourly average concentration.

[2] Based on daily average concentration.

[3] Based on 30-min average concentration.

[4] Incremental percentage change is listed in the ( ).

Carcinogenic Health Risk

17.2.6.7 The predicted incremental carcinogenic risks (i.e. 3RS – 2RS), total risk for 3RS, total risk for 2RS at the representative receivers are listed in **Appendix 17.2.5**. The maximum incremental carcinogenic risks are summarised in **Table 17.2.35**. The risks have been compared with the risk criteria stated in **Section 17.2.5.11**. The predicted highest total incremental carcinogenic risk occur at Sha Lo Wan (i.e., SLW-1), with a calculated value of 1.14 $x10^{-5}$. According to the risk criteria presented in the above-mentioned section, the increase in risk is within the tolerable range.

17.2.6.8 Apart from comparing the incremental risk to the total carcinogenic risk in urban area (**Appendix 17.2.5**), which is around 9 – 16 x 10^{-4}, the maximum increase in carcinogenic risk due to the 3RS is less than 1.5%. Hence, it is considered that the operation of the 3RS would not cause an unacceptable risk to the representative human receptors.

Table 17.2.35: Maximum incremental life time carcinogenic health risk

Major Area	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung
1,3-butadiene	9.82E-08 (1.0%)	3.67E-07 (3.1%)	2.07E-07 (2.0%)	1.49E-07 (1.4%)	9.43E-08 (0.9%)	1.05E-07 (1.0%)
Acetaldehyde	1.93E-08 (0.6%)	6.97E-08 (1.9%)	4.03E-08 (1.2%)	2.83E-08 (0.8%)	1.84E-08 (0.6%)	2.06E-08 (0.6%)
Benzene	2.21E-08 (0.3%)	7.96E-08 (0.9%)	4.62E-08 (0.5%)	3.41E-08 (0.4%)	2.02E-08 (0.2%)	2.88E-08 (0.3%)

Major Area	Siu Ho Wan	Sha Lo Wan	San Shek Wan	San Tau	Sham Wat	Tung Chung
Benz(a)anthracene	6.62E-12 (0.0%)	4.39E-11 (0.1%)	1.75E-11 (0.0%)	5.40E-11 (0.1%)	5.43E-12 (0.0%)	3.48E-11 (0.1%)
Benzo(a)pyrene	7.13E-09 (0.0%)	4.88E-08 (0.2%)	1.96E-08 (0.1%)	6.24E-08 (0.3%)	5.85E-09 (0.0%)	3.76E-08 (0.2%)
Benzo(bk)fluoranthene	1.72E-11 (0.0%)	1.23E-10 (0.2%)	4.53E-11 (0.1%)	1.66E-10 (0.3%)	1.24E-11 (0.0%)	9.67E-11 (0.2%)
Chrysene	8.76E-13 (0.0%)	5.49E-12 (0.1%)	2.28E-12 (0.0%)	6.71E-12 (0.1%)	7.22E-13 (0.0%)	4.56E-12 (0.1%)
Dibenz(a,h)anthracene	6.62E-12 (0.0%)	1.07E-11 (0.0%)	1.14E-11 (0.0%)	3.39E-12 (0.0%)	5.77E-12 (0.0%)	6.82E-12 (0.0%)
Ethylbenzene	8.96E-10 (0.0%)	3.73E-09 (0.1%)	2.11E-09 (0.0%)	1.52E-09 (0.0%)	9.62E-10 (0.0%)	9.40E-10 (0.0%)
Formaldehyde	3.27E-07 (0.7%)	1.19E-06 (2.2%)	6.85E-07 (1.4%)	4.81E-07 (0.9%)	3.12E-07 (0.7%)	3.49E-07 (0.7%)
Indeno(1,2,3-cd)pyrene	5.14E-12 (0.0%)	3.74E-11 (0.1%)	1.35E-11 (0.0%)	5.11E-11 (0.2%)	3.63E-12 (0.0%)	2.93E-11 (0.1%)
Naphthalene	4.12E-08 (0.2%)	1.53E-07 (0.6%)	8.55E-08 (0.3%)	6.77E-08 (0.3%)	3.88E-08 (0.1%)	8.37E-08 (0.3%)
Arsenic	1.05E-09 (0.0%)	7.57E-10 (0.0%)	1.70E-09 (0.0%)	1.13E-09 (0.0%)	1.13E-09 (0.0%)	1.05E-09 (0.0%)
Beryllium	1.46E-10 (0.2%)	4.51E-10 (0.5%)	2.96E-10 (0.3%)	5.70E-10 (0.6%)	1.29E-10 (0.1%)	2.05E-10 (0.2%)
Cadmium	1.09E-10 (0.0%)	3.38E-10 (0.0%)	2.22E-10 (0.0%)	4.28E-10 (0.0%)	9.66E-11 (0.0%)	1.53E-10 (0.0%)
Chromium VI	8.69E-08 (0.1%)	1.09E-06 (1.0%)	1.16E-07 (0.1%)	3.20E-07 (0.3%)	8.45E-08 (0.1%)	1.75E-07 (0.2%)
Diesel Particulate Matters	2.21E-06 (0.3%)	8.58E-06 (1.0%)	4.90E-06 (0.6%)	8.86E-06 (1.0%)	1.80E-06 (0.2%)	6.97E-06 (0.8%)
Nickel	1.03E-09 (0.1%)	1.24E-08 (0.7%)	1.37E-09 (0.1%)	3.67E-09 (0.2%)	1.00E-09 (0.1%)	2.02E-09 (0.1%)
Lead	3.68E-11 (0.0%)	4.33E-10 (0.1%)	5.02E-11 (0.0%)	1.31E-10 (0.0%)	3.58E-11 (0.0%)	7.12E-11 (0.0%)
TCDD	8.98E-10 (0.0%)	3.66E-09 (0.1%)	2.03E-09 (0.0%)	3.80E-09 (0.0%)	7.22E-10 (0.0%)	3.09E-09 (0.0%)
Total	2.82E-06 (0.2%)	1.14E-05 (0.6%)	6.11E-06 (0.4%)	9.99E-06 (0.6%)	2.37E-06 (0.1%)	7.65E-06 (0.4%)

Note:

[1] Incremental percentage change is listed in the ( ).

Criteria Pollutants

Short-Term Health Effects

17.2.6.9 The predicted CO, SO_2 and NO_2 concentrations for the 3RS and 2RS scenarios and their incremental change in concentrations are shown in **Appendix 17.2.6**.

17.2.6.10 As shown in **Appendix 17.2.6**, the predicted highest cumulative 1-hr average CO concentrations at the identified human receptors for 3RS scenario comply with the corresponding AQO for CO, i.e., 30,000 $\mu g/m^3$. Therefore, adverse short-term health impact of CO due to the 3RS project is considered as acceptable.

17.2.6.11 As shown in **Appendix 17.2.6**, the predicted highest cumulative 10-min average SO_2 concentrations at the identified human receptors for 3RS scenario comply with the corresponding AQO for SO_2, i.e., 500 $\mu g/m^3$. Therefore, the associated short-term health impact (less than 24 hours) due to the 3RS project is considered as acceptable. Nevertheless, the maximum daily SO_2 at all HSRs is greater than 20 $\mu g/m^3$ due to the high background level.

17.2.6.12 As shown in **Appendix 17.2.6**, the predicted 19th highest cumulative 1-hr average NO_2 concentrations at the identified human receptors for the 3RS scenario comply with the NO_2 AQO of 200 µg/m^3. While it can be noted that the maximum cumulative 1-hr average NO_2 at some HSRs is greater than 200 µg/m^3, it should be noted that the derivation of the short-term NO_2 criteria (200 µg/m^3) is not based on mortality.

Quantitative Analyses for Health Risk Associated with Short-term Exposure of Air Pollutants

17.2.6.13 To further determine the short-term health risk associated with short-term exposure of criteria air pollutants, the change in daily pollutant concentrations shall be adopted, since the daily values are used to derive the RR values of hospital admission and mortality. **Table 17.2.36** shows the incremental change of maximum daily concentrations of criteria pollutants for different human receptors. The maximum daily concentration changes for majority of HSRs are below 5% when comparing to those of the 2RS scenario. The short-term health impact due to the 3RS project for these HSRs is considered as acceptable.

17.2.6.14 The receptors with maximum 1-hr NO_2 concentration greater than 200 µg/m^3 and incremental daily NO_2 concentration changes greater than 5% include those HSRs on airport island. Similarly, the receptors with maximum daily SO_2 concentration on airport island higher than 20 µg/m^3 and incremental daily SO_2 concentration changes greater than 5% include those HSRs on airport island BCF and Siu Ho Wan. Apart from the residential type HSRs, as AI-C1 and AI-C4 are the two hotels on airport island with the exposure time of their visitors longer than 24hr, they were included to determine the short-term hospital admission and mortality in subsequent sections.

Table 17.2.36: Incremental change of maximum daily average concentrations of criteria pollutant at different representative human receptors

Area	HSR	Incremental (3RS - 2RS) Changes of Daily-avg. Conc. (µg/m^3)			
		NO_2	RSP	FSP	SO_2
Tung Chung	TC-1	-0.10 (-0.1%)	-0.12 (-0.1%)	0.00 (0.0%)	0.20 (0.5%)
	TC-2	-0.08 (-0.1%)	-0.17 (-0.1%)	0.00 (0.0%)	0.19 (0.5%)
	TC-3	-0.05 (-0.1%)	-0.22 (-0.2%)	0.00 (0.0%)	0.19 (0.5%)
	TC-4	-0.05 (-0.1%)	-0.23 (-0.2%)	0.01 (0.0%)	0.19 (0.5%)
	TC-5	-0.21 (-0.2%)	-0.20 (-0.2%)	-0.01 (0.0%)	0.20 (0.5%)
	TC-6	-0.16 (-0.2%)	-0.17 (-0.1%)	-0.01 (0.0%)	0.20 (0.5%)
	TC-7	-0.03 (0.0%)	-0.22 (-0.2%)	0.02 (0.0%)	0.19 (0.5%)
	TC-8	-0.05 (-0.1%)	0.23 (0.2%)	0.11 (0.1%)	0.01 (0.0%)
	TC-9	-0.06 (-0.1%)	0.27 (0.2%)	0.12 (0.1%)	0.00 (0.0%)
	TC-10	-0.24 (-0.3%)	0.31 (0.3%)	0.11 (0.1%)	0.01 (0.0%)
	TC-11	-0.20 (-0.2%)	0.25 (0.2%)	0.10 (0.1%)	0.01 (0.0%)
	TC-12	-0.18 (-0.2%)	0.17 (0.2%)	0.09 (0.1%)	0.01 (0.0%)
	TC-13	-0.09 (-0.1%)	0.44 (0.4%)	0.13 (0.1%)	0.01 (0.0%)
	TC-14	-0.11 (-0.1%)	0.39 (0.3%)	0.13 (0.1%)	0.01 (0.0%)
	TC-15	-0.10 (-0.1%)	0.34 (0.3%)	0.13 (0.1%)	0.01 (0.0%)
	TC-16	-0.02 (0.0%)	0.26 (0.2%)	0.15 (0.2%)	0.02 (0.0%)

Area	HSR	Incremental (3RS - 2RS) Changes of Daily-avg. Conc. (μg/m^3)			
		NO_2	RSP	FSP	SO_2
	TC-17	0.10 (0.1%)	0.24 (0.2%)	0.14 (0.2%)	0.01 (0.0%)
	TC-18	-0.17 (-0.2%)	0.28 (0.2%)	0.16 (0.2%)	0.01 (0.0%)
	TC-19	-0.17 (-0.2%)	-0.22 (-0.2%)	0.02 (0.0%)	0.19 (0.4%)
	TC-20	-0.30 (-0.3%)	0.31 (0.3%)	0.16 (0.2%)	0.01 (0.0%)
	TC-21	0.34 (0.4%)	0.15 (0.1%)	0.14 (0.2%)	-0.01 (0.0%)
	TC-22	0.52 (0.6%)	0.18 (0.2%)	0.14 (0.2%)	-0.01 (0.0%)
	TC-23	0.81 (0.9%)	-0.25 (-0.2%)	0.03 (0.0%)	0.25 (0.6%)
	TC-24	1.21 (1.4%)	-0.21 (-0.2%)	0.03 (0.0%)	0.24 (0.6%)
	TC-25	1.20 (1.4%)	-0.22 (-0.2%)	0.03 (0.0%)	0.24 (0.6%)
	TC-26	1.11 (1.3%)	-0.22 (-0.2%)	0.03 (0.0%)	0.24 (0.6%)
	TC-27	1.26 (1.4%)	-0.12 (-0.1%)	0.02 (0.0%)	0.23 (0.6%)
	TC-28	1.28 (1.5%)	-0.17 (-0.2%)	0.03 (0.0%)	0.23 (0.6%)
	TC-29	1.16 (1.3%)	-0.07 (-0.1%)	0.02 (0.0%)	0.24 (0.6%)
	TC-30	1.14 (1.3%)	-0.02 (0.0%)	0.01 (0.0%)	0.24 (0.6%)
	TC-31	0.88 (1.0%)	-0.15 (-0.1%)	0.02 (0.0%)	0.25 (0.6%)
	TC-32	0.35 (0.4%)	-0.02 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-33	0.05 (0.1%)	-0.03 (0.0%)	0.02 (0.0%)	0.25 (0.6%)
	TC-34	0.37 (0.4%)	-0.14 (-0.1%)	0.02 (0.0%)	0.26 (0.6%)
	TC-35	0.44 (0.5%)	-0.23 (-0.2%)	0.03 (0.0%)	0.26 (0.6%)
	TC-36	0.35 (0.4%)	-0.17 (-0.2%)	0.03 (0.0%)	0.26 (0.6%)
	TC-37	-0.16 (-0.2%)	-0.02 (0.0%)	0.02 (0.0%)	0.26 (0.6%)
	TC-38	0.65 (0.8%)	0.00 (0.0%)	0.01 (0.0%)	0.27 (0.7%)
	TC-39	0.74 (0.9%)	0.01 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-40	0.80 (0.9%)	0.01 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-41	0.75 (0.9%)	-0.02 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-42	0.78 (0.9%)	-0.05 (0.0%)	0.01 (0.0%)	0.26 (0.6%)
	TC-43	0.73 (0.9%)	-0.07 (-0.1%)	0.01 (0.0%)	0.26 (0.6%)
	TC-44	0.63 (0.8%)	-0.08 (-0.1%)	0.01 (0.0%)	0.27 (0.7%)
	TC-45	0.55 (0.7%)	-0.04 (0.0%)	0.01 (0.0%)	0.28 (0.7%)
	TC-46	0.91 (1.1%)	0.01 (0.0%)	0.01 (0.0%)	0.23 (0.6%)
	TC-47	0.86 (1.0%)	0.01 (0.0%)	0.00 (0.0%)	0.24 (0.6%)
	TC-48	0.75 (0.9%)	-0.02 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-49	0.72 (0.9%)	-0.03 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-50	0.61 (0.7%)	-0.05 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-51	0.45 (0.5%)	-0.01 (0.0%)	0.01 (0.0%)	0.16 (0.4%)
	TC-52	-0.14 (-0.2%)	0.03 (0.0%)	0.01 (0.0%)	0.00 (0.0%)

Area	HSR	Incremental (3RS - 2RS) Changes of Daily-avg. Conc. (µg/m^3)			
		NO_2	RSP	FSP	SO_2
	TC-53	0.01 (0.0%)	0.01 (0.0%)	0.01 (0.0%)	0.09 (0.2%)
	TC-54	0.58 (0.7%)	-0.02 (0.0%)	0.01 (0.0%)	0.27 (0.7%)
	TC-55	-0.01 (0.0%)	0.03 (0.0%)	0.01 (0.0%)	0.00 (0.0%)
	TC-56	-0.10 (-0.1%)	0.03 (0.0%)	0.01 (0.0%)	0.00 (0.0%)
	TC-57	-0.04 (0.0%)	0.02 (0.0%)	0.01 (0.0%)	0.00 (0.0%)
	TC-58	-0.08 (-0.1%)	0.02 (0.0%)	0.01 (0.0%)	0.00 (0.0%)
	TC-59	-0.01 (0.0%)	0.02 (0.0%)	0.01 (0.0%)	0.00 (0.0%)
	TC-P1	0.60 (0.7%)	0.02 (0.0%)	0.01 (0.0%)	0.25 (0.6%)
	TC-P2	0.31 (0.3%)	0.22 (0.2%)	0.13 (0.2%)	-0.02 (-0.1%)
	TC-P5	-0.12 (-0.1%)	-0.07 (-0.1%)	0.02 (0.0%)	0.10 (0.2%)
	TC-P6	0.25 (0.3%)	-0.02 (0.0%)	0.02 (0.0%)	0.30 (0.7%)
	TC-P7	0.37 (0.4%)	0.33 (0.3%)	0.13 (0.1%)	0.01 (0.0%)
	TC-P8	-0.23 (-0.3%)	-0.24 (-0.2%)	0.00 (0.0%)	0.23 (0.6%)
	TC-P9	1.21 (1.3%)	0.09 (0.1%)	0.06 (0.1%)	0.27 (0.7%)
	TC-P10	1.49 (1.5%)	0.37 (0.3%)	0.08 (0.1%)	1.54 (3.4%)
	TC-P11	2.85 (3.0%)	0.32 (0.3%)	0.09 (0.1%)	1.31 (2.9%)
	TC-P12	-0.14 (-0.2%)	-0.15 (-0.1%)	0.06 (0.1%)	0.03 (0.1%)
	TC-P13	-0.54 (-0.6%)	-0.30 (-0.3%)	-0.01 (0.0%)	0.22 (0.5%)
	TC-P14	-0.26 (-0.3%)	-0.20 (-0.2%)	-0.01 (0.0%)	0.20 (0.5%)
	TC-P15	-0.64 (-0.7%)	-0.11 (-0.1%)	0.00 (0.0%)	0.21 (0.5%)
	TC-P16	0.41 (0.4%)	-0.06 (-0.1%)	0.00 (0.0%)	0.22 (0.5%)
	TC-P17	0.58 (0.7%)	-0.02 (0.0%)	0.01 (0.0%)	0.26 (0.6%)
San Tau	ST-1	-1.61 (-1.7%)	0.03 (0.0%)	0.01 (0.0%)	0.02 (0.1%)
	ST-2	-1.36 (-1.4%)	0.03 (0.0%)	0.01 (0.0%)	0.02 (0.0%)
	ST-3	-1.17 (-1.3%)	0.02 (0.0%)	0.01 (0.0%)	0.03 (0.1%)
Sha Lo Wan	SLW-1	-12.08 (-9.9%)	-0.11 (-0.1%)	-0.01 (0.0%)	1.07 (2.3%)
	SLW-2	-11.63 (-10.2%)	0.01 (0.0%)	0.00 (0.0%)	0.64 (1.4%)
	SLW-3	-2.59 (-2.3%)	-0.34 (-0.3%)	-0.03 (0.0%)	0.19 (0.4%)
	SLW-4	-1.12 (-1.0%)	-0.41 (-0.4%)	-0.04 (0.0%)	0.17 (0.4%)
San Shek Wan	SSW-1	2.10 (2.2%)	-0.17 (-0.2%)	-0.01 (0.0%)	0.09 (0.2%)
Sham Wat	SW-1	0.22 (0.3%)	0.00 (0.0%)	0.00 (0.0%)	0.04 (0.1%)
	SW-2	0.99 (1.1%)	0.01 (0.0%)	0.00 (0.0%)	0.93 (1.8%)
Siu Ho Wan	SHW-1	0.19 (0.2%)	0.22 (0.2%)	0.10 (0.1%)	0.00 (0.0%)
	SHW-2	-0.27 (-0.3%)	-0.03 (0.0%)	0.01 (0.0%)	-0.20 (-0.5%)
	SHW-3	3.72 (4.2%)	0.11 (0.1%)	0.06 (0.1%)	2.76 (6.4%)
	SHW-4	2.65 (2.9%)	0.13 (0.1%)	0.06 (0.1%)	0.17 (0.4%)

Area	HSR	Incremental (3RS - 2RS) Changes of Daily-avg. Conc. (μg/m^3)			
		NO_2	RSP	FSP	SO_2
	SHW-5	-0.38 (-0.4%)	-0.02 (0.0%)	0.00 (0.0%)	-0.22 (-0.5%)
Proposed Lantau Logistic Park	LLP-P1	3.13 (3.5%)	0.14 (0.1%)	0.06 (0.1%)	0.11 (0.3%)
	LLP-P2	-0.95 (-1.0%)	0.11 (0.1%)	0.04 (0.0%)	-0.71 (-1.5%)
	LLP-P3	-2.52 (-2.7%)	0.13 (0.1%)	0.04 (0.0%)	-0.92 (-2.0%)
	LLP-P4	-1.22 (-1.3%)	0.07 (0.1%)	0.03 (0.0%)	-0.62 (-1.3%)
Tuen Mun	TM-7	0.08 (0.1%)	-0.03 (0.0%)	0.09 (0.1%)	0.01 (0.0%)
	TM-8	0.07 (0.1%)	-0.07 (-0.1%)	0.13 (0.1%)	0.03 (0.1%)
	TM-9	0.10 (0.1%)	-0.04 (0.0%)	0.13 (0.1%)	0.03 (0.1%)
	TM-10	0.08 (0.1%)	-0.26 (-0.2%)	0.17 (0.2%)	0.03 (0.1%)
	TM-11	-0.02 (0.0%)	-0.05 (0.0%)	0.11 (0.1%)	-0.02 (0.0%)
	TM-12	0.05 (0.1%)	0.11 (0.1%)	0.11 (0.1%)	0.03 (0.1%)
	TM-13	-0.09 (-0.1%)	0.11 (0.1%)	0.09 (0.1%)	0.10 (0.2%)
	TM-14	0.35 (0.4%)	0.05 (0.0%)	0.06 (0.1%)	-0.05 (-0.1%)
	TM-15	-0.18 (-0.2%)	0.06 (0.1%)	0.06 (0.1%)	-0.05 (-0.1%)
	TM-16	0.06 (0.1%)	-0.22 (-0.2%)	0.15 (0.2%)	0.03 (0.1%)
	TM-17	0.06 (0.1%)	0.03 (0.0%)	0.11 (0.1%)	0.02 (0.0%)
	TM-18	0.05 (0.0%)	0.02 (0.0%)	0.10 (0.1%)	0.02 (0.0%)
	TM-19	-0.02 (0.0%)	0.03 (0.0%)	0.10 (0.1%)	-0.02 (0.0%)
Airport	AI-C1	16.39 (14.4%)	-2.55 (-2.0%)	0.12 (0.1%)	11.12 (19.5%)
	AI-C2	5.79 (5.7%)	-2.38 (-1.9%)	-0.05 (-0.1%)	3.92 (7.6%)
	AI-C3	6.06 (5.9%)	-1.67 (-1.3%)	0.03 (0.0%)	4.21 (8.1%)
	AI-C4	7.20 (6.8%)	-0.32 (-0.3%)	0.14 (0.2%)	5.31 (10.1%)
	AI-C5	10.97 (10.0%)	-0.31 (-0.2%)	0.20 (0.2%)	7.57 (13.9%)
	AI-C6	11.58 (10.4%)	0.22 (0.2%)	0.21 (0.2%)	7.71 (14.1%)
Hong Kong Boundary Crossing Facilities	BCF-1	6.80 (6.5%)	0.28 (0.2%)	-0.01 (0.0%)	7.12 (13.0%)

Notes:
[1] Values in ( ) indicate the percentage change with reference to 2RS.
[2] Incremental change of max. daily-avg. conc. = max. daily conc. of 3RS – max. daily conc. of 2RS.

17.2.6.15 Due to the variation of daily concentrations of 3RS and 2RS scenarios, both health impact and health benefits will be present (as their maximum may not necessary occur at the same day). Consequently, the annual average concentration changes of criteria pollutants are more representative in determining the average daily hospital admission and mortality. If the mean daily concentrations have increased to a certain level under the 3R scenario, the short-term effects on mortality would be estimated by means of the RRs (derived from CM Wong's study). The predicted incremental unit risk of hospital admissions attributable to NO_2, RSP, and SO_2 per annum have been derived and are summarised in **Table 17.2.37**. The unit risk of hospital admission and mortality due to the change in maximum daily concentrations per annum are also presented. The detailed calculation is shown in **Appendix 17.2.7**.

Table 17.2.37: Incremental unit risk of hospital admission per annum attributable to NO_2, RSP and SO_2

Major Area	Incremental Unit Risk per Annum of Hospital Admission [2] & [3]		Incremental Unit Risk per Annum of Hospital Admission [2] & [4]	
	Cardiovascular Disease	Respiratory Disease	Cardiovascular Disease	Respiratory Disease
Tung Chung	8.20E-06 (5.69E-06 – 1.06E-05)	5.75E-06 (3.28E-06 – 8.21E-06)	2.93E-05 (1.94E-05 – 3.84E-05)	1.71E-05 (7.66E-06 – 2.65E-05)
San Tau	1.15E-05 (7.66E-06 – 1.51E-05)	7.45E-06 (3.73E-06 – 1.11E-05)	-2.89E-05 (-3.62E-05 – -2.13E-05)	-2.39E-05 (-3.16E-05 – -1.61E-05)
Sha Lo Wan	-4.17E-05 (-5.22E-05 – -3.08E-05)	-3.36E-05 (-4.47E-05 – -2.26E-05)	-2.07E-04 (-2.58E-04 – -1.54E-04)	-1.80E-04 (-2.34E-04 – -1.26E-04)
San Shek Wan	-1.86E-05 (-2.30E-05 – -1.41E-05)	-1.62E-05 (-2.09E-05 – -1.15E-05)	-1.18E-05 (-1.49E-05 – -8.83E-06)	-1.30E-05 (-1.61E-05 – -9.80E-06)
Sham Wat	7.59E-06 (5.00E-06 – 1.00E-05)	5.10E-06 (2.60E-06 – 7.60E-06)	3.16E-05 (2.03E-05 – 4.20E-05)	1.60E-05 (5.33E-06 – 2.66E-05)
Siu Ho Wan	6.31E-06 (4.41E-06 – 8.11E-06)	4.53E-06 (2.65E-06 – 6.39E-06)	7.62E-06 (5.22E-06 – 9.95E-06)	6.88E-06 (4.49E-06 – 9.27E-06)
Airport	4.65E-05 (3.17E-05 – 6.02E-05)	2.68E-05 (1.25E-05 – 4.10E-05)	4.52E-04 (2.97E-04 – 5.95E-04)	2.34E-04 (8.67E-05 – 3.81E-04)

Notes:

[1] The estimates of the effect of one pollutant will include the effects of another, if the two pollutants are correlated. This is especially true for NO_2 and PM in according to WHO. Hence, there will be overlaps in the estimation of hospital admissions and deaths by adding up the effects of all the criteria pollutants, resulting in an overestimation of health risk.

[2] The unit risk on number of hospital admission = the number of hospital admission in the predicted year / the population in the concerned area.

[3] With reference to incremental change of annual-avg. concentration for averaged daily concentration determination.

[4] With reference to incremental change of max. daily-avg. concentration.

[5] The incremental unit risks are estimated with references to the average values of RR. The values in the brackets indicate the 95% confidence intervals of RR.

17.2.6.16 The hospital admissions due to cardiovascular (ICD10:I00-I99) and respiratory diseases (ICD10: J00-J99) in Hong Kong are around 2171 and 2363 per 100,000 population respectively in Year 2012. For the short-term exposure to NO_2 RSP and SO_2, the estimated largest yearly increase in risk of average daily hospital admissions due to cardiovascular and respiratory diseases under the operation of 3RS against 2RS are around 4.65×10^{-5} and 2.68×10^{-5} (i.e., 4.65 and 2.68 per 100,000) for the identified areas. The increase in risk of average daily hospital admission per annum is considered as relatively small.

17.2.6.17 The predicted unit risk of premature deaths (short term mortality) due to all-causes per annum attributable to NO_2, RSP and SO_2 were derived and are summarised in **Table 17.2.38.** The detailed calculation is shown in **Appendix 17.2.7**.

Table 17.2.38: Incremental unit risk of premature deaths (short-term mortality) due to all-causes per annum attributable to NO_2, RSP and SO_2

Major Area	Incremental Unit Risk per Annum of All-cause Premature Deaths (Short-Term Mortality) [2] & [3]	Incremental Unit Risk per Annum of All-cause Premature Deaths (Short-Term Mortality) [2] & [4]
Tung Chung	2.23E-06 (1.40E-06 – 3.06E-06)	7.86E-06 (4.67E-06 – 1.10E-05)
San Tau	3.07E-06	-8.07E-06

Major Area	Incremental Unit Risk per Annum of All-cause Premature Deaths (Short-Term Mortality) [2] & [3]	Incremental Unit Risk per Annum of All-cause Premature Deaths (Short-Term Mortality) [2] & [4]
	(1.81E-06 – 4.32E-06)	(-1.06E-05 – -5.47E-06)
Sha Lo Wan	-1.17E-05 (-1.54E-05 – -8.01E-06)	-5.81E-05 (-7.62E-05 – -3.99E-05)
San Shek Wan	-5.27E-06 (-6.83E-06 – -3.70E-06)	-3.23E-06 (-4.32E-06 – -2.15E-06)
Sham Wat	2.00E-06 (1.15E-06 – 2.85E-06)	8.39E-06 (4.79E-06 – 1.20E-05)
Siu Ho Wan	1.73E-06 (4.53E-07 – 1.09E-06)	2.00E-06 (1.18E-06 – 2.83E-06)
Airport	1.27E-05 (7.92E-06 – 1.75E-05)	1.22E-04 (7.18E-05 – 1.71E-04)

Notes:

[1] The estimates of the effect of one pollutant will include the effects of another, if the two pollutants are correlated. This is especially true for NO_2 and PM in according to WHO. Hence, there will be overlaps in the estimation of hospital admissions and deaths by adding up the effects of all the criteria pollutants, resulting in an overestimation of health risk.

[2] The unit risk on number of deaths = the number of deaths in the predicted year / the population in the concerned area.

[3] With reference to incremental change of annual-avg. concentration for averaged daily concentration determination.

[4] With reference to incremental change of max. daily-avg. concentration.

[5] The incremental unit risks are estimated with references to the average values of RR. The values in the brackets indicate 95% confidence intervals of RR.

17.2.6.18 The total mortality due to all-causes of disease (exclude external causes of morbidity and mortality) in Hong Kong is around 587 per 100,000 population in Year 2012. For the short-term exposure to NO_2, RSP and SO_2, the estimated largest yearly maximum increase in average daily risk of premature death (short-term mortality) due to all-causes under the operation of 3RS against 2RS is around 1.27 x 10^{-5} (i.e. 1.27 per 100,000 population) for the identified areas. The increase in such short-term mortality risk of premature death per annum is considered as relatively small.

Long Term Health Effect

17.2.6.19 **Appendix 17.2.6** shows the annual concentrations of NO_2, RSP, and FSP. The annual concentrations at residential type of human receptors outside airport comply with the annual average AQO criteria. **Table 17.2.39** shows the incremental change of annual concentrations of criteria pollutants for different human receptors.

Table 17.2.39: Incremental annual average concentrations of criteria pollutant at different representative human receptors

Area	HSR	Incremental (3RS - 2RS) Changes of Annual-avg. Conc. (μg/m^3)			
		NO_2	RSP	FSP	SO_2
Tung Chung	TC-1	0.34 (1.2%)	0.03 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-2	0.36 (1.3%)	0.04 (0.1%)	0.02 (0.1%)	0.06 (1.1%)
	TC-3	0.34 (1.2%)	0.04 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-4	0.32 (1.1%)	0.04 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-5	0.27 (1.0%)	0.04 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-6	0.27 (1.0%)	0.04 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-7	0.32 (1.1%)	0.04 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-8	0.38 (1.4%)	0.05 (0.1%)	0.02 (0.1%)	0.08 (1.4%)

Area	HSR	Incremental (3RS - 2RS) Changes of Annual-avg. Conc. (μg/m^3)			
		NO_2	RSP	FSP	SO_2
	TC-9	0.38 (1.3%)	0.05 (0.1%)	0.02 (0.1%)	0.08 (1.4%)
	TC-10	0.30 (1.1%)	0.04 (0.1%)	0.02 (0.1%)	0.08 (1.4%)
	TC-11	0.31 (1.1%)	0.04 (0.1%)	0.02 (0.1%)	0.08 (1.4%)
	TC-12	0.31 (1.1%)	0.04 (0.1%)	0.02 (0.1%)	0.09 (1.4%)
	TC-13	0.33 (1.2%)	0.05 (0.1%)	0.03 (0.1%)	0.09 (1.4%)
	TC-14	0.34 (1.2%)	0.05 (0.1%)	0.03 (0.1%)	0.08 (1.4%)
	TC-15	0.35 (1.2%)	0.05 (0.1%)	0.02 (0.1%)	0.08 (1.4%)
	TC-16	0.58 (1.9%)	0.06 (0.1%)	0.04 (0.1%)	0.09 (1.4%)
	TC-17	0.55 (1.8%)	0.05 (0.1%)	0.03 (0.1%)	0.08 (1.4%)
	TC-25	0.36 (1.4%)	0.03 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-26	0.34 (1.3%)	0.03 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-27	0.29 (1.1%)	0.02 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-28	0.30 (1.2%)	0.02 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-29	0.28 (1.1%)	0.02 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-30	0.27 (1.1%)	0.02 (0.1%)	0.01 (0.1%)	0.05 (0.8%)
	TC-31	0.30 (1.2%)	0.03 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-32	0.25 (0.9%)	0.02 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-33	0.26 (0.9%)	0.02 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-34	0.28 (1.1%)	0.02 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-35	0.29 (1.1%)	0.03 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-36	0.25 (0.9%)	0.02 (0.1%)	0.02 (0.1%)	0.05 (0.9%)
	TC-37	0.21 (0.7%)	0.01 (0.0%)	0.02 (0.1%)	0.05 (0.9%)
	TC-38	0.23 (1.0%)	0.00 (0.0%)	0.01 (0.1%)	0.05 (0.8%)
	TC-39	0.24 (0.9%)	0.00 (0.0%)	0.01 (0.0%)	0.04 (0.8%)
	TC-40	0.24 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.04 (0.8%)
	TC-41	0.25 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.04 (0.8%)
	TC-42	0.25 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.05 (0.8%)
	TC-43	0.25 (1.1%)	0.00 (0.0%)	0.01 (0.0%)	0.05 (0.8%)
	TC-44	0.24 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.05 (0.8%)
	TC-45	0.21 (0.9%)	-0.01 (0.0%)	0.02 (0.1%)	0.05 (0.9%)
	TC-46	0.23 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.04 (0.7%)
	TC-47	0.23 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.04 (0.7%)
	TC-48	0.24 (1.0%)	0.01 (0.0%)	0.01 (0.0%)	0.04 (0.8%)
	TC-49	0.24 (1.1%)	0.01 (0.0%)	0.01 (0.0%)	0.04 (0.8%)
	TC-50	0.24 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.04 (0.8%)
	TC-51	0.24 (1.1%)	0.01 (0.0%)	0.01 (0.0%)	0.05 (0.8%)

Area	HSR	Incremental (3RS - 2RS) Changes of Annual-avg. Conc. (μg/m^3)			
		NO_2	RSP	FSP	SO_2
	TC-52	0.40 (1.5%)	0.02 (0.1%)	0.02 (0.1%)	0.09 (1.6%)
	TC-53	0.32 (1.2%)	0.02 (0.0%)	0.01 (0.1%)	0.07 (1.2%)
	TC-54	0.24 (1.0%)	0.00 (0.0%)	0.01 (0.0%)	0.05 (0.9%)
	TC-55	0.43 (1.7%)	0.04 (0.1%)	0.02 (0.1%)	0.11 (1.9%)
	TC-56	0.40 (1.6%)	0.03 (0.1%)	0.01 (0.1%)	0.09 (1.6%)
	TC-57	0.39 (1.6%)	0.03 (0.1%)	0.01 (0.1%)	0.09 (1.6%)
	TC-58	0.38 (1.5%)	0.02 (0.1%)	0.01 (0.0%)	0.08 (1.4%)
	TC-59	0.37 (1.5%)	0.02 (0.1%)	0.01 (0.0%)	0.08 (1.4%)
	TC-P1	0.23 (0.9%)	0.00 (0.0%)	0.01 (0.0%)	0.05 (0.8%)
	TC-P5	0.31 (1.1%)	0.01 (0.0%)	0.02 (0.1%)	0.08 (1.3%)
	TC-P6	0.15 (0.6%)	-0.05 (-0.1%)	0.03 (0.1%)	0.05 (0.8%)
	TC-P7	0.20 (0.7%)	0.02 (0.1%)	0.03 (0.1%)	0.08 (1.3%)
	TC-P8	0.25 (1.0%)	0.03 (0.1%)	0.02 (0.1%)	0.07 (1.2%)
	TC-P9	0.31 (1.3%)	0.01 (0.0%)	0.01 (0.0%)	0.08 (1.3%)
	TC-P10	0.39 (1.5%)	0.02 (0.1%)	0.02 (0.1%)	0.11 (1.9%)
	TC-P11	0.38 (1.4%)	0.04 (0.1%)	0.02 (0.1%)	0.11 (1.9%)
	TC-P13	0.12 (0.4%)	0.04 (0.1%)	0.02 (0.1%)	0.07 (1.2%)
	TC-P14	0.26 (1.0%)	0.04 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-P15	-0.04 (-0.1%)	0.03 (0.1%)	0.02 (0.1%)	0.07 (1.1%)
	TC-P16	0.36 (1.3%)	0.02 (0.1%)	0.02 (0.1%)	0.06 (1.1%)
	TC-P17	0.25 (1.1%)	0.01 (0.0%)	0.01 (0.0%)	0.05 (0.8%)
San Tau	ST-1	0.33 (1.1%)	0.09 (0.2%)	0.03 (0.1%)	0.16 (2.5%)
	ST-2	0.28 (0.9%)	0.08 (0.2%)	0.02 (0.1%)	0.17 (2.6%)
	ST-3	0.38 (1.3%)	0.08 (0.2%)	0.02 (0.1%)	0.15 (2.3%)
Sha Lo Wan	SLW-1	-2.65 (-6.9%)	0.22 (0.5%)	0.03 (0.1%)	-0.04 (-0.5%)
	SLW-2	-1.76 (-5.1%)	0.17 (0.4%)	0.03 (0.1%)	0.04 (0.5%)
	SLW-3	-1.30 (-4.1%)	0.09 (0.2%)	0.02 (0.1%)	0.06 (0.8%)
	SLW-4	-1.45 (-4.5%)	0.10 (0.2%)	0.02 (0.1%)	0.05 (0.6%)
San Shek Wan	SSW-1	-0.26 (-1.0%)	0.05 (0.1%)	0.02 (0.1%)	0.16 (2.3%)
Sham Wat	SW-1	0.13 (0.6%)	0.03 (0.1%)	0.01 (0.0%)	0.11 (1.8%)
	SW-2	0.21 (1.0%)	0.13 (0.3%)	0.01 (0.0%)	0.10 (1.6%)
Siu Ho Wan	SHW-1	0.24 (1.0%)	0.01 (0.0%)	0.01 (0.0%)	0.05 (0.8%)
	SHW-2	0.24 (1.0%)	0.02 (0.0%)	0.01 (0.0%)	0.06 (1.0%)
	SHW-3	0.25 (1.1%)	0.03 (0.1%)	0.01 (0.0%)	0.10 (1.8%)
	SHW-5	0.22 (0.9%)	0.02 (0.0%)	0.01 (0.0%)	0.05 (0.9%)

Note:
[1] Values in ( ) indicate the percentage change with reference to 2RS.

17.2.6.20 The highest cumulative annual average SO_2 concentrations predicted at the residential HSRs would range from 5.7 to 8.8 μg/m^3. The annual average contribution by the 3RS would range from -0.04 to 0.17 μg/m^3 (i.e., -0.5 to 2.6%). Nevertheless, there is still considerable scientific uncertainty as to whether SO_2 is the pollutant responsible for the observed adverse effects or, rather a surrogate for particulate matters. While it is not possible to totally rule out its adverse health effects, the potential additional health effects are likely to be small.

17.2.6.21 For RSP, the highest cumulative annual average RSP concentrations predicted at the residential HSRs would range from 37.7 to 40.0 μg/m^3. The average contributions by the 3RS would be from -0.05 to 0.22 μg/m^3 (i.e. -0.1 to 0.5%). The associated additional risk for adverse health effects of RSP due to the 3RS are likely to be very small and are unlikely to be quantifiable.

17.2.6.22 For FSP, the highest cumulative annual average FSP concentrations predicted at the residential HSRs would range from 26.8 to 28.1 μg/m^3. The average contributions by the 3RS would be from 0.01 to 0.04 μg/m^3 (i.e., 0.0 to 0.1%.). The associated additional risk for adverse health effects of FSP due to the 3RS are likely to be very small and are unlikely to be quantifiable.

17.2.6.23 For NO_2, the highest cumulative annual average NO_2 concentrations at the residential HSRs would range from 21.5 to 35.9 μg/m^3. The average contribution by the 3RS would range from -2.65 to 0.58 μg/m^3 (i.e., -6.9% to 1.9%). The associated additional risk for adverse health effects of NO_2 due to the 3RS are likely to be very small. It is very unlikely that the NO_2 generated by the 3RS will cause significant long-term adverse health effects.

Quantitative Analyses for Health Risk associated with Long-term Exposure of FSP

17.2.6.24 To further determine the long-term risk, the predicted long-term mortality attributable to FSP has been derived and are summarised in **Table 17.2.40**. The detailed calculation is shown in **Appendix 17.2.7**.

Table 17.2.40: Incremental unit risk of premature deaths (long-term mortality) due to all-causes per annum attributable to FSP

Major Area	Incremental Unit Risk of All-cause Premature Deaths per Annum (Long-Term Mortality) [1]
Tung Chung	3.99E-07 (1.03E-07 - 7.68E-07)
San Tau	5.65E-07 (1.45E-07 - 1.09E-06)
Sha Lo Wan	6.61E-07 (1.70E-07 - 1.27E-06)
San Shek Wan	5.20E-07 (1.34E-07 - 1.00E-06)
Sham Wat	2.94E-07 (7.56E-08 - 5.66E-07)
Siu Ho Wan	2.26E-07 (5.83E-08 - 4.36E-07)

Notes:
[1] The unit risk on number of deaths = the number of deaths in the predicted year / the population in the concerned area.
[2] The incremental unit risks are estimated with references to the average values of RR. The values in the brackets indicate 95% confidence intervals of RR.

17.2.6.25 The total mortality due to all-causes of disease (exclude external causes of morbidity and mortality) in Hong Kong is around 587 per 100,000 population in Year 2012. For long-term exposure to FSP, the estimated maximum yearly increase in risk of premature death (long-term mortality) due to all-

causes under the operation of 3RS against 2RS is around 6.61 x 10^{-7} (i.e., 0.0661 per 100,000 population) for the identified areas. The increase in such long-term mortality risk of premature death is considered as relatively small.

17.2.7 Means to Reduce Health Impact by Air Emissions and Recommendation of Reasonably Practicable Measures

17.2.7.1 As discussed in above sections, it is considered that the health risks due to TAP and criteria pollutants arising from the operation of the project are acceptable. As described in **Chapter 5**, AAHK has also been implementing a number of new measures and initiatives aimed at further reducing air emissions from airport activities and operations. These include:

- Banned all idling vehicle engines on the airside since 2008, except for certain vehicles that are exempted;
- Banning the use of APU for all aircraft at frontal stands by end 2014;
- Requiring all saloon vehicles as electric vehicles by end 2017;
- Increasing charging stations for electric vehicles and electric GSE to a total of 290 by end 2018;
- Conducting review on existing GSE emission performance and explore measures to further control air emissions;
- Exploring with franchisees feasibility of expediting replacement of old airside vehicles and GSE with cleaner ones during tender or renewal of contracts;
- Requiring all new airside vehicles to be fuel-efficient and making it a prerequisite for the licensing process;
- Providing the cleanest diesel and gasoline at the airfield;
- Requiring all of the AAHK's diesel vehicles to use biodiesel (B5);
- Promoting increased use of electric vehicles and electric ground service equipment at HKIA by provision of charging infrastructure; and
- Providing a liquefied petroleum gas (LPG) fuelling point for airside vehicles and ground service equipment.

17.2.7.2 The above measures will effectively reduce VOC (i.e. TAP), PM, NO_2, SO_2 and CO. This will further reduce the associated health risk.

17.2.8 Uncertainty Analysis

17.2.8.1 The risk estimates presented in the section above are based on estimates of concentrations of TAP and criteria pollutants predicted at representative human receptors obtained through emissions and dispersion modeling. These are subject to uncertainties as emissions estimates

are sensitive to values used to represent the numerous emission source variables and emission factors of TAP for each source. The key sources of uncertainties are summarised below:

- The Year 2011 monitoring data has been adopted as background for TAP. This cannot reflect the improvement in air quality due to implementation of short-term and long-term control measures by the Government. This may overestimate the associated health risks;
- The modeling verification suggests that the model results will over-estimate the predicted pollutant concentrations, which in turn will overestimate the health risks. Hence, it is considered that the assessments of the health impact would be conservative;
- In determining the total risks of hospital admissions and mortalities, the hospital admissions and mortalities risk associated with each individual criteria pollutant are summed. The RR for short-term health effects (both hospital admissions and deaths) of each criteria pollutant was derived from a single-pollutant statistical model. Therefore, the estimates of the effect of one pollutant will include the effects of another, if the two pollutants are correlated. This is especially true for NO_2 and PM according to WHO. Hence, there will be overlaps in the estimation of hospital admissions and deaths by adding up the effects of all the criteria pollutants, resulting in an overestimation of health risks; and
- The toxicity criteria adopted from agencies (WHO, IRIS, etc.) would introduce uncertainty to the risk assessment. These toxicity criteria are used as single-point estimates throughout the analysis with uncertainty and variability associated with them. The application of safety factor to LOAEL or NOAEL for derivation of toxicity criteria for long-term chronic toxicity is another source of uncertainty. This uncertainty may overestimate or underestimate the risk.

17.2.9 Conclusion

17.2.9.1 The short-term (i.e 1-hour / 24-hour) and long-term (i.e. annual) TAP concentrations due to the operation of 3RS modeled at all potential human receptors would comply with the respective acute and chronic non-carcinogenic risk criteria. The acute risk and non-carcinogenic chronic risk due to 3RS are considered as acceptable.

17.2.9.2 The maximum increase in carcinogenic health risk due to TAP is around 1.14×10^{-5} for the 3RS. The increase in carcinogenic health risk due to the 3RS is considered as acceptable.

17.2.9.3 For short-term exposure to criteria pollutants, the short-term concentrations of CO (1-hour), NO_2 (1-hour) and SO_2 (10-minute) are well below the AQO in the assessment areas. Moreover, the estimated largest yearly increases in risks of hospital admission and premature death (short-term mortality risk) associated with short-term exposure to NO_2, RSP and SO_2 due to the operation of the 3RS compared with 2RS are relatively small. Hence, the short-term health risk associated with short-term exposure of the concerned criteria pollutants is considered as acceptable.

17.2.9.4 The incremental change arising from the operation of 3RS against 2RS for annual concentrations of NO_2 (-2.65 $\mu g/m^3$ to 0.58 $\mu g/m^3$, i.e., -6.9 to 1.9%), RSP (-0.05 $\mu g/m^3$ to 0.22 $\mu g/m^3$, i.e., -0.1 to 0.5%), FSP (0.01 $\mu g/m^3$ to 0.04 $\mu g/m^3$, i.e., 0.0 to 0.1%) and SO_2 (-0.04 $\mu g/m^3$ to 0.17 $\mu g/m^3$, i.e., -0.5 to 2.6%), in the assessment areas. Besides, the estimated largest yearly increase in premature death (long-term mortality risk) associated with long-term exposure to FSP due to the operation of the 3RS compared with 2RS is relatively small. Hence, the long-term health impact

associated with long-term exposure of the concerned criteria pollutants is considered as acceptable.

17.3 Health Impact Assessment for Aircraft Noise

17.3.1 Technical Requirements

17.3.1.1 In accordance with the technical requirements set out in section II of Appendix C of the EIA Study Brief, the HIA of aircraft noise shall be conducted taking into account the findings of the aircraft noise impact assessment (see **Section 7.3**) and include the following key steps:

a) Identification of the health impact from aircraft noise during the operation of the project;

b) Assessment of the likelihood and consequences of exposure to the aircraft noise;

c) Identification of means by which the health impact could be further reduced;

d) Recommendations of reasonably practical measures, if any, to reduce the health impact during the operation of the project.

17.3.1.2 Clause 3.4.14.2 of the EIA Study Brief also requires a literature search to determine the best approach and methodology for the HIA, including any codes of practices, guidelines, etc. applied locally in Hong Kong and elsewhere in the world. This has been undertaken accordingly and the findings of the Literature Review are presented in **Section 17.3.2** below.

17.3.2 Literature Review

17.3.2.1 A literature search was carried out which covered a review of a wide range of research studies and other documents, including available guidelines and codes of practices, on non-auditory health effects of environmental noise. This outputs a summary of studies related to environmental noise and non-auditory health effects published in recent years (mostly between years 2006 and 2012). Non-auditory health effects are focused in this health impact assessment, not auditory ones because the former generally occurs at moderate noise intensity in the environment, whilst the latter is normally encountered only in occupational and workplace settings.

17.3.2.2 The search of applicable literature has also made reference to previous literature reviews of environmental noise and non-auditory health effects.

17.3.2.3 A search of applicable literature was performed on the impact of aircraft noise on human health. Published documents from previous major airport planning inquiries in various countries, relating to accepted approaches and methodologies used in the performance of an aircraft noise health impact assessment, were also reviewed.

Objectives

17.3.2.4 The purpose of the literature review is to identify key issues and research findings relating to non-auditory effects caused by aircraft noise, focusing on those effects on health and well-being which are caused by exposure to aircraft noise, with the exclusion of effects on the hearing organ and the effects which are due to the masking of auditory information. The review findings were taken

on board as appropriate in developing the methodology for the assessment of potential aircraft noise health impact arising from the operation of the project. To accomplish this objective, the scope of this literature review involves the following tasks:

a) Collection of information on medical and academic research on non-auditory effects caused by aircraft noise, making reference to studies in European countries, Asian countries, the United States, Canada and Australia;

b) Summarization of the major findings of each study and provide information on the study methodology, the type of noise, the nature of exposed population, and any limitations of the study;

c) Analysis of the information collected and the collation of any conclusions made on exposure-response relationships between aircraft noise and non-auditory health effects, and take into account other confounding factors such as age, gender, occupation, etc.

Literature Review Process

Sources of Data

17.3.2.5 The literatures evaluated in this review were identified systematically based on (a) manual search from the lists of references in review articles and other research papers on aircraft noise and its non-auditory health effects; (b) manual search for the journals available in the worldwide web for the most up-to-date articles/papers on related topics; and (c) manual search from libraries of local universities. In the selection of publications for review, emphasis was placed on reviews which have themselves looked over a number of studies such as good practice guidelines.

17.3.2.6 As mentioned in **Section 17.3.2.2**, this literature review has also made incorporated references to the previous literature reviews of environmental noise and non-auditory health effects.

Criteria for Articles / Publications to be Reviewed

17.3.2.7 Literatures obtained in this review included original research articles and review papers published in academic journals in recent years mainly from years 2006 through 2012. Reports, guidelines and other documents from the World Health Organization (WHO) and the European Union (EU) on aircraft noise were also included. The literatures mainly assessed and described the relationship between aircraft noise exposure and human non-auditory responses.

17.3.2.8 A “Literature Evaluation Form” was developed which included criteria by which each document was scored from 0 (none or too few requirements are met) to 4 (nearly all requirements are met), or determined to be not applicable (NA). Examples of criterion included: clear purpose and statement of the problem, evidence is given that the research design/methodology is appropriate for answering study’s questions, sufficient background material is presented, etc. In addition, documents that were published for some years (prior to 2006) were given a lower score if more recent similar documents were available.

Data Extraction and Summary

17.3.2.9 An initial list containing about 70 reports, guidance documents and published papers dealing with environmental noise and non-auditory health effects was developed (see **Appendix 17.3.1**). As an initial step in the review, all reports were first reviewed and categorised into different topics on environmental noise and non-auditory health effects. A preliminary screening of these documents was then performed to identify those studies and reports which were published in peer reviewed journals or recognized research organisations such as the WHO, and which contained adequate information pertaining to specific research methods such as sample size, effect size, and statistical significance of the reported results. The review also included synthesis reports such as European Environment Agency (EEA) Technical Report No. 11/2010: Good Practice Guide on Noise Exposure and Potential Health Effects, October 2010. Based on this initial review, a list of 44 reports, guidelines and published papers was developed for detailed review and analysis. Contents in each paper selected for detailed analysis were extracted in a tabular form including the following information: author(s), title, subjects, study type, confounding factors, influencing covariates, threshold values, major findings, as well as weaknesses of study.

Key Findings of Literature Review

17.3.2.10 The literature review provides a summary of available findings of non-auditory health effects caused by environmental noise. Results of the literature review for the selected documents are summarised in **Appendix 17.3.2**, whilst major findings are presented below.

Health End Points of Key Interest

17.3.2.11 In the assessment of aircraft noise health impact, the WHO's definition of health as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO 1946) is adopted. Two major health endpoints related to environmental noise - annoyance and self-reported sleep disturbance are identified. They are not diseases but represent deviations from the WHO's definition of health. Positive associations between exposure to environmental noise, including aircraft noise and annoyance and sleep disturbance have been well-documented in the literature. These are further described below.

17.3.2.12 For annoyance, positive association was found between self-reported annoyance and aircraft noise exposure in a number of studies. Annoyance is source dependent and generally used to characterise negative emotional responses to aircraft noise. These negative emotional reactions might also be described as anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation or exhaustion (Job 1993; Fields et al 1997, 1998 in Berglund et al 1999). Annoyance as an emotional response is linked with the disturbance of everyday activities such as communication and sleep. Vulnerable groups generally include noise sensitive individuals, and those who have fears with aircraft safety. Exposure to noise or vibration also intensifies annoyance and individuals experiencing stress or mental illness such as anxiety and depression (at clinical or subclinical levels) are likely to report higher levels of annoyance. Measured by means of defined questionnaire, exposure-response relationships between aircraft noise and self-reported annoyance had been developed by some researchers for estimation of proportion of annoyed population with respect to noise exposure levels.

17.3.2.13 With regards to sleep disturbance, again, positive association was found between aircraft noise exposure and self-reported sleep disturbance in many studies. The WHO-Night Noise Guidelines (2009) discusses the relations between noise, sleep quality and health. Exposure-response relationships between aircraft noise and self-reported sleep disturbance had been developed by

researchers for estimation of proportion of sleep-disturbed population with respect to night-time noise levels. Vulnerable individuals include elderly, children and those suffering from physical and mental illness. However, evidence for effects of noise on different sleep stages and habituation of sleep to noise was limited.

17.3.2.14 For both health end points of annoyance and sleep disturbance from aircraft noise, it is noted that well established correlations are observed with reference to the following key literature references:

- Good Practice Guide on Noise Exposure and Potential Health Effects. EEA Technical Report No. 11/2010, October 2010.
- World Health Organization, 2012. Methodological Guidance for Estimating the Burden of Disease from Environmental Noise. Copenhagen, WHO Regional Office for Europe.

17.3.2.15 The first of these (the EEA Technical Report), produced by the Expert Panel on Noise (EPoN) of the European Environment Agency is relevant, since it brings together the best available information on exposure-response relationships for self-reported health effects of annoyance and sleep disturbance. In the scope of work discussion contained in the EPoN document, the following is stated:

"The emphasis is first of all to provide end users with practical and validated tools to calculate health impacts of noise in all kinds of strategic noise studies such as the action plans required by the Environmental Noise Directive (END) or any environmental impact statements. The basis of this is a number of recent reviews carried out by well-known institutions like WHO, National Health and Environment departments and professional organizations."

17.3.2.16 The exposure-response relationships given in the report for both annoyance and sleep disturbance are primarily derived from studies in Europe. Therefore, these exposure-response relationships that are based on self-reported health effects may not be applicable to Asian countries and cities including Hong Kong. Nevertheless, this assessment is to evaluate the effect to the key health endpoints associated to aircraft noise by the project relative to the baseline situation without the project, rather than the absolute number of population to be affected. Therefore, such uncertainties would be reduced and flattened by comparison. Also, such assessment approach and model are widely accepted and employed internationally for the purpose of similar assessments.

Other Potential Health Effects

17.3.2.17 It is noted that other potential health effects associated with exposure to environmental noise discussed in the literature include cardiovascular diseases and cognitive effects on children. The key findings of the literature review are presented below.

17.3.2.18 Regarding cardiovascular effects, it is noted that although the EEA Technical Report and WHO's Methodological Guidance mentioned above have proposed exposure-response curves, there is a lack of overall consistency in findings of reported studies, even often with statistically insignificant results due to a number of confounding factors and thus there is yet sufficient scientific evidence on reliable exposure-response relationship for quantitative nor qualitative analysis.

17.3.2.19 Many studies have been published between years 1980 and 2009 on the relationship between environmental noise, including aircraft noise, and cardiovascular diseases. Two major diseases – ischaemic heart disease (IHD) and hypertension have been studied extensively for their associations with aircraft noise. Some studies show positive associations between the level of noise exposure and the risk of these diseases. However, there is a lack of consistency in the findings of these studies, some with statistically insignificant results, and this applies to aircraft noise in particularly. There are a number of confounding factors (risk factors that may influence the conclusion of a study) for cardiovascular diseases. Part of these studies on hypertension and IHD and aircraft noise did not adjust for these confounding factors that are strong risk factors (e.g., smoking and dietary intake) for these diseases. In some studies, the biologically observable effects, such as a transient increase in blood pressure are small and have little clinical relevance (W. Babisch, 2002). In addition, methodology differences in the assessment of exposure and outcome between studies make it difficult to combine results from different studies (W. Babisch et al. 2009). To sum up, the scientific evidence for a cause-effect relationship between heart diseases and aircraft noise is weak, with statistically insignificant findings at low noise levels. There is more evidence of an association between hypertension and noise in some studies. Specifically, there is growing evidence of a link between high aircraft noise and hypertension in more recent studies and these have been reviewed and discussed in evaluating the potential cardiovascular effects as part of the HIA study. The WHO's overall conclusion is that association of aircraft noise to cardiovascular effect is weak (WHO, 2009). The potential cardiovascular effects were also discussed in the recent ENNAH (European Network on Noise and Health) Final Report 2013, which critically reviewed a number of papers from 1980 to 2013 with an expert survey on strength of evidence for various health outcomes against transportation noise sources including aircraft noise revealing no consensus for cardiovascular diseases and hypertension.

17.3.2.20 On cognitive effects for children, this focuses on primary schoolchildren, some studies concluded effects of aircraft noise on cognitive performance of children but not other groups of the general population.

17.3.2.21 Some reports suggest that the impact of environmental noise is more obvious to children than to adults since the former are more vulnerable to noise and have different perceptions of its dangers (WHO, 2012). Other studies focused on the reversibility of the possible cognitive effects. Based on the Policy Interpretation Network on Children's Health and Environment (PINCHE) research (M. Zuurnier et al. 2007), transport noise was classified as of medium priority. There is evidence to suggest that high aircraft noise level has significant potential to affect children's learning. Learning impacts include reading, memory, auditory discrimination, speech perception and academic performance (WHO, 2009). It should be noted that learning impacts are not directly linked to the health of children. Nevertheless, an assessment of the number of schools that are likely to be affected in their cognitive performance by aircraft noise is made as part of the HIA, as learning plays an important role in the intellectual development of children. The long-term follow-up study shows a non-significant effect on reading comprehension (Clark et al, 2013).

17.3.2.22 A number of laboratory studies indicate that noise may influence learning and performance, but the relationship is complex, as people usually try to keep performance up. This kind of research was primarily carried out in schoolchildren because the potential mechanism of the impact of environmental noise on children's cognitive performance is mainly by affecting the intelligibility of speech communication via loss of meaning or concentration in the content of teachers' instruction (Kempen, 2005 and W. Babisch, 2002). A study on Aircraft and Road Traffic Noise and Children (RANCH study), which involved a cross-national and cross-sectional study of school children in

the age of 9-10 years, was one of the first studies that convincingly demonstrated in a multinational field study that there is a relationship between learning (measured as reading ability) at school and noise exposure. Only a few studies, eg. Matsui et al, 2004, examined the additional effect of noise exposure at home. Moreover, it is considered that learning / studying at home is relatively short compared to that in school. In addition, a variable number of confounding factors are present in the home environment, that may affect learning. For these reasons, there is insufficient evidence of a link between cognitive effects and environmental noise at home.

17.3.2.23 The RANCH study reflects that reading comprehension begins to fall below average at aircraft noise exposure greater than the range around 55 to 60dB Leq. Moreover, WHO provides guidelines on noise levels for school (outdoor playground) of 55dB for effect of annoyance. Hence, Leq of 55dB is selected as a starting point for an effect on the cognitive function of school.

17.3.2.24 The cognitive effects for children were also discussed in the ENNAH Final Report 2013, for various health outcomes against transportation noise sources including aircraft noise. This study did not establish a strong relationship between the aircraft noise and cognitive performance.

17.3.2.25 For hearing impairment, there is evidence that exposure to excessive noise has negative impacts on auditory health, which include hearing and sensory effects. However, hearing impairment only occurs at high intensity (in excess of 85 dBA 8-hour average) which is more common in the workplace, rather than in the general environment. The effect of aircraft noise on hearing impairment is restricted to occupational exposure at the airport, including airline workers, who may be subjected to high noise levels at work and hearing protection would be provided as appropriate. Therefore, it is not considered relevant or of importance in relation to exposure of general population among sensitive receivers in the vicinity of the airport.

17.3.2.26 For speech interference, communication interference would result with the masking effect of noise over the speech signal, causing extraction of the speech sound more difficult for the listener and this may require the speaker to use a raised voice. This would be mainly an effect of communication disturbance. Literatures suggest that speech interference would pose concern under occurrence of flight with noise levels exceeding 70dBA in places with low background noise where communication is important.

17.3.2.27 For effect on task performance, noise would impair a person's ability to perform complex tasks by disrupting communication and impairing cognitive functions relating to recall and memory, as discussed above. Secondary effects of noise exposure might also affect task performance. Performance effects related to noise exposure are complex and are often the results of a primary disturbance such as sleep disturbance. It has been documented in some studies that noise adversely affects cognitive task performance, but the direct link between noise and performance of tasks is not conclusive even for exposure to noise levels in the workplace.

17.3.2.28 For mental illness, from the literature review, there is insufficient evidence that exposure to noise is directly linked to mental health illness. It is suspected that noise may accelerate and intensify the development of latent mental disorder; however, environmental noise such as aircraft noise is not believed to be a direct cause of mental illness.

17.3.3 Methodology for Health Impact Assessment due to Aircraft Noise

17.3.3.1 The steps presented in the following sections are based on the EEA Technical Report described earlier in **Section 17.3.2.15** with respect to annoyance and sleep disturbance for which there are established exposure-response relationships for Europe. The exposure-response relationships may not be applicable to Asian cities including Hong Kong. Nevertheless, as already pointed out in **Section 17.3.2.16**, this assessment is to evaluate the effect to the key health endpoints associated with aircraft noise by the project relative to the baseline situation without the project, rather than the absolute number of population to be affected. Therefore, such uncertainties would be reduced and flattened by comparison.

17.3.3.2 According to the EEA Technical Report, when a defined population is exposed to aircraft noise, a sub-set of that population may notice aircraft noise and develop adverse feelings, such as stress reactions, self-reported sleep disturbance, and other effects. These factors or life stressors may increase health risks in vulnerable members of the population; and smaller sub-set of the vulnerable population may then develop clinical symptoms.

17.3.3.3 Local population data have been retrieved from Planning Department and Census and Statistics Department, especially the future population data is based on TPEDM. For specific sites such as the proposed Tung Chung East and West Development, information on population was based on Theme 1 in the Stage 2 public consultation digest released in May 2013 (available at http://www.tung-chung.hk/tung-chung_PE2-digest_final%20output_20130520.pdf).

17.3.3.4 In order to be responsive to the EIA Study Brief with respect to the required HIA, the following approach and methodology for the HIA have been developed, and these are described in the following sections. Noise data and analyses used in development of the HIA were based upon, and accomplished in tandem with the Aircraft Noise Impact Assessment presented in **Section 7.3**.

Definition of Health End Points

17.3.3.5 Given the broad definition of health referring as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity, the HIA has addressed all relevant health endpoints, including annoyance and sleep disturbance for which there are established exposure-response relationships based on self-reported health effects. Another potential health effect relating to cognitive effects on children has also been considered taking into account the findings of the literature review. Some of these endpoints may be defined as intermediary effects that lead to clinical symptoms. For the purpose of this assessment, “exposure-response” approach, rather than a “threshold-criteria” approach, was adopted because calculations based on exposure-response relationships would give a more complete picture than an assessment based on whether certain thresholds are exceeded. In particular, it is not adequate to assume there should be no incidence of adverse health effects where the noise exposure is below the threshold-criteria. Indeed, there is an increasing incidence of effect with increasing exposure, and this is the aforesaid “exposure-response” relationship. This assessment is to evaluate the effect to the key health endpoints associated with aircraft noise by the project compared to the baseline situation without the project, rather than the absolute number of population that is estimated to be affected.

Selection of Noise Metrics

17.3.3.6 This assessment has made reference to Annex V of the EEA's Technical Report for the criteria for selecting aircraft noise metrics.

17.3.3.7 L_{den} satisfies most of these criteria and is widely adopted for assessment of annoyance and other health endpoints. In addition, research indicated that the night-time period merits special attention. Therefore, L_{night} is selected to characterise noise levels in the night-time period (Laszloa, et al., 2012) and is commonly used to evaluate disturbance to sleep. In addition, $L_{Aeq,16h}$ has been used in studies related to cognitive effects on children. Details of these noise metrics are elaborated below:

- L_{den}: This is the day-evening-night noise level. It is a descriptor of noise level based on energy equivalent noise level (L_{eq}) over a whole day with a penalty of 10 dB(A) for night time noise (2300 to 0700) and a penalty of 5 dB(A) for evening noise (1900 to 2300).

- L_{night}: This is the equivalent continuous noise level over the night-time period (2300 to 0700), which does not contain any night-time noise weighting.

- $L_{Aeq,\ 16hr}$: This is the equivalent sound level (often called equivalent continuous sound level) averaged over 16 hours (0700 to 2300).

17.3.3.8 Annoyance has been assessed using the exposure-response relationships shown in **Appendix 17.3.3**. The L_{den} noise metric is adopted because it is one of the most commonly referenced noise metrics in current research studies for annoyance. It is also the metric used in the exposure-response relationships for annoyance (the Miedema's equations).

17.3.3.9 Self-reported sleep disturbance has been assessed using the exposure-response relationships shown in **Appendix 17.3.3**.This is the relationship given in the EEA Technical Report. The L_{night} noise metric is adopted because it differentiates from noise during the day and it is also one of the most commonly referenced noise metrics in current research studies for sleep disturbance. This is also because it is the metric used in the exposure-response relationship, which is derived from such research.

17.3.3.10 Cognitive effect for children has been assessed based on the results of the RANCH study. It is used to estimate the number of schools subject to such significant increases in noise exposure. The $L_{eq,16hr}$ noise metric is adopted because it is one of the most commonly referenced noise metrics in current research studies.

Prediction of HIA Noise Metrics

17.3.3.11 The Federal Aviation Administration (FAA) Integrated Noise Model (INM) analysis described in the aircraft noise impact assessment (**Chapter 7**) formed the basis of and provided the required noise metrics for the aircraft noise HIA. The planned future standard HKIA operating procedures (as primary operation mode involving a number of aircraft noise mitigation measures including (i) putting the existing south runway on standby where possible at night between 2300 and 0659; (ii) requiring departures to take the southbound route via West Lamma Channel during east flow at night from 2300 to 0659, subject to acceptable operational and safety consideration; (iii) new arrival Required Navigation Performance (RNP) Track 6 designed for preferential use in the

runway 25 direction between 2300 and 0659; and (iv) implementing a preferential runway use programme when wind conditions allow, were used in the analysis. The noise analysis focused on the three-runway system (3RS) scenario, during the future year with the maximum total noise emission as the worst assessment year (termed the "worst operation mode"). This has been determined to be Year 2030 (detailed in **Section 7.3.3.8**). Accordingly, the aircraft noise HIA has been undertaken based on the same assessment year, and the results of the analysis were used to predict the noise exposure metrics associated with the health endpoints described above.

Assessment Area

17.3.3.12 Taking into account the aircraft noise standard adopted in Hong Kong and the findings of the aircraft noise assessment presented in **Section 7.3**, in particular **Drawing MCL/P132/EIA/7-3-007** and **Table 7.3.19**, the noise sensitive populated districts/regions located adjacent to the NEF 25[1] contour line in Year 2030, including Sha Lo Wan, Tung Chung, Northern Lantau, Ma Wan and Siu Lam (including village houses in Tai Lam Chung), are identified as the locations of interest (hereafter collectively referred to as the assessment area) for a quantitative comparison of the 3RS scenario with the two-runway system (2RS) scenario with respect to potential health effects. There are other districts/regions named in the EIA Study Brief for the related aircraft noise impact assessment presented in **Section 7.3**, including Tuen Mun, Tsing Lung Tau, Shatin, Ma On Shan, Tsuen Wan, Sham Tseng, Tsing Yi, Tai Kok Tsui, Yuen Long and Kwai Chung; however, as shown in **Table 7.3.19** which is reproduced in **Table 17.3.1** below, the modeled noise levels at these districts/ regions are substantially below the EIAO-TM aircraft noise criterion of NEF 25 (i.e., << 25).

17.3.3.13 The potential health impacts associated with exposure to aircraft noise are evaluated by estimating the overall change of population potentially affected in the assessment area when the 3RS operation is compared with the "without-project" 2RS scenario in year 2030. Future population data within the assessment area is based on Territorial Population and Employment Data Matrices (TPEDM) provided by the Planning Department and with consideration of relevant site-specific information where available such as the Stage 2 Public consultation digest for Tung Chung New Town Development. The change in population affected within the assessment area under the 3RS and 2RS scenarios are estimated based on future population data, aircraft noise results in terms of noise bands in associated noise metrics, as well as the aforesaid exposure-response relationships given in **Appendix 17.3.3**.

Table 17.3.1: Approximate NEF Ranges at Various Areas in Year 2030 (3RS)

Areas	Approx. NEF Range based on INM Modeling Results in Year 2030
1. Ma Wan	**< 25**
2. Tuen Mun	<< 25
3. Tsing Lung Tau	<< 25
4. Shatin	<< 25
5. Ma On Shan	<< 25
6. Tsuen Wan	<< 25
7. Sham Tseng	<< 25
8. Tsing Yi	<< 25

[1] *Noise Exposure Forecast (NEF) 25 is the noise standard for domestic premises stipulated in Annex 5 of EIAO-TM. Reviewed papers and guidelines adopted for airport planning show that noise levels above NEF25 would be associated with negative impacts on health, including community annoyance and sleep disturbance.*

9. Tung Chung	**< 25**
10. Tai Kok Tsui	<< 25
11. Siu Lam	**< 25 (except a portion in Lok On Pai)**
12. Yuen Long	<< 25
13. Kwai Chung	<< 25
14. Sha Lo Wan	**25 to 30**
15. North Lantau Villages	**25 to 30**

Remarks: **Bolded** areas are identified as locations of interest (hereafter collectively referred to as assessment area) for a quantitative comparison of the 3RS and 2RS scenarios in the HIA. Other areas are subject to aircraft noise levels substantially below the EIAO-TM criterion of NEF25 (i.e. << 25).

17.3.4 Evaluation and Assessment of Potential Health Impacts due to Aircraft Noise

17.3.4.1 Taking into account the findings of the aircraft noise impact assessment in **Section 7.3**, the literature review and methodology presented above, the HIA focused on both annoyance and self-reported sleep disturbance in the assessment area identified in **Section 17.3.3.12**.

Annoyance

17.3.4.2 A Technical Note that presents the approach and key assumptions and data adopted in the calculation of change in affected population within the assessment area is given in **Appendix 17.3.4**. As discussed in **Section 7.3.2.10**, village houses/licensed structures affected by aircraft noise in Northern Lantau areas will be offered the provision of indirect noise mitigation measures in the form of window insulation and air-conditioning before the operation of the third runway and therefore these village houses / licensed structures have been excluded in the calculation of change in affected population within the assessment area.

17.3.4.3 Based on the estimated future population within the assessment area, the change in affected population within the assessment area between the 3RS and the 2RS scenarios in 2030 is estimated for each noise band in the L_{den} noise metric using the exposure-response relationship for annoyance presented in **Appendix 17.3.3**. **Table 17.3.2** presents the findings of the analysis.

Table 17.3.2: Analysis of Annoyance

Predicted Noise Levels, L_{den} (dB)	Estimated Change in Population that might be Highly Annoyed, 3RS - 2RS
45 – 50	-600
50 – 55	7,800
55 – 60	-10,700
60 – 65	-200
65 – 70	0
70 – 75	0
Overall Change in Population	**-3,700**
Overall % Change in Population	**-9.6%**

17.3.4.4 As presented above, the quantitative analysis has revealed that within the assessment area, there would be an overall reduction in future population of about 10% who might be highly annoyed with the implementation of 3RS as compared to the 2RS in year 2030. The assessment results reflected, from the HIA perspective, the effectiveness of the aircraft noise mitigation

measures described in **Section 7.3.3.11** in reducing the population that would be subject to potential annoyance from exposure to aircraft noise.

Self-reported Sleep Disturbance

17.3.4.5 As described above, the Technical Note in **Appendix 17.3.4** has presented the approach and key assumptions and data adopted in the calculation of change in affected population within the assessment area and this is also applicable in the analysis of self-reported sleep disturbance. As indicated above, the village houses / licensed structures in Northern Lantau areas that will be offered the provision of indirect mitigation measures in the form of window insulation and air-conditioning before the operation of the third runway were excluded in the calculation of change in affected population.

17.3.4.6 The approach in estimating the change in affected population within the assessment area with respect to self-reported sleep disturbance between the 3RS and 2RS scenarios is similar to that undertaken for annoyance. **Table 17.3.3** presents the findings of the analysis.

Table 17.3.3: Analysis of Self-reported Sleep Disturbance

Predicted Noise Levels, L_{night} (dB)	Estimated Change in Population who might be Highly Sleep Disturbed, 3RS - 2RS
45 – 50	-900
50 – 55	-8,100
55 – 60	0
60 – 65	0
65 – 70	0
70 – 75	0
Overall Change in Population	**-9,000**
Overall % Change in Population	**-47.8%**

17.3.4.7 The quantitative analysis as presented above has revealed that there would be an overall reduction in population of about 50% who might be highly sleep disturbed among the future population within assessment area with implementation of the 3RS comparing to the 2RS in 2030. As for annoyance presented above, the assessment results reflected, from the HIA perspective, the effectiveness of the aircraft noise mitigation measures described in **Section 7.3.3.11** in reducing the population that would be subject to potential sleep disturbance from exposure to aircraft noise.

Cognitive Effects for Children

17.3.4.8 On cognitive effects for children, a similar analysis of the predicted noise levels in $L_{Aeq, 16hr}$ and consideration of available information on school locations have revealed that no schools would be affected in all noise bands, except down to 55 to 60 dB $L_{Aeq, 16hr}$, within which one educational institute (kindergarten, although studies mainly focus on primary schoolchildren) is observed within the assessment area in Siu Lam under the 3RS scenario. However, it is considered that cognitive effects to students associated with aircraft noise in this institute would unlikely be significant, as this would be masked by the typical outdoor background noise levels of L_{eq} 60 dB, which was the noise level measured during daytime onsite.

Other Potential Noise Effects

17.3.4.9 The other potential health effects associated with exposure to aircraft noise, including cardiovascular diseases, hearing impairment, speech interference, task performance and mental health effects are not likely to reach exposure levels that have been identified as having potential effects, or there is a lack of overall consistency or statistically insignificant results that would provide sufficient scientific evidence on reliable exposure-response relationship for analysis.

17.3.5 Health Impact Reduction by Aircraft Noise Mitigation Measures

17.3.5.1 As per the EIA Study Brief requirements, an identification of means by which the health impact could be further reduced is required.

17.3.5.2 Concerning the key health end points of annoyance and self-reported sleep disturbance as identified in the above, the health impact assessments for both have reflected overall positive improvements under the 3RS operation, comparing to the without project scenario (with about 10% and 50% reduction of population that would be highly annoyed and highly sleep disturbed, respectively) within the assessment area.

17.3.5.3 Moreover, as indicated in **Section 17.3.3.11**, the assessment scenario takes into account the planned future standard HKIA operation procedures (as primary operation mode involving a number of aircraft noise mitigation measures as listed below):

- Putting the existing south runway on standby where possible at night between 2300 and 0659;
- Requiring departures to take the southbound route via West Lamma Channel during east flow at night from 2300 to 0659, subject to acceptable operational and safety consideration. This is an arrangement that is consistent with the existing requirement in the operation of the two-runway system at night;
- A new arrival Required Navigation Performance (RNP) Track 6 has been designed for preferential use in the west flow direction (i.e., runway 25 direction) between 2300 and 0659 and it is assumed that up to 95% of flights may preferentially use this new Track 6 instead of the existing straight-in tracks by year 2030; and
- Implementing a preferential runway use programme when wind conditions allow such that west flow is used when departures dominate while east flow is used when arrivals dominate during night-time.

17.3.5.4 Besides, as discussed in **Section 7.3.4.9** of the aircraft noise impact assessment, it is considered that all practicable direct mitigation measures have been evaluated, adopted and exhausted (such as control of night flight movement over residential area, restriction of aircraft type in night-time period, use of Required Navigation Performance system etc.). Therefore, the aircraft noise impact and associated health impact are considered to be reduced as much as practicable during the operation of the project.

17.3.6 Recommendation of Reasonably Practicable Measures, if any

17.3.6.1 As mentioned above, it is considered that the planned future airport operation has already exhausted all reasonably practicable measures to reduce health impact associated with aircraft noise.

17.3.7 Uncertainties and Limitations

17.3.7.1 Uncertainties and limitations are considered in this health impact assessment, which might include but not limited to, air traffic and population forecast, assumed operation mode, occurrence / variability of human experience / responses to aircraft noise, and the annoyance and sleep disturbance exposure-response relationships occurrence / variability of human experience / responses to aircraft noise.

17.3.7.2 Having considered the above possible uncertainties and limitations, it is considered that most of the possible uncertainties and limitations are introduced by assumptions in forecasts, which have been set in a conservative manner.

17.3.7.3 The exposure-response relationships may not be applicable to Asian cities including Hong Kong. Nevertheless, this assessment is to evaluate the effect to the key health endpoints associated with aircraft noise by the project relative to the baseline situation without the project. The uncertainties of the assessment methodology would be reduced and flattened by the comparison of the 3RS with the 2RS scenario. Based on the information, technology and resources available during the course of assessment, the approach is considered appropriate with reasonably acceptable uncertainties and limitations in place.

17.3.8 Conclusions

17.3.8.1 As discussed in **Section 17.3.4** above, there will be an overall reduction in future population that would be subject to annoyance and self-reported sleep disturbance (with about 10% and 50% reduction of population affected respectively), with implementation of the third runway in the assessment area.

17.3.8.2 Considering cognitive effect on children by aircraft noise, it is noted that one kindergarten is within the noise band of 55 to 60 dB within the assessment area in Siu Lam under the three-runway scenario. However, it is considered that cognitive effects on students in this institute would unlikely be significant, as the aircraft noise levels would be masked by the background noise level of 60 dB measured onsite.

17.3.8.3 Considering the overall improvements in the identified assessment area with respect to both annoyance and self-reported sleep disturbance by implementation of the third runway while cognitive effect on children arising from the operation of the project is not apparent, it is concluded that the overall health impact associated with aircraft noise from the project in the assessment area is minimal.

17.4 References

Aircraft and Airport-Related Hazardous Air Pollutants: Research Needs and Analysis, ACRP, 2008.

A Method to Estimate the Chronic Health Impact of Air Pollutants in .US. Residences, Ernest Orlando Lawrence Berkeley National Laboratory, 2011.

Air Pollution and Health: A European Information System (APHEIS), Health Impact Assessment of Air Pollution and Communication Strategy.

Airport Cooperative Research Program Report 7: Aircraft and Airport-Related Hazardous Air Pollutants: Research Needs and Analysis, Transport Research Board, 2008.

Air Quality Manual, ICAO, 2011.

Agreement No. CE 29/2008 (EP) Engineering Investigation and Environmental Studies for Integrated Waste Management Facilities Phase 1 – Feasibility Study, EPD, 2011.

Agreement No CE 57/2006 (EP) Review of Air Quality Objectives and Development of a Long Term Air Quality Strategy for Hong Kong - Feasibility Study, ARUP/EPD, 2009.

APHES: Health Impact Assessment of Air Pollution and Communication Strategy. Third Year Report. ISBN: 2-11-094838-8; Institut de Veille Sanitaire; June 2005. Downloaded from: http://www.apheis.org/vfbisnvsApheis.pdf]

Assessment of Toxic Air Pollutant Measurements in Hong Kong, EPD, 2003.

CAFÉ Program Methodology for the Cost-Benefit analysis for CAFE: Volume 2: Health Impact Assessment, AEA Technology Environment / European Commission DG Environment, 2005.

EIA report for development of the Integrated Waste Management Facilities Phase 1, EPD, 2011.

EIS / MDP for the New Parallel Runway (NPR) of Brisbane Airport Volume D7 Health Impact Assessment, 2007:
http://www.bne.com.au/corporate/major-projects/new-parallel-runway-project/eismdp

Environmental Impact Statement for Fort Lauderdale – Hollywood International Airport, FAA, 2008.

Evaluation and use of epidemiological evidence for environmental health risk assessment, WHO, 2000.

General Aviation Airport Air Monitoring Study, USEPA , 2010.

Health Impact Assessment for Finningley Airport, Doncaster Health Authority, 2005.

Health Impact Assessment for Schiphol Airport, National Institute of Public Health, Bilthoven, 1994.

Hospital statistical Yearbook 2011 – 2012, Hospital Authority Hong Kong

High-Priority Compounds Associated with Aircraft Emissions PARTNER Project 11 final report on subtask: Health Risk Prioritization of Aircraft Emissions Related Air Pollutants, Partnership for AiR Transportation Noise and Emissions Reduction An FAA/NASA/Transport Canada sponsored Center of Excellence, 2008.

Human Health Risk Assessment Technical Report for LAX Master Plan EIS / EIR, Camp Dresser & Mckee Inc., 2001

Human Health risk Assessment of air emissions from proposed biosolids incinerator, The City of Hamilton, US, 2010.

Huijbregts, MAJ, Rombouts, LJA, Ragas, AMJ, van de Meent, D. 2005. Human-toxicological effect and damage factors of carcinogenic and noncarcinogenic chemicals for life cycle impact assessment. Integrated Environmental Assessment and Management 1(3): 181-244.

McDonnell WF *et al.* Relationships of mortality with the fine and coarse fractions of long-term ambient PM10 concentrations in nonsmokers. Journal of Exposure Analysis and Environmental Epidemiology 2000;10:427-436;

O'Hare Modernization Final Environmental Impact Statement, Chicago O'Hare International Airport, 2005.

Pope, A.C., et al., Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution, Journal of the American Medical Association, 2002.

Santa Monica Airport Health Impact Assessment (HIA), UCLA, 2010.

Select Resource Materials and Annotated Bibliograpjy on the Topic of Hazardous Air Pollutants (HAPs) Associated with Aircraft, Airport, and Aviation, FAA, 2003.

Source Identification and Base Year 1990 Emission Inventory Guidance for Mobile Sources HAPs on the OAQPS List of 40 Priority HAPs, USEPA, 1997.

The Stansted Generation 2 Project – A health Impact Assessment, ERM, 2008.

Health Impact Assessment Schiphol Airport, The National Institute of Public Health and Environmental Protection (RIVM), 1994.

Health Assessment Document For Diesel Engine Exhaust, USEPA, 2002

USEPA's Air Toxics Risk Assessment Reference Library - Volume 2 Facility-Specific Assessment (EPA-453-K-04-001B), 2004.

Wong, C.M., McGhee, S.M., Yeung, R.Y.T., et al, Final Report for the Provision of Service for Study of Short-Term Health Impact and Costs due to Road Traffic Air Pollution. EPD Study Report, 2002a.

Wong, C.M., Atkinson, R.W., Anderson, R., Hedley, A.J., and Ma, S., Chau, P.Y.K., Lam, T.H., A Tale of Two Cities: Effects of Air Pollution on Hospital Admissions in Hong Kong and London Compared, Environmental Health Perspectives, Volume 110, 2002b.

Wong, CM, Thach TQ, Chau PYK, Chan EKP, Chung RYN, Ou C-Q, Yang L, Peiris JSM, Thomas GN, Lam TH, Wong TW, Hedley AJ. Public Health and Air Pollution in Asia (PAPA): Coordinated studies of short-term exposure to air pollution and daily mortality in four cities. Part 4: Interaction between air pollution and respiratory viruses: Time series study of daily mortality and hospital admissions in Hong Kong. Research Report No. 154, Health Effects Institute, Boston, MA, U.S.A. November 2010.

Wong TW, Tam W, Yu ITS, Wun YT, Wong AHS, Wong CM. Association between Air Pollution and General Practitioner Visits for Respiratory Diseases in Hong Kong. Thorax 2006; 61:585-591.