Environmental Research Consultancy Department

Aircraft Noise and Health Effects – a six-month update

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Chapter 1

Introduction

1.1 This report is an update on recent work and findings in the field of aircraft noise and health effects. It covers published research between March and September 2025, and includes findings presented in published academic papers and relevant conferences during that period.

1.2 The aim of the report is to provide a succinct overview of new work relating to aviation noise and health, and such updates are published on a six-monthly basis. This report has been published to provide the public and the aviation industry with a concise and accessible update on recent noise and health developments. It should be noted that the CAA has not validated any of the analysis reported at the conferences, nor takes any view on their applicability to UK policy making. With thanks to Ben Fenech from the UK Health Security Agency for his assistance with some of the source material.

1.3 The findings in the following chapters are grouped by conference and subject area.

Chapter 2

Euronoise 2025

2.1 The Euronoise congress was held in Malaga, Spain, in June 2025. This chapter includes the relevant findings relating to aircraft noise and health effects presented at the Congress.

Annoyance

2.2 Two papers were focused on the effects of aircraft noise on annoyance. The first was by **Merino-Martínez and Buzețelu** and examined the analysis of aircraft noise-induced annoyance using psychoacoustic listening experiments. The aim of the study was to identify sound metrics that best predict aircraft noise-induced annoyance, using psychoacoustic listening experiments and correlation analysis between annoyance ratings and various sound metrics.

2.3 The data from 60 aircraft flyover recordings (30 take-offs, 30 landings) from Schiphol Airport were scaled to simulate an overhead altitude of 1500m (with sound pressure levels between 49.4- 70dBA). The selected audio files for the listening experiment had a duration of 16 seconds for take-offs and 10 seconds for landings. 30 participants were included in the study at Delft University, Netherlands, and each rated 40 audio recordings using the ICBEN 11-point annoyance scale (0 = not annoyed, 10 = extremely annoyed).

2.4 The metrics analysed included conventional noise metrics such as Effective Perceived Noise Level (EPNL), Sound Exposure Level (SEL), Sound Pressure Level (SPL), Maximum noise level (L_{max}), together with Sound Quality Metrics (SQMs) such as Loudness, Sharpness, Tonality, Roughness, and Fluctuation Strength. The data was analysed alongside various psychoacoustic annoyance models including those by Zwicker, Di, More et al.

2.5 The results indicated that take-offs were perceived as slightly more annoying than the landings, with overall average annoyance ratings of 6.11 and 5.75, respectively. It was also noticed that the spread in the obtained annoyance ratings is larger in the case of take-offs, with most of the responses generally concentrated between 5 and 7, suggesting that aircraft noise caused medium to high annoyance in the study. The psychoacoustic annoyance metrics were found to outperform conventional noise metrics and individual sound quality metrics in predicting annoyance, particularly those by Zwicker and Di. Although loudness is a strong individual predictor, combined models are superior in terms of explaining the variance in annoyance findings. In addition, non-linear functions (logarithmic, logistic, hyperbolic tangent) further improve the annoyance prediction performance, and better capture annoyance trends.

2.6 The authors suggest that future work should increase the dataset to further validate these findings in a larger sample size.

2.7 **Reedijk et al** presented findings on the exposure relationships between aircraft noise, annoyance and sleep disturbance around Dutch military and civil airports. The study was conducted by the National Institute for Public Health and the Environment (RIVM) in the Netherlands and collected questionnaire data on annoyance and sleep disturbance from various environmental sources including aircraft. The study was conducted in autumn 2020 on residents aged 18 years or older. The objective was to derive exposure-response (ER) relationships between aircraft noise and high annoyance/sleep disturbance for 14 Dutch airports (civil and military).

2.8 The data was obtained from the Municipal Public Health Service Health Monitor 2020 with 262,310 participants. Aircraft noise levels were modelled by the Netherlands Aerospace Centre using a 100x100m grid, using L_{den} (daytime annoyance) and L_{night} (sleep disturbance) metrics.

2.9 Participants rated annoyance and sleep disturbance on an 11-point ISO scale (0-10); scores ≥ 8 were considered “high”. Residential addresses were linked to modelled noise exposure. Population-weighted logistic regression models (both linear and non-linear spline models[1]) were used to derive ER relationships.

2.10 The airports included 14 civil and military airports:

- Civil: Schiphol, Rotterdam, Lelystad, Groningen, Maastricht Aachen.
- Military/Civil: Eindhoven, De Kooy.
- Military: Gilze-Rijen, Volkel, Leeuwarden, Woensdrecht, Deelen, De Peel, Geilenkirchen.

2.11 For Airbase De Peel (no flights in 2020), Airbase Deelen, Maritime Air Base de Kooy, Groningen Airport Eelde and Lelystad Airport it was not possible to derive airport specific ER relationships due to exposure distribution (too low or too small range) and or limited number of residents at higher exposure levels.

2.12 The results suggested that there was a higher rate of annoyance and sleep disturbance in 2020, and that compared to the 2002 Schiphol study, more residents reported high annoyance and sleep disturbance at similar noise levels. There was significant variation in ER relationships across airports, which the authors suggest is likely due to both acoustic and non-acoustic factors.

2.13 The regression analysis indicated that non-linear spline models had a better fit than linear models, especially for Schiphol Airport in this study, possibly due to the non-linear model being more flexible and therefore able to predict the relationship more accurately. The authors suggested that the COVID-19 pandemic may have impacted the responses in this study due to the reduced

[1] A spline model allows for adaptation to the data, rather than assuming a straight line as with a linear model.

civil air traffic in 2020 affecting exposure levels and therefore the survey responses. It is also highlighted that the analysis of non-acoustic factors such as noise sensitivity, attitudes towards the airport etc was not possible due to factors such as these not being included in the original questionnaire. The authors suggest a repeat data collection post-COVID (e.g. for 2024) to improve estimates with the inclusion non-acoustic factors for deeper understanding. They also plan to expand this research to rail and road traffic noise.

Other papers

2.14 The other relevant presentations are described in this section. The first was by **Clark et al** which was a review and discussion of the evidence, gaps and research needs for quantifying transportation noise impacts on mortality. The purpose of the review, commissioned by the UK Health Security Agency, was to assess the exposure-response relationships between road, rail, and aircraft noise and all-cause mortality, and to support health impact assessments and burden of disease models in the UK and Europe.

2.15 The umbrella+ review conducted combined systematic reviews (Search 1) and recent original studies (Search 2) for studies published between January 2015 and November 2024. The inclusion criteria focused on residential noise exposure to the three transportation noise sources, and all-cause mortality outcomes.

2.16 Three review papers and one original research study were identified, providing evidence from eight prospective cohort (longitudinal) epidemiological studies on the effects of transportation noise on all-cause mortality (7=road, 2=rail, and 1=aircraft). The sample sizes were approximately 17 million for road traffic, 13 million for railway noise, and 117,000 for aircraft noise. Most were conducted in Europe (seven), with three of the studies in Denmark and the aircraft review was conducted in the US by Grady et al. The Grady study was a prospective follow-up of nurses in the Nurses Health Study (n=53,306) and the Nurses Health Study II (n=60,058) in the USA from 1994-2014. Aircraft noise was modelled for 90 airports.

2.17 Road traffic noise showed the strongest and most association with all-cause mortality with a relative risk (RR) of 1.055 [1.026-1.084] and the certainty of evidence being rated as high. Aircraft noise evidence was limited and less conclusive, with a RR of 1.03 [0.94-1.12] and very low certainty of evidence, and railway noise showed a small but statistically significant association RR=1.004 [1.001-1.007] and low certainty of evidence.

2.18 The authors stress there are limitations with this review. The low ratings for all reviews were assigned due to methodological reporting issues (not necessarily study quality). There was a lack of control for multiple noise sources in most studies, with the risk of double-counting. There is a risk of geographical bias due to most studies being conducted in Europe with limited data from other regions. Aircraft noise evidence is sparse and uncertain.

2.19 The authors suggest that road traffic noise RR could be used in burden of disease models (e.g., EEA assessments) based on the evidence in this review. More research is needed on aircraft and railway noise, non-European populations, and combined noise exposures and confounding factors.

2.20 **Van Kempen et al** presented work exploring whether existing meta-analyses on the relationship between transportation noise and stroke should be updated and proposed a framework to guide such decisions. The study was commissioned by the UK's Interdepartmental Group on Costs and Benefits noise subject group (IGCB(N)). The purpose was to examine whether to update the WHO Environmental Noise Guidelines meta-analyses on stroke and diabetes. The study used stroke outcome as a case study to develop a decision-making framework.

2.21 The rationale was that meta-analyses are powerful tools for summarising evidence but updating them is time consuming and resource intensive. Reasons to update meta-analyses include the addition of new studies with better methods or more data, changes in effect estimates, and policy needs or scientific advancements. However, no standard framework exists for deciding when an update is necessary, especially in environmental epidemiology. Due to a lack of an existing standard, the authors developed a framework that could be used to decide upon an update of a meta-analysis, in this case using transportation noise (road, rail and aircraft) and stroke.

2.22 The framework considered the following elements:

- Availability of newer systematic reviews/meta-analyses.
- Quality of these newer reviews.
- Methodological quality of included meta-analyses.
- Currency of the information (how up-to-date it is).
- Discordance between old and new findings.
- Likelihood of changed conclusions if new evidence is included.

2.23 The original review by Van Kempen et al (2018) was included in the WHO guidelines and was used as the basis for the study. New studies and meta-analyses have emerged since 2014, and nine more recent systematic reviews/meta-analyses were identified (2016–2024). Improvements in exposure assessment and confounder control were noted by the authors, and preliminary analysis indicated variation in quality, methods, and findings.

2.24 The authors found that some newer reviews registered protocols and used robust methods in their methodology, though quality assessment tools varied, and meta-analysis techniques and bias detection methods were inconsistent. The study is still ongoing, and the next steps include evaluating how much new evidence was missed by older reviews. An assessment of whether new evidence would change conclusions will also be undertaken and then if justified, the authors will proceed with an updated meta-analysis on transportation noise and stroke.

2.25 **Röösli et al** presented up-to-date epidemiological evidence on health effects from transportation noise for burden of disease assessment. This is a comprehensive review and meta-analysis of recent studies on the health impacts of transportation noise (road, railway and aircraft) with the aim to update exposure-response functions (ERFs) for use in European burden of disease assessments.

2.26 The objectives of the study were to review recent epidemiological evidence on transportation noise and health, rate the certainty of evidence for causal associations, conduct meta-analyses for key health outcomes, and then derive updated ERFs, including effect thresholds and relative risks.

2.27 Although aircraft noise was included in this review, the evidence base was more limited compared to road traffic noise. For cardiovascular disease, five original studies on aircraft noise and cardiovascular outcomes were identified. These studies were included in the meta-analysis, but detailed pooled estimates were not provided in the main paper. The authors suggest that effect estimates for aircraft noise differ from those for road and rail noise, possibly due to different diurnal patterns (e.g. night flights), and lower population exposure, leading to reduced statistical power. In addition, exposure misclassification may occur, especially where aircraft noise overlaps with road traffic noise.

2.28 For all-cause mortality the authors noted that little research has been conducted on aircraft noise and all-cause mortality. The certainty of evidence for aircraft noise was classed as lower than for road traffic noise, possibly due to methodological challenges such as limited sample sizes, lower exposure prevalence and possible masking by road noise.

2.29 The health outcomes, and RRs from meta-analysis of road traffic studies on adults were as follows: (Relative risks refer to a 10 dB increase in L_{den}).

- All-cause mortality: RR = 1.055 (strong evidence)
- Ischemic heart disease: RR = 1.041
- Heart failure: RR = 1.041
- Stroke: RR = 1.046

- Arrhythmia: RR = 1.006
- Hypertension: RR = 1.045 (low certainty)
- Diabetes: RR = 1.062 (strong evidence)
- Depression: RR = 1.038 (moderate to strong evidence)
- Dementia: RR = 1.052 (moderate to strong evidence)

2.30 For children, the following RRs were found:

- Total behavioural difficulties: RR = 1.073
- Hyperactivity/inattention: RR = 1.047
- Overweight/obesity: RR = 1.063
- Cognition: Moderate evidence (meta-analysis not feasible due to heterogeneous measures)
- Birth outcomes: Low/very low certainty (e.g., low birth weight, preterm birth)

2.31 The authors propose that the effect threshold for the quantification of negative health impacts from transportation noise be reduced to 45 dB L_{den}. New evidence shows effects at such levels in about 50% of the studies. It is also proposed that exposure–response relationships derived for road traffic noise may be used as proxies for aircraft and railway noise in burden of disease assessments, until more empirical data is available from these noise sources.

2.32 **Song et al** presented findings on the measurement of noise sensitivity. It is well understood that noise sensitivity is an important non-acoustic factor that requires consideration in noise effects research, yet the main resources for measuring sensitivity have not been statistically compared in the same data sample. The aim of this study was to provide psychometric evaluation of three major noise sensitivity questionnaires:

- WNSS (Weinstein Noise Sensitivity Scale) 21 items.
- LEF (Individual Noise Sensitivity Questionnaire) 52 items.
- NoiSeQ (Noise Sensitivity Questionnaire) 35 items.

2.33 The study was conducted via an online survey with 305 UK participants. The questionnaires were analysed for internal consistency, factor structure, and correlations with long-term annoyance from six noise sources, including aircraft noise.

2.34 The results showed high internal consistency for the three noise sensitivity questionnaires and strong inter-correlations (all r > .83) of their overall scores. The three questionnaires showed weak to moderate positive correlations with long-term noise annoyance ratings. Participants rated their annoyance from six noise sources over the past 12 months, including aircraft noise, using the 11-point ISO scale. Correlations between noise sensitivity and aircraft noise annoyance were found to be positive, but weak to moderate (ranging from 0.11 to 0.43 across instruments). The authors suggest that this is lower than expected compared to previous studies due to the retrospective nature of the annoyance ratings and lack of actual exposure data. The study confirms that noise sensitivity is a distinct psychological trait, measurable with high reliability, and provides support for the psychometric quality of the established noise-sensitivity questionnaires.

Chapter 3

Internoise 2025

3.1 The Internoise 2025 was held in Sao Paulo, Brazil in August. There was a paucity of presentations on aircraft noise and health effects this year, but the following two paper was presented.

3.2 **Clark et al** presented some preliminary findings from a pilot study on the impact of Heathrow's noise insulation scheme on quality of life (QoL) and health. The pilot study commenced in 2023 and evaluated the health and quality of life impacts of Heathrow Airport's Residential Insulation Scheme (RIS), which aims to mitigate aircraft noise exposure for residents living near the airport. The study was designed as a before-and-after longitudinal assessment, with data collected at baseline, 3 months, and 12 months post-intervention.

3.3 The health and QoL outcomes assessed included annoyance, sleep disturbance, self-reported general health, QoL, wellbeing and noise sensitivity. The pilot study included 41 participants who were eligible for the RIS. The majority were male (61%) and Asian (59%). Most lived in semi-detached or end-terrace homes, with 98% being homeowners and 61% had lived in their homes for over 10 years.

3.4 The findings after the three month follow up suggested there was improvements in noise annoyance, with mean scores decreased significantly across 24-hour, daytime, and nighttime periods, and the proportion of Highly Annoyed (HA) residents dropped from ~68% to ~15-22%. In relation to sleep disturbance, mean scores reduced and the proportion of Highly Sleep Disturbed (HSD) dropped from 46% to 15%. There was no significant change found for QoL, general health, or wellbeing (life satisfaction, anxiety, happiness).

3.5 The results suggest that annoyance and sleep disturbance are sensitive and responsive to noise insulation interventions, but QoL and wellbeing may require longer follow-up periods, or be influenced by broader life factors. The authors propose that the large effect sizes found for annoyance and sleep disturbance suggest strong potential for public health benefit if replicated in larger samples. The study supports policy relevance of noise insulation as a health intervention, especially in aviation contexts.

3.6 Limitations included a small sample size, a relatively short follow-up period (3 months), the inclusion of English-speaking participants only, few flats included in the study, and the outcomes being self-reported.

3.7 The next steps of the study are to complete a 12-month follow-up and expand the sample size and diversity. The authors also intend to explore cost-effectiveness and user experience and further assess the equity of effects across demographic groups with differing socioeconomic, health, and ethnic vulnerability to the effects of noise on health.

Chapter 4

Aircraft Noise and Other findings

4.1 This chapter outlines the other findings from published research on aircraft noise and health effects during the six months between March and September 2025.

Sleep disturbance

4.2 **Wicki et al** published findings on the associations of exposure to transportation noise with sleep and cardiometabolic health. This was a cross-sectional study with the aim to investigate how chronic exposure to different types of transportation noise (road traffic, railway, and aircraft) affects cardiometabolic health in healthy adults, with a focus on sleep efficiency as a potential mediator.

4.3 527 healthy Swiss adults (aged 20-89) participated in the study. Data was collected on cardiometabolic biomarkers, accelerometer-based sleep and physical activity, and noise exposure data was modelled at participants' home address. For each noise source, the following conceptual model was tested using Structural Equation Modelling (SEM)[2]: Noise was assumed to affect the two latent constructs *metabolic risk* (defined by waist-to-hip ratio and HDL-cholesterol) and *cardiovascular risk* (defined by systolic blood pressure and pulse wave velocity) directly, as well as indirectly via sleep efficiency.

4.4 The findings indicated that road traffic noise was directly associated with increased cardiovascular risk (β = 0.16, 95% CI: 0.01–0.31). There was no significant association with sleep efficiency or metabolic risk. The authors explain this suggests non-sleep-mediated pathways are present (e.g., autonomic arousal, oxidative stress).

4.5 Railway Noise was associated with reduced sleep efficiency (β = 0.15, 95% CI: 0.23–0.06) and was also associated with increased metabolic risk (β = 0.14, 95% CI: 0.05–0.32). Sleep efficiency partially mediated the effect on metabolic risk (10% mediation), and no meaningful association with cardiovascular risk was evident.

4.6 Aircraft noise showed no significant associations with sleep, metabolic, or cardiovascular risk. The authors concluded that road traffic noise increases cardiovascular risk independently of sleep, and railway noise affects metabolic health via sleep disruption. They propose that these findings underscore the need for source-specific noise mitigation strategies and further research into non-sleep pathways like stress and annoyance.

[2] Structural Equation Modelling (SEM) is a multivariate statistical method that combines aspects of factor analysis and multiple regression to examine causal relationships among variables. It allows researchers to test theoretical models that include both direct and indirect effects.

4.7 **Uyhelji et al** investigated biomarkers for noise-induced sleep disruption. This FAA-funded study investigated molecular biomarkers associated with sleep disruption caused by aviation noise, using Ribonucleic Acid (RNA) sequencing of blood samples from human participants exposed to simulated aircraft noise. It also evaluated mitigation strategies like earplugs and pink noise (broadband sound) at 40 and 50 dBA.

4.8 The study included 26 healthy adult participants, who each spent 7 consecutive nights in a sleep laboratory. The first was an adaptation night (no data collected).

4.9 They were then exposed to six randomised exposure nights with the following auditory conditions:

- No noise (control group)
- Aircraft Noise (AN)
- AN + earplugs
- AN + pink noise at 40 dBA
- AN + pink noise at 50 dBA
- Pink noise alone at 50 dBA

4.10 Participants were exposed to 93 events per night, including noise from jets, helicopters, drones, sonic booms, alarms, etc played at 45, 55, or 65 dBA. The objective measurements taken included polysomnography for analysis of sleep stages, awakenings, and arousals. Blood pressure and heart rate variability were also measured. Subjective measures of sleep quality were assessed by surveys. Performance was measured by a driving simulation task and cognitive performance tests. Morning blood samples were provided for the RNA sequencing.

4.11 The gene expression was analysed by noise exposure category, and 1,246 genes were found to be differentially expressed across all conditions. The results indicated that only Aircraft noise + pink noise at 40 dBA versus the control group showed significant changes (1,306 genes). No significant changes were found between: AN versus control, AN vs. AN + earplugs or AN + pink noise at 50 dBA, or pink noise alone at 50 dBA versus the control group.

4.12 When analysed alongside the sleep metrics, 2,181 genes were associated with awakenings per hour during noise periods. No other polysomnographic metrics (e.g., REM, sleep efficiency) showed associations.

4.13 Gene expression was also analysed against the physiology/performance variables tested and the results revealed that very few genes were associated with blood pressure, heart rate variability, driving performance, or cognitive speed.

4.14 The authors concluded that analyses of gene expression changes did not reveal an impact of simulated aircraft noise as compared to a control night without noise exposure, with zero genes differentially expressed between these two conditions. They propose that further research is needed to understand the reason the condition aircraft noise + pink noise at 40 dBA (and not aircraft noise without mitigation) resulted in an impact on gene expression. They explain that additional work is also required to understand the absence of a response in analyses with 50 dBA pink noise, and more testing with a larger sample size would be required.

4.15 It is suggested that the participants were given an 8-hour sleep opportunity, which might have meant the noise exposure used in this study did result in severe enough sleep disruption to have a notable molecular impact. A large number of genes changed in association with awakenings during noise events, which could indicate that awakenings during noise are a strong trigger for molecular changes when noises are sufficient to cause a participant to awaken from sleep. The authors suggest that these biological indicators can complement traditional sleep metrics in evaluating noise effects. It is also possible that pink noise may be a potential, low-cost noise mitigation strategy.

4.16 **Garg** published a narrative review article on environmental noise and cardiovascular health. The review discusses current knowledge on how environmental noise, particularly from transportation sources including aircraft, affects cardiovascular health. It covers molecular mechanisms, sleep disruption, epidemiological evidence, and public health implications.

4.17 The acute and chronic physiological mechanisms of noise exposure effects are discussed. Acute effects occur when noise activates the autonomic nervous system and HPA axis, leading to increased stress hormones (catecholamines, glucocorticoids), elevated blood pressure, and vascular changes. Chronic Effects occur as a result of long-term exposure, which promotes oxidative stress, inflammation, and vascular dysfunction, contributing to atherosclerosis and other cardiovascular effects.

4.18 During sleep, noise at night impairs sleep architecture, reducing Rapid Eye Movement (REM) and slow-wave (restorative) sleep, increasing arousals, and elevating nocturnal blood pressure. These disruptions are strongly linked to increased cardiovascular risk.

4.19 The epidemiological evidence for aircraft noise was discussed:

- Hypertension: Aircraft noise exposure is associated with a 3% increased risk per 10 dB increase in nighttime levels. The HYENA study and DEBATS study confirmed stronger associations for night-time exposure than daytime. Gender differences suggest men may be more susceptible, and annoyance is a potential modifier of risk.

- Coronary Heart Disease (CHD): Aircraft noise contributes to myocardial infarction (MI) risk, especially at levels above 65 dBA. Risk increases with duration of residence and cumulative exposure.
- Stroke: Aircraft noise is linked to haemorrhagic stroke, particularly when multiple night-time events exceed 50 dB. This suggests that event frequency and pattern (not just average levels) are important.
- Atrial Fibrillation & Heart Failure: Long-term aircraft noise exposure is associated with increased risk of atrial fibrillation and congestive heart failure, independent of air pollution.

4.20 Combined exposures were discussed, and the challenge of aircraft noise often co-occurring with air pollution, complicating causal attribution of health effects. The author explained that studies show independent and synergistic effects on cardiovascular outcomes, and that oxidative stress is a shared pathway, contributing to cardiovascular disease.

4.21 Garg concluded that environmental noise is a significant cardiovascular risk factor, especially during the night, and in sensitive individuals. Its effects are mediated through stress responses, sleep disruption, and vascular inflammation. Public health strategies should prioritise noise reduction, particularly in urban and airport-adjacent communities, to mitigate long-term health impacts.

Annoyance

4.22 This section outlines the main findings relating to aircraft noise and annoyance that have been published in the six months between March to September 2025. The first paper is by **Nguyen et al** and examines annoyance and sleep disturbance related to changes in aircraft noise in Vietnam. This was a longitudinal study (2019–2023), investigating how aircraft noise exposure affects annoyance and sleep disturbance among residents near Tan Son Nhat International Airport (TSN) in Ho Chi Minh City, Vietnam. The aim was to examine the effects of noise reduction during the COVID-19 pandemic and its subsequent recovery, while also exploring the role of non-acoustic factors.

4.23 The study ran in four survey waves: 2019 (pre-pandemic, N = 502), June 2020 (N = 145), September 2020 (N = 519), and August 2023 (N = 329). There were 12 survey sites located under landing/take-off paths, peripheral zones, and control areas. The surveys were completed during face-to-face interviews with one adult per household and included the ISO 11-point scale for annoyance and a scale for insomnia.

4.24 The Integrated Noise Model (INM) was used to estimate L_{den} and L_{night} metrics, validated with flight data and on-site measurements. Noise contours were also mapped for each survey period. Flight numbers dropped from 728 per day in 2019 to 299 per day in Sept 2020.

4.25 Figure 1 illustrates the data for Annoyance (%HA) versus L_{den} for each of the study periods.

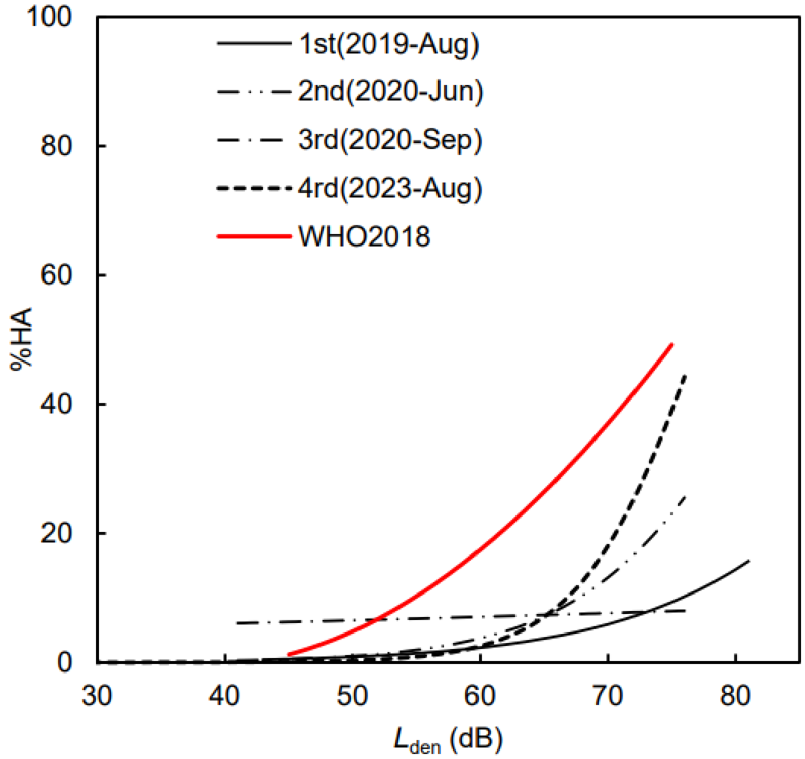

Figure 1: Comparison of L_{den} and % HA relationships for each survey.

4.26 The results indicate an unexpectedly high level of annoyance in June 2020 despite reduced noise. The lowest annoyance level was found in September 2020, possibly due to adaptation. Annoyance then increased again in 2023 with noise recovery, although this is notably lower than the WHO 2018 guideline curve. The authors suggest that non-acoustic factors (e.g. poor housing, stress, time at home) influenced annoyance more than noise level during the pandemic. The authors suggest that the WHO curves may not fully apply in dense urban Southeast Asian contexts, where local tolerance to noise may be higher due to urban noise saturation.

4.27 Multiple logistic regression tests revealed that annoyance was significantly associated with: L_{den}, short residence duration, poor view and small floor area. Insomnia was significantly associated with L_{night}, heat sensitivity, time spent at home and poor view.

4.28 Structural Equation Modelling (SEM) was also used in order to further analyse the data. The latent variables were found to be living conditions, health, sensitivity, and time at home. For the annoyance model, the results indicated that pre-pandemic there was a strong link to L_{den} and sensitivity. During the pandemic time at home became dominant, and post-pandemic revealed a hybrid model whereby noise level and time at home were both important.

4.29 For the insomnia model, during the pandemic L_{night} directly affected insomnia, and post-pandemic L_{night}, sensitivity, and time at home were all significant. The authors interpreted these results as annoyance having an immediate response to contextual changes, whereas insomnia exhibited a delayed response, with a marked increase emerging only in the post-pandemic phase (2023), likely influenced by shifts in circadian routines, a return to regular work hours, and residual stress.

4.30 In terms of non-acoustic factors, indoor environment, stress, and sensitivity were found to mediate the relationships between aircraft noise and annoyance, and aircraft noise and insomnia. Time at home during lockdowns amplified exposure and reactions.

4.31 The authors concluded that public responses to noise are not solely determined by decibel levels but are strongly mediated by contextual and personal factors. Therefore, effective noise management and urban planning must incorporate both acoustic and non-acoustic dimensions such as environmental and psychological variables in areas undergoing urbanisation.

4.32 **Schäffer et al** published their findings on the association between residential greenery and noise annoyance from different sources. The aim of the study was to investigate how residential greenery (measured via normalized difference vegetation index, (NDVI) and land-use data) influences noise annoyance from road traffic, railway, and aircraft noise, using data from the NORAH study (Germany, n = 26,607). The study explored whether greenery reduces annoyance and whether this effect varies by noise source, exposure level, and environmental context. A recent study using the Swiss nation-wide SiRENE survey data suggested that residential greenery was associated with reduced road traffic and railway noise annoyance, but increased aircraft noise annoyance, a finding which was unexpected and which the authors were looking to validate with this study.

4.33 For road traffic noise, the results indicated that annoyance decreases with increasing NDVI up to approximately 60 dB L_{den}. Above 60 dB L_{den}, the trend reversed, more greenery was associated with higher annoyance. It was suggested that this was a moderating effect, and that NDVI interacts with L_{den}, and the effect is not just additive. The authors suggested that the results indicate residential greenery may buffer annoyance at low exposure levels. At high exposure, greenspaces may also be noisy, thereby reducing restorative benefits.

4.34 For railway noise, there was no consistent pattern observed across regions. Some regions show reduced annoyance with more greenery at high exposure. It was explained that this may be due to greenspaces near railways often being non-accessible or private allotments.

4.35 For aircraft noise, annoyance increased with NDVI up to approximately 55 dB L_{den}. Above 55 dB L_{den}, NDVI was shown to have little effect. This confirmed the SiRENE study findings, and the authors suggested that possible reasons include that aircraft noise is intrusive and alien, especially over greenspaces. There may be visual-auditory incongruence, whereby tranquil landscapes are disrupted by aircraft. In terms of visual intrusion, aircraft not masked by vegetation.

4.36 The authors concluded that residential greenery moderates noise annoyance, but the effects depend on the noise source, exposure level, and environmental context. They suggested that greenery is beneficial for road traffic noise, but not a substitute for traditional noise mitigation, and that urban planning should integrate greenery with acoustic considerations. They noted that the results indicated that at high road traffic noise exposure (> 60 dB L_{den}), the beneficial link of annoyance with greenery may disappear.

4.37 **Dzhambov et al** published findings on the first large-scale socio-acoustic survey in Bulgaria to derive exposure-response relationships (ERRs) for road, rail, and aircraft noise and high noise annoyance (HNA). It also estimated the health burden (in Disability Adjusted Life Years - DALYs) and economic costs of noise annoyance across five major Bulgarian cities. In 2023, 4640 adults were cross-sectionally sampled from the five largest cities in Bulgaria. Annoyance was rated on the ICBEN 5-point scale.

4.38 Aircraft noise exposure ranged from 37.5-57.5 dB L_{den}. The increase in annoyance began at 40-45 dB L_{den}, which aligned with the WHO's threshold. However, the exposure-response relationship for aircraft noise was relatively flat, as seen in Figure 2.

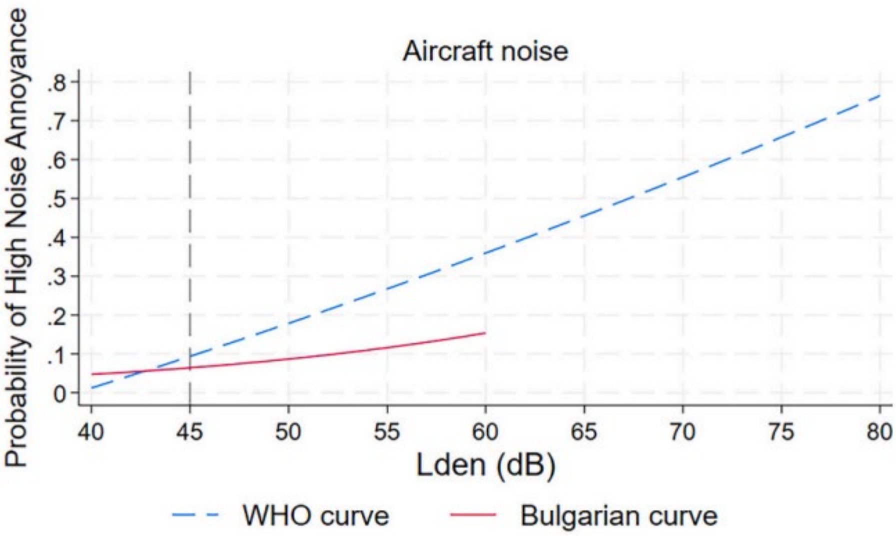

Figure 2: Probability of being highly noise annoyed (HNA) by aircraft noise according to exposure-response curves developed by the World Health Organization and the Bulgarian study.

4.39 An explanation is suggested that the flattening of the line is possibly due to low exposure levels in the sample (with fewer participants exposed to >60 dB). Also, there may be potential underestimation due to limitations in noise mapping and exposure assignment.

4.40 The authors used health burden exposure-response relationships (ERRs) to calculate the DALYs. When they used Bulgarian ERRs aircraft noise contributed 166 DALYs across all cities. The monetised cost was ~$1.87 million USD or ~€1.41 million EUR. When they used the WHO ERRs the aircraft noise burden was higher, indicating that local ERRs may underestimate the true impact. The WHO curves are recommended for policy and burden estimation until better local data are available.

Cardiovascular disease

4.41 This section highlights recent findings on the cardiovascular implications of aircraft noise. The first study was by **Mihalikova et al**, which aimed to determine if and how aircraft noise exposure worsens cardiovascular and metabolic outcomes in the presence of diabetes, given the overlapping mechanisms and the increasing prevalence of both noise pollution and metabolic disease in modern societies.

4.42 Experimental studies in both humans and animals have shown that noise exposure can cause cardiovascular damage, primarily through mechanisms involving oxidative stress and inflammation. The authors explain that diabetes itself is a major risk factor for cardiovascular disease, and both diabetes and noise exposure share common pathological mechanisms (e.g., endothelial dysfunction, oxidative stress, inflammation). However, the interaction between noise exposure and diabetes, specifically, whether noise has additive or synergistic adverse effects in the context of pre-existing diabetes, had not been systematically studied.

4.43 The study aimed to fill this gap by investigating whether aircraft noise exposure would exacerbate cardiovascular and metabolic complications in mouse models of type 1 diabetes, type 2 diabetes, and metabolic syndrome.

4.44 Three mouse models were used to represent:

- Type 1 diabetes (induced)
- Type 2 diabetes (induced insulin resistance)
- Metabolic syndrome/Type 2 diabetes (high-fat diet, HFD)

4.45 The mice were exposed to aircraft noise for 4 days, (maximum sound pressure level of 85 dBA, average sound pressure level of 72 dBA). Physiological measures of insulin signalling, glucose metabolism, blood pressure, vascular function, oxidative stress, inflammation, mitochondrial function, and gene expression were taken.

4.46 The main findings indicated that aircraft noise exposure alone increased blood pressure and oxidative stress, even in non-diabetic mice.

4.47 In diabetic mice, noise exposure:

- Further impaired insulin signalling (lowered insulin in all models, increased insulin resistance in HFD).
- Aggravated hypertension: There was an additive increase in blood pressure in Type 1 and HFD models.
- Worsened endothelial dysfunction: Both macro- (aorta) and microvascular function were further impaired.
- Increased oxidative stress: Additive increases in superoxide in aorta, heart, and brain.
- Adipose tissue inflammation: Especially in HFD mice, noise + diabetes led to higher expression of oxidative and inflammatory markers.
- Mitochondrial dysfunction: Diabetes was the main driver, but noise further increased mitochondrial superoxide in some models.
- Gene expression: RNA showed additive upregulation (increased activity) of inflammatory and metabolic dysregulation pathways (e.g. circadian rhythm disruption).

4.48 The authors concluded that aircraft noise acts as a cardiovascular risk factor, especially in those with pre-existing metabolic disease. Noise and diabetes have additive effects on vascular dysfunction, oxidative stress, and inflammation.

4.49 For public health protection they highlight that vulnerable populations (e.g., people with diabetes or metabolic syndrome) are at higher risk from environmental noise exposure, and as a result urban planning and stricter noise regulations are needed to protect at-risk groups.

4.50 **Kränkel** published an editorial article referring to this study, and addressed how the findings may translate to humans. The study indicated that noise may accelerate disease progression in those with pre-diabetes/metabolic syndrome.

4.51 Kränkel suggested that perceived noise-induced annoyance may be more relevant to health than objective noise levels, and calls for holistic approaches including infrastructure planning, behavioural interventions, and technical solutions (sound barriers, speed limits). The paper emphasised the need for further research on duration, reversibility, and mitigation of noise-induced damage. It was concluded that translational studies in humans are needed, especially in pre-diabetic/metabolic syndrome populations.

4.52 **Valar et al** published a review on the impact of noise and air pollution on the cardiovascular system through the brain-heart pathway. The brain-heart axis is a central pathway through which environmental stressors impact cardiovascular health.

4.53 The authors provide a discussion on how the aircraft noise affects the cardiovascular system. In brief, this comprised of three elements:

1. Central Stress Pathways

- Aircraft noise acts as a psychological stressor.
- Activation of the sympathetic nervous system (SNS) and the hypothalamic–pituitary-adrenal (HPA) axis.
- Leads to release of stress hormones (catecholamines, cortisol).
- Triggers neuroinflammation and oxidative stress in the brain.

2. Brain–Heart Axis

- The efferent pathway: From the brain (prefrontal cortex, amygdala, hypothalamus) to the heart, modulating autonomic and hormonal output.
- The afferent pathway: From cardiovascular sensors (baroreceptors, chemoreceptors) to the brain, influencing central processing.
- Noise-induced stress disrupts this axis, leading to:

  a) Increased blood pressure

  b) Endothelial dysfunction

  c) Vascular inflammation

3. Oxidative Stress and Inflammation.

- Noise exposure (especially aircraft noise) increases oxidative stress in both the brain and cardiovascular system.
- Chronic noise leads to persistent inflammation, endothelial dysfunction, and can exacerbate pre-existing cardiovascular conditions.

4.54 The authors discussed the key evidence in human studies, such as those from epidemiological studies (e.g., HYENA cohort) indicated that aircraft noise exposure is associated with increased saliva cortisol, especially in women.

4.55 Nighttime aircraft noise has been found to impair endothelial function and increases morning adrenaline levels (Schmidt et al., 2013), and amygdala activation (central stress processing) has been linked to arterial inflammation and increased risk of major adverse cardiovascular events in people exposed to transportation noise (Osborne et al., 2020).

4.56 The authors explain that perceived annoyance and sleep disturbance are important mediators of the health effects of aircraft noise.

4.57 The evidence from animal studies is also referred to, for example findings that suggested short and long-term aircraft noise exposure in mice leads to SNS and HPA axis activation, increased blood pressure, endothelial dysfunction, neuroinflammation and blood–brain barrier disruption with effects persisting with chronic exposure indicating the lack of tolerance development. Interventions (e.g., β-blockers, antioxidants like vitamin C) may mitigate some of the adverse effects, highlighting the role of adrenergic signalling and oxidative stress.

4.58 The review describes how noise and air pollution exist as co-exposures and may have additive or synergistic effects on the brain–heart axis and cardiovascular health. Both act via oxidative stress, inflammation, and stress hormone pathways, but noise primarily acts centrally (brain), while air pollution acts peripherally (lung, systemic inflammation). Current exposure limits are frequently exceeded in urban areas, and the authors conclude that mitigation strategies (urban planning, stricter regulations, noise barriers, personal interventions) are urgently needed, especially for the protection of vulnerable populations (those with pre-existing CVD, diabetes, or high stress), who are at particular risk from aircraft noise.

4.59 **Minkin et al** revisited the association between transportation noise and heart disease reported in the WHO Environmental Noise Guidelines, which included findings from studies to 2015. The authors conducted a systematic review and meta-analysis to update the evidence base used in the 2018 WHO Environmental Noise Guidelines for the European Region and to examine whether the quantitative relationship was changed with the addition of new findings. The review focused on transportation noise road, railway and aircraft) in studies published up to the end of 2023 and with the heart disease outcomes: ischaemic heart disease (IHD), atrial fibrillation (AF), and heart failure (HF).

4.60 The inclusion and exclusion criteria followed the eligibility criteria using a PECCOS (Population, Exposure, Comparator, Confounders, Outcome, and Study) framework. The criteria included adults (≥18 years), experiencing long-term residential exposure to transport noise. Results were adjusted for age, sex, and socioeconomic status. Fifty-three studies were included in the systematic review: 85% investigated associations for road, 23% for rail and 30% for aircraft noise exposure.

4.61 The results of the meta-analysis revealed that for incidence of heart disease, the following relative risks (RRs) were found:

- Road traffic noise: RR = 1.02 (95% CI: 1.01–1.04) per 10 dB increase in L_{den}
- Aircraft noise: RR = 1.03 (95% CI: 0.99–1.07)
- Rail noise: RR = 1.00 (95% CI: 0.98–1.03)
- Combined transport noise: RR = 1.03 (95% CI: 1.01–1.04).

4.62 For mortality from heart disease the RRs were:

- Road traffic noise: RR = 1.03 (95% CI: 1.01–1.05)
- Rail noise: RR = 1.02 (95% CI: 1.02–1.03)
- Aircraft noise: RR = 1.07 (95% CI: 1.01–1.14).

4.63 The prevalence of heart disease revealed a RR = 1.09 (95% CI: 0.95–1.26), with insufficient data for aircraft and railway noise.

4.64 For aircraft noise the starting point for incidence of heart disease was 45 L_{den}, and for mortality it was 42 L_{den}. Higher risk estimates were observed in studies with older populations (≥ 50 years), studies with more precise exposure assignment (e.g., residential address with floor height) and adjustment for lifestyle factors (e.g., smoking, alcohol) reduced the observed risk in mortality models.

4.65 79% of studies were rated as having a low risk of bias. It was suggested that aircraft noise studies had higher bias due to ecological designs and limited exposure precision. The quality of the evidence was rated using the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) framework. The ratings varied, for road and rail noise the evidence was rated as high for mortality and moderate for incidence. For aircraft noise the ratings were low to very low.

4.66 It was concluded that road and aircraft noise were associated with increased risk of heart disease incidence and mortality. Railway noise was associated with mortality, but not incidence.

4.67 Evidence strength varies by source and outcome. The authors explained that current evidence gaps include studies from outside Western Europe (87% of studies were European, especially from Denmark), studies for railway and aircraft noise with sufficient numbers of people exposed to higher noise levels (> 55 dB L_{den}), and studies with precise exposure assignment calculated at the residential address, taking into account floor height. These results have implications on quantitative health risk assessments that inform policy and decision making, although the authors recommend caution if located outside of Europe.

4.68 **Marques et al** published their findings on short-term aircraft noise stress and metabolic shift in heart proteome and metabolome. A proteome is the entire set of proteins expressed by an organism, a biological system, or a tissue at a specific time and under particular conditions. The metabolome is the complete collection of small-molecule compounds, called metabolites, found within a biological sample at a particular moment in time.

4.69 The rationale for the study was to examine how short-term noise stress affects cardiac and vascular metabolism at the molecular level. The study design included mice exposed to aircraft noise (average sound pressure level of 72 dBA), for nine hours per day for 3 days. Changes in the heart and blood vessels were examined using advanced molecular techniques.

4.70 The key findings indicated that:

- Aircraft noise causes heart stress quickly. Just 3 days of aircraft noise exposure (72 dB(A)) led to noticeable changes in the heart metabolism in mice. This shift is reminiscent of failing or ischemic hearts, suggesting early signs of cardiovascular dysfunction.
- Energy production shifts in the heart. The heart switched from using fats (via β-oxidation and mitochondrial respiration) to using sugar (via glycolysis), which the authors explain is similar to what happens in heart disease.
- Signs of oxidative stress and inflammation. Noise exposure increased markers of oxidative stress and inflammation, which are both known contributors to cardiovascular disease.
- ROS[3] (reactive oxygen species) seem to drive the harmful changes. When ROS production was blocked, the heart's metabolism improved.
- Metabolic bottlenecks appear. A build-up of succinate suggests a blockage in energy pathways, forcing the heart to rely on less efficient glycolysis.
- Lipid metabolism is disrupted. Fat breakdown and transport were impaired, with reduced levels of key enzymes and molecules.
- Glycolysis increased, indicating a shift toward sugar-based energy production.
- Data suggests the heart responds as if it's in a low-oxygen state, further promoting glycolysis.
- Serotonin levels rise Noise stress increased serotonin and tryptophan in the heart, which may influence metabolism and stress responses.

[3] ROS (Reactive Oxygen Species) are a group of unstable, oxygen-containing molecules produced during normal metabolism that are highly reactive and can damage cells but also act as signalling molecules to regulate cellular processes.

- Blocking ROS helps. Mice lacking NOX2 (a ROS generator) showed less metabolic disruption, suggesting a potential therapeutic target.

4.71 The study concluded that the study provides molecular-level evidence that even short-term aircraft noise can induce cardiometabolic dysfunction, supporting epidemiological links between noise exposure and cardiovascular disease. Limitations of the study include that translational relevance to humans is constrained by species differences in auditory sensitivity and stress resilience. In addition, short exposure duration may not fully capture chronic noise effects but is valuable for modelling acute stress responses. They propose the findings from this study underscores the urgency of integrating noise mitigation strategies into public health frameworks and exploring ROS-targeted therapies to protect vulnerable populations.

4.72 **Münzel et al** published their review of transportation noise and cardiometabolic risk. The aim of the review was to summarise the epidemiological evidence on chronic transportation noise exposure and risk of cardiovascular disease, as well as provide insight on the pathophysiological mechanisms. The review included road traffic, aircraft and railway noise. Aircraft noise is identified as a significant environmental stressor contributing to cardiometabolic diseases, including ischemic heart disease (IHD), stroke, heart failure, diabetes, and hypertension. Although road traffic noise has stronger epidemiological evidence, aircraft noise shows acute and chronic effects, particularly during nighttime exposure.

4.73 The review covers the epidemiological evidence with respect to various cardiovascular endpoints. For cardiovascular mortality, aircraft noise exposure (especially >50 dBA at night) was found to be linked to increased odds of cardiovascular death within 2 hours of exposure. Weak, but suggestive associations were found between aircraft noise and ischemic heart disease (IHD); RR of 1.01 per 10 dBA. There was limited and inconsistent data for aircraft noise and stroke and heart failure. For arrythmias, aircraft noise showed a higher RR (1.21) than road noise (1.01), though based on sparse data. For mental health outcomes, aircraft noise was more strongly associated with depression and annoyance than other sources. The review found that for arterial hypertension, the evidence was mixed and the authors suggested more longitudinal research is required.

4.74 The mechanisms for these findings in humans were discussed. Nighttime aircraft noise impairs endothelial function, increases adrenaline, and elevates oxidative stress markers. Sleep disruption from aircraft noise leads to vascular dysfunction and non-dipping blood pressure patterns. Vitamin C has been found to improve endothelial function, suggesting a role for reactive oxygen species (ROS).

4.75 In animal studies, the evidence has suggested that aircraft noise exposure (L_{eq} 72 dBA) in mice elevates blood pressure, stress hormones, and vascular oxidative stress. There is evidence indicating that noise exacerbates diabetes-related vascular damage in multiple diabetic mouse models. Noise had also been found to disrupts circadian rhythms, amplifying neuroinflammation and metabolic dysfunction.

4.76 The review also referred to the impacts of co-exposures. Aircraft noise and air pollution have additive effects on oxidative stress and inflammation. It was concluded that combined exposures worsen vascular and cerebral damage, especially when sleep and circadian rhythms are disrupted.

4.77 The authors discussed mitigation strategies for noise reduction. Aircraft-specific interventions included night flight bans, optimised flight paths, and steeper descent approaches. In terms of knowledge gaps, it was concluded that there is an underrepresentation of aircraft noise in cardiovascular prevention guidelines, and a need for longitudinal studies and intervention trials to establish clearer relationships.

Noise effects on children

4.78 This section discusses recent findings on aircraft noise and children. The first study focused on the impact of noise on learning in children and adolescents **(Fretes and Paulau).** This was a meta-analysis investigating the effects of environmental and classroom noise on learning, with a focus on cognitive and academic performance in elementary and secondary school students. The meta-analysis consisted of 21 studies, on non-university students aged 6-18 years.

4.79 The overall effect size was achieved using the Restricted Maximum Likelihood (REML) method, resulting in a value of −0.46 (95% CI: −0.54 to −0.38), indicating a moderate negative impact of noise on performance. The most vulnerable group was found to be children aged 6-12 years, suggesting developmental sensitivity to noise.

4.80 The study categorised noise-related impacts into 8 domains:

- Language comprehension: Reading fluency, listening, writing, spoken word perception.
- Processing speed: Slower response times in noisy conditions.
- Executive control: Impaired cognitive flexibility and interference management.
- Attention: Disruption of selective and sustained attention.
- Mathematical skills: Arithmetic and reasoning performance decline.
- Academic performance: Lower standardised test scores.
- Visuospatial skills: Less affected, possibly due to reduced auditory dependence.
- Memory: Working memory and recall negatively impacted.

4.81 For aircraft noise, the results suggested that chronic exposure is linked to reduced reading comprehension, impaired sustained attention and lower reasoning and perception scores. Younger children were found to be especially vulnerable and the authors suggested that acoustic interventions should be prioritised in early education, and aircraft noise should be considered in school positioning and urban planning.

4.82 **Fernández-Quezada et al** published a meta-analysis on the Influence of noise exposure on cognitive function in children and adolescents. The review included eight primary studies published between 2001 and 2023 in children and adolescents aged 8-16 years. The sample size was 3,385 participants (1,687 noise-exposed, 1,698 controls) from noise sources including aircraft, road traffic, environmental, and white noise. Exposure levels ranged between 55-95 dB, primarily in schools and homes of participants.

4.83 The overall effect of noise revealed a standardised mean difference (SMD) of -0.544 (95% CI: [−0.616, −0.472]), $z = -14.85$, $p < 0.0001$, indicating a highly significant effect of noise on cognitive performance. Aircraft noise was the primary exposure source (four of eight studies included), with studies from Korea, South Africa, and the UK revealing that there was an IQ reduction in children exposed to 75-80 dB aircraft noise, reading comprehension deficits were found in children near airports, and attention and memory impairments were linked to chronic aircraft exposure.

4.84 The mechanisms of the impact of noise on cognition were discussed in the review. Examples of these direct pathways include the damage to auditory hair cells from high sound pressure levels, and the impact of sleep disturbance leading to impairment of neural restoration and memory consolidation. Indirect pathways include a neuroendocrine stress response, leading to increased cortisol levels and glucose dysregulation. Oxidative stress results in neuronal damage, with noise levels above 65 dB being linked to increased oxidative markers, resulting in increased inflammation, impairing cognitive performance.

4.85 The authors proposed the use of longitudinal designs with larger, more diverse cohorts in future work and the standardisation of noise exposure metrics and cognitive tests across studies. Future research should control for socioeconomic status, air pollution, sleep quality, and educational context, and include subjective noise sensitivity and individual noise exposure history.

4.86 **Bartels et al** examined the perception of aircraft noise and its impact among children. The study investigated how children aged 8-10 years perceived aircraft noise in their home environment, particularly in terms of annoyance and disturbance of daily activities and compared their self-reports with parental assessments. It also explored predictors of annoyance, both acoustical and non-acoustical. The authors explained that children are more vulnerable to environmental stressors like aircraft noise due to their developmental stage and longer sleep durations, especially during shoulder hours (evening and early morning). Despite known cognitive and psychological impacts, annoyance, a key mediator in noise-health pathways, is under-explored in children. Most existing data rely on parental reports, potentially missing children's subjective experiences.

4.87 51 healthy children (8-10 years) and one parent each, living near Cologne/Bonn Airport (24/7 operations) were included in the study. Noise exposure was classed as moderate to high (50-70 dBA L_{den}; 45-65 dBA L_{night}). Separate surveys for children (interview-based) and parents (paper-pencil) were completed, with child-appropriate adaptations to scales and terminology. The authors explained this was an exploratory study, as part of a larger study into the effects of aircraft noise on sleep and annoyance.

4.88 The findings indicated that according to the children, passive communication (TV, radio) was the activity most frequently disturbed (median, Mdn = 3, “once a month or more often disturbed”). Children reported the lowest disturbance for playing indoors and outdoors (Mdn = 1, “never disturbed”). With regard to sleep, children reported highest disturbance while trying to fall asleep (Mdn = 3 „once a month or more often”). Parents rated children’s disturbance of falling asleep significantly lower (p = 0.029). Disturbance during the night’s sleep and while sleeping late on free days was less pronounced according to the children (Mdn = 2) and they rated disturbance lower than their parents (p = 0.052 and p = 0.038, respectively). Parents underestimated disturbance during falling asleep and passive communication, and they overestimated disturbance in outdoor active communication and night sleep.

4.89 In terms of annoyance, children reported low to moderate annoyance (mean ≈ 2.3 on a 5-point scale). There was no significant difference between children's self-reports and parents’ assessments of their children, although parents reported higher personal annoyance than children.

4.90 The strongest predictor of annoyance was perceived loudness of aircraft, and disturbance while relaxing, falling asleep, and sleeping late. There was no significant correlation with noise exposure, or non-acoustic factors (e.g., noise sensitivity, attitudes, satisfaction with living environment).

4.91 The authors concluded that children’s annoyance is not well predicted by traditional adult-based models. Perceived loudness and disturbance of sleep-related activities are more relevant than measured noise levels. Metrics such as L_{den} and L_{night} may be insufficient for capturing children's subjective experiences, highlighting the need for more nuanced exposure metrics (e.g., number and peak levels of flyovers). The authors also suggested that survey instruments for children need further validation, especially for abstract concepts like annoyance and noise sensitivity.

Chapter 5

Summary

5.1 This update report has summarised the main findings in the field of aircraft noise and health effects research over the six-month period between and March and September 2025.

5.2 The aim of this report was to provide an overview of the recently published findings on aircraft noise and health effects, including new findings presented at the Internoise and Euronoise congresses.

5.3 The main findings from the work on annoyance and sleep disturbance suggest that psychoacoustic metrics (e.g., loudness, sharpness) outperform conventional noise metrics in predicting annoyance. Exposure-response relationships (ERRs) vary significantly across airports and populations, with non-acoustic factors (e.g., housing quality, time at home) continuing to be important.

5.4 In the area of cardiovascular and metabolic health, aircraft noise has been linked to increased risks of hypertension, coronary heart disease, stroke, atrial fibrillation, and heart failure, especially during night-time exposure. Experimental studies in mice indicate that aircraft noise exacerbates cardiovascular damage in diabetic models, with additive effects on blood pressure, oxidative stress, and inflammation. Molecular studies have revealed that short-term aircraft noise exposure induces metabolic shifts in the heart, resembling early signs of cardiovascular disease.

5.5 Noise sensitivity is a measurable psychological trait that correlates with annoyance and sleep disturbance. Findings suggest that residential greenery may buffer annoyance from road and rail noise but may increase annoyance from aircraft noise due to visual-auditory incongruence.

5.6 Aircraft noise negatively affects children's cognitive performance, especially reading comprehension, attention, and memory. Meta-analyses have confirmed impacts on learning, with younger children being most vulnerable. One of the findings indicated that children's annoyance and sleep disturbance are better predicted by perceived loudness and activity disturbance than by standard noise metrics.

5.7 Noise insulation schemes (e.g., Heathrow RIS) show promising reductions in annoyance and sleep disturbance, though broader health and wellbeing impacts require longer-term evaluation.

5.8 The next update report is due at the end of March 2026. The evidence base for aircraft noise and health effects continues to grow, with increasing recognition of the role of non-acoustic factors, vulnerable populations, and the need for refined exposure metrics. The findings support the development of more targeted mitigation strategies, updated exposure-response relationships, and integrated public health policies to address the multifaceted impacts of aviation noise.

Chapter 6

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