Memorandum

To:	Gareth Gretton, Energy Efficiency and Conservation Authority (EECA)
Date:	28 January 2026
From:	Gerda Kuschel & Lou Wickham (EIL)
Re:	**Response to Dr Brian Cox Peer Review of the Indoor Combustion Study**
cc:	Andrew Neal (EECA)

The Energy Efficiency and Conservation Authority (EECA) commissioned Emission Impossible Ltd and Resource Economics to prepare a study into the health effects and costs of indoor combustion in New Zealand homes. Our report, published in September 2025 on EECA's website here, estimates the health-related costs of emissions attributed to wood burners, gas stoves, and unflued gas heaters in New Zealand (Metcalfe *et al* 2025).[1] Following the report's release, the Gas Association of New Zealand engaged Dr Brian Cox (Public Health Research and Consulting Services) to comment on the epidemiological assumptions underpinning the EIL/Resource Economics study (Cox 2025).[2]

Dr Cox's primary concern regarding our report is the lack of evidence (in his opinion) supporting causality for the harmful effects of nitrogen dioxide – specifically linked to the use of domestic gas in New Zealand. The following memorandum discusses key issues (only) raised by Dr Cox (***in bold italics***) and our response. We have not responded to all matters raised noting that many of the methodological issues are discussed in detail in the technical volumes supporting HAPINZ 3.0.

We have, however, taken the opportunity to provide additional clarification in the report with an updated (January 2026) version. Please note this update does not include any change in the methodology, findings or conclusions.

Thank you for the opportunity to respond to this review.

Ngā mihi / Regards,

Dr Gerda Kuschel
Director

Lou Wickham
Director

[1] Metcalfe J, Kuschel G, Wickham L, and Denne T (2025). *Indoor Combustion: Health Effects and Costs*. Report prepared by Emission Impossible Ltd and Resource Economics for Energy Efficiency & Conservation Authority, September 2025

[2] Cox B (2025). *The imputation of health outcomes from the domestic use of gas in the EECA report*. Prepared for the Gas Association of New Zealand, 15 December 2025

Issue: Lack of evidence for causality linked to domestic gas use in New Zealand

Response: We agree with Dr Cox on the importance of rigour to establish causality.

Establishing causality for air pollution causing disease in epidemiology relies on frameworks like Bradford Hill criteria (temporality, strength, consistency, specificity, dose-response, plausibility, coherence, experiment and analogy), distinguishing it from mere association, and requires robust study designs (like randomized control trials) to show an intervention changes an outcome, not just that they occur together.

However, it is worth pointing out that epidemiology is only one of multiple lines of scientific enquiry necessary to provide a sufficient body of evidence for a causal relationship. In reaching consensus that particulate pollution *causes* lung cancer, the World Health Organisation considered (WHO 2013):

- Epidemiological studies show strong, consistent, dose-related associations;
- Toxicology and mechanistic studies demonstrate biological pathways that plausibly lead to disease;
- Evidence is coherent across countries, methods, and pollutant mixtures;
- Reductions in the pollutant lead to measurable improvements in health; and
- Alternative explanations do not account for the observed patterns.

Assessing causality linked to domestic gas use in New Zealand relies on establishing the links between gas combustion and increases in NO_2 concentrations and then NO_2 concentrations and adverse health outcomes (discussed further below),

The link between exposure to air pollutants and adverse health outcomes

Major public health agencies, including the World Health Organization (**WHO**) and the United States Environmental Protection Agency (**US EPA**), state that several air pollutants have been extensively studied, with sufficient evidence to establish a causal link to specific adverse health effects, as opposed to a mere statistical association. These include:

Particulate matter ($PM_{2.5}$ and PM_{10}):

- Health effects shown to be caused: Premature death, development of cardiovascular diseases (such as stroke and ischemic heart disease), respiratory diseases (such as chronic obstructive pulmonary disease and lung cancer), and adverse birth outcomes like low birth weight.
- Mechanism: Fine particles ($PM_{2.5}$) are small enough to penetrate deep into the lungs and enter the bloodstream, causing systemic inflammation, oxidative stress, and damage to various organs and tissues.

Nitrogen dioxide (NO_2):

- Health effects shown to be caused: Increased susceptibility to respiratory infections, chronic lung disease from long-term exposure, and aggravation of existing respiratory conditions, such as asthma.

- Mechanism: NO_2 irritates the respiratory system, leading to inflammation and cellular damage in the lungs.

The 2021 WHO Global Air Quality Guidelines reflect global consensus that there is a causal relationship between long-term $PM_{2.5}$ exposure and mortality.[3] WHO (2021) further noted that the relationship between long-term NO_2 exposure and mortality is suggestive of causality, and likely causal for respiratory effects. Consequently, all health outcomes assessed in our study are either causal, likely to be causal or suggestive of causality.

We have provided additional clarification to our report to ensure the above is clear.

The link between domestic gas use and air pollution

Pollution increments were established for appliance types assessed in the study. For the domestic gas appliances – gas stoves and unflued gas heaters – likely pollution increments due to their use were developed for NO_2 only.

Dr Cox does not dispute that gas appliances used indoor release NO_2 – he is concerned about the increments selected and the use of exposure response functions developed for other sources of NO_2 (transport).

As mentioned in our report, our priority was sourcing robust indoor air quality data, ideally from New Zealand studies. Our increments were based on Gillespie-Bennett et al (2008), with cross-checking against Sun et al (2025), Kashtan et al (2024), Jacobs et al (2023) and Kornatit et al (2010).

The increments used in the modelling were the central values, but low and high values were provided for sensitivity testing. The central values chosen were consistent with overseas values, allowing for differences in climate and home heating requirements between various countries.

The uncertainties associated with using exposure response functions (ERFs) developed from outdoor sources for indoor sources are well-canvassed in a recent position statement on this subject by the UK Committee on the Medical Effects of Air Pollutants (COMEAP 2025).[4] This statement cautions against adapting studies from different countries or regions – which has not been done in our study which uses New Zealand-derived ERFs.

With respect to COMEAP's other recommendations (*in italic blue font below*) as applied to NO_2, we note:

(i) *Modifying ERFs to be expressed in terms of integrated personal exposure* – our model does something quite different – it considers incremental exposure attributable to the

[3] WHO (2021). *WHO global air quality guidelines: particulate matter ($PM_{2.5}$ and PM_{10}), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide*. World Health Organization, Geneva, September 2021. Available at: https://www.who.int/publications/i/item/9789240034228

[4] Committee on the Medical Effects of Air Pollutants (2025). *COMEAP statement on possible approaches to estimating the health effects attributable to exposure to $PM_{2.5}$ and NO_2 indoors, using epidemiological studies on outdoor air pollution*. UK Health Security Agency, United Kingdom, December 2025. Available at: https://www.gov.uk/government/publications/estimating-health-effects-of-pm25-and-no2-indoors/comeap-statement-on-possible-approaches-to-estimating-the-health-effects-attributable-to-exposure-to-pm25-and-no2-indoors-using-epidemiological-stud

appliance on a per household basis (only). We have not modified the ERF – noting our approach is consistent with WHO (2024) in attributing outdoor ERFs to indoor exposure.

(ii) *Modifying an ERF using time-activity and infiltration data relevant to the population from which the ERF was derived* - this is infeasible as studies of time-activity and infiltration data populations are a tiny subset of the national HAPINZ 3.0 population;

(iii) *Applying the modified ERF using time-activity and concentration data relevant to the target population of interest* – our model achieves this through consideration of incremental exposure;

(iv) *Conceptually, the modified ERF could be applied in a range of assessments as long as the exposure being evaluated is expressed in terms of integrated personal exposure (μg/m^3) taking into account the proportion of time spent, and the pollutant concentrations, in the microenvironment(s) concerned.* As noted above, our model considers seasonalised household exposure. This approach supports EECAs intended use of the model to evaluate different scenarios; and

(v) *We draw attention to the increased uncertainty when applying the modified ERF to quantify the effects attributable to pollution from indoor sources. Uncertainty arises because of differences in the temporal pattern and co-pollutant mixtures of emissions indoors and outdoors.* This is true and our report has been careful to state limitations and uncertainties throughout. Ultimately, however, our approach is consistent with the World Health Organisation and other jurisdictions undertaking research in this area, being based upon the best available evidence.

Issue: Use of specific respiratory diseases instead of cardiac and respiratory hospitalisations

Response: The ERFs used in the indoor study for respiratory and cardiac hospitalisations were those derived for HAPINZ 3.0 by Hales et al (2021).[5] They were developed using data on public hospital discharges reported with ICD10 codes for **cardiovascular** disease overall (G45, I011, I012, I05-I13, I159, I20-I51, I60-I99, M30-M31); **respiratory** causes overall (J22-J65, J668, J67-J98) as well as ischaemic heart disease (I20-I25); stroke (G45, I60-I69); lung cancer (C33-C34) and asthma (J45-J46).

Therefore, the ERFs shown in our report are correct for overall hospitalisations.

Issue: Confusion over whether incidence or prevalence of asthma was assessed

Response: We agree that some of the descriptions in the report were not clear and have revised the text accordingly.

We confirm that the modelling was done on <u>prevalence</u> of asthma as per HAPINZ 3.0.

[5] Hales et al (2021). Long term exposure to air pollution, mortality and morbidity in New Zealand: Cohort study. S Hales, J Atkinson, J Metcalfe, G Kuschel & A Woodward, *Sci Tot Env* **801**:149660, December 2021. doi.org/10.1016/j.scitotenv.2021.149660