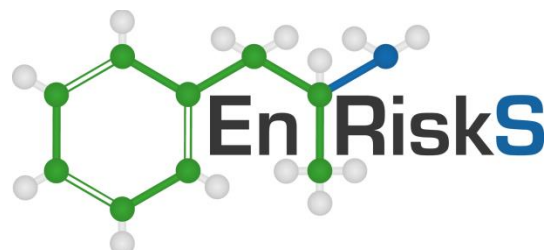


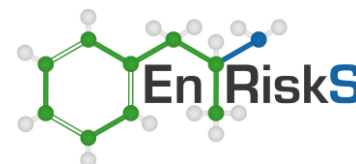


# Review of In-Cabin Carbon Dioxide Levels

*Prepared for: NSW RMS*

8 September 2017





## Document History and Status

<b>Report Reference</b>	RMS/17/CO2R001
<b>Revision</b>	C – Revised Final
<b>Date</b>	8 September 2017
<b>Previous Revisions</b>	A – Issued on 19 February 2017 B – Final issued on 29 May 2017

## Limitations

Environmental Risk Sciences has prepared this report for the use of NSW RMS in accordance with the usual care and thoroughness of the consulting profession. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report.

It is prepared in accordance with the scope of work and for the purpose outlined in the Section 1 of this report.

The methodology adopted and sources of information used are outlined in this report. Environmental Risk Sciences has made no independent verification of this information beyond the agreed scope of works and assumes no responsibility for any inaccuracies or omissions. No indications were found that information contained in the reports for use in this assessment was false.

This report was prepared from December 2016 to May 2017 and revised in September 2017 and is based on the information provided and reviewed at that time. Environmental Risk Sciences disclaims responsibility for any changes that may have occurred after this time.

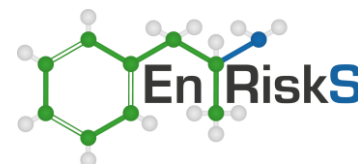
This report should be read in full. No responsibility is accepted for use of any part of this report in any other context or for any other purpose or by third parties. This report does not purport to give legal advice. Legal advice can only be given by qualified legal practitioners.

# Table of Contents

---

## Executive Summary

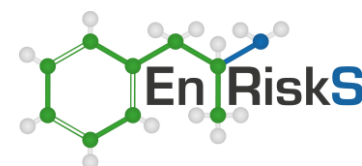
<b>Section 1. Introduction .....</b>	<b>1</b>
1.1 Context.....	1
1.2 Objectives and Scope of Works.....	2
1.3 Methodology .....	3
1.4 Literature Review.....	3
<b>Section 2. Accumulation of CO<sub>2</sub> in Vehicles.....</b>	<b>4</b>
2.1 RMS 2016 Study .....	4
2.2 Other Studies.....	6
2.2.1 General.....	6
2.2.2 Australian studies .....	7
2.2.3 International studies.....	8
2.3 Modelling .....	10
<b>Section 3. Health Effects of Exposure to CO<sub>2</sub> .....</b>	<b>15</b>
3.1 General.....	15
3.2 Acute and subchronic toxicity .....	16
3.3 CO <sub>2</sub> Effects on Cognitive Performances and Productivity .....	18
3.4 Sensitive Populations .....	19
3.5 Guidelines for Short-Term Exposure to CO <sub>2</sub> .....	20
3.6 Summary of health effects associated with short-term exposures.....	21
3.7 Relevance of Studies to Evaluating Driver Safety .....	23
3.7.1 General.....	23
3.7.2 Background on issues related to driver distraction.....	23
3.7.3 Studies related to distraction/fatigue .....	24
3.7.4 Identification of Criteria .....	26
<b>Section 4. Management Measures for CO<sub>2</sub> Levels in Vehicles.....</b>	<b>27</b>
<b>Section 5. Assessment of Short-Duration CO<sub>2</sub> Exposures.....</b>	<b>30</b>
5.1 Approach.....	30
5.2 Evaluation of CO <sub>2</sub> Exposures.....	31
<b>Section 6. Risk Management .....</b>	<b>43</b>
<b>Section 7. Conclusions .....</b>	<b>44</b>
<b>Section 8. References .....</b>	<b>45</b>



## Glossary of Terms

---

Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days).
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems.
AER	Air exchange rate
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
Biodegradation	Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).
Carcinogen	A substance that causes cancer.
Chronic exposure	Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure].
CO <sub>2</sub>	Carbon dioxide
DECCW	Department of Environment, Climate Change and Water
Detection limit	The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.
Dose	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
EPA	Environment Protection Authority
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.
Exposure pathway	The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed) to it. An exposure pathway has five parts: a source of contamination (such as chemical leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receiver population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.



Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the NSW Department of Environment and Conservation (DEC) or institutions such as the National Health and Medical Research Council (NHMRC), Australia and New Zealand Environment and Conservation Council (ANZECC) and World Health Organisation (WHO)), that is used to identify conditions below which no adverse effects, nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter- and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines will have different names, such as investigation level, trigger value, ambient guideline etc.
Inhalation	The act of breathing. A hazardous substance can enter the body this way [see route of exposure].
LOAEL	Lowest-observed-adverse-effect-level - The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.
LOR	Limit of Reporting
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NOAEL	No-observed-adverse-effect-level - The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.
NSW	New South Wales
OEH	Office of Environment and Heritage
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA)
PM <sub>10</sub>	Particulates with an aerodynamic diameter of 10 microns (or $\mu\text{m}$ ) or less. Particles of this size are of importance to health as they are small enough to enter the respiratory system. The small particles (PM <sub>2.5</sub> ) penetrate deeper into the lungs than the larger particles in this fraction size.
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].
Risk	The probability that something will cause injury or harm.
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact]
STEL	Short-term exposure limit, relevant to occupational environments
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
Toxicology	The study of the harmful effects of substances on humans or animals.
TWA	Time-weighted average, typically relevant to the assessment of occupational exposures where the exposure concentrations are and average over a workday.





Uncertainty factor	Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].
USEPA	United States Environmental Protection Agency
WHO	World Health Organisation

## Executive Summary

---

Several major road infrastructure developments are currently planned for Sydney that include road tunnels of various lengths. To minimise exposures to elevated levels of nitrogen dioxide (NO<sub>2</sub>) (and fine particles) inside these tunnels advice has been provided to keep windows up and ventilation on recirculation.

The Advisory Committee on Tunnel Air Quality (the Advisory Committee) commissioned a study investigating NO<sub>2</sub> inside vehicles traversing road tunnels in Sydney. Results related to levels of NO<sub>2</sub> within vehicles, from this study are presented in the report: *“Road tunnels: reductions in nitrogen dioxide concentrations in-cabin using vehicle ventilation systems”* prepared by Pacific Environment Limited (PEL) for the NSW Roads and Maritime Services (RMS) dated 16 February 2016 (referred to as the 2016 RMS Study). The study showed that when ventilation is switched to recirculate NO<sub>2</sub> levels inside the vehicle can be significantly reduced, well below the levels measured outside of the vehicles inside the tunnel. The data collected during the study also included carbon dioxide levels (CO<sub>2</sub>), principally as a basis for calculating the air exchange rate within the vehicles. This data indicated that while the ventilation was on recirculation during the study, CO<sub>2</sub> levels increased with time. The potential for increasing levels of CO<sub>2</sub> inside vehicles has been further evaluated in this report.

The overall objective of the review and assessment presented in this report is to evaluate potential exposures to CO<sub>2</sub> within vehicles when ventilation is on recirculation and identify risks to health, including driver safety, associated with short-duration exposures that may occur when using tunnels. More specifically the report has been prepared to address the following:

- provide a preliminary characterisation of the risk to vehicle occupants due to CO<sub>2</sub> accumulation in vehicle cabins when the windows are closed and the ventilation system is set to recirculate over periods of 15 minutes, 30 minutes and 60 minutes;
- consider any identified CO<sub>2</sub> exposure risks in the context of any other relevant road safety risks;
- identify implications of any identified CO<sub>2</sub> exposure risks for the use of re-circulation to reduce exposure to in-cabin NO<sub>2</sub> on the proposed Sydney road tunnel network; and
- provide recommendations for any further actions that may be required to appropriately characterise and/or address the risks due to CO<sub>2</sub> exposure.

Health effects associated with short-term exposures to high concentrations of CO<sub>2</sub> are well understood, and form the basis for a number of occupational guidelines. However, recent studies indicate that subtle adverse effects of even short-term low level CO<sub>2</sub> exposure can be measured. Subtle cognitive effects identified at exposures levels of 1,000 ppm and lower are not considered to be relevant to driver behaviour. Other adverse effects including fatigue, headaches and visual disturbances effects have been reported at CO<sub>2</sub> levels above 0.3% (3,000 ppm). For very short-duration exposures to CO<sub>2</sub>, acute physiological effects are of more importance.

The available literature from Australia and International sources all confirm that CO<sub>2</sub> levels increase in-cabin where the ventilation is on recirculation, with the maximum levels of CO<sub>2</sub> in-cabin varying depending on a wide range of vehicle and driving factors. However, where the vehicle air exchange



rate has been measured, it is possible to estimate the concentration of CO<sub>2</sub> in-cabin over time. This has been verified through the use of the 2016 RMS study data.

Based on the data collected during the 2016 RMS study, the model established to predict in-cabin CO<sub>2</sub> levels when ventilation is on recirculation and the potential health effects associated with exposure to CO<sub>2</sub> in-cabin over time periods up to an hour (relevant to exposures that may occur in tunnels) for 1 to 5 occupants, the levels of CO<sub>2</sub> that may be present are not expected to adversely affect driver safety.

Assessment of potential exposures that may occur for periods over 1 hour, where ventilation is left on recirculation indicates that there may be levels of CO<sub>2</sub> where there are 1 or more passengers that may affect an already fatigued driver.

It is noted that there is a general lack of guidance or regulations in terms of the design or use of ventilation systems in vehicles in Australia. Hence there is currently no advice to drivers on the suitable use of ventilation in various circumstances, to minimise the potential for effects on already fatigued drivers.

Where RMS provides specific advice to drivers entering road tunnels to put ventilation on recirculation, it would also be necessary to provide advice that recirculation should be switched off and not left on for an extended period of time.



## Section 1. Introduction

---

### 1.1 Context

Several major road infrastructure developments are currently planned for Sydney. Tunnels feature prominently amongst these developments, notably in the NorthConnex (<http://northconnex.com.au/>) and WestConnex (<http://www.westconnex.com.au/>) projects. If it is approved, WestConnex will form the longest single network of road tunnels in Australia.

Air quality has historically been a factor that influences the acceptance of road tunnels by the Sydney community, particularly ambient (outdoor) air quality and the effects of tunnel ventilation outlets. However, given the likely lengths and traffic volumes of the planned tunnels for NorthConnex and WestConnex, the potential exposure of vehicle occupants to elevated levels of air pollutants within tunnels is increasingly being scrutinised.

Historically carbon monoxide has been the basis of in-tunnel air quality criteria, relevant to designing tunnel ventilation requirements. With improvements in vehicle technology, carbon monoxide is no longer the key issue. However, with the increase in use of diesel vehicles, and the relatively high emissions of NO<sub>x</sub> (and hence nitrogen dioxide (NO<sub>2</sub>) from these vehicles, NO<sub>2</sub> has become the key pollutant of concern for in-tunnel air quality.

High concentrations of NO<sub>2</sub> are of concern in relation to human health, however the data that is available to evaluate the impacts on human health are limited. Hence, for projects such as NorthConnex and WestConnex, advice has been provided to keep vehicle windows up and ventilation on recirculation to minimise exposures to NO<sub>2</sub> while using the tunnels.

The Advisory Committee on Tunnel Air Quality (the Advisory Committee) commissioned a study investigating nitrogen dioxide (NO<sub>2</sub>) inside vehicles traversing road tunnels in Sydney. Results related to levels of NO<sub>2</sub> within vehicles, from this study are presented in the report: *“Road tunnels: reductions in nitrogen dioxide concentrations in-cabin using vehicle ventilation systems”* prepared by Pacific Environment Limited (PEL) for the NSW Roads and Maritime Services (RMS) dated 16 February 2016 (referred to in this report as the 2016 RMS Study). The study has also been published (Martin et al. 2016). The study showed that when ventilation is switched to recirculate NO<sub>2</sub> levels inside the vehicle can be significantly reduced, well below the levels measured outside of the vehicles inside the tunnel. The data collected during the study also included carbon dioxide levels (CO<sub>2</sub>), principally as a basis for calculating the air exchange rate within the vehicles. This data indicated that while the ventilation was on recirculation during the study, CO<sub>2</sub> levels increased with time. The potential for increasing levels of CO<sub>2</sub> inside vehicles requires further evaluation, particularly in terms of potential effects on the health of vehicle occupants and driver safety.

Environmental Risk Sciences Pty Ltd (enRiskS) has been commissioned by NSW RMS to conduct a review of risk issues related to CO<sub>2</sub> levels in vehicles that may be associated with the use of ventilation on recirculation within tunnels.

## 1.2 Objectives and Scope of Works

The overall objective of the review and assessment presented in this report is to evaluate potential exposures to CO<sub>2</sub> within vehicles when ventilation is on recirculation and identify risks to health, including driver safety, associated with short-duration exposures that may occur when using tunnels. More specifically the report has been prepared to address the following:

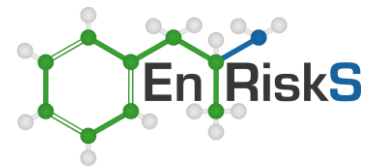
- provide a preliminary characterisation of the risk to vehicle occupants due to CO<sub>2</sub> accumulation in vehicle cabins when the windows are closed and the ventilation system is set to recirculate over periods of 15 minutes, 30 minutes and 60 minutes;
- consider any identified CO<sub>2</sub> exposure risks in the context of any other relevant road safety risks;
- identify implications of any identified CO<sub>2</sub> exposure risks for the use of re-circulation to reduce exposure to in-cabin NO<sub>2</sub> on the proposed Sydney road tunnel network; and
- provide recommendations for any further actions that may be required to appropriately characterise and/or address the risks due to CO<sub>2</sub> exposure.

To address the above, the review has been undertaken on the basis of the following scope of works:

- An initial meeting was held between NSW RMS and NSW Health on the 8 November 2016, where the scope of works was discussed and refined (as outlined below).
- Undertake a literature review in relation to:
  - short term effects of CO<sub>2</sub>, including determining the dose-response relevant to exposure and how these effects may affect driver safety
  - how CO<sub>2</sub> accumulates in vehicles during limited ventilation (recirculation), with consideration of different driving environments (i.e. within tunnels or on open roads)
  - measures undertaken by other relevant bodies globally to characterise or address such risks.

The literature review is presented in **Section 2**.

- Utilise available data collected by RMS, as well as data available from literature to characterise short-term risks to vehicle users over different exposure periods, namely 15 minutes, 30 minutes and 60 minutes. Issues associated with leaving ventilation systems on recirculate for longer periods of time have also been addressed. This evaluation is presented in **Section 3**.
- Based on the findings of the risk characterisation, the implications of the possible risks due to exposure to CO<sub>2</sub> in vehicles during times of limited ventilation will be considered and discussed, particularly in regard to existing advice to reduce in cabin NO<sub>2</sub> exposure by limiting ventilation while driving through tunnels.
- Discussion of any other findings relevant to managing/characterising CO<sub>2</sub> exposure inside vehicles.



### 1.3 Methodology

The characterisation of health risks has been undertaken on the basis of the following guidance:

- enHealth (2012) “Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards” (enHealth 2012)
- Health Impact Assessment Guidelines. Published by the Environmental Health Committee (enHealth), which is a subcommittee of the Australian Health Protection Committee (AHPC) (enHealth 2001)
- NSW Health, Healthy Urban Development Checklist, A guide for health services when commenting on development policies, plans and proposals, 2009.

Other guidance has also been considered, where relevant, and referenced within this report.

### 1.4 Literature Review

A literature review has been undertaken using on-line search tools available from Scopus, PubMed, Google Scholar and Google to identify publications and reports (including industry reports) relevant to addressing the following issues:

- short term effects of CO<sub>2</sub>, including determining the dose-response relevant to exposure and how these effects may affect driver safety
- how CO<sub>2</sub> accumulates in vehicles during limited ventilation (recirculation), with consideration of different driving environments (i.e. within tunnels or on open roads)
- measures undertaken by other relevant bodies globally to characterise or address such risks

The literature review has been supplemented by information and data collected and reported in the 2016 RMS Study. Review of this study has been included in the discussion presented.

The following sections present the information and data obtained from the literature review, with some additional discussion and evaluation, where relevant.

## Section 2. Accumulation of CO<sub>2</sub> in Vehicles

### 2.1 RMS 2016 Study

The focus of the 2016 RMS Study was the collection of data on NO<sub>2</sub> levels outside and inside vehicles while using existing tunnels in Sydney. CO<sub>2</sub> was measured within, and outside, the vehicles for the purpose of determining in-cabin air exchange rates (AER). Data collected on CO<sub>2</sub> levels during the study have been provided by RMS for the purpose of this review.

The 2016 RMS Study involved the use of 9 petrol vehicles, considered representative of the Sydney fleet, that were driven over a 30 km travel route in Sydney. The vehicles included 3 manufactured in Europe, 3 manufactured in Japan or Korea and 3 manufactured in the US or Australia (which had different expected AER performance), with different age vehicles selected in each group.

**Table 1** presents a summary of the vehicles included in the study, and notes in relation to the CO<sub>2</sub> data available.

**Table 1 Summary of vehicles included in RMS 2016 study**

Make and model	Model year	Engine size (litres)	Odometer reading (km)	Age band	Size band	Expected AER performance band (manufacturer region)	Notes
Ford Fiesta	2004	1.4	21,000	Old	Small	Worst (US/AU)	In-vehicle CO <sub>2</sub> data lost due to incorrect file names
Audi A3	2002	1.8	35,000	Old	Medium	Best (EU)	Door seals visibly degraded
Subaru Outback	2007	2.5	139,000	Old	Large	Intermediate (JP/KO)	
Fiat Punto	2007	1.4	60,000	Intermediate	Small	Best (EU)	
Toyota Corolla	2007	1.8	75,000	Intermediate	Medium	Intermediate (JP/KO)	
Holden Astra	2008	1.8	80,000	Intermediate	Large	Worst (US/AU)	Data collected for scenario with recirculation off, limited other data
Hyundai i30	2014	1.8	20,000	New	Small	Intermediate (JP/KO)	
Holden Cruze	2011	1.8	37,790	New	Medium	Worst (US/AU)	
BMW X3	2014	2	15,000	New	Large	Best (EU)	No in-cabin CO <sub>2</sub> data available

The travel route was completed over a 45 to 60 minute period and included the major tunnels currently in use within Sydney. This included the Land Cove tunnel (3.6 km in length), Sydney Harbour tunnel (2.3 km in length), Eastern Distributor tunnel (1.7 km in length) and M5 East tunnel (4 km in length). The travel route also included some shorter tunnels which were less than 600m in length and some major surface roads in Sydney. Each vehicle was tested over a 2 day period resulting in 495 pass throughs of all major tunnels (in total), with most of the travel occurring during the inter-peak period, 9am to 4:30pm. It is understood that each vehicle had 2 adults (1 driver and 1 passenger).

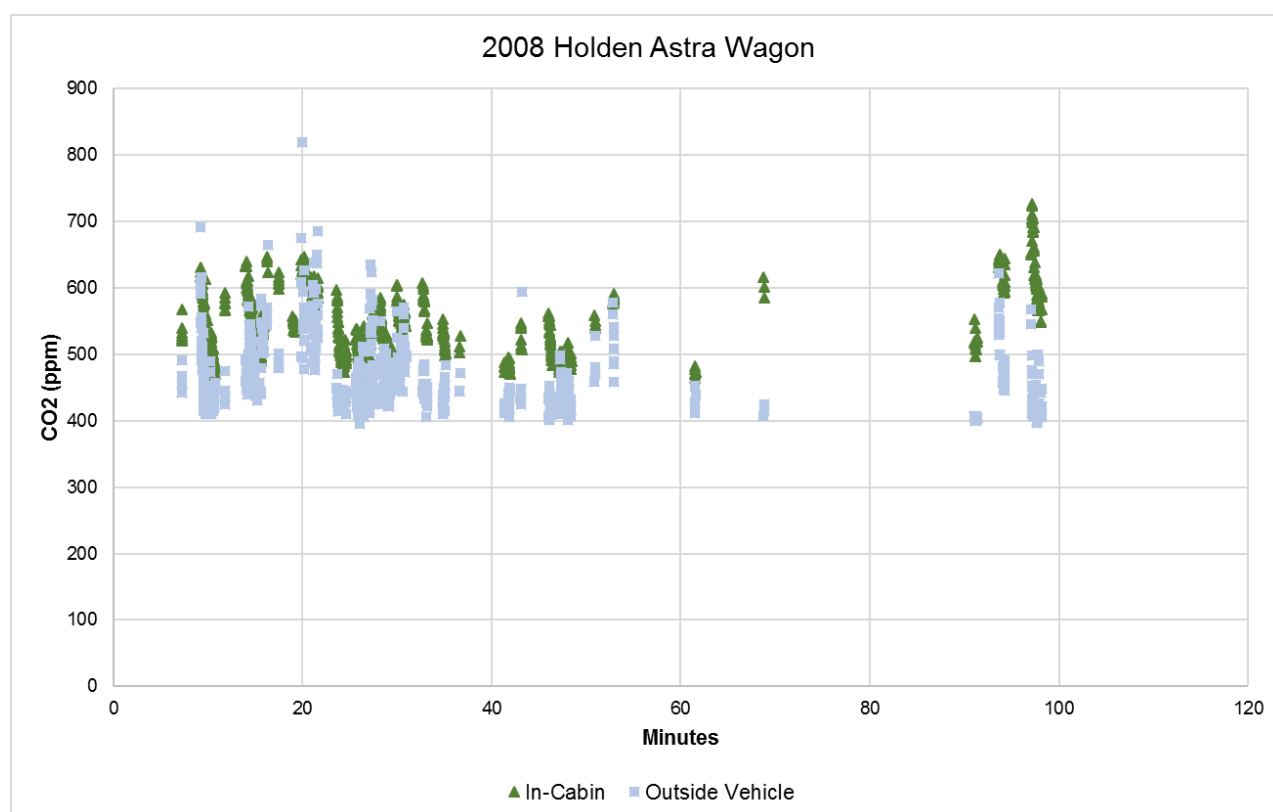
The study considered several different ventilation modes including the vehicle ventilation system either on or off recirculation, opening windows following transit through a tunnel, use of air

conditioning and different fan speeds. **Table 2** presents a summary of the ventilation modes evaluated in the study.

**Table 2** Summary of study ventilation modes

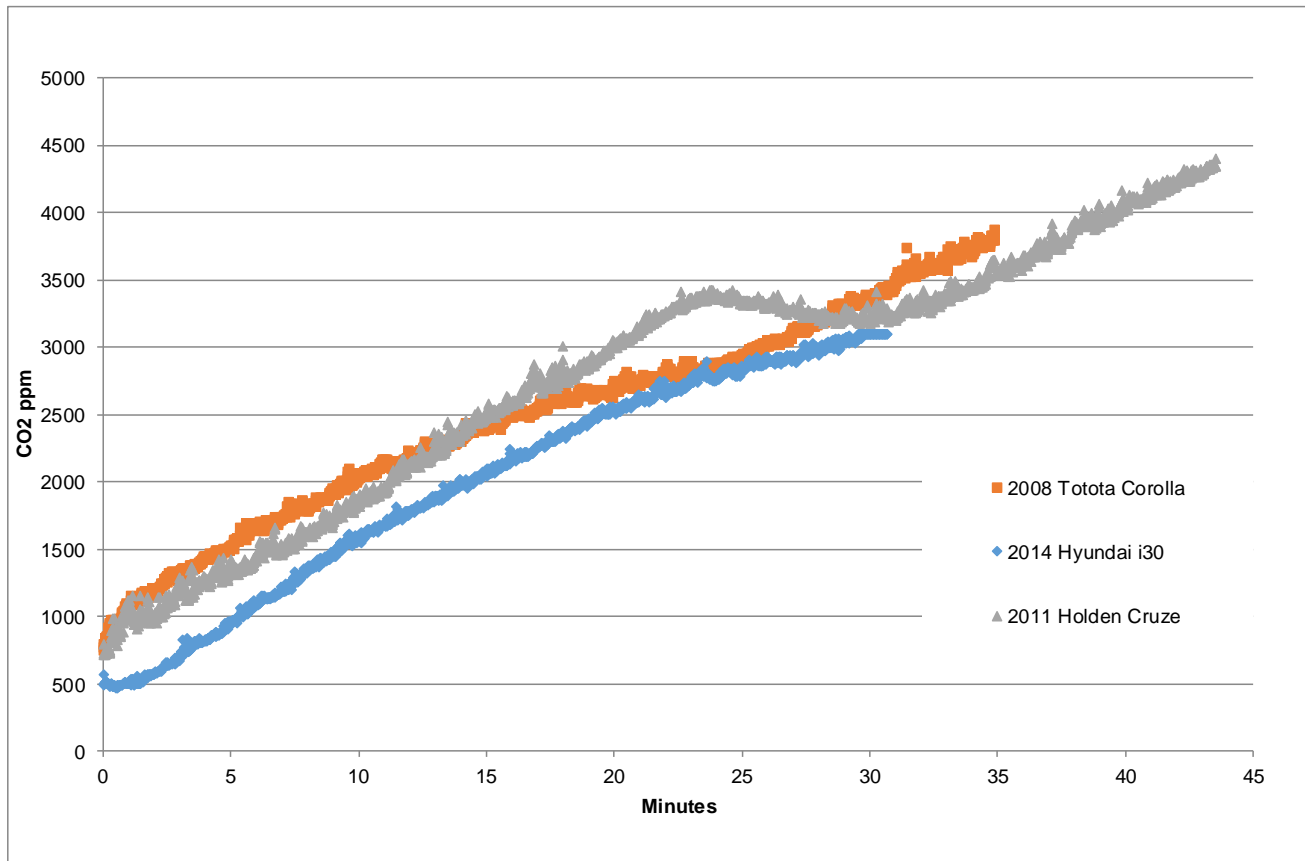
Ventilation mode	Air recirculation (RC)	Air -conditioning	Fan speed (% of maximum)
M1	On	On	50%
M2	On	Off	50%
M3	On/off <sup>(a)</sup>	Off	50%
M4	Off	Off	50%
M5	Off	Off	0%
M6	Off	Off	100%

With recirculation off, the concentration of CO<sub>2</sub> inside the vehicle was generally consistent with that reported outside the vehicle. CO<sub>2</sub> levels reported outside the vehicles was typically in the range of 400 to 600ppm. This is illustrated in **Figure 1** for the 2008 Holden Astra Wagon.



**Figure 1:** CO<sub>2</sub> Concentrations In-Cabin and Outside Vehicle, Recirculation off, air conditioning off, fan speed 100%

Where the ventilation was set on recirculate the CO<sub>2</sub> levels were observed to rise in all the vehicles where CO<sub>2</sub> data was collected. **Figure 2** presents the CO<sub>2</sub> levels measured inside 3 of the vehicles over the duration of the travel route, where ventilation was left on recirculation with the fan speed set at 50%. The rise in CO<sub>2</sub> levels, from a baseline or outdoor level appears generally linear over the duration of the sampling time. This relationship has been observed for all the vehicles included in the study.



**Figure 2: In-Cabin CO<sub>2</sub> Concentrations, recirculate on, fan speed 50%**

The duration of the study is limited to travel times between 30 and 45 minutes. The increase in CO<sub>2</sub> concentrations beyond this time period has not been determined in this study.

## 2.2 Other Studies

### 2.2.1 General

Levels of CO<sub>2</sub> have been evaluated in a number of environments where people spend time and where ventilation requirements are of importance. This includes office spaces, classrooms and more confined workplaces such as submarines and aircraft.

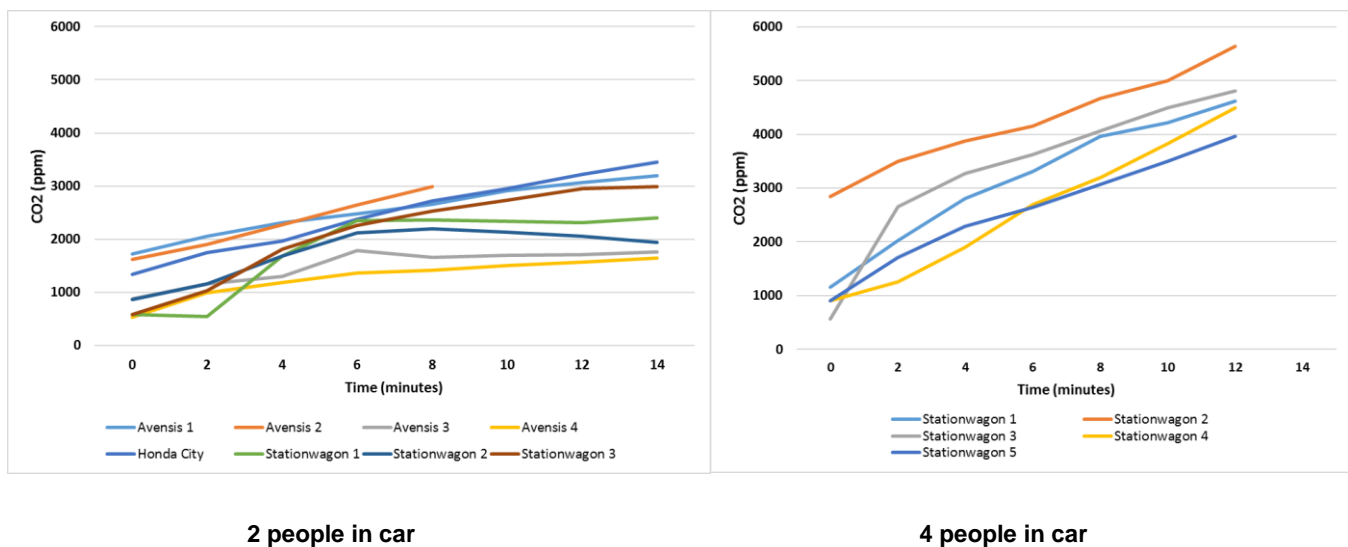
There are also a number of studies that have been undertaken to measure or model CO<sub>2</sub> levels inside vehicles.



### 2.2.2 Australian studies

A limited study conducted in Sydney in 2014<sup>1</sup> (Torres 2014) involved use of 3 different vehicles (date of manufacture unknown) to evaluate the concentrations of CO<sub>2</sub> inside vehicles with 2, 3 and 4 passengers over a 12 to 15 minute period, when the ventilation was set to recirculate. Data was also collected for a longer period of time (approximately 1 hour). This study presents limited information about the vehicles used, test conditions or the calibration of equipment used.

The short-term tests indicated rises in CO<sub>2</sub> in all the vehicles tests, as indicated in **Figure 3**. The results are generally similar for both 2 and 4 people in the vehicle.



**Figure 3: CO<sub>2</sub> Concentrations In-Cabin, Ventilation on Recirculation (Torres 2014)**

Where longer duration data was collected (up to 1 hour), CO<sub>2</sub> levels were found to continue to increase to levels in excess of 8500 to 9000 ppm (with 4 people in the vehicle), before the levels dropped significantly before rising again. This observation was noted on all the longer duration sampling events, where it was inferred by the author that the ventilation system in the vehicles had an automatic cycle that brought in fresh air after a certain amount of time, before switching back to recirculation. Insufficient information is available to determine in which vehicle this occurred, and if this observation related to all the vehicles tested. It does, however suggest that the operation of ventilation systems, particularly on recirculation, may differ between different vehicles.

<sup>1</sup> This study was conducted by a Year 10 Student, as a result the level of reporting and interpretation of the data collected is limited. The study has been included in this review as it includes data collected from vehicles travelling on Sydney roads, where limited other published data is available.

### 2.2.3 International studies

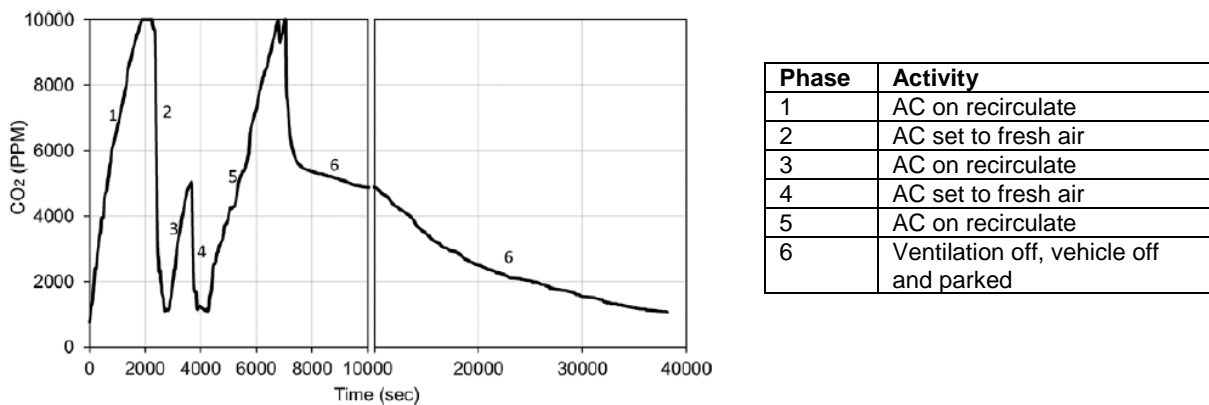
A number of published studies on CO<sub>2</sub> levels in vehicles where ventilation is on recirculation are derived from tropical countries as use of recirculation is very common in those countries to maximise use of air conditioning while minimising fuel use (to run the air conditioning).

CO<sub>2</sub> levels inside a vehicle in Malaysia (Goh et al. 2016) was undertaken with the ventilation of recirculation, and 1 or 2 occupants, travelling at different speeds (50, 70 and 90 km/hr). CO<sub>2</sub> levels were collected over a period of 90 minutes. The data collected indicated that the number of occupants was important to the levels of CO<sub>2</sub> in the vehicle and the higher the vehicle speed the lower the concentration of CO<sub>2</sub>. It was inferred that the higher speeds resulted in greater external air pressure on the vehicle, and more leakage of fresh air into the vehicle.

There was no dip in CO<sub>2</sub> levels (as was observed by Torres 2014), however CO<sub>2</sub> levels were observed to level off inside the vehicles after a period of approximately 20 to 30 minutes. No levelling off of CO<sub>2</sub> concentrations was observed in the RMS study.

An earlier study conducted in Malaysia (Mohd Sahril Mohd Fouzi, Mohamad Asyraf Othoman & Sulaiman 2014) involved the measurement of CO<sub>2</sub> inside a vehicle travelling on a country road with the air conditioning on recirculation, with CO<sub>2</sub> levels measured over a 60 minute period as well as during more intermittent events (4 x 15 minute periods). The data reported was compromised by the upper limit of the instrument (2500 ppm) clipping much of the data. However, the data collected did show increasing CO<sub>2</sub> levels over the intermittent 15 minute periods, and for the 1 hour duration, elevated CO<sub>2</sub> levels were reported for most of the sampling period (noting that no flush of fresh air occurred at the start of the test), with a drop in CO<sub>2</sub> levels reported approximately 25 minutes into the test. No reason is provided for the drop.

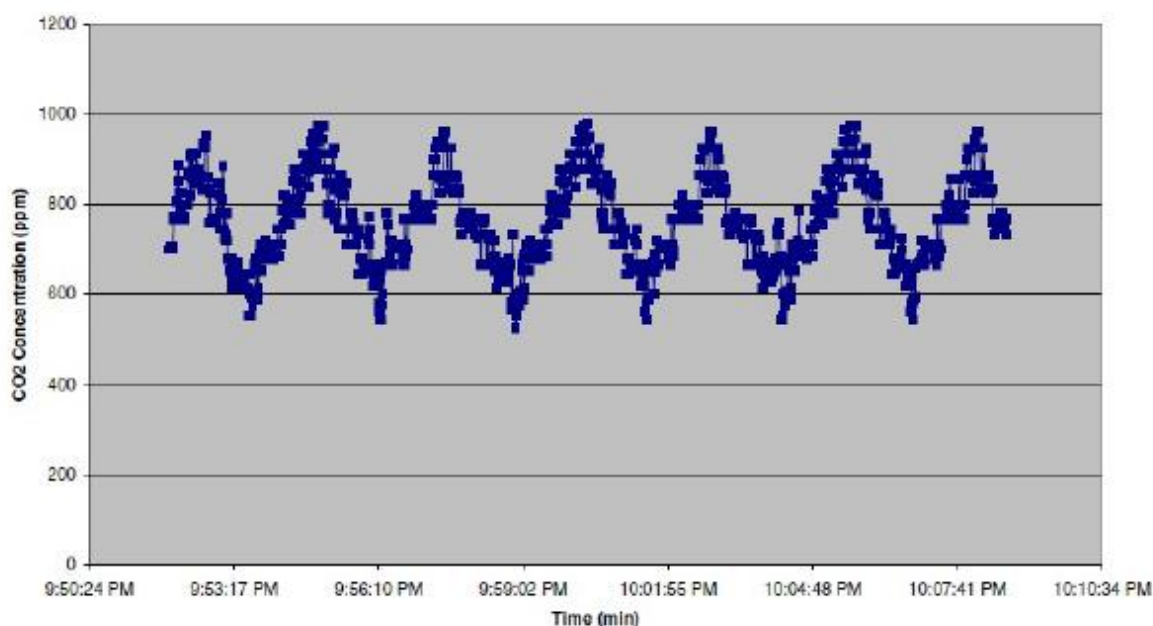
CO<sub>2</sub> levels inside vehicles in Thailand were evaluated (Luangprasert et al. 2016) with the aim of comparing the measured levels with those modelled. The study included 3 vehicles manufactured in 2007, 2010 and 2012. The data collected also evaluated how long CO<sub>2</sub> levels take to reduce in a parked vehicle. The study confirmed the previously observed rise in CO<sub>2</sub> levels when the ventilation is on recirculation, while driving. Levels of CO<sub>2</sub> reached the instrument maximum of 10,000 ppm inside the vehicle where there are 4 occupants. When parked, it was found that CO<sub>2</sub> levels returned to ambient/outdoor levels within 10 hours. This is illustrated in **Figure 4**. When the vehicle was on recirculation the increase in CO<sub>2</sub> levels measured agreed with the modelling undertaken and presented in the paper.



**Figure 4:** CO<sub>2</sub> Levels In-Cabin during different AC Usage, 4 occupants in vehicle (Luangprasert et al. 2016)

Monitoring of CO<sub>2</sub> levels in vehicles in the US (Mathur 2008, 2009b, 2009a) and Poland (Gładyszewska-Fiedoruk 2011; Gładyszewska-Fiedoruk 2011) also confirmed the increase in CO<sub>2</sub> levels in-cabin with ventilation on recirculation, with peak levels reported in these studies of 4025 ppm in-cabin. In both studies the CO<sub>2</sub> levels were found to increase over time, with data collected over different time periods ranging from 18-20 minutes to 90 minutes, and the number of occupants varying between 1 and 4 people. The rate of increase was found to occur more quickly with more occupants. The work by Mathur (2008) concluded that the ventilation or air conditioning system should not be operated in recirculation mode for extended periods of time, due to the observed build-up of CO<sub>2</sub> inside the vehicle.

Where the ventilation setting was varied between recirculation and fresh air over a 3 minute cycle, with 4 people in the vehicle, the average CO<sub>2</sub> level in-cabin was stable, as illustrated in **Figure 5**.

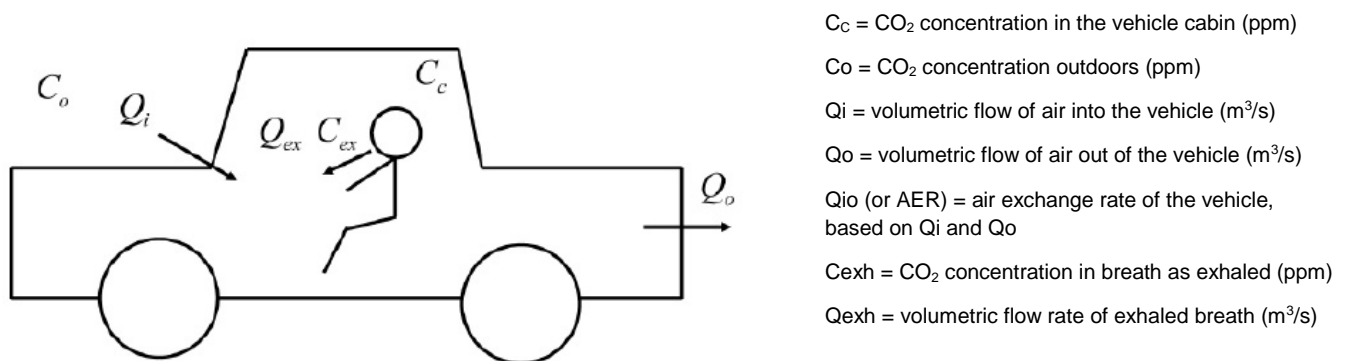


**Figure 5:** CO<sub>2</sub> in-cabin where ventilation cycled recirculate/fresh air each 3 minutes (4 occupants) in city traffic (Mathur 2008).

The field tests undertaken (Mathur 2008, 2009b, 2009a) determined that the CO<sub>2</sub> concentrations were a function of the number of occupants, vehicle speed, ambient temperatures and cabin leakage.

## 2.3 Modelling

The concentration of CO<sub>2</sub> within a vehicle can be estimated on the basis of a model. The most commonly used model for the estimation of CO<sub>2</sub> levels inside vehicles is the model derived from Jung (Jung 2013). This model describes that the CO<sub>2</sub> concentration reaches equilibrium due to the balance between CO<sub>2</sub> source (exhale of passengers), and the leakage in and out of the vehicle cabin. It is noted that vehicle and people vary and hence estimating CO<sub>2</sub> levels in all situations is difficult. However, the model described by Jung (2013) is general and considered applicable in most situations. **Figure 6** illustrates the basis for the model.



**Figure 6: Schematic of In-Cabin CO<sub>2</sub> Model (Jung 2013)**

The occupants of the vehicle are the primary source of CO<sub>2</sub> (noting that CO<sub>2</sub> is also present in outdoor air) which will depend on (Jung 2013; Matton 2015):

- The number of occupants
- The CO<sub>2</sub> concentration as exhaled, which is reported to vary from 38,000 to 56,000 ppm, per person (Scott, Kraemer & Keller 2009)
- The breathing rate of the occupants when driving
- The volume of the cabin

The movement of air into and out of the cabin, i.e. the AER, will vary has been found to depend on (Fruin et al. 2011; Hudda et al. 2012; Jung 2013; Matton 2015):

- The external air concentration
- the fraction of air that is recirculated in the vehicle, which is typically 100% for recirculation mode or 0% for the use of fresh air
- vehicle age, where the quality of seals around the vehicle body deteriorate, resulting in greater leakage
- ventilation speed, with higher ventilation speeds resulting in lower the equilibrium CO<sub>2</sub> concentration. This occurs as a result of a larger pressure difference across recirculation door in the ventilation system, resulting in more leakage of fresh air

- vehicle speeds, with higher speeds resulting in lower equilibrium CO<sub>2</sub> concentrations, as a result of higher pressure differentials between inside the cabin and the exterior of the vehicle, resulting in more outdoor air leaking into the vehicle at higher speeds

The model presented by Jung (2013) recommends that the AER be based on measurements relevant to the vehicles being evaluated under conditions relevant to the exposure (i.e. city vs freeway driving). In the RMS 2016 study, the AER was measured for the different vehicles, for 2 different speeds with and without ventilation on recirculation. Hence these data can be used to model CO<sub>2</sub> concentrations in the vehicle.

The equation presented by Jung (2013), where solved to determine the in-cabin air concentration of CO<sub>2</sub> over time, is as follows:

$$C_C = \left( C_{t0} - \left( C_0 + n \times C_{\text{exh}} \times \frac{Q_{\text{exh}}}{Q_l} \right) \right) \times \exp \left( \frac{-Q_l}{V_c} \times t \right) + \left( C_0 + n \times C_{\text{exh}} \times \frac{Q_{\text{exh}}}{Q_l} \right)$$

$C_C$  = CO<sub>2</sub> concentration in the vehicle cabin (ppm)

$C_{t0}$  = CO<sub>2</sub> concentration in the vehicle at t=0, assumed to be equal to the outdoor concentration unless otherwise stated

$C_0$  = CO<sub>2</sub> concentration outdoors (ppm)

$C_{\text{exh}}$  = CO<sub>2</sub> concentration in breath as exhaled (ppm)

$n$  = number of occupants in the vehicle

$Q_{\text{exh}}$  = volumetric flow rate of exhaled breath (m<sup>3</sup>/s)

$Q_l$  = body leakage volumetric flow rate, calculated as AER x  $V_c$  (m<sup>3</sup>/s)

AER = air exchange rate for the cabin of the vehicle, either measured (preferred) or modelled (1/s)

$V_c$  = volume of the cabin (m<sup>3</sup>)

$t$  = time (s)

This equation is consistent with that presented by Cornak et al (Cornak, Horak & Chaladek 2012). The work undertaken by Mather has been further extended to develop a model for estimating CO<sub>2</sub> levels in-cabin<sup>2</sup>. The paper related to this model is not yet available, hence it cannot be compared with the model described above (or below).

Other models have been developed to evaluate the movement of outdoor pollutants into the cabin of vehicles, including the model developed to evaluate the movement of particulates into vehicles (Knibbs, de Dear & Atkinson 2009; Knibbs, de Dear & Morawska 2010) adopted and used in the assessment of NO<sub>2</sub> in the 2016 RMS study.

<sup>2</sup> <http://papers.sae.org/2017-01-0163/>

Much of the work conducted on the movement of pollutants into and out of vehicles, relates to measuring or modelling the AER. The most comprehensive study of AER was undertaken in California (Fruin et al. 2011), where 59 vehicles were tested under different conditions, and builds on an earlier study conducted using 4 different vehicles (Ott, Klepeis & Switzer 2008). Other studies have also been undertaken in Australia, using 6 different vehicles (Knibbs, de Dear & Atkinson 2009; Knibbs, de Dear & Morawska 2010).

These studies have been compiled and reviewed (Hudda et al. 2012), to form the basis of a generalised estimating equation for calculating AER with recirculation on and off. These relationships calculate the AER, based on vehicle speed, the age of the vehicle, fan strength (significant for fresh air ventilation only), volume of cabin and manufacture of vehicle. The model presented by Hudda et al (2012) was evaluated in the 2016 RMS study.

A review has been undertaken to determine how well the model as described by Jung (2013) correlates with the CO<sub>2</sub> data collected in the 2016 RMS study. The model adopted for evaluating NO<sub>2</sub> within the RMS 2016 study has also been evaluated.

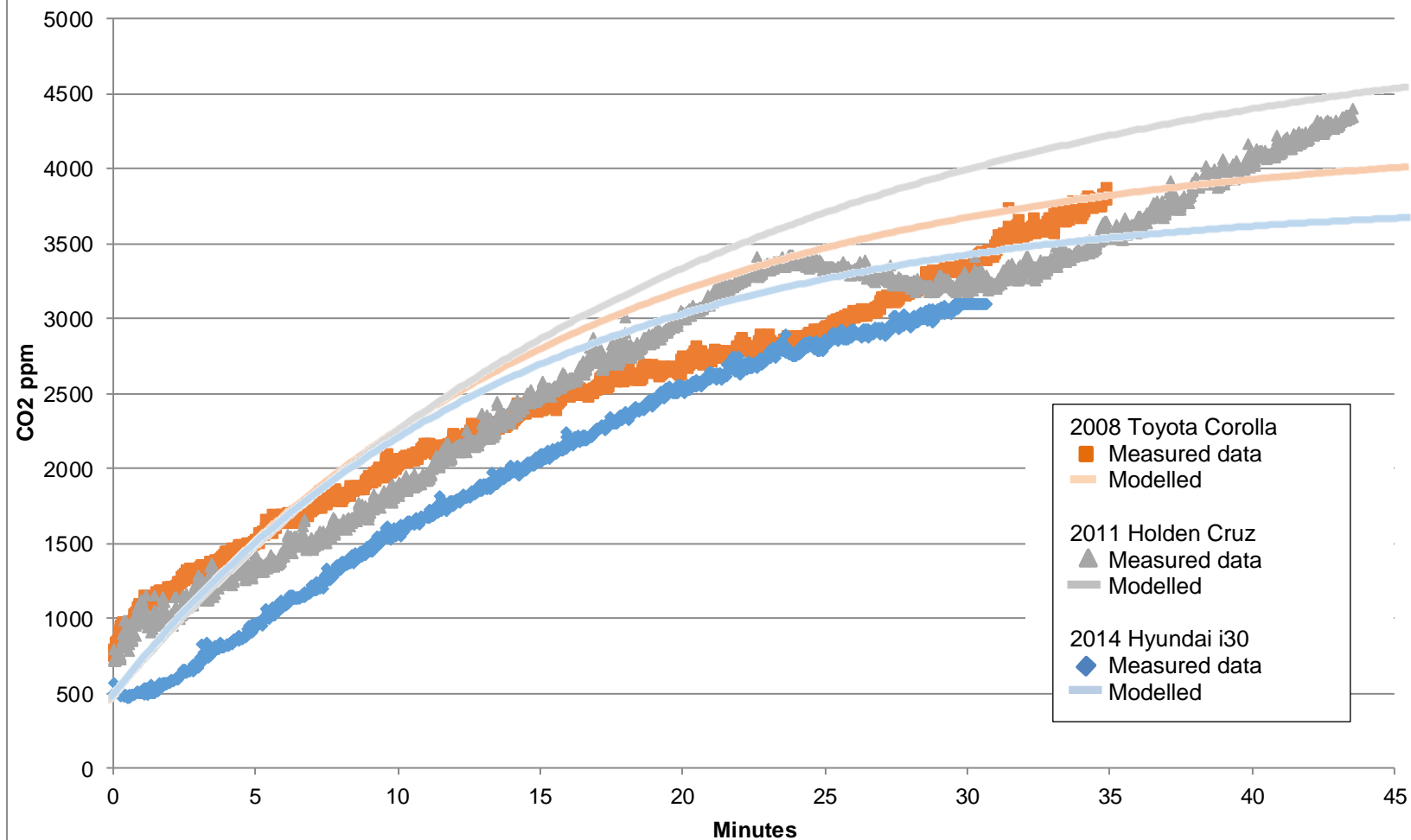
For comparison, the CO<sub>2</sub> data collected in the 2016 RMS study for the 2008 Toyota Corolla, 2011 Holden Cruz and 2014 Hyundai i30 (which provided the most continuous data sets for the scenario where recirculation is on), has been compared with modelled CO<sub>2</sub> concentrations. The modelled concentrations are based on the vehicle parameters relevant to each of the vehicles as well as the measured AER for the vehicles, at 60 km/hr. **Table 3** presents a summary of the input parameters adopted for the modelling of CO<sub>2</sub> in the cabins of these 3 vehicles.

**Table 3** Summary of Parameters Adopted for Modelling CO<sub>2</sub> Accumulation In-Cabin, Recirculation On

Parameter	Units	Value adopted for vehicles considered			Notes
		2008 Toyota Corolla	2011 Holden Cruz	2014 Hyundai i30	
C <sub>t0</sub>	ppm	500	500	500	Assume equal to outdoor value
C <sub>o</sub>	ppm	500	500	500	Typical value reported in 2016 RMS study (noting the concentration increases the distance in the tunnel, varying from 400 to 600 ppm)
C <sub>exh</sub>	ppm	35,000	35,000	35,000	Value adopted by Cornak et al (2012). The value is lower than the range 38,000 to 56,000 ppm previously noted (Scott, Kraemer & Keller 2009). The lower value has been adopted as this provides the better correlation with the measured data from the 2016 RMS study
n	-	2	2	2	2 adults (driver and passenger) were present in the vehicles used in the 2016 RMS study
Q <sub>exh</sub>	m <sup>3</sup> /s	0.00016	0.00016	0.00016	Breathing rate for adults driving as presented by the USEPA (USEPA 2011)
AER	1/s	0.0011	0.0008	0.0012	Measured in the 2016 RMS study, recirculation on, travelling at 60 km/hr
V <sub>c</sub>	m <sup>3</sup>	2.81	3.04	2.81	Relevant to each vehicle evaluated
Q <sub>l</sub>	m <sup>3</sup> /s	0.0031	0.0025	0.0035	Calculated based on AEC and V <sub>c</sub>

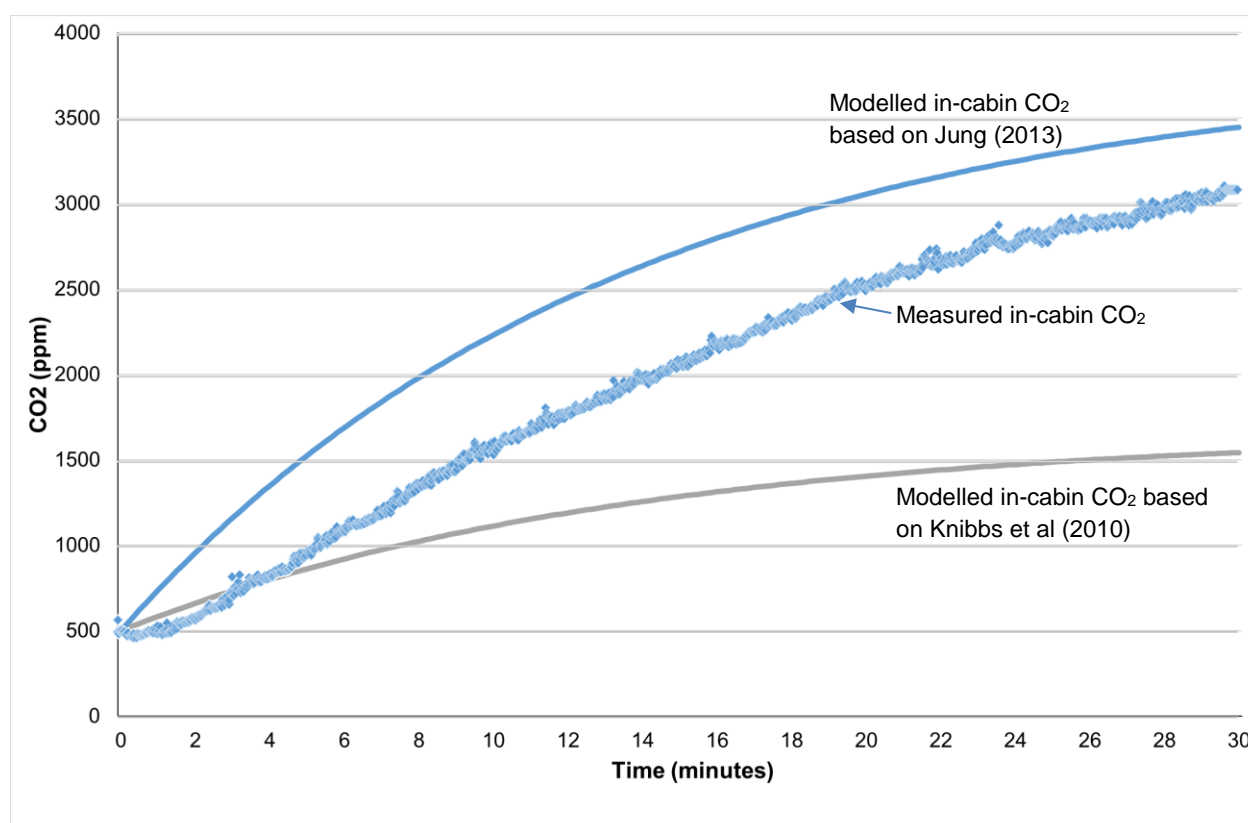


**Figure 7: Comparison of Measured and Modelled\* CO<sub>2</sub> Concentrations, 2016 RMS Study Data**



Based on the above parameters, **Figure 7** presents a comparison of the measured and modelled CO<sub>2</sub> concentrations for a period of time up to 45 minutes. CO<sub>2</sub> concentrations are modelled/estimated to increase a little faster, and be a little higher than those measured in the field trial. The modelled results, after 35 to 45 minutes, are either consistent with or slightly higher than measured. The shape of the modelled CO<sub>2</sub> concentrations is not as linear as measured, however the modelling does start to show that the increase in CO<sub>2</sub> levels starts to fall off with time. It is expected that at some point an equilibrium CO<sub>2</sub> concentration would be established, where the input from occupants would equal the removal due to leakage out of the vehicle. It is noted that the CO<sub>2</sub> concentrations are predicted to be higher than presented in **Figure 7**, where higher concentrations of CO<sub>2</sub> in exhaled breath (up to 58,000 ppm) are considered. This may be relevant when considering the presence of different occupants in the vehicles.

It is noted that where the Knibbs et al (2010) model is used CO<sub>2</sub> concentrations are significantly underestimated, by a factor of 2-fold. Hence the Knibbs model is not considered suitable for the estimation of CO<sub>2</sub> concentrations and exposures in-cabin. This difference of predicted CO<sub>2</sub> concentrations is illustrated in **Figure 8**.



**Figure 8:** Comparison of modelled CO<sub>2</sub> in-cabin for Hyundai i30 where ventilation is on recirculation, Knibbs et al (2010) and Jung (2013)

## Section 3. Health Effects of Exposure to CO<sub>2</sub>

---

### 3.1 General

CO<sub>2</sub> is an important trace gas in Earth's atmosphere. Currently it constitutes about 0.04% (equal to 400 parts per million; ppm) by volume of the atmosphere. The average indoor concentration of CO<sub>2</sub> is 0.08% to 0.1%. At normal temperature and pressure, CO<sub>2</sub> is an odourless, colourless, and heavier than air gas, with a faintly pungent odour (at high concentrations).

CO<sub>2</sub> has numerous applications. It is used in food freezing and chilling, beverage carbonation, chemical manufacture, fire prevention and extinction, metal working, and oil and gas recovery. It is produced on combustion of all carbonaceous fuels and is a product of animal metabolism. It is also known to be produced during putrefaction, and fermentation. In air, carbon dioxide is a very stable and non-flammable compound.

CO<sub>2</sub> is a normal constituent of the human body generated by cellular respiration. CO<sub>2</sub> diffuses from cells into the surrounding capillaries and is carried by the blood either bound to haemoglobin or dissolved as CO<sub>2</sub>, carbonic acid, or bicarbonate ion. Dissolved CO<sub>2</sub> in bodily fluids undergoes hydration according to the following reaction:



This reaction can interfere with the acid-base balance in humans. A minor amount of CO<sub>2</sub> can be bound to plasma proteins to form carbamino compounds.

Humans produce CO<sub>2</sub> via oxidative metabolism of carbohydrates, fatty acids, and amino acids; the production rate is dependent on the caloric expenditure of the individual and its partial pressure under normal conditions in pulmonary capillary blood (almost 7% or 46 mmHg) is greater than that in alveolar air (6% or 40 mmHg). The gas is exchanged freely through the alveolar membrane and is thus released from the lungs by diffusion because of the concentration gradient existing between blood and air in the alveoli.

Its free diffusion through the lipid cell membranes allows it to be one of the main regulators of intracellular pH acting as a stimulant or a brake in numerous cellular processes. Because of its free diffusion through tissue membranes, the toxicological effects of CO<sub>2</sub> appear very rapidly and are mainly observed in the blood pH, lungs, heart and central nervous system.

Hypercapnia, also known as hypercarbia and CO<sub>2</sub> retention, is a condition of abnormally elevated CO<sub>2</sub> levels in the blood. Possible causes for hypercapnia include direct CO<sub>2</sub> exposure such as the use of dry ice, food and floral preservation; living in closed and restrained environments such as spacecraft, submarines, or using SCUBA diving re-breathing apparatus; exposure to combustion gases such as tobacco smoke or vehicle emissions; pathological conditions such as pulmonary diseases and sleep apnea. Hypercapnia may have systemic effects as well as pathologies of various organs including lung, heart, CNS, neuroendocrine system, bladder, liver, kidney and reproductive organs.

The following sections present additional detail more specific to the assessment of health effects that may occur as a result of acute or short-duration exposures to CO<sub>2</sub>.

### 3.2 Acute and subchronic toxicity

CO<sub>2</sub> is a simple asphyxiant and lethal asphyxiations have been reported at concentrations as low as 11% (Hamilton and Hardy 2015). Loss of consciousness can occur within a minute of exposure at 30% and within 5-10 minutes (min) of exposure at 10% (HSDB 2004). The effects of concentrations of CO<sub>2</sub> between 0.7% and 30% in humans and animals and include tremor, headaches, chest pain, respiratory and cardiovascular effects, and visual and other central nervous system (CNS) effects.

The respiratory, cardiovascular, and CNS effects of CO<sub>2</sub> are related to the decreases in blood and tissue pH that result from exposures (HSDB 2004). Changes in pH act directly and indirectly on those systems. The pH changes also trigger various compensatory mechanisms, including increased ventilation to reduce excess CO<sub>2</sub> in the bloodstream, increased renal acid excretion to restore acid-base balance, and sympathetic nervous system stimulation to counteract the direct effects of pH changes on heart contractility and vasodilation (HSDB 2004). The key effects for setting Emergency and Continuous Exposure Guidance Levels (EEGL and CEGL) values are tremor, headache, hyperventilation, visual impairment, and CNS impairment (NRC 2007).

The following key acute effects have been identified in studies conducted:

**Tremor** – seen in 10 of 12 subjects exposed at 0.7-1.4% for 10-20 minute (Sechzer et al. 1960)

**Dyspnea** – is a commonly reported end point and can be induced by acute exposures to CO<sub>2</sub> at >3% (NRC 2007). Hyperventilation without dyspnea occurs at exposure concentrations as low as 1% (NRC 2007). Dyspnea attributable to CO<sub>2</sub> is aggravated by increasing the level of exertion. The bulk of the data indicate a no-observed-adverse-effect level (NOAEL) for CO<sub>2</sub> of about 2.8% ppm (for both acute and chronic exposure) on the basis of the findings on dyspnea and intercostal pain (NRC 2007).

**Headaches** - are commonly associated with increased CO<sub>2</sub> concentrations in inspired air, but there is conflicting data on the concentrations reliably associated with that end point. There may also be an effect of exertion, because CO<sub>2</sub> seems to cause more headaches at lower concentrations during exercise than it does during rest. Concentrations tested ranged from 1-8% and headaches induced by CO<sub>2</sub> seem to be both mild and reversible (NRC 2007). NASA researchers using health reports from astronauts have used a benchmark dose approach to derive a 1% risk level (for headaches) of about 2.3 mm Hg (0.35% CO<sub>2</sub>) with some variation between models (James, Meyers & Alexander 2011).

**Hyperventilation** – is seen as a physiological compensatory mechanism to reduce excess CO<sub>2</sub> in the blood. Various studies report that it takes an exposure concentration of at least 1% to increase minute-volume after a plateau in the hyperventilatory response has been reached, usually after a few hours.

**Acidosis** - exposures to CO<sub>2</sub> at concentrations much higher than those in ambient air lead to increased partial pressure of CO<sub>2</sub> in alveoli and blood. That causes a lowering of blood pH, which is eventually buffered by blood proteins and bicarbonate. Exposures as low as 0.7% can lower blood pH by up to 0.05 units, but even at high exposures, renal compensation seems to occur in healthy subjects. Compensation occurs over a variable period of time, but effects of lowered pH on clinical status or performance have not been reported either experimentally or operationally (NRC 2007).

**Cardiovascular endpoints** – few changes in electrocardiographic measurements have been reported in subjects at CO<sub>2</sub> concentrations lower than 3% (NRC, 2007).

**Sensory effects** - it is well established that CO<sub>2</sub> acutely impairs vision and hearing at concentrations exceeding about 2.5% (NRC 2007).

**CNS effects** – most studies summarised by the NRC (2007) reported minimal neurobehavioral effects (learning tasks) at CO<sub>2</sub> concentrations between 1.5-4% with exposure periods of 2 weeks; the NRC (2007) adopted a NOAEL of 3%.

NASA has conducted considerable research on CO<sub>2</sub> levels and astronaut health. James et al (2011) reports that some astronauts are experiencing adverse health effects from on-orbit exposure to CO<sub>2</sub> levels well below the current Spacecraft Maximum Allowable Concentration (SMAC), which is 0.8% CO<sub>2</sub> for 180 days of exposure. Headaches were reported at CO<sub>2</sub> concentrations as low as 0.3% but neuro-cognitive effects were not. NASA also reports an interesting genetic variation in CO<sub>2</sub> response and notes that pigs have widely variable response to high doses of CO<sub>2</sub> and that humans with panic disorder (PD) respond differently to high levels of CO<sub>2</sub> based on PD subtype (James, Meyers & Alexander 2011). Investigations by Singh (Singh 1984a, 1984b) reported a positive correlation between the hyper-ventilatory response to CO<sub>2</sub> (ranging between 6-10%) and neurotic personality traits in a group of male and female normal subjects using a re-breathing apparatus for 4 minutes.

A study by Vehviläinen et al (Vehvilainen et al. 2016) measured physiological stress reactions, alertness, and subjective symptoms during simulated office work using volunteer male subjects during 3 lots of 4 hour work meetings in an office room, both in a ventilated and a non-ventilated environment. The environmental parameters measured included CO<sub>2</sub>, temperature, and relative humidity. The physiological test battery consisted of measuring autonomic nervous system functions, salivary stress hormones, blood CO<sub>2</sub> level and oxygen saturation, skin temperatures, thermal sensations, vigilance, and sleepiness. The subjects were isolated for the 4 hours per day in a sealed room with both ventilation and no ventilation achieving mean CO<sub>2</sub> levels between 0.1% and 0.27% (max. 0.5%) respectively. The higher CO<sub>2</sub> concentrations were associated with higher CO<sub>2</sub> concentrations in tissues, changes in heart rate variation, and an increase of peripheral blood circulation. Subjective responses were reported as loss of concentration, dizziness, headache, cold feet, shivering, and irritation of the eyes, all of which resolved after 24 hours. Apart from increased CO<sub>2</sub> levels, uncontrolled and possibly contributing factors to the adverse effects were increases in the concentrations of total volatile organic compounds (VOCs) and fine particles, and increased temperature and relative humidity.

Vercruyssen et al (Vercruyssen, Kamon & Hancock 2007) tested six highly trained male participants who performed psychomotor tests while breathing for 60 minutes, 3 different CO<sub>2</sub> mixtures (room air, 3% CO<sub>2</sub>, or 4% CO<sub>2</sub>) prior to, between, and following two 15 minute treadmill exercise bouts (70% VO<sub>2max</sub>). Each individual was extensively practiced (at least 4 days) before testing began, and both gas conditions and order of tasks were counterbalanced. Results showed physiological reactions and work-related psychomotor effects, but no effects of gas concentration on addition, multiplication, grammatical reasoning, or dynamic postural balance.

A review by Seppanen et al (Seppänen, Fisk & Mendell 1999) summarised the associations of building ventilation rates and CO<sub>2</sub> concentrations in non-residential and non-industrial buildings (primarily offices) with health and other human outcomes using the amelioration of symptoms associated with 'Sick Building Syndrome' or SBS. The authors report that ventilation rates below 10L/sec were associated with SBS and a weak association between CO<sub>2</sub> concentrations above 0.08% and SBS. A more recent study (Lu et al. 2015) of 417 office workers in high-rise buildings studied the association between self-reported symptoms of SBS and indoor measurements of CO<sub>2</sub> concentration and VOCs. Mean indoor CO<sub>2</sub> was 0.12% (range 0.04-0.28%) and increasing CO<sub>2</sub> was associated with increasing reports of SBS symptoms.

The body compensates for high levels of CO<sub>2</sub>, through a combination of increased breathing, blood pH buffering, kidney and bone adaptations depending on the length of continuous exposure, until we can resume breathing lower levels of CO<sub>2</sub>. There are very few studies that indicate what level of CO<sub>2</sub> in the air will induce the longer-term compensation activities. Kidney involvement has been documented to occur in animals at 2,000 ppm (Schaefer, K. E. et al. 1979) and 7,000 ppm in humans (Halperin 2007) although no lower limits were defined. One author (Robertson 2006) suggests that the body compensates for CO<sub>2</sub> exposures at levels as low as 430 ppm. These compensation mechanisms can produce health effects including kidney calcification and bone loss, with short-term studies indicating health effects association with body compensation for blood acidity at CO<sub>2</sub> levels around 4000 ppm (Bierwirth 2016).

### **3.3 CO<sub>2</sub> Effects on Cognitive Performances and Productivity**

In a carefully controlled study, Allen et al (Allen et al. 2016) investigated the effects of several indoor environmental quality parameters on an objective measure of cognitive function. They used a double-blinded study design that included repeated measures of cognitive function on the same individual, characterisation of potential confounding indoor environmental quality (IEQ) variables, and midweek testing to avoid Monday/Friday effects. The 3 test parameters that were experimentally controlled were ventilation with outdoor air, CO<sub>2</sub>, and VOCs. All participants in the simulated office environments received the same exposures on each day, with exposures varying each day. The cognitive assessment was performed daily using the Strategic Management Simulation (SMS) software tool, a computer-based test that has been designed to assess higher-order decision making. Testing at two CO<sub>2</sub> concentrations (0.0945% [945 ppm] and 0.14% [1400 ppm]) and with two ventilation rates reported, on average, a 0.044% increase in CO<sub>2</sub> was associated with a 21% decrease in a typical participant's cognitive scores across all domains after adjusting for participant.

In a well-designed study investigating the effect of CO<sub>2</sub> concentration on human decision making, Satish et al (Satish et al. 2012) exposed twenty-two subjects to CO<sub>2</sub> at 0.06% (600 ppm), 0.1% (1000 ppm), and 0.25% (2500 ppm) in an office-like chamber, in 6 groups. Each group was exposed to these conditions in 3 lots of 2.5 hour sessions, all on 1 day, with exposure order balanced across groups. Under each condition, participants completed a computer-based test of decision-making performance as well as questionnaires on health symptoms and perceived air quality. Relative to 0.06%, at 0.1% CO<sub>2</sub>, moderate and statistically significant decrements occurred in 6 of 9 scales of decision-making performance (basic activity, applied activity, task orientation, initiative, information usage, breadth of approach, and basic strategy). At 0.25%, large and statistically significant reductions occurred in seven scales of decision-making performance. Compared with mean raw



scores at 0.06% CO<sub>2</sub>, mean raw scores at 0.1% CO<sub>2</sub> were 11–23% lower, and at 0.25% CO<sub>2</sub> were 44–94% lower.

Air quality studies in vehicles include work by Constantin et al (Constantin et al. 2016) who measured CO<sub>2</sub> concentrations inside different standing vehicles with up to five occupants, with and without circulating air. Measures of perceived air quality were assessed but no measures of cognitive performance were undertaken. CO<sub>2</sub> measurements performed in a stationary vehicle show that reaching the experimental limit of 0.5% (5000 ppm) ranges from 13 minutes (for four people) to 56 minutes (for 1 person). Participants reported a complex response to perception of air quality air recirculation. The authors suggest that there is an accommodation or tolerance with polluted air and it is perceived as being of a higher quality than it actually is and that at 0.5% CO<sub>2</sub> the air is not perceived as being of a low quality.

In chamber studies (Kajtar & Herczeg 2012) with human volunteers exposed to CO<sub>2</sub> concentrations of 0.06% - 0.5% (600 to 5000 ppm) measures of mental function and objective and perceived measures of physical comfort were taken during 2-3 hour sessions of exposure. Increased fatigue was reported at levels of 0.3% CO<sub>2</sub> (3000 ppm) compared with 0.06% (600 ppm). This study also measured some physiological parameters with heart rate analysis suggesting significantly increased mental effort at 0.3% - 0.4% (3000 to 4000 ppm).

### 3.4 Sensitive Populations

The following populations have been identified (Rice 2003) as potentially sensitive to the effects of CO<sub>2</sub>:

- Individuals Performing Complex Tasks: CO<sub>2</sub> can significantly diminish performance on tasks requiring psychomotor coordination, visual perception, attention, and rapid response.
- Infants & Children: Infants and children breathe more air than adults relative to their body size and thus they tend to be more susceptible to respiratory exposures.
- Medicated Patients: Respiratory centre stimulation by CO<sub>2</sub> is depressed by anoxia and by various drugs such as alcohol, anaesthetics, morphine, barbiturates, etc. In these cases, compensatory mechanisms do not protect and symptomology does not alert the individual to the presence of high CO<sub>2</sub> levels.
- Panic Disorder Patients: Panic disorder patients experience an increased frequency of panic attacks at 5% CO<sub>2</sub>. Anxiety and somatic symptoms also are significantly increased and are similar to those experienced by healthy subjects exposed to 7.5% CO<sub>2</sub>. Panic attack and significant anxiety can affect the ability of the individual to exercise appropriate judgment in dangerous situations.
- Individuals with pre-existing medical conditions, including:
  - Cerebral Disease & Trauma Patients: CO<sub>2</sub> is a very potent cerebrovascular dilator. CO<sub>2</sub> exposure can seriously compromise patients in coma or with head injury, increased intracranial pressure or bleeding, or expanding lesions.
  - Pulmonary & Coronary Disease Patients: CO<sub>2</sub> exposure can increase pulmonary pressure as well as systemic BP and should be avoided in individuals with systemic or pulmonary hypertension.

### 3.5 Guidelines for Short-Term Exposure to CO<sub>2</sub>

Most guidelines available for CO<sub>2</sub> relate to occupational exposures (including confined space guidelines) or guidelines relevant to office, classrooms or other public spaces. The guidelines for indoor air are also commonly set to manage comfort, such as the perception of body odours and other pollutants.

Safe Work Australia<sup>3</sup> has established the following guidelines for CO<sub>2</sub> in occupational environments:

- TWA (8 hour workday average) = 5,000 ppm, with a higher limit of 12,500 ppm established for coal mines
- STEL (15 minute short term exposure level) = 30,000 ppm for all environments including coal mines

The above guidelines are based on direct effects on acidification of the blood and are consistent with occupational guidelines from the US, available from ACGIH.

NIOSH, also included in Standards Australia document TS ISO 16976.3:2015, in relation to CO<sub>2</sub> levels and effects in humans. This is based on levels related to metabolic acidosis.

**Table 4 Potential effects and limitations on human tolerance imposed by exposure to CO<sub>2</sub>**

Average CO <sub>2</sub> in air (% [ppm])	At rest		Extremely high work rate	
	Potential effects and/or limitations	Exposure limit (time)	Potential effects and/or limitations	Exposure limit (time)
1.5 [15,000 ppm]	No restrictions on activity	Indefinite	Increase in ventilation	Unknown
2.5 [25,000 ppm]	Increase in ventilation	Unknown	Increase in ventilation	2 hours
3 [30,000 ppm]	Increase in ventilation, no restrictions within the exposure limit	15 hours	Increase in ventilation	30 minutes
5 [50,000 ppm]	Increase in ventilation, no restrictions within the exposure limit	8 hours	Increase in ventilation, collapse/unconsciousness	15 minutes
7 [70,000 ppm]	Increase in ventilation, severe limitations on activity	<30 minutes	Collapse/unconsciousness	NA
10 [100,000 ppm]	Increase in heart rate, collapse/unconsciousness	<2 minutes	Collapse/unconsciousness	NA

In relation to aircraft, the US Federal Aviation Authority has adopted a guideline of 0.5% (5000 ppm) as the maximum level permitted in the aircraft cabin, consistent with the occupational TWA. The same guideline is adopted for long-term exposures in spacecraft. It is noted that a short-duration guideline of 2% (20,000 ppm) is also established based on tolerance to mild headache and hypertension over a 1 hour exposure period that is insignificant to performance (Law, Watkins & Alexander 2010).

<sup>3</sup> <http://hcis.safeworkaustralia.gov.au/ExposureStandards>

The American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE) have set a standard of 1000 ppm for CO<sub>2</sub> in indoor spaces. In this guideline, CO<sub>2</sub> is used as a surrogate for the presence of other bioeffluents that cause odours that may be viewed as unacceptable by others, rather than based on the health effects of CO<sub>2</sub>. The guideline is established to enable appropriate ventilation controls to be designed. This guideline relates to continuous exposures indoors.

The UK (EFA 2014) has established a guideline of 1500 ppm for CO<sub>2</sub> in classrooms, also based on maintaining suitable ventilation levels. The guideline notes that the maximum level in a classroom should not exceed 5000 ppm, based on the occupational TWA.

Health Canada (Health Canada 1987) established an indoor air guideline of 3500 ppm for long-term exposures indoors, derived from the lowest effect where blood acidity has been observed with a 2 fold safety factor.

The Massachusetts Department of Public Health<sup>4</sup> has adopted an indoor air guideline for CO<sub>2</sub> of 800 ppm, with a lower value of 600 ppm adopted for classrooms to protect more sensitive children.

There are no health based guidelines established for in-cabin areas in vehicles or other modes of transport.

### **3.6 Summary of health effects associated with short-term exposures**

This summary review is focussed on the acute human health effects of short-term exposure to elevated CO<sub>2</sub> levels.

The acute and chronic toxicity of CO<sub>2</sub> is well studied in animals and humans. At high concentrations, CO<sub>2</sub> is an asphyxiant and causes unconsciousness and death at concentrations ranging from 10% to 30%.

At low concentrations, up to 3% (30,000 ppm) under chronic exposure conditions, CO<sub>2</sub> seems to be physically tolerated in humans over short term periods with various physiological adaptations to the induced hypercapnia (high blood CO<sub>2</sub>). Various agencies have set occupational exposure levels of 0.5% (5000 ppm) CO<sub>2</sub> to guard against health effects, principally acidification of blood. Most indoor guidelines are based on the use of CO<sub>2</sub> as a surrogate for general indoor air comfort and ventilation requirements, not the protection of health.

However, recent studies indicate that subtle adverse effects of even short-term low level CO<sub>2</sub> exposure can be measured. Such adverse effects include headaches and visual disturbances effects at CO<sub>2</sub> levels around 0.3% (3000 ppm).

The more recent studies have described disease symptoms at levels around 0.1% (1000 ppm) CO<sub>2</sub> and reduced cognitive ability in humans at around 0.08% (800 ppm) CO<sub>2</sub>. The studies of cognitive effects were conducted at CO<sub>2</sub> levels that represent typical conditions currently present in offices, classrooms and apartments and report modest reductions in multiple aspects of decision making at

---

<sup>4</sup> [www.mass.gov/eohhs/docs/dph/environmental/iaq/appendices/carbon-dioxide.doc](http://www.mass.gov/eohhs/docs/dph/environmental/iaq/appendices/carbon-dioxide.doc)

exposures as low as 0.095% (950 ppm). It is possible that such effects may not be noticed in daily life. Such subtle CO<sub>2</sub> induced decline in cognitive ability is possibly due to increased cerebral blood flow (CBF) and the resulting effects on central nervous system and brain cortical function.

**Table 4** presents a summary of selected studies relevant to low level CO<sub>2</sub> concentrations (below 1% (10,000 ppm) is given in the table below, adapted from Bierwirth (Bierwirth 2016). The available guidelines for CO<sub>2</sub> are also included in the table for reference (presented in blue text).

**Table 4 Summary of health effects from breathing CO<sub>2</sub> at concentrations below 1%**

CO <sub>2</sub> Level	Health effect	Exposure duration	Source
10,000 ppm (1%)	Kidney calcification, decreased bone formation and increased bone resorption in guinea pigs	6 weeks	(Schaefer, K. E. et al. 1979)
8500 ppm	Increased lung dead space volume	20 days	(Rice 2004)
7000 ppm	35% increase in cerebral blood flow (implications for cognitive effects seen in other studies)	23 days	(Sliwka et al. 1998)
5000-6600 ppm	Headaches, lethargy, moodiness, mental slowness, emotional irritation, sleep disruption	Short-term	(Cronyn, Watkins & Alexander 2012; Law, Watkins & Alexander 2010)
5000 ppm	Kidney calcification, bone degradation in guinea pigs	8 weeks	(Schaefer, K. E. et al. 1979)
5000 ppm	Current allowable levels for continuous exposure in aircraft, submarines and spacecraft	Operational continuous	(Cronyn, Watkins & Alexander 2012; Halperin 2007)
5000 ppm	Occupational exposure limit (TWA) for a work day, based on acidification of blood	8 hours	Safe Work Australia, ACGIH, OSHA
2000 ppm	Kidney effects in animals (likely calcification) - incomplete study	Chronic studies	(Schaefer, Karl E. 1982)
1400-3000 ppm	Significant impairment of cognitive function including fatigue, headache and visual disturbance	2.5 – 8 hours	(Allen et al. 2016; Kajtar & Herczeg 2012; Satish et al. 2012)
1000 ppm	Harmful changes in respiration, circulation, and the cerebral cortex	Assumed to be 8 hours	(Goromosov 1968)
1000 ppm	Oxidative stress and damage to DNA in bacteria (implications for cancer diseases in humans)	3 hours	(Ezraty et al. 2011)
1000 ppm	Level associated with respiratory diseases, headache, fatigue, difficulty concentrating in classrooms	School or workday	(Carreiro-Martins et al. 2014; Ferreira & Cardoso 2014; Seppänen, Fisk & Mendell 1999)
1000 - 1500 ppm	Indoor air guidelines – not based on the protection of health, but used as a surrogate for indoor comfort and ventilation design	8 hours	ASHRAE and UK (EFA 2014)
950-1000 ppm	Low to moderate impairment of cognitive function	2.5 to 8 hours	(Allen et al. 2016; Satish et al. 2012)
800 ppm	Level associated with Sick Building Syndrome headaches, dizziness, fatigue, respiratory tract, eye, nasal and mucous membrane symptoms	8 hours	(Lu et al. 2015; Seppänen, Fisk & Mendell 1999; Tsai, Lin & Chan 2012)
400 ppm	Current average outdoor air concentration – no known effect	Lifetime	Carbon Dioxide Information Analysis Center 2015 <sup>5</sup>

**Notes:**

Blue text

Available guidelines

<sup>5</sup> <http://cdiac.ornl.gov/>

## 3.7 Relevance of Studies to Evaluating Driver Safety

### 3.7.1 General

Whether the effects reported at low levels of exposure to CO<sub>2</sub> have the potential to affect driver behaviour and driver safety requires further consideration, particularly when evaluating exposures that may occur over short periods of time, varying from 15 minutes to 1 hour and potentially up to 2 hours.

Clearly, exposures to very high levels of CO<sub>2</sub> are associated with significant acute health effects that will affect driver safety.

In terms of exposures to lower levels of CO<sub>2</sub>, issues of driver attention/distraction and fatigue/drowsiness are relevant and are a major contributor to vehicle crashes (Stutts et al. 2001).

### 3.7.2 Background on issues related to driver distraction

Driver distraction is defined as "when a driver is delayed in the recognition of information needed to safely accomplish the driving task because some event, activity, object or person within or outside the vehicle compelled or tended to induce the driver's shifting attention away from the driving task". There are wide range of distractions that can affect a driver that can be grouped as: visual, cognitive, auditory, and biomechanical/physical (Young, Regan & Hammer 2003).

Fatigue is quite different from distraction. The term *fatigue* refers to a combination of symptoms, such as impaired performance and subjective feelings of drowsiness. The term of fatigue still does not have a universally accepted definition. Different individuals show different symptoms and the degrees vary, so there is no concrete method to measure the level of fatigue (Sahayadhas, Sundaraj & Murugappan 2012; Stork 2014).

The driving task is considered to be complex, requiring the interaction and coordination of various cognitive, physical, sensory and psychomotor skills. It also requires a substantial degree of attention and concentration on the part of the driver. Despite these complexities, it is not unusual to see drivers engaged in various other activities while driving (Young, Regan & Hammer 2003), hence some level of distraction or inattention is part of normal driving behaviour, and drivers can self-regulate their driving to compensate for any decrease in attention (Young, Regan & Hammer 2003). In particular, it is noted that for experienced drivers the complexity and level of cognitive performance required to drive is low. However, for young, inexperienced drivers and the elderly, the cognitive requirements for driving are expected to be higher. Most research relates to the factors that affect attention, not on how well the driver manages those factors and compensates to enable safe driving. There are a range of compensation factors that drivers use to deal with inattention and/or fatigue (EC 2009). The levels at which drivers may effectively compensate for minor or very small changes in inattention or cognitive function, that may occur as a result of low levels of CO<sub>2</sub> exposures, is not well understood as most research relates to factors that significantly and immediately affect attention such as mobile phone use, conversations or changing radio stations (Tay & Knowles 2004).

In relation to fatigue it is difficult to determine the level of fatigue related accidents or the level of drowsiness that would affect driver safety. There are different kinds of fatigue from the view of individual organ functionality, such as local physical fatigue, general physical fatigue, central

nervous fatigue and mental fatigue. Central nervous fatigue and mental fatigue are the most dangerous types for driving (the driver is less conscious of these types of fatigue) and these will eventually cause sleepiness and increase the probability of accidents (Stork 2014). Drowsiness principally depends on the quality of the last sleep; the circadian rhythm (i.e. time of day) and the duration of the driving task, particularly if the driving task is monotonous (Sahayadhas, Sundaraj & Murugappan 2012). The effects of fatigue are also related to time, as it depends on the type and duration of the factor that is causing fatigue and the ability of the driver to compensate for these factors. A person has the ability to immediately react or compensate for increased fatigue when it is first noticed. However, if the trigger for fatigue is ongoing, the extra effort required to compensate for fatigue can result in impacts on performance (EC 2009), including driving. The influence of CO<sub>2</sub> on drowsiness, compared to these factors, is not clear. It is likely that effects associated with CO<sub>2</sub> exposures are of less significance/influence than the above key factors.

### **3.7.3 Studies related to distraction/fatigue**

The available studies associated with health effects of short-term exposures to CO<sub>2</sub> do not consider very short duration exposures of 1 hour or less. Hence application of these studies, in terms of the potential to affect driver safety, needs to be undertaken with some caution. It is noted that the occupational guideline of 5,000 ppm, is currently applied to pilots or aircraft, those operating submarines and spacecraft, which require higher levels of cognitive performance and decision making than driving. This guideline relates to longer-term average exposures, during a workday, flight or for longer periods of time in spacecraft. The guideline does not specifically address short-duration exposures, but has been included as a point of reference in this evaluation.

While the more recent studies have described disease symptoms at levels around 0.1% (1000 ppm) CO<sub>2</sub> and reduced cognitive ability in humans at around 0.08% (800 ppm) CO<sub>2</sub>, these effects are subtle. This means that while effects were identified, the effects were small and did not change outcomes of key indicators from competent to incompetent. In addition, it is noted that the cognitive tests performed related to a range of tasks relevant in an office type environment, such as proof reading and mathematical problem solving (Allen et al. 2016; Satish et al. 2012). The categories used to evaluate cognitive performance are not easily relatable to driving ability. Measures where there were no dose-related effects observed for CO<sub>2</sub> were focused activity (i.e. ability to pay attention or alertness) and information searching (capacity to gather information from various sources). The cognitive measures used in these studies did not specifically address reaction times which would have been relevant for driving. Therefore, the cognitive effects reported at 1,000 ppm and lower are not relevant to evaluating driver behaviour.

A further study (Zhang et al. 2017) over a 4.25 hour duration of exposure did not identify any changes to performance measures relevant to office activities, including alertness and response time, both of which are relevant to driving, at CO<sub>2</sub> exposures up to 3,000 ppm.

The available studies suggest that fatigue or drowsiness, that may be relevant to driving behaviour, are unlikely occur below 3,000 ppm, for exposure periods of 2.5 hours to 8 hours. For the purpose of this assessment, a threshold of 3,000 ppm derived from studies over 2.5 to 8 hour durations has been considered to be a threshold for effects associated with potential driver fatigue. This is lower than the occupational value of 5,000 ppm over an 8 hour averaging period, which is applied to higher risk long-haul drivers, pilots and spacecraft operators.



The relevance of this threshold for evaluating shorter duration exposures, such as 15 minutes, 30 minutes and an hour, that may occur inside tunnels, is not clear. How quickly the body responds to and starts compensating for increased levels of CO<sub>2</sub> is not well understood, particularly for lower level exposures to CO<sub>2</sub>. Studies on shorter duration exposures to CO<sub>2</sub> found that for 1 hour exposures to 4% (40,000 ppm) CO<sub>2</sub> (Vercruyssen, Kamon & Hancock 2007) and 10 minute exposures to up to 5% (50,000 ppm) CO<sub>2</sub> (Sheehy, Kamon & Kiser 1982), there was no significant cognitive or motor performance impairment, however there were measurable physiological changes such as increased ventilation and reports of headaches. A small study associated with 30 minute exposures to CO<sub>2</sub>, with exercise found no effects on basic cognitive functions, but effects on short-term memory, long-term memory, working memory, performing mathematical tasks at 1.5% (15,000 ppm) CO<sub>2</sub> (Selkirk, Briggs & Shykoff 2010). These studies only involved small numbers of fit and healthy individuals which may limit the relevance for the wider population. These studies suggest that for shorter duration exposures, cognitive effects may not be of significance, rather physiological effects are of more importance. When considering effects that may influence driving safety over very short periods of exposure these studies suggest that cognitive effects are not likely to be of significance.

In relation to acute effects other than cognitive effects, the available studies indicate these effects occur at increasing concentrations as exposures get shorter, as summarised in **Table 5**.

**Table 5**                      **Physiological tolerance time for CO<sub>2</sub> exposures and acute health effects (Law, Watkins & Alexander 2010; US EPA 2000)**

Physiological Tolerance			Acute Health Effects	
CO <sub>2</sub> (%)	CO <sub>2</sub> (ppm)	Maximum Exposure Limit (minutes)	Duration of Exposure	Effects
0.5	5000	Indefinite		
1.0	10000	Indefinite		
1.5	15000	480		
2.0	20000	60	Several hours	Headache, dyspnoea upon mild exertion
3.0	30000	20	1 hour	Headache, sweating, dyspnoea at rest
4.0	40000	10	(4-5%)	Headache, dizziness, increased blood pressure, uncomfortable dyspnoea
5.0	50000	7	Within a few minutes	
6.0	60000	5	1-2 minutes ≤16 minutes Several hours	Hearing, visual disturbances Headache, dyspnoea Tremors
7.0	70000	<3	(7-10%)	Unconsciousness, near-unconsciousness Headache, increased heart rate, shortness of breath, dizziness, sweating, rapid breathing  <i>Lowest published lethal concentration</i>
9	90000	N/A	Few minutes 1.5 minutes to 2 hours  <i>9% for 5 minutes</i>	
10	100000	N/A	(>10-15%)	
15	150000	N/A	1 minute to several minutes	Dizziness, drowsiness, severe muscle twitching, unconsciousness
17	170000	N/A	(17-30%) Within 1 minute	Loss of control and purposeful activity, unconsciousness, convulsions, coma, death

### 3.7.4 Identification of Criteria

Adjusting CO<sub>2</sub> criteria to address shorter periods of exposure is complex as it needs to take into account the nature of the effects observed (discussed above), the physiological responses associated with such exposures and consideration of exposures that may occur to the general public, that include elderly drivers or drivers with pre-existing medical conditions. Applying a linear exposure duration time adjustment to the longer-term criteria (outlined below) results in criteria that are similar to those in **Table 5**, for the sub 1 hour time periods. The values presented in **Table 5**, however may not adequately address exposures and effects on all members of the population.

For this assessment, the following criteria have been considered for CO<sub>2</sub> in-cabin:

- **Exposures of longer than 1 hour – CO<sub>2</sub> average of 3,000 ppm** based on the level of CO<sub>2</sub> exposure that is unlikely to affect driver behaviour. This is based on studies for longer durations of exposure, namely 2.5 to 8 hours. No studies are available for exposures of longer than 1 hour but less than 2.5 hours that address cognitive performance. Hence this value has been adopted for exposures over an hour.
- **Exposures of over 30 minutes and up to 1 hour – CO<sub>2</sub> average of 7,500 ppm**, based on extrapolation of the physiological effects criteria adopted for exposures less than or equal to 30 minutes (below) as well as extrapolation<sup>6</sup> of the criteria based on driver behaviour listed above. This criterion is considered to be conservative as the studies indicate that effects associated with very short duration exposures occur at level of 1.5% (15,000 ppm) to 2% (20,000 ppm) and higher.
- **Exposures of less than or equal to 30 minutes – CO<sub>2</sub> average of 15,000 ppm**, based on the highest level listed in **Table 5** with no physiological effects, consistent with the data from the limited 30 minute exposure study (Selkirk, Briggs & Shykoff 2010).

---

<sup>6</sup> Extrapolation of this criteria is based on 3000 ppm for 2.5 hours to 7,500 ppm for 1 hour.

## Section 4. Management Measures for CO<sub>2</sub> Levels in Vehicles

---

There are no jurisdictions in Australia or internationally that have established management levels for CO<sub>2</sub> concentrations in passenger vehicles.

In relation to ventilation requirements in vehicles in Australia, the Vehicle Standard (Australian Design Rule 42/01 – General Safety requirements) 2006 states the following:

42.17. Windows and Ventilation

42.17.1 General Requirements

At least half the number of windows must be capable of being opened or the vehicle must be provided with an alternative method of ventilation.

42.17.2 Ventilation

Omnibuses and N-group vehicles must be provided with a means of ventilation other than by means of windows and door openings.

42.17.2.1 Except in the case of omnibuses equipped with flow-through ventilation or refrigerated air-conditioning, the provision of an inlet air vent and at least 2 rotary vents or a hatch in the roof towards the rear of the passenger compartment as a means of ventilation is deemed to meet the provisions of this clause.

Section 42.17.2.3 relates to Power Operated Windows, and is not specifically relevant to the operation of any ventilation system.

There are no other rules for the ventilation of passenger vehicles in Australia.

Most vehicle manufacturers include a standard ventilation system, with flow through fresh air or recirculation, changed manually by the driver. Some individual manufacturers have included ventilation systems that include other features to better manage air quality inside the cabin:

**Lexus**<sup>7</sup>: The air conditioning system is designed to allow up to approximately 10% of the air coming from the air vents to be fresh air from the outside. The exact amount of fresh air entering the vehicle on recirculation mode will vary. If the A/C system is turned on in the AUTO mode, then the A/C ECU switches the air intake ducting between "fresh," "fresh and recirculation mix" or "recirculation" according to the required outlet air temperature that the ECU calculates. This calculation is based on the temperature that is dialled in with the control knob, the interior air temperature and the operating conditions.

---

<sup>7</sup> [http://lexus2.custhelp.com/app/answers/detail/a\\_id/8309/~when-the-system-is-set-on-recirculation-mode,-does-fresh-air-still-enter-the](http://lexus2.custhelp.com/app/answers/detail/a_id/8309/~when-the-system-is-set-on-recirculation-mode,-does-fresh-air-still-enter-the)

**BMW**<sup>8</sup>: An Automatic Air Recirculation (AAR) system is installed. This system is designed to minimise pollutants outside the vehicle from entering the vehicle. Information available indicates that the system recognises gases in the air from outside the cabin - carbon monoxide, nitrogen oxide and other pollutants in the surrounding atmosphere. If their concentrations are too high, the system automatically switches to recirculation for a time. The air in the cabin is processed and filtered by the climate control system. Air from outside is only reintroduced when the system detects that the level of pollutants has fallen. There is no information available in relation to the sensors used or the thresholds at which the ventilation is automatically changed to recirculation or fresh-air.

**Hyundai**: The manufacturer has indicated<sup>9</sup> that it offers a CO<sub>2</sub> sensor-controlled ventilation system in the 2015 Genesis model. The system has a CO<sub>2</sub> sensor that switches the ventilation to bring in more fresh-air when CO<sub>2</sub> levels reach 2500 ppm. It is not clear if this feature is offered on vehicles in Australia.

**VW**: The manufacturer indicates<sup>10</sup> they have an AAR system which includes an air quality sensor as part of the "Climatronic" automatic air conditioning system. It measures pollutants, in the form of oxidisable or reducible gases, in the air outside your car. Oxidisable gases include carbon monoxide (CO), hydrocarbons (vapours from benzene or petrol) and other incompletely burnt fuel components. Nitrogen oxides (NO<sub>x</sub>) are reducible gases. If the quality of the outside air drops – in a traffic jam or when driving through a tunnel, for example – the control system activates the Climatronic's air recirculation mode, stopping polluted air from coming in and reducing the quality of air inside your car. No more details are available on this system, and it is not clear if this system is present in Australian VW vehicles.

There are a few studies where different techniques have been evaluated to determine ways in which CO<sub>2</sub> levels may be managed or minimised within vehicles.

Matton (2015) evaluated a number of strategies that may be considered to manage or control CO<sub>2</sub> levels in-cabin. This was undertaken using modelling. The evaluation considered potential compliance with the ASHRAE Standard 62 (which is an indoor air quality standard in the US) of 1100 ppm for CO<sub>2</sub>. The assessment considered the following strategies:

- The use of a CO<sub>2</sub> sensor to control whether recirculation is on or off. This strategy was shown to effectively manage CO<sub>2</sub> levels however temperature and humidity issues were identified. In addition, the cycle time to maintain lower levels of CO<sub>2</sub>, ranged from 1 minute (slow speed and full cabin) to 10 minutes (high speed with only the driver). This cycle time would mean that in a tunnel, air from outside of the cabin would be brought into the cabin on a number of occasions (depending on speed, occupants and length of tunnel) while the

<sup>8</sup> [http://www.bmw.com/com/en/insights/technology/technology\\_guide/articles/automatic\\_air\\_recirculation.html?source=index&article=automatic\\_air\\_recirculation](http://www.bmw.com/com/en/insights/technology/technology_guide/articles/automatic_air_recirculation.html?source=index&article=automatic_air_recirculation)

<sup>9</sup> <http://www.co2meter.com/blogs/news/23987521-high-co2-levels-in-your-car>

<sup>10</sup> [http://en.volkswagen.com/en/innovation-and-technology/technical-glossary/umlufschaltung\\_automatisch.html](http://en.volkswagen.com/en/innovation-and-technology/technical-glossary/umlufschaltung_automatisch.html)

vehicle remains in the tunnel. In addition, the durability of the recirculation flap in the ventilation system to enable this type of control to be effective needed to be considered.

- Timed recirculation control, where fresh air is introduced into the ventilation system (when on recirculate) after a set period of time. This would not require the use of a CO<sub>2</sub> sensor, however the various time periods evaluated (ranging from 15 minutes recirculation and 1 minute of 50% fresh air to 3.5 minutes recirculation and 1 minute 100% fresh air) did not manage CO<sub>2</sub> levels below the ASHRAE standard.
- Fractional recirculation control where there is some percentage of fresh air in the ventilation system, when on recirculation. The modelling showed that a small amount of fresh air, between 5% and 10%, introduced into the ventilation system when on recirculation was sufficient to maintain CO<sub>2</sub> levels below the ASHRAE standard. This concept has also been evaluated by Grady et al (Grady 2013; Grady et al. 2013), where CO<sub>2</sub> levels in-cabin were shown to be maintained to less than 2000 ppm on recirculation where the air includes around 15% fresh air. Such a technique may not be desirable within tunnels where the use of recirculation is required to minimise exposure to NO<sub>2</sub> in the air within the tunnel.

## Section 5. Assessment of Short-Duration CO<sub>2</sub> Exposures

### 5.1 Approach

The information and data obtained in the 2016 RMS Study has been utilised, in conjunction with the modelling of CO<sub>2</sub> levels in-cabin, described in **Section 2.1**, to provide estimates of CO<sub>2</sub> levels in the range of different vehicles. The levels of CO<sub>2</sub> inside these vehicles have then been compared with the health based criteria discussed in **Section 3.7**.

The review has considered the use of ventilation on recirculation for the following time periods:

- 15 minutes
- 30 minutes
- 60 minutes
- 2 hours – this has been included to evaluate CO<sub>2</sub> levels inside vehicles if recirculating is left on for a longer period of time. The 2 hour time period was chosen as it represents current advice to drivers to take driving breaks every 2 hours.

Following these time periods, it is then assumed that the ventilation system is changed to fresh air for a period of time. The model has then been used to evaluate the change (lowering) of CO<sub>2</sub> levels in-cabin.

These scenarios were evaluated for vehicles with 1 to 5 occupants, where the average CO<sub>2</sub> in exhaled breath for the occupants has been estimated to be 40,000 ppm.

The 2016 RMS Study involved the use of 9 different vehicles, considered representative of the Sydney vehicle fleet (refer to **Section 2.1**). For 8 of the vehicles the study obtained measured, and modelled (using the model described by Hudda et al (2012), values for the AER when operating with ventilation on recirculation (RC on) as well as fresh air (RC off). The CO<sub>2</sub> data was lost for 1 of the vehicles (the 2004 Ford Fiesta) which meant that no measured AERs could be obtained.

**Table 6** presents a summary of the vehicles evaluated, the interior volume and the measured and modelled AERs.

**Table 6 Measured and Modelled AERs from 2016 RMS Study**

Vehicle	Cabin volume (m <sup>3</sup> )	Ventilation mode	Vehicle speed (km/hr)	Measured AER (per hour)	Modelled AER (per hour)
2008 Holden Astra	3.40	RC on	60	7	57
			100	10	66
		RC off	60	62	21
			100	105	25
2014 BMW X3	3.64	RC on	60	8	42
			100	11	67
		RC off	60	16	17
			100	50	21
2014 Hyundai i30	2.81	RC on	60	4	8
			100	14	13
		RC off	60	57	33
			100	63	39



Vehicle	Cabin volume (m <sup>3</sup> )	Ventilation mode	Vehicle speed (km/hr)	Measured AER (per hour)	Modelled AER (per hour)
2007 Fiat Punto	2.52	RC on	60	6	5
			100	7	6
		RC off	60	39	41
			100	55	49
2007 Toyota Corolla	2.81	RC on	60	4	12
			100	5	12
		RC off	60	51	33
			100	45	39
2007 Subaru Outback	3.71	RC on	60	4	99
			100	4	104
		RC off	60	36	16
			100	58	20
2002 Audi A3*	2.74	RC on	60	14	11
			100	13	6
		RC off	60	10	35
			100	19	42
2011 Holden Cruze	3.03	RC on	60	3	6
			100	8	8
		RC off	60	11	60
			100	26	73

Shaded cells are the AER values considered in this review

\* Data collected from the Audi A3 is noted to be problematic as the AER is higher with ventilation on recirculation compared with recirculation off (i.e. fresh air in). The vehicle is noted to be old with very poor door seals, which may be the reason why the measured AER values are unusual. There is no indication that there were any equipment problems during the measurements, hence this data has not been excluded. However the data is only considered relevant to the vehicle tested and not representative of other vehicles that are of similar age or style.

For a number of the vehicles there was little correlation between the modelled and measured AERs. This may be a limitation of the 2016 RMS Study, in that only a small number of experiments were undertaken on each vehicle to calculate the AER, as well as limitations with the regression models used to estimate the AERs. For the purpose of this review the measured AERs, for vehicles travelling at 60 km/hr have been utilised. The speed adopted is considered more representative of average speeds likely to be travelled on the road network in urban areas, including inside tunnels.

For each of the scenarios evaluated in this review, the following criteria have been considered for CO<sub>2</sub> exposures:

- Exposures to up to and equal to 30 minutes: 15,000 ppm (as an average over exposure period)
- Exposures over 30 minutes, up to and equal to 1 hour: 7500 ppm (as an average over exposure period)
- Exposures over 1 hour: 3000 ppm (as an average over exposure period)

## 5.2 Evaluation of CO<sub>2</sub> Exposures

Figures 9 to 16 present graphs that show the changes in CO<sub>2</sub> concentrations inside the 8 vehicles evaluated, for the different periods of time where ventilation is on recirculation, then switched back to fresh air, for 1 to 5 occupants. The Figures also present the average concentration of CO<sub>2</sub> for the duration of the travel time, assumed within a tunnel, and comparison against the adopted criteria.

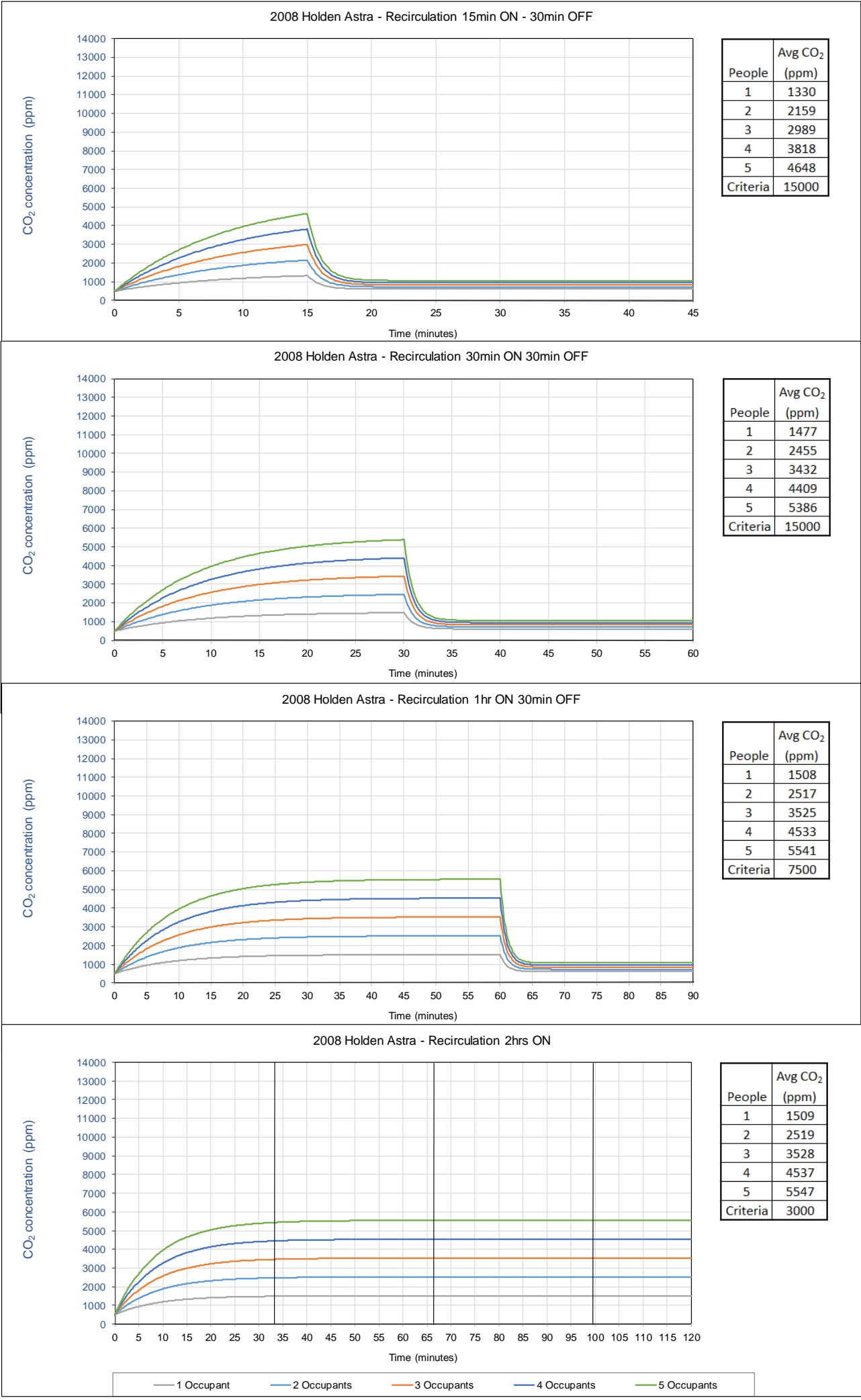
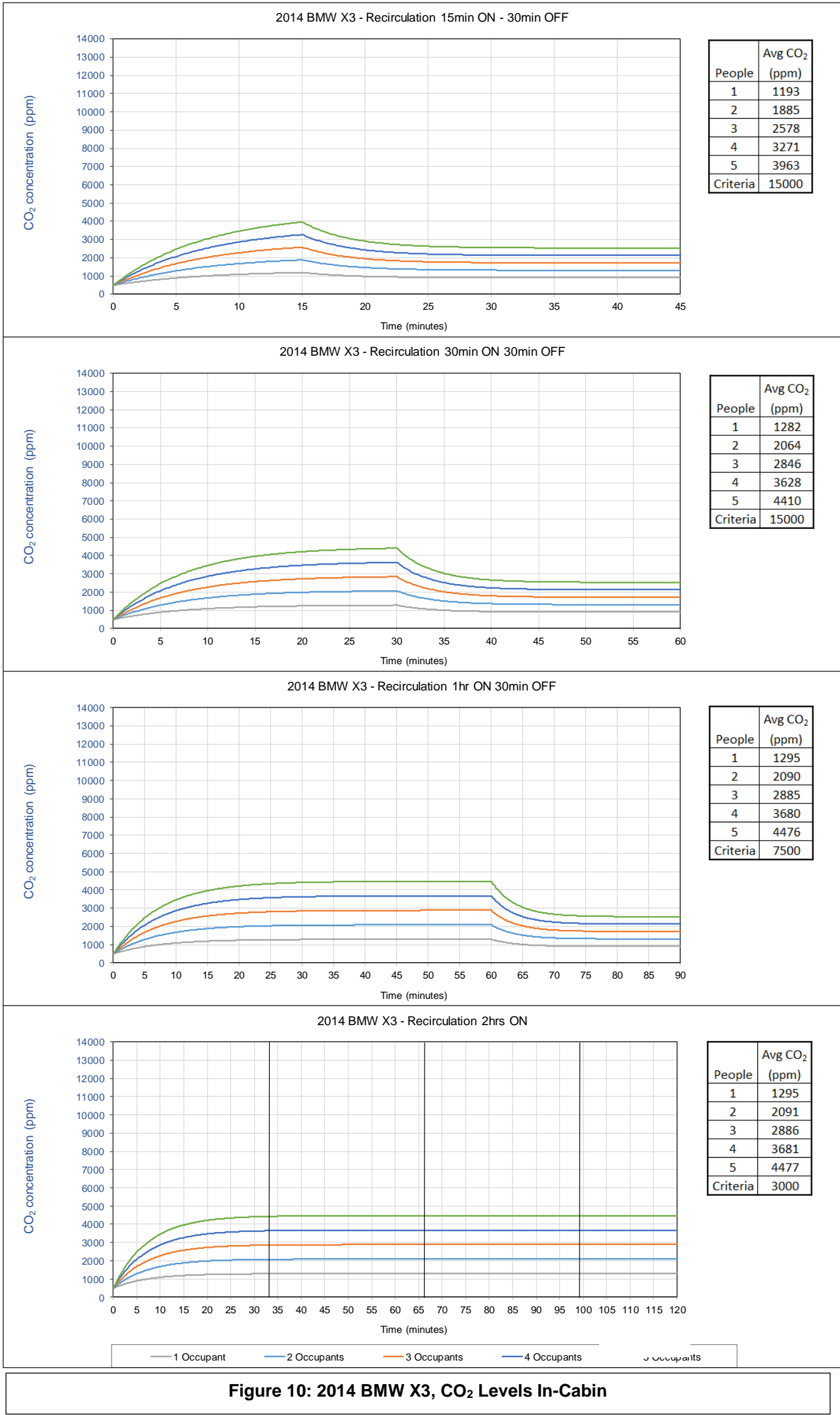


Figure 9: 2008 Holden Astra, CO<sub>2</sub> Levels In-Cabin



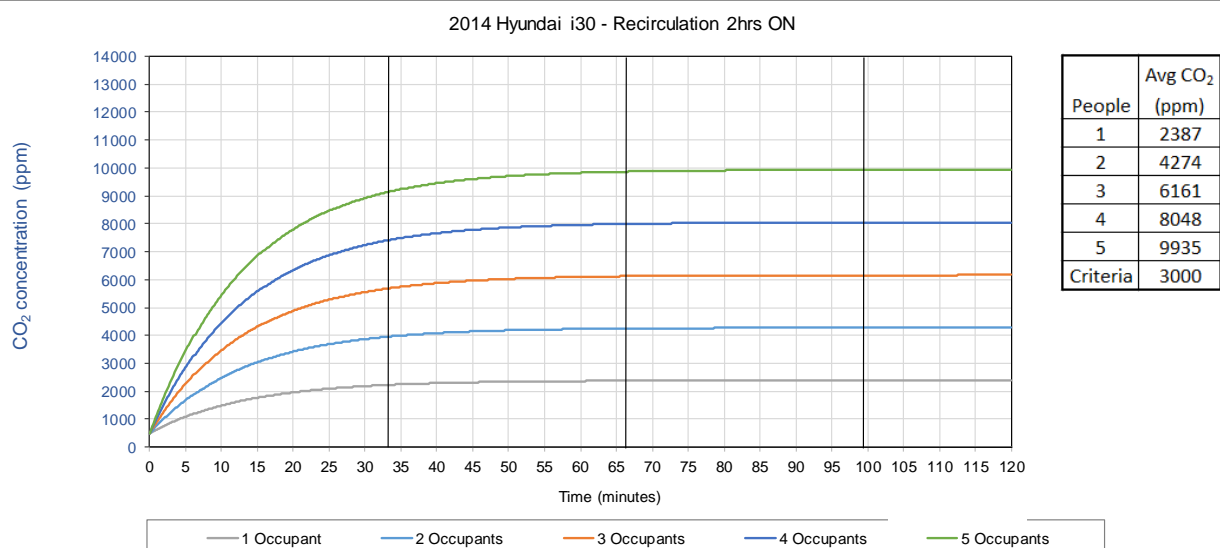
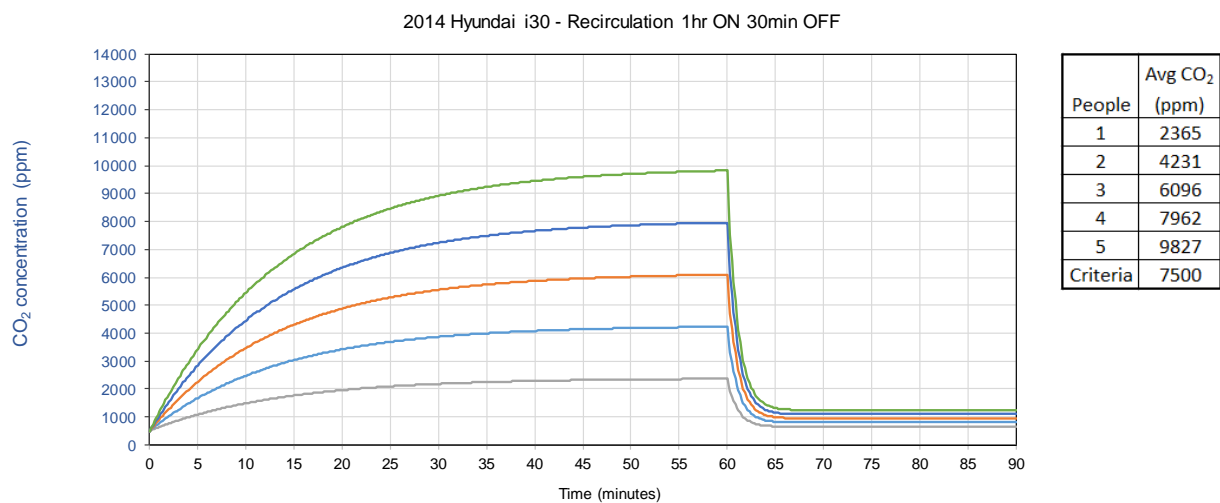
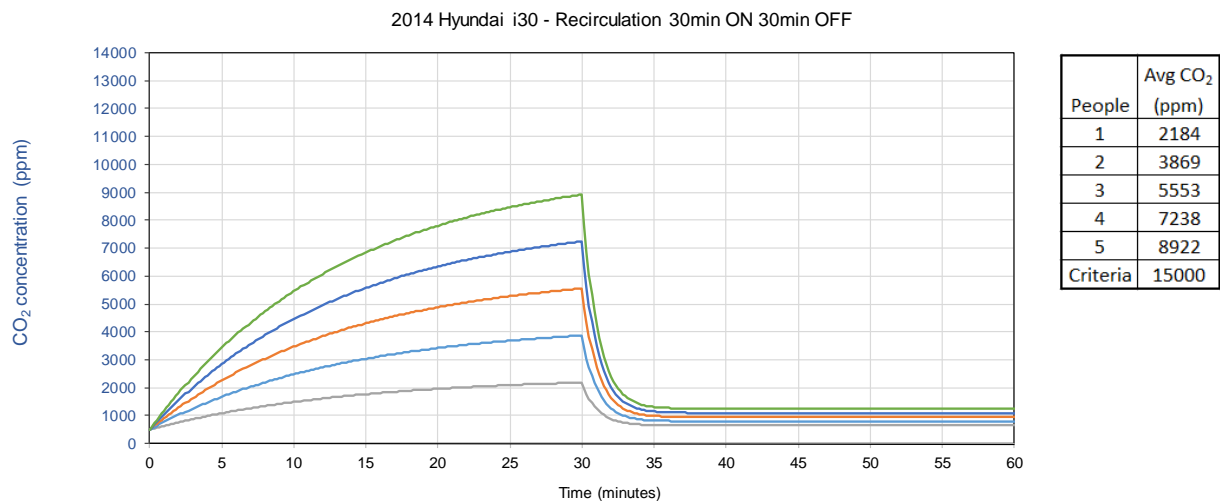
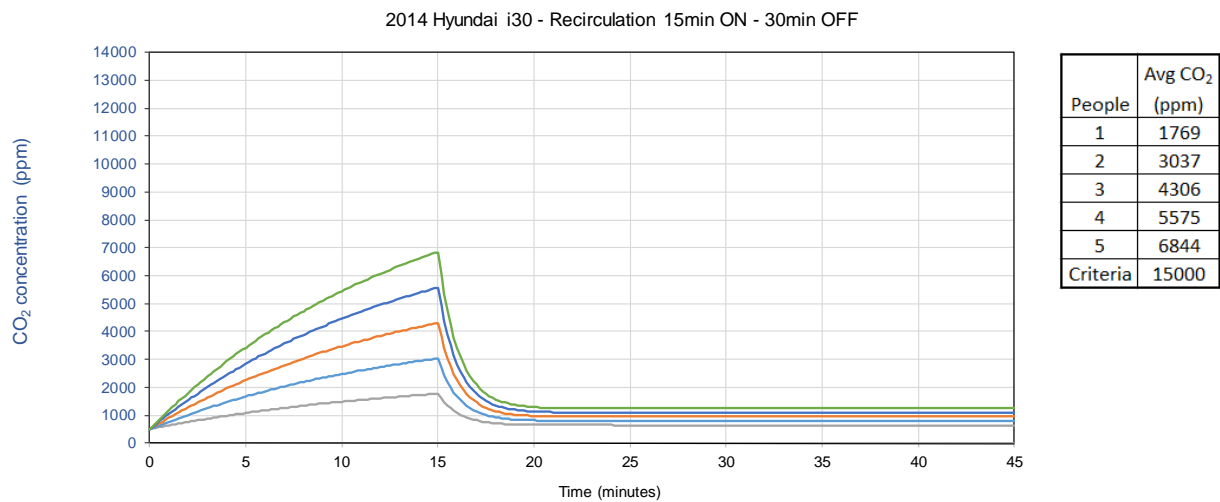


Figure 11: 2014 Hyundai i30, CO<sub>2</sub> Levels In-Cabin

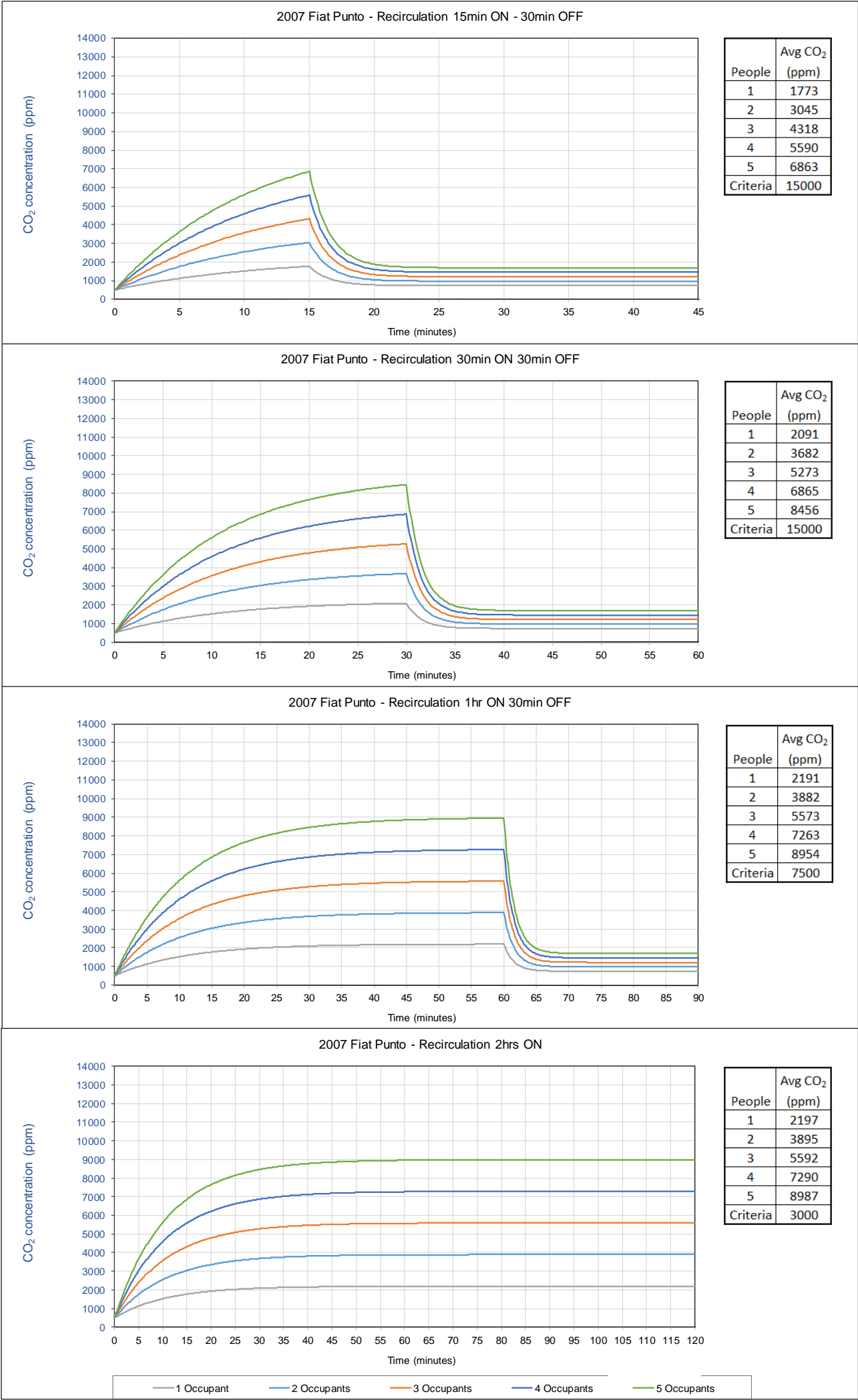


Figure 12: 2007 Fiat Punto, CO<sub>2</sub> Levels In-Cabin

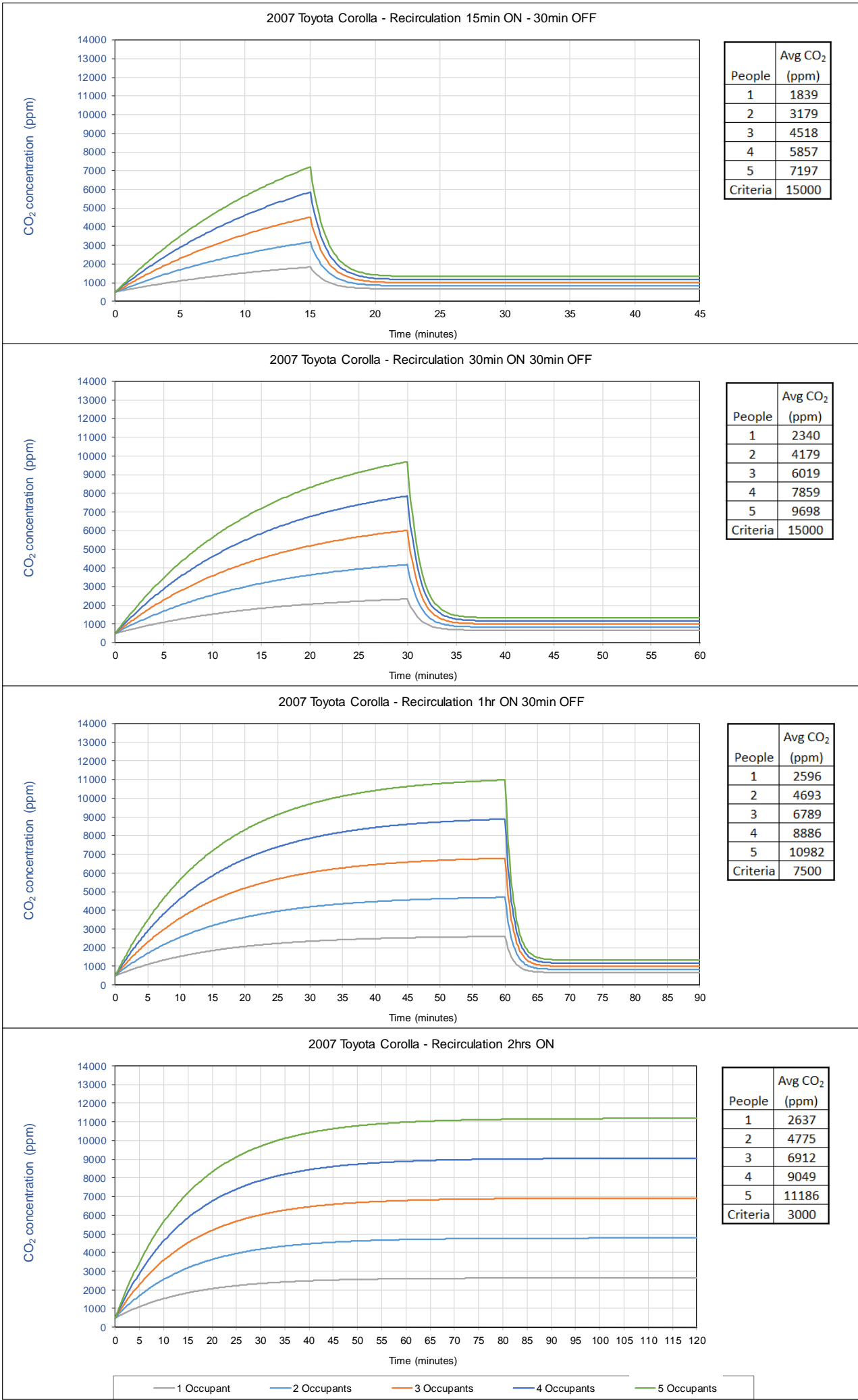


Figure 13: 2007 Toyota Corolla, CO<sub>2</sub> Levels In-Cabin



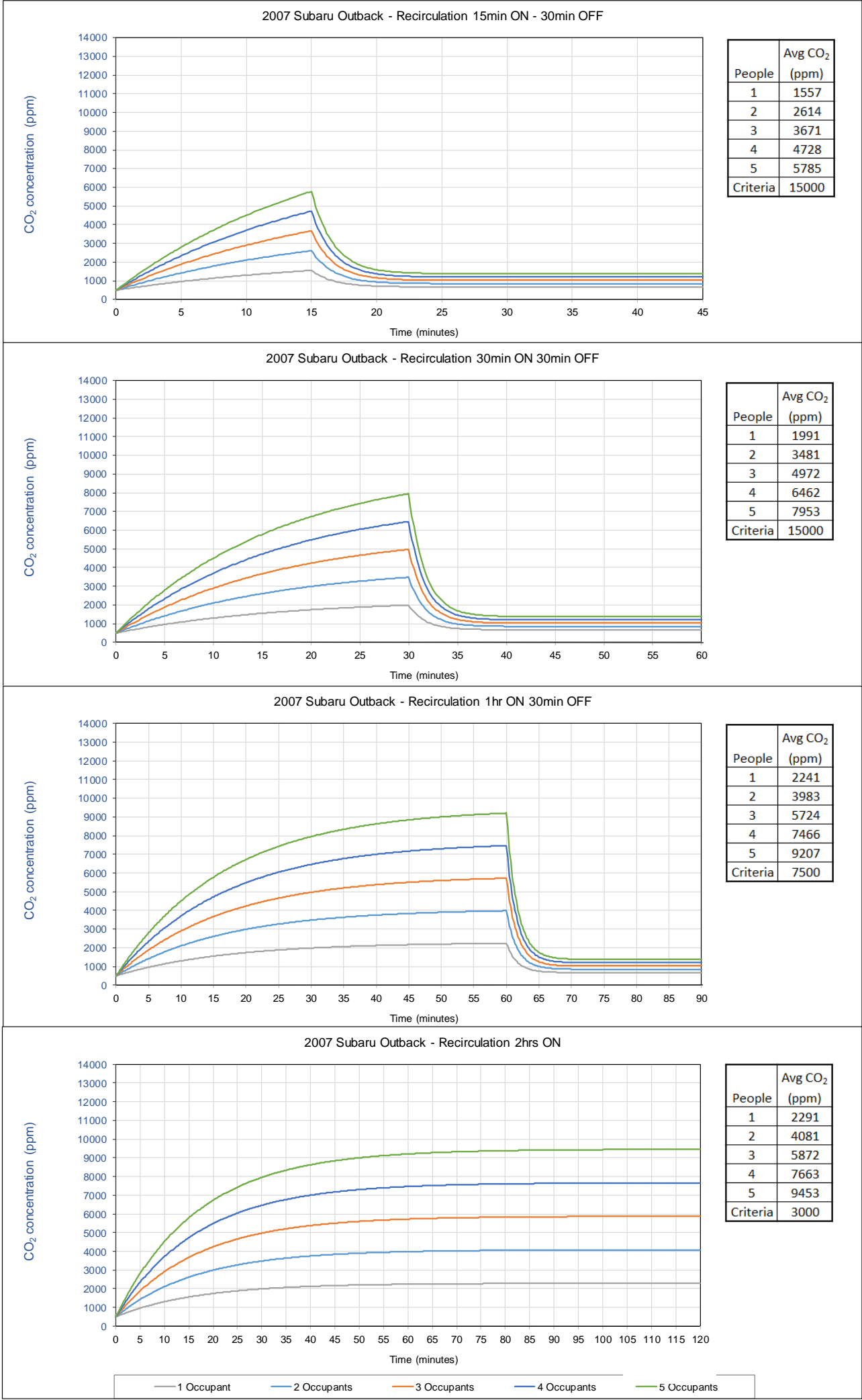


Figure 14: 2007 Subaru Outback, CO<sub>2</sub> Levels In-Cabin

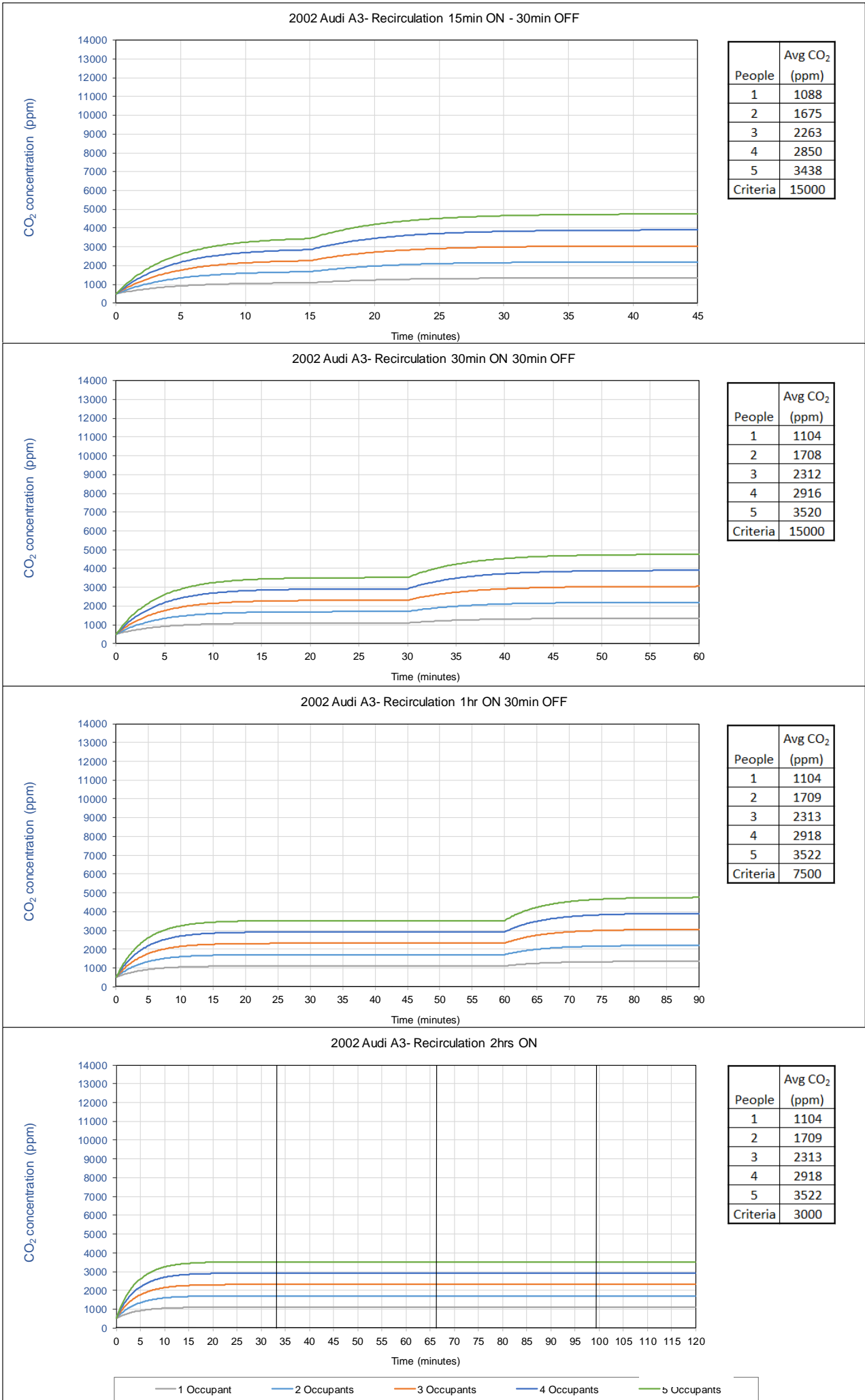
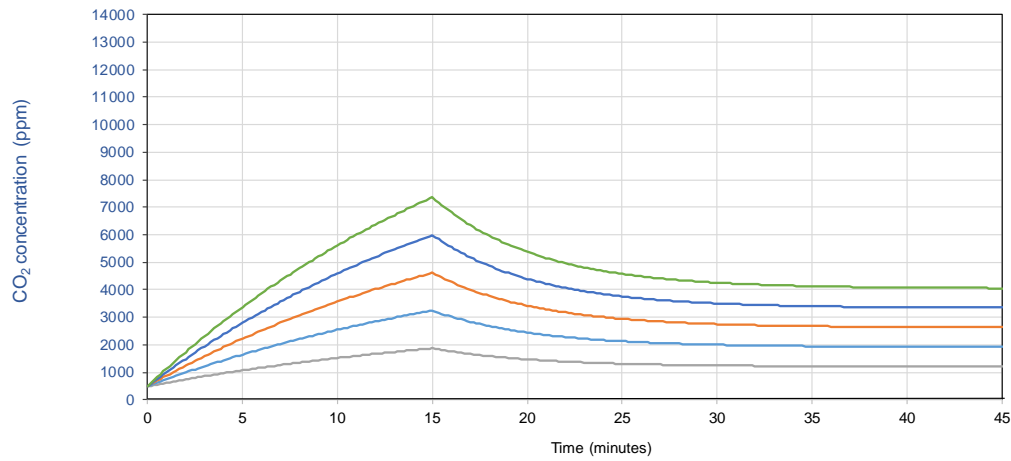


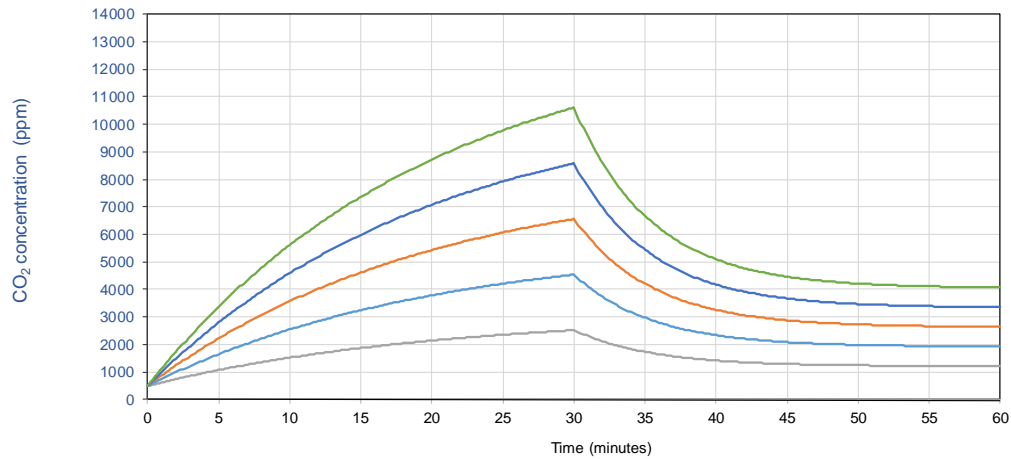
Figure 15: 2002 Audi A3, CO<sub>2</sub> Levels In-Cabin

2011 Holden Cruze - Recirculation 15min ON - 30min OFF



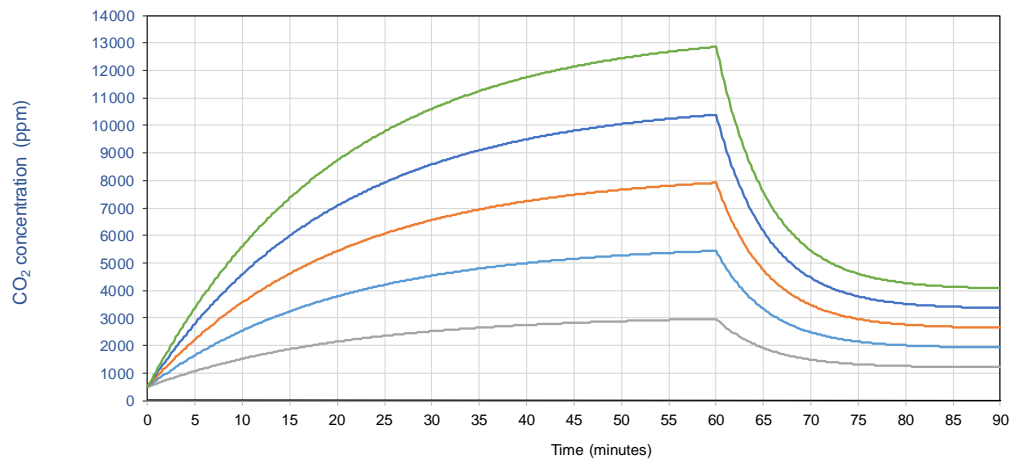
People	Avg CO <sub>2</sub> (ppm)
1	1873
2	3245
3	4618
4	5991
5	7364
Criteria	15000

2011 Holden Cruze - Recirculation 30min ON 30min OFF



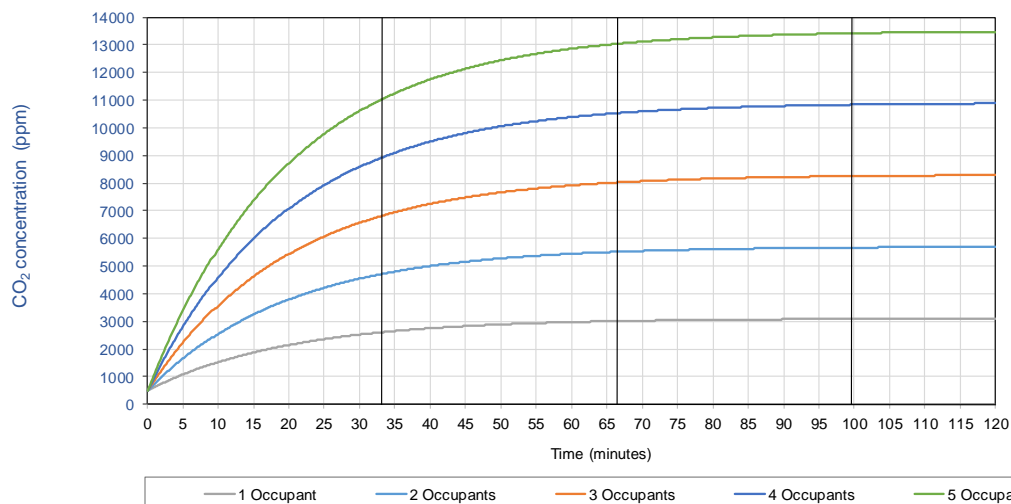
People	Avg CO <sub>2</sub> (ppm)
1	2521
2	4542
3	6563
4	8584
5	10606
Criteria	15000

2011 Holden Cruze - Recirculation 1hr ON 30min OFF



People	Avg CO <sub>2</sub> (ppm)
1	2972
2	5444
3	7916
4	10388
5	12860
Criteria	7500

2011 Holden Cruze - Recirculation 2hrs ON



People	Avg CO <sub>2</sub> (ppm)
1	3095
2	5690
3	8285
4	10880
5	13476
Criteria	3000

Figure 16: 2011 Holden Cruze, Changes in CO<sub>2</sub> Levels In-Cabin

**Table 7 Summary of In-Vehicle CO<sub>2</sub> Concentrations**

	CO <sub>2</sub> concentrations in-vehicle for 1 to 5 occupants (ppm)																			
	15 minute average concentration (criteria = 15,000 ppm)					30 minute average concentration (criteria = 15,000 ppm)					60 minute average concentration (criteria = 7,500 ppm)					120 minute average concentration (criteria = 3000 ppm)				
	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
2008 Holden Astra	1330	2159	2989	3818	4648	1477	2455	3432	4409	5386	1508	2517	3525	4533	5541	1509	2519	3528	4537	5547
2014 BMW X3	1193	1885	2578	3271	3963	1282	2064	2846	3628	4410	1295	2090	2885	3680	4476	1295	2091	2886	3681	4477
2014 Hyundai i30	1769	3037	4306	5575	6844	2184	3869	5553	7238	8922	2365	4231	6096	7962	9827	2387	4274	6167	8048	9935
2007 Fiat Punto	1773	3045	4318	5590	6863	2091	3682	5273	6865	8456	2191	3882	5575	7263	8954	2197	3895	5592	7290	8987
2007 Toyota Corolla	1839	3179	4518	5857	7197	2340	4179	6019	7859	9698	2396	4693	6789	8886	10982	2637	4775	6912	9049	11186
2007 Subaru Outback	1557	2614	3671	4728	5785	1991	3481	4972	6462	7953	2241	3983	5724	7466	9207	2291	4081	5872	7663	9453
2002 Audi A3*	1088	1675	2263	2850	3438	1104	1708	2312	2916	3520	1104	1709	2313	2918	3522	1104	1709	2313	2918	3522
2011 Holden Cruz	1873	3245	4618	5991	7364	2521	4542	6563	8584	10606	2972	5444	7916	10388	12860	3095	5690	8285	10880	13476

\* ACH measured for the Audi A3 noted to be problematic (i.e. do not follow the expected trends in ACH inside vehicles on different ventilation conditions). Hence this data, and the modelled CO<sub>2</sub> levels only relates to the vehicle tested. It should not be considered representative of vehicles in the Australian vehicle fleet of a similar age and build.

**2000** Shaded cells are concentrations in excess of the adopted guideline

**Table 7** presents a summary of the average in-cabin CO<sub>2</sub> concentrations predicted for the vehicles and scenarios evaluated, with comparison against the adopted criteria.

Review of **Figures 9 to 16** and **Table 7** indicates the following in relation to potential CO<sub>2</sub> exposures in-cabin that relate to travel periods that may occur during journeys within tunnels (up to an hour):

- For most vehicles, when the ventilation is changed to fresh air, from recirculation, this results in a drop in CO<sub>2</sub> levels over a relatively short period of time. The exception to this observation is for the 2002 Audi A3, where the data is not considered representative of similar vehicles.
- Most vehicles used in Sydney are occupied by 1 person (with the average ranging from 1.08 during commuting to 1.47 for social/recreational purposes and 2.18 when a vehicle is used to serve passengers (Corpuz 2006)). Where the CO<sub>2</sub> levels for all vehicles with 1 occupant are considered, all CO<sub>2</sub> levels predicted, regardless of the duration of the time ventilation is on recirculation, are less than or equal to the adopted fatigue criteria. All predicted concentrations inside vehicles with 1 occupant are also below the occupational guideline of 5,000 ppm. On this basis, the levels of CO<sub>2</sub> in-cabin through the use of recirculation is not expected to affect fatigue levels of drivers where there are no passengers.
- In relation to trips where recirculation is used for a 15 minute period, all predicted concentrations of CO<sub>2</sub> in-cabin for 1 to 5 occupants are below the relevant criteria. Hence the use of recirculation for a short period of time of 15 minutes is not expected to affect the health (including fatigue) of drivers and occupants.
- In relation to trips where recirculation is used for a 30 minute period, all predicted concentrations of CO<sub>2</sub> in-cabin for 1 to 5 occupants are below the relevant criteria. Hence the use of recirculation for a short period of time of 30 minutes is not expected to affect the health (including fatigue) of drivers and occupants.
- In relation to trips where recirculation is used for a 1 hour period, the level of CO<sub>2</sub> in-cabin has reached or close to the maximum level likely to be achieved (based on the number of passengers and AER). There are some vehicles where the 1 hour average CO<sub>2</sub> levels are below the adopted criteria for 1 to 5 occupants (2008 Holden Astra, 2014 BMW X3 and 2002 Audi A3). For all other vehicles, the 1 hour average level of CO<sub>2</sub> in-cabin exceeds the adopted criteria where the number of occupants increases to 3 or more. It is noted that the criteria adopted for evaluating potential exposures to CO<sub>2</sub> for periods of over 30 minutes and up to an hour are conservative as the available data suggests that effects of concern may only occur at exposure to levels higher than 1.5% (15,000 ppm) or 2% (20,000 ppm). Hence the potential for adverse effects to occur that may affect driver behaviour is considered to be low.

In relation to exposures that may occur for 2 hours or longer if recirculation has been left on, the following is noted:

- Where recirculation was left on for periods of 2 hours (or longer) the level of CO<sub>2</sub> in-cabin has reached the maximum level likely for all vehicles (based on the number of passengers and AER).



- For these situations, the presence of passengers (i.e. a total of 2 or more occupants) has the potential to result in CO<sub>2</sub> levels that exceed the adopted fatigue criteria and the long-term occupational guideline.
- The number of passengers that result in exceedance of the criteria varies depending on the individual vehicle, with the most significant exposures predicted within the 2011 Holden Cruz. While it is not possible to determine if the CO<sub>2</sub> levels that exceed these criteria will actually affect driver safety, there may be situations where an already fatigued driver (from a poor night's sleep or driving late at night) may be affected by the levels of CO<sub>2</sub> in-cabin where there are a number of passengers also present.

Overall, for the vehicles evaluated, where ventilation is placed on recirculation the levels of CO<sub>2</sub> that may be present are not expected to adversely affect driver safety. There may, however, be some situations for some vehicles (namely where recirculation is left on and the driver spends 1 or more hours in the vehicle with a number of passengers) where the levels of CO<sub>2</sub> may affect an already fatigued driver.



## Section 6. Risk Management

---

Advice is provided for motorists entering tunnels in the Sydney network to wind the windows up and place the ventilation on recirculation. This advice is provided to minimise exposures to NO<sub>2</sub>, and other pollutants such as fine particulates, while inside the tunnel environment.

There are currently no Australian Vehicle Design requirements that relate to the use of ventilation systems, particularly when on recirculate, that would address exposures to CO<sub>2</sub> in-cabin. There are no other jurisdictions that provide regulation in relation to managing CO<sub>2</sub> levels inside vehicle.

As discussed in **Section 4** some vehicle manufacturers have developed ventilation systems that automatically manage recirculated and fresh air to address outside pollutants, or even CO<sub>2</sub> in-cabin. However, there are very few of these, and for many of the technologies described by manufacturers it is not clear if these relate to vehicles on sale in Australia.

Review of advice provided by driving organisations indicates that they often recommend the use of air conditioning on recirculation to save of running costs (fuel costs) when cooling in hot conditions or heating in cold conditions. Very little advice is provided about how long a system should be run on recirculation before allowing fresh air into the cabin. Some note that the use of recirculation may result in fogging up inside the vehicle, which is typically the trigger to bring in fresh air.

It is unlikely that changes to vehicle design standards or rules on ventilation would occur in Australia.

Given the potential for CO<sub>2</sub> levels inside some vehicles to increase significantly when on recirculation, with levels potentially exceeding criteria that may result in some level of fatigue, particularly for longer duration trips with passengers, it would be appropriate that advice is provided that leaving ventilation on recirculation for extended periods of time should not be undertaken.

## Section 7. Conclusions

---

To manage exposures to exterior pollutants within tunnels, advice is being provided to drivers to wind up windows and change ventilation to recirculation. This assessment has evaluated the available literature and data obtained from the 2016 RMS Study in relation to the accumulation of CO<sub>2</sub> inside vehicles over different periods of time where ventilation is on recirculation.

Health effects associated with short-term exposures to high concentrations of CO<sub>2</sub> are well understood, and form the basis for a number of occupational guidelines. However, recent studies indicate that subtle adverse effects of even short-term low level CO<sub>2</sub> exposure can be measured. Subtle cognitive effects identified at exposures levels of 1,000 ppm and lower are not considered to be relevant to driver behaviour. Other adverse effects including fatigue, headaches and visual disturbances effects have been reported at CO<sub>2</sub> levels above 0.3% (3,000 ppm). For very short-duration exposures to CO<sub>2</sub>, acute physiological effects are of more importance.

The available literature from Australia and International sources all confirm that CO<sub>2</sub> levels increase in-cabin where the ventilation is on recirculation, with the maximum levels of CO<sub>2</sub> in-cabin varying depending on a wide range of vehicle and driving factors. However, where the vehicle air exchange rate has been measured, it is possible to estimate the concentration of CO<sub>2</sub> in-cabin over time. This has been verified through the use of the 2016 RMS study data.

Based on the data collected during the 2016 RMS study, the model established to predict in-cabin CO<sub>2</sub> levels when ventilation is on recirculation and the potential health effects associated with exposure to CO<sub>2</sub> in-cabin over time periods up to an hour (relevant to exposures that may occur in tunnels) for 1 to 5 occupants, the levels of CO<sub>2</sub> that may be present are not expected to adversely affect driver safety.

Assessment of potential exposures that may occur for periods over 1 hour, where ventilation is left on recirculation indicates that there may be levels of CO<sub>2</sub> where there are 1 or more passengers that may affect an already fatigued driver.

It is noted that there is a general lack of guidance or regulations in terms of the design or use of ventilation systems in vehicles in Australia. Hence there is currently no advice to drivers on the suitable use of ventilation in various circumstances, to minimise the potential for effects on already fatigued drivers.

Where RMS provides specific advice to drivers entering road tunnels to put ventilation on recirculation, it would also be necessary to provide advice that recirculation should be switched off and not left on for an extended period of time.

## Section 8. References

---

- Allen, JG, MacNaughton, P, Satish, U, Santanam, S, Vallarino, J & Spengler, JD 2016, 'Associations of Cognitive Function Scores with Carbon Dioxide, Ventilation, and Volatile Organic Compound Exposures in Office Workers: A Controlled Exposure Study of Green and Conventional Office Environments', *Environmental health perspectives*, vol. 124, no. 6, Jun, pp. 805-812.
- Bierwirth, P 2016, 'Carbon dioxide toxicity and climate change: a serious unapprehended risk for human health', *Working Paper, Web Published: ResearchGate DOI:10.13140/RG.2.2.16787.48168*.
- Carreiro-Martins, P, Viegas, J, Papoila, AL, Aelenei, D, Caires, I, Araujo-Martins, J, Gaspar-Marques, J, Cano, MM, Mendes, AS, Virella, D, Rosado-Pinto, J, Leiria-Pinto, P, Annesi-Maesano, I & Neuparth, N 2014, 'CO(2) concentration in day care centres is related to wheezing in attending children', *Eur J Pediatr*, vol. 173, no. 8, Aug, pp. 1041-1049.
- Constantin, D, Mazilescu, C-A, Nagi, M, Draghici, A & Mihartescu, A-A 2016, 'Perception of Cabin Air Quality among Drivers and Passengers', *Sustainability*, vol. 8, no. 9, p. 852.
- Cornak, S, Horak, V & Chaladek, Z 2012, 'Prediction of Vehicle Cabin Air Quality', *Advances in Military Technology*, vol. 7, no. 2, pp. 23-32.
- Corpuz, G 2006, *Analysis of Peak Hour Travel Using the Sydney Household Travel Survey Data* 29th Australasian Transport Research Forum. <[http://atrf.info/papers/2006/2006\\_Corpuz.pdf](http://atrf.info/papers/2006/2006_Corpuz.pdf)>.
- Cronyn, PD, Watkins, S & Alexander, DJ 2012, *Chronic Exposure to Moderately Elevated CO2 During Long-Duration Space Flight*, National Aeronautics and Space Administration (NASA). Houston. <<https://ntrl.ntis.gov/NTRL/dashboard/searchResults/titleDetail/N20120006045.xhtml>>.
- EC 2009, *Fatigue, SafetyNet*, European Commission, Directorate-General Transport and Energy.
- EFA 2014, *Building Bulletin 101, Ventilation of School Buildings, Regulations Standards Design Guidance*, Education Funding Agency. <<https://www.gov.uk/government/publications/building-bulletin-101-ventilation-for-school-buildings>>.
- enHealth 2001, *Health Impact Assessment Guidelines*, Commonwealth Department of Health and Aged Care.
- enHealth 2012, *Environmental Health Risk Assessment, Guidelines for assessing human health risks from environmental hazards*, Commonwealth of Australia. Canberra. <[http://www.health.gov.au/internet/main/publishing.nsf/content/804F8795BABFB1C7CA256F1900045479/\\$File/DoHA-EHRA-120910.pdf](http://www.health.gov.au/internet/main/publishing.nsf/content/804F8795BABFB1C7CA256F1900045479/$File/DoHA-EHRA-120910.pdf)>.
- Ezraty, B, Chabaliere, M, Ducret, A, Maisonneuve, E & Dukan, S 2011, 'CO2 exacerbates oxygen toxicity', *EMBO Rep*, vol. 12, no. 4, Apr, pp. 321-326.
- Ferreira, AM & Cardoso, M 2014, 'Indoor air quality and health in schools', *J Bras Pneumol*, vol. 40, no. 3, May-Jun, pp. 259-268.

Fruin, SA, Hudda, N, Sioutas, C & Delfino, RJ 2011, 'Predictive Model for Vehicle Air Exchange Rates Based on a Large, Representative Sample', *Environmental science & technology*, vol. 45, no. 8, 2011/04/15, pp. 3569-3575.

Gładyszewska-Fiedoruk, K 2011, 'Concentrations of carbon dioxide in a car', *Transportation Research Part D: Transport and Environment*, vol. 16, no. 2, 3//, pp. 166-171.

Gładyszewska-Fiedoruk, K 2011, 'Concentrations of carbon dioxide in the cabin of a small passenger car', *Transportation Research Part D: Transport and Environment*, vol. 16, no. 4, 6//, pp. 327-331.

Goh, CC, Kamarudin, LM, Shukri, S, Abdullah, NS & Zakaria, A 2016, *Monitoring of carbon dioxide (CO<sub>2</sub>) accumulation in vehicle cabin*.

Goromosov, MS 1968, *The physiological basis of health standards for dwellings*, World Health Organization. Geneva.

Grady, ML 2013, 'On-Road Air Quality and the Effect of Partial Recirculation on In-Cabin Air Quality for Vehicles', Mechanical Engineering, University of California.

Grady, ML, Jung, H, Kim, Yc, Park, JK & Lee, BC 2013, 'Vehicle Cabin Air Quality with Fractional Air Recirculation', SAE International. <<http://dx.doi.org/10.4271/2013-01-1494>>.

Halperin, WE 2007, *National Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants. Vol. 1*, National Research Council of the National Academies.

Hamilton and Hardy 2015, *Hamilton and Hardy's Industrial Toxicology, 6th Edition*, Wiley,

Health Canada 1987, *Exposure Guidelines for Residential Indoor Air Quality, A Report of the Federal-Provincial Advisory Committee on Environmental and Occupational Health*, Environmental Health Directorate, Health Protection Branch, Health Canada.

HSDB 2004, *Carbon dioxide*, TOXNET, Specialized Information Services, U.S. National Library of Medicine, Hazardous Substances Data Bank. Bethesda.

Hudda, N, Eckel, SP, Knibbs, LD, Sioutas, C, Delfino, RJ & Fruin, SA 2012, 'Linking In-Vehicle Ultrafine Particle Exposures to On-Road Concentrations', *Atmospheric environment (Oxford, England : 1994)*, vol. 59, 06/06, pp. 578-586.

James, JT, Meyers, VM & Alexander, D 2011, *Assessing the Health and Performance Risks of Carbon Dioxide Exposures*, Presentation at Aerospace Medical Association Meeting 9 May 2011.

Jung, H 2013, 'Modeling CO<sub>2</sub> Concentrations in Vehicle Cabin', SAE International. <<http://dx.doi.org/10.4271/2013-01-1497>>.

Kajtar, L & Herczeg, L 2012, 'Influence of carbon-dioxide concentration on human well-being and intensity of mental work', *Q. J. Hung. Meteorol. Serv*, vol. 116, pp. 145-169.

Knibbs, LD, de Dear, RJ & Atkinson, SE 2009, 'Field study of air change and flow rate in six automobiles', *Indoor air*, vol. 19, no. 4, Aug, pp. 303-313.

Knibbs, LD, de Dear, RJ & Morawska, L 2010, 'Effect of cabin ventilation rate on ultrafine particle exposure inside automobiles', *Environmental science & technology*, vol. 44, no. 9, May 1, pp. 3546-3551.

Law, J, Watkins, S & Alexander, D 2010, *In-Flight Carbon Dioxide Exposures and Related Symptoms: Association, Susceptibility, and Operational Implications*, NASA Center for AeroSpace Information.

<<http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.694.8269&rep=rep1&type=pdf>>.

Lu, CY, Lin, JM, Chen, YY & Chen, YC 2015, 'Building-Related Symptoms among Office Employees Associated with Indoor Carbon Dioxide and Total Volatile Organic Compounds', *Int J Environ Res Public Health*, vol. 12, no. 6, May 27, pp. 5833-5845.

Luangprasert, M, Vasithamrong, C, Pongratananukul, S, Chantranuwathana, S, Pumrin, S & De Silva, IPD 2016, 'In-vehicle CO<sub>2</sub> concentration in commuting cars in Bangkok, Thailand', *Journal of the Air & Waste Management Association*, pp. null-null.

Martin, AN, Boulter, PG, Roddis, D, McDonough, L, Patterson, M, Rodriguez del Barco, M, Mattes, A & Knibbs, LD 2016, 'In-vehicle nitrogen dioxide concentrations in road tunnels', *Atmospheric environment*, vol. 144, 11//, pp. 234-248.

Mathur, GD 2008, 'Field Tests to Monitor Build-up of Carbon Dioxide in Vehicle Cabin with AC System Operating in Recirculation Mode for Improving Cabin IAQ and Safety', *SAE Int. J. Passeng. Cars – Mech. Syst.*, vol. 1, no. 1, pp. 757-767.

Mathur, GD 2009a, 'Measurement of Carbon Dioxide in Vehicle Cabin to Monitor IAQ during Winter Season with HVAC Unit Operating In OSA Mode', SAE International.

<<http://dx.doi.org/10.4271/2009-01-0542>>.

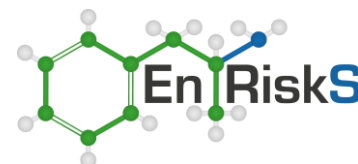
Mathur, GD 2009b, 'Field Monitoring of Carbon Dioxide in Vehicle Cabin to Monitor Indoor Air Quality and Safety in Foot and Defrost Modes', SAE International. <<http://dx.doi.org/10.4271/2009-01-3080>>.

Matton, TJP 2015, 'Simulation and Analysis of Air Recirculation Control Strategies to Control Carbon Dioxide Build-up Inside a Vehicle Cabin', Mechanical, Automotive and Materials Engineering, University of Windsor, Windsor, Ontario.

Mohd Sahril Mohd Fouzi, Mohamad Asyraf Othoman & Sulaiman, SA 2014, 'Effect of Recirculation on Air Quality in a Car Compartment', *Australian Journal of Basic and Applied Sciences*, vol. 8, no. 4, pp. 466-470.

NRC 2007, *Carbon Dioxide. Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants*, National Research Council Washington, DC.

Ott, W, Klepeis, N & Switzer, P 2008, 'Air change rates of motor vehicles and in-vehicle pollutant concentrations from secondhand smoke', *Journal of exposure science & environmental epidemiology*, vol. 18, no. 3, May, pp. 312-325.



Rice, SA 2003, *Health Effects of Acute and Prolonged CO<sub>2</sub> Exposure in Normal and Sensitive Populations*, Second Annual Conference on Carbon Sequestration, May 5-8, Alexandria, Virginia. <<http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.464.2827&rep=rep1&type=pdf>>.

Rice, SA 2004, *Human health risk assessment of CO<sub>2</sub>: Survivors of acute high-level exposure and populations sensitive to prolonged low-level exposure*, Third Annual Conference on Carbon Sequestration. 3-6 May 2004, Alexandria, Virginia. <<https://www.netl.doe.gov/publications/proceedings/04/carbon-seq/169.pdf>>.

Robertson, DS 2006, 'Health effects of increase in concentration of carbon dioxide in the atmosphere', *Current Science*, vol. 90, pp. 1607-1609.

Sahayadhas, A, Sundaraj, K & Murugappan, M 2012, 'Detecting Driver Drowsiness Based on Sensors: A Review', *Sensors (Basel, Switzerland)*, vol. 12, no. 12, pp. 16937-16953.

Satish, U, Mendell, MJ, Shekhar, K, Hotchi, T, Sullivan, D, Streufert, S & Fisk, WJ 2012, 'Is CO<sub>2</sub> an indoor pollutant? Direct effects of low-to-moderate CO<sub>2</sub> concentrations on human decision-making performance', *Environmental health perspectives*, vol. 120, no. 12, Dec, pp. 1671-1677.

Schaefer, KE, Douglas, WH, Messier, AA, Shea, ML & Gohman, PA 1979, 'Effect of prolonged exposure to 0.5% CO<sub>2</sub> on kidney calcification and ultrastructure of lungs', *Undersea Biomed Res*, vol. 6 Suppl, pp. S155-161.

Schaefer, KE 1982, 'Effects of increased ambient CO<sub>2</sub> levels on human and animal health', *Experientia*, vol. 38, no. 10, pp. 1163-1168.

Scott, JL, Kraemer, DG & Keller, RJ 2009, 'Occupational hazards of carbon dioxide exposure', *Journal of Chemical Health and Safety*, vol. 16, no. 2, 3//, pp. 18-22.

Sechzer, PH, Egbert, LD, Linde, HW, Cooper, DY, Dripps, RD & Price, HL 1960, 'Effect of carbon dioxide inhalation on arterial pressure, ECG and plasma catecholamines and 17-OH corticosteroids in normal man', *J Appl Physiol*, vol. 15, May, pp. 454-458.

Selkirk, A, Briggs, JF & Shykoff, B 2010, *Cognitive Effects of Hypercapnia on Immersed Working Divers*, Navy Experimental Diving Unit.

Seppänen, OA, Fisk, WJ & Mendell, MJ 1999, 'Association of Ventilation Rates and CO<sub>2</sub> Concentrations with Health and Other Responses in Commercial and Institutional Buildings', *Indoor air*, vol. 9, no. 4, pp. 226-252.

Sheehy, JB, Kamon, E & Kiser, D 1982, 'Effects of Carbon Dioxide Inhalation on Psychomotor and Mental Performance during Exercise and Recovery', *Human Factors*, vol. 24, no. 5, pp. 581-588.

Singh, BS 1984a, 'Ventilatory response to CO<sub>2</sub>. I. A psychobiologic marker of the respiratory system', *Psychosom Med*, vol. 46, no. 4, Jul-Aug, pp. 333-345.

Singh, BS 1984b, 'Ventilatory response to CO<sub>2</sub>. II. Studies in neurotic psychiatric patients and practitioners of transcendental meditation', *Psychosom Med*, vol. 46, no. 4, Jul-Aug, pp. 347-362.





Sliwka, U, Krasney, JA, Simon, SG, Schmidt, P & Noth, J 1998, 'Effects of sustained low-level elevations of carbon dioxide on cerebral blood flow and autoregulation of the intracerebral arteries in humans', *Aviat Space Environ Med*, vol. 69, no. 3, Mar, pp. 299-306.

Stork, M 2014, 'Some Methods Systems and Sensors which are Possible for Driver's Drowsiness Estimation', *Latest Trends in Circuits, Automatic Control and Signal Processing*.

Stutts, JD, Reinfurt, DW, Staplin, L & Rodgman, EA 2001, *The Role of Driver Distraction in Traffic Crashes*, University of North Carolina, prepared for AAA Foundation for Traffic Safety.

Tay, R & Knowles, D 2004, 'DRIVER INATTENTION: Drivers' Perception of Risks and Compensating Behaviours', *IATSS Research*, vol. 28, no. 1, //, pp. 89-94.

Torres, C 2014, *Breathe Easy, A study on the levels of CO2 in vehicle cabins*.

Tsai, DH, Lin, JS & Chan, CC 2012, 'Office workers' sick building syndrome and indoor carbon dioxide concentrations', *Journal of occupational and environmental hygiene*, vol. 9, no. 5, pp. 345-351.

US EPA 2000, *Carbon dioxide as a fire suppressant: examining the risks*, US Environmental Protection Agency.

USEPA 2011, *Exposure Factors Handbook*, US Environment Protection Agency.

<<http://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252>>.

Vehvilainen, T, Lindholm, H, Rintamaki, H, Paakkonen, R, Hirvonen, A, Niemi, O & Vinha, J 2016, 'High indoor CO2 concentrations in an office environment increases the transcutaneous CO2 level and sleepiness during cognitive work', *Journal of occupational and environmental hygiene*, vol. 13, no. 1, pp. 19-29.

Vercruyssen, M, Kamon, E & Hancock, PA 2007, 'Effects of carbon dioxide inhalation on psychomotor and mental performance during exercise and recovery', *Int J Occup Saf Ergon*, vol. 13, no. 1, pp. 15-27.

Young, KJ, Regan, M & Hammer, M 2003, *Driver Distraction: a review of the Literature*, Report No. 206 Monash University. Victoria, Australia.

Zhang, X, Wargocki, P, Lian, Z & Thyregod, C 2017, 'Effects of exposure to carbon dioxide and bioeffluents on perceived air quality, self-assessed acute health symptoms, and cognitive performance', *Indoor air*, vol. 27, no. 1, pp. 47-64.