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links between cooking aerosols exposure and acute cardiopulmonary and neurological responses: A clinical investigation

by

Motahareh Naseri

Submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Science Engineering and Technology

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Declaration

I declare that the research contained in this thesis, unless otherwise formally indicated within the text, is the original work of the author's original work. The thesis has not been previously submitted to this or any other university for a degree, and does not incorporate any material already submitted for a degree.

Motahareh Naseri

Date

Abstract

Indoor air quality is a growing public health concern, particularly due to the increasing time individuals spend indoors, where they are exposed to a variety of aerosols and pollutants generated by everyday household activities. The prevalence of cooking as a daily activity has raised significant concerns regarding the associated health impacts of cooking aerosols, particularly ultrafine particles (UFPs) and particulate matter (PM) emissions. These pollutants can penetrate deep into the lungs and bloodstream, potentially causing adverse cardiovascular, respiratory, and neurological health effects. The complex composition of cooking aerosols varies significantly depending on the type of stove used, the cooking methods employed, and the specific food items prepared. For instance, frying and grilling are known to produce higher concentrations of UFPs compared to boiling or steaming. Furthermore, the combustion process can generate not only particulate matter but also a variety of gaseous pollutants, including nitrogen dioxide (NO_2) and carbon monoxide (CO), which can further exacerbate health risks. Despite the well-documented risks associated with solid fuel cooking, there is a lack of controlled human exposure studies on the health impacts of gas and electric stove emissions. In this study, we present a new exposure design that addresses several key shortcomings in prior human exposure studies. By carrying out tightly controlled exposure sessions, we reduced the risk of confounding by post-exposure environments, especially in the essential time frame leading up to a 24-hour follow-up measure and exposure during commuting. To our knowledge, this is the first time that the particulate and gaseous fractions of cooking emission effects on cardiopulmonary outcomes have been isolated in a comprehensive manner, enabling a finer estimate of their individual adverse impacts on health. In this study, we introduce a unique intervention method using P100 respirators to isolate the effects of particles and gases, decoupling their respective contributions to health outcomes. Furthermore, this is the first study to measure acute cognitive function responses to gaseous components of cooking air pollution, which is a long-standing gap in the scientific literature. The research presented in this thesis was aimed at addressing these gaps by investigating the health effects of cooking aerosols from gas and electric stoves, post short-term (30 minutes and 2 hours) and medium-term (24-hour) exposure. A series of controlled human exposure studies were conducted, employing novel methodologies to assess cardiopulmonary and neurological responses while accounting for exposure to

different particle sizes and gaseous pollutants. To achieve this, a cohort of healthy adult participants was recruited for controlled exposure studies, where they were subjected to cooking emissions in a monitored environment. Using a novel exposure setup, healthy adults were exposed to emissions under controlled conditions while minimizing post-exposure confounding. A unique intervention using P100 respirators was employed to decouple particle and gas effects. This investigation employed a range of assessment techniques, including cognitive tests to evaluate neurocognitive responses, as well as measurements of blood pressure, electrocardiogram (ECG), fractional exhaled nitric oxide (FeNO), and peak flow meter readings to assess cardiopulmonary effects. The findings reveal that cooking generates significant amounts of UFPs, which have immediate and sustained impacts on lung and heart function. Analysis of the data indicated that cooking-generated aerosols—comprising both particles and gases—had no statistically significant impact on PEFR, SBP, HR, or SpO_2 up to 24 hours post-exposure; however, significant effects were observed for FeNO and DBP, indicating inflammatory and vascular responses. A significant 9.35 % decrease in DBP was observed immediately after cooking, while FeNO rose by 73.24 % right after cooking and remained 60.27 % higher 30 minutes later. The gas-only exposure intervention (from electric stove emissions) revealed no significant changes in DBP, SBP, HR, SpO_2, or PEFR, yet cognitive assessments suggested a transient disruption in memory and attention, likely due to neurocognitive distraction caused by cooking-related gases. These findings underscore the dominant role of particulate matter—rather than gaseous by-products—in driving acute physiological responses to cooking emissions, highlighting the need for targeted mitigation strategies to reduce indoor particle exposure during cooking activities. This research provides valuable insights into how cooking-related pollutants affect human health and offers recommendations for future studies to enhance understanding and policy development in this critical area of public health.

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Above all, I extend my deepest appreciation to my supervisors, Dr. Dhawal Shah, Dr. Mehdi Amouei Torkmahalleh, Dr. Byron Crape, and Dr. Flemming Cassee, for their invaluable mentorship, encouragement, and unwavering support throughout my PhD journey. Their expertise and insightful guidance played a crucial role in shaping my research and ensuring the successful completion of this dissertation.

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My heartfelt appreciation goes to my mother, whose unwavering love and sacrifices have profoundly shaped my path. I am forever indebted to her strength, wisdom, and boundless faith in me. In remembrance of my late father, who left us many years ago, I dedicate this achievement to his memory—his influence continues to guide and inspire me.

To my dear husband, Behzad, and our wonderful daughter, Lara, I am endlessly grateful for your love, patience, and encouragement. Behzad, your steadfast support has been my greatest strength, and Lara, your joyful spirit has been a constant source of motivation throughout this journey.

I would also like to acknowledge my colleagues and friends who have shared this academic path with me. Their camaraderie, encouragement, and moral support have been invaluable, and I deeply cherish the moments of collaboration and shared experiences.

Lastly, I extend my gratitude to Nazarbayev University for their financial support, which enabled me to dedicate myself fully to my research and contribute meaningfully to my field of study.

Sincerely,
Motahareh Naseri

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List of Abbreviations and Acronyms

ABB	Alveolar-blood barrier	LRT	Lower respiratory tract
BBB	Blood-brain barrier	LPG	Liquefied petroleum gas
CH_4	Methane	MCI	Mild cognitive impairment
CH_2O	Formaldehyde	MMSE	Mini-Mental State Examination
CI	Confidence interval	NMHCs	Non-methane hydrocarbons
CNS	Central nervous system	NO	Nitric Oxide
CO_2	Carbon dioxide	NO_2	Nitrogen dioxide
COPD	Chronic obstructive pulmonary disease	OR	Odd ratio
CPC	Condensation particle counter	P	P-Value
CVD	Cardiovascular disease	PAHs	Polycyclic aromatic hydrocarbons
DBP	Diastolic blood pressure	PERF	Peak expiratory flow rate
ECG	Electrocardiogram	PM	Particulate matter
EEG	Electroencephalogram	PNC	Particle number concentration
FeNO	Fractional exhaled nitric oxide	RP	Relative power
FEV1	Forced Expiratory Volume in one second	RH	Relative humidity
FVC	Forced Vital Capacity	RMSSD	Root Mean Square of the Successive Differences
fMRI	Functional Magnetic Resonance Imaging	SBP	Systolic blood pressure
HazRat	Hazard ratio	SDNN	Standard Deviation of NN intervals
HF	High-Frequency Power	SMPS	Scanning Mobility Particle Sizer
HR	Heart rate	SOA	Secondary organic aerosol
HRV	Heart rate variability	SpO_2	Blood oxygen saturation
HVLT-R	Hopkins Verbal Learning Test–Revised	UFPs	Ultrafine particles
IAQ	Indoor air quality	URT	The upper respiratory

IHD	Ischemic heart disease	VOC	Volatile organic compounds
LF	Low-Frequency Power		

Chapter 1: Introduction

1.1. Cooking emissions and health

Individuals, particularly in developed countries, spend approximately 87% of their time indoors, leading to continuous exposure to indoor aerosols and pollutants (Klepeis et al., 2001). Household activities, including cooking, cleaning, cigarette smoking, and burning candles or wood, release aerosol particles (Lachowicz et al., 2022, Massey et al., 2012). Previous research has demonstrated that these aerosols can have harmful effects on almost every organ in the human body (Schraufnagel et al., 2019b, Schraufnagel, 2020). Particulate matter (PM), which refers to the suspended particles in aerosol, is a significant factor among the various indoor pollutants due to its potential health impacts. Cooking, a seemingly innocuous daily activity, has emerged as one of the primary sources of indoor gases and PM, surpassing many other everyday activities in terms of emission levels. PM is typically categorized based on its size, including PM_{10} (particles with a diameter of ≤10 μm), $PM_{2.5}$ (fine particles with a diameter of ≤2.5 μm), and $PM_{0.1}$ (ultrafine particles, UFPs, with a diameter of ≤0.1 μm) (Miller and Newby, 2019) (Figure 1-1).

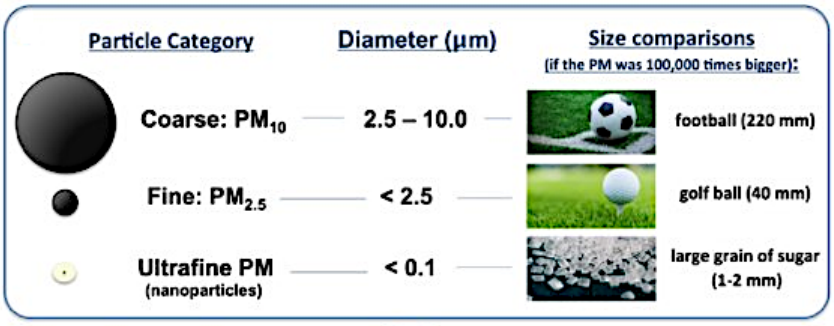


Figure 0-1 . Size-based categorization of PM in Air Pollution. The diagram illustrates the classification of PM by aerodynamic diameter, including coarse particles (PM_{10}), fine particles ($PM_{2.5}$), and ultrafine particles (UFPs; <100 nm). [adapted from (Miller and Newby, 2019)]

Particular matter generated during cooking has been found to exceed the levels encountered during most other routine activities. Kumar et al. (2013a) investigated $PM_{2.5}$ and submicron particle emission rates and compared indoor activities, including

smoking, floor sweeping, vacuuming, candle burning, hair drying, etc. Their findings highlighted cooking as the primary source of indoor air pollution among different activities. Notably, particles generated from just one minute of cooking on a gas stove can reach levels comparable to those produced by 10 minutes of cigarette smoking or one minute of driving heavy-duty vehicles (Kumar et al., 2013a).

Cooking is acknowledged as a short-term event capable of also generating high concentrations of particles in the UFP range (Abdullahi et al., 2013, Amouei Torkmahalleh et al., 2017b, Géhin et al., 2008, Wallace and Ott, 2011, He et al., 2004, Hussein et al., 2006). During cooking, people can be exposed to exceptionally high levels of UFPs, with concentrations reaching up to 550 times higher than background levels (Zhang et al., 2010b). Sources of UFPs during cooking, such as heating an electric stove, a gas-burning in a stove, and frying food, generate particles with different timeframes for reaching peak particle concentrations, ranging from just a few seconds to a few minutes (Afshari et al., 2005). Wallace et al. (2008) reported that the dominant size diameter of particles during 159 cooking episodes was 5 nm. The authors observed that the number concentration of UFPs below 10 nm was ten times higher than that of UFPs above 10 nm. Gas stove flames with no food produced particle sizes between 5-7 nm and an emission rate ranging from 10^{12} to 10^{13} particles/min.

Particles generated during cooking contain various hazardous chemical species, including trace elements, polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), and secondary organic aerosol (SOA) (Abdullahi et al., 2013, Wang et al., 2017, Zhao and Zhao, 2018, Li et al., 2003, Zhang et al., 2019, Klein et al., 2016). However, the composition also depends on the source of the particles. Chromium (Cr), selenium (Se), vanadium (V), and zinc (Zn) were the most abundant metals identified in barbeque restaurants' fine particles (Taner et al., 2013). Furthermore, Amouei Torkmahalleh et al. (2017a) found that V, lead (Pb), and Manganese (Mn) were emitted during grilling beef under poor ventilation. Investigators assessing the carcinogenic health hazards for kitchen staff revealed a significant reduction in the respiratory functions of cooks. This decline was linked to increased exposure to trace elements such as Pb, Cr, copper (Cu), potassium (K), nickel (Ni), and Mn (Varlı et al., 2024). The chemical composition analysis also revealed the presence of toxic elements such as Zn, Cr, barium (Ba), and aluminum (Al) in deep-frying cooking-generated PM (Saini and Sharma, 2023).

Lu et al. (2021) reported that the total potential carcinogenic risk level associated with VOCs emitted from six residential cooking methods decreased as follows: deep-frying (risk level = 5.75) > stir-frying (risk level = 3.95) > quick-frying (risk level = 2.94) > stewing (risk level = 1.99) > boiling (risk level = 1.73) > steaming (risk level = 1.48). The incremental lifetime cancer risk associated with the inhalation of VOCs was estimated to be 1.4×10^{-5} in barbecue restaurants (Arı et al., 2020). Exposure to PAHs can increase oxidative stress levels, contributing to the development of degenerative and non-degenerative ailments such as cardiovascular and pulmonary diseases, diabetes, and Alzheimer's disease (Deligannu and Muniandy, 2024, Verma et al., 2022). Heating soybean oil on a gas stove, the incremental lifetime cancer risk values associated with PAHs exposure exceeded the acceptable thresholds set by the United States Environmental Protection Agency (Luo et al., 2024). Prolonged exposure to carcinogenic PAHs, particularly in the form of respirable suspended PM, may induce acute pulmonary conditions, exacerbate asthma, increase susceptibility to pulmonary tuberculosis, and elevate the risk of developing lung cancer among individuals engaged in cooking activities (Goel et al., 2019).

The presence of other pollutants in cooking fumes, including respiratory irritants such as nitrogen dioxide and acrolein, can have harmful effects on health (Seltenrich, 2014, Lee and Gany, 2013, Hecht et al., 2010). Exposure to these pollutants, including PM and aldehydes, can induce lung and respiratory inflammation, thereby contributing to chronic respiratory symptoms (Olloquequi and Silva O, 2016). Results from a recent study indicated that individuals who cooked 21 times or more per week have a higher risk of chronic bronchitis compared to those who cooked less than 14 times per week (Odds Ratio (OR) = 4.73; 95% Confidence Interval (CI) 1.65–13.53) (Chen et al., 2018). Cooking inside a home was associated with respiratory symptoms such as dyspnea (OR = 1.85; CI 1.05–3.24), runny nose (OR = 1.58; 95% CI 1.05–3.24), coughing (OR = 1.68; 95% CI 1.20–2.36), and sore throat (OR = 1.91; 95% CI 1.25–2.92) (Juntarawijit and Juntarawijit, 2019). Even with clean fuels like natural gas or an electric stove, cooking can significantly increase the risk of respiratory difficulties and symptoms. Since cooking is a daily activity in every household, health effect related to cooking represents a public health issue requiring greater attention.

1.2. Ultrafine particles health effects

Exposure to UFPs, in particular, has been associated with a wide array of health effects, including cardiopulmonary morbidity and mortality (Schwarz et al., 2023, Samoli et al., 2020, Liu et al., 2018, Lanzinger et al., 2016), exacerbation of inflammatory conditions such as asthma, acute bronchitis, and chronic obstructive pulmonary disease (COPD) (Moreno-Ríos et al., 2022), cardiovascular diseases (CVD) (Xiao et al., 2016, Bourdrel et al., 2017, Yang et al., 2017, Møller et al., 2020, Liu et al., 2018), neurodegenerative diseases (Heusinkveld et al., 2016, Wei et al., 2017), diabetes (Chen et al., 2016), and cancer (Marabini et al., 2017). Evidence from global investigations indicates that greater UFP exposure produces both immediate and long-term systemic and neurological consequences, including elevated blood pressure (Cory-Slechta et al., 2018, Liu et al., 2018).

Furthermore, UFP exposure has been linked to systemic inflammation, endothelial dysfunction, and coagulation abnormalities, all of which heighten the risk of ischemic cardiovascular disability and hypertension (Hong and Jee, 2020). UFPs can travel along the olfactory nerves to the brain, potentially leading to cerebral and autonomic dysfunction. Due to their small size, UFPs have a high propensity for deposition in the alveoli and possess a large surface area that can harbor toxins (Kwon et al., 2020). Additionally, they can penetrate blood vessels, potentially triggering oxidative stress and inflammation. This process could catalyze the progression of atherosclerosis, ultimately leading to thrombus formation and exerting genotoxic effects (Møller et al., 2020, Brown et al., 2013, Meng et al., 2016). The role of genotoxicity in carcinogenic processes is of particular significance, highlighting the potential for these nanoparticles to contribute to cancer development (Leikauf et al., 2020, Miller and Newby, 2020, Stone et al., 2017).

1.3. Toxicological evidence of UFPs translocation to cardiopulmonary organs

Inhaled UFPs have the potential to penetrate deeply into the lungs, reaching the alveoli due to their tiny size (1- 2 and 1- 3) (Moreno-Ríos et al., 2022, Castelo, 2017). With a larger specific surface area, UFPs can elicit intense reactions or inflammatory responses in the body than larger particles (Kulkarni et al., 2011, Marval and Tronville, 2022, Thomas, 2013). Various mechanisms, such as inertial impaction, Brownian diffusion, gravitational sedimentation, and electrostatic effects, contribute to particle deposition in the respiratory tract (Wang, 2005).

Toxicological evidence showed that UFPs can translocate beyond the respiratory tract within a relatively short time. For instance, within just 1 hour of exposure, 24% of the inhaled titanium dioxide particles had penetrated the rats' lung cells and entered the bloodstream. Interestingly, there was no significant difference in the distribution of particles between the 1-hour and 24-hour samples across the different lung compartments (Weinhold, 2005). Animal studies indicate that UFPs can reach interstitial sites in the respiratory system and extrapulmonary organs such as the liver within 4 to 24 hours after inhalation exposure (Oberdörster et al., 2002). Moreover, studies involving manganese oxide nanoparticles have revealed that while the uptake of particles into the olfactory bulb was minimal (0.2%) within the first 30 minutes of intranasal exposure, it increased significantly to 6.8% by 24 hours (Elder et al., 2006). In terms of lung retention, the concentration of ^{13}C in the lungs decreased from 1.39 µg/g on day 1 to 0.59 µg/g by day 7 post-exposure. However, in the olfactory bulb, ^{13}C levels exhibited a persistent increase, rising from 0.35 µg/g on day 1 to 0.43 µg/g by day 7 (Oberdörster et al., 2004).

Fine particles in the 1–3 µm range can deeply penetrate lung tissue and settle in the alveoli. In contrast, larger coarse particles, over 8 µm, impact the respiratory airways and deposit in the larger bronchioles due to their greater inertia (Oberdörster, 2000, Tsuda et al., 2002). Ultrafine particles, smaller than 0.1 µm, remain suspended in the air for extended periods and can easily penetrate the alveoli. While cells are generally capable of engulfing particles of various sizes, UFPs have a unique ability to translocate across alveolar epithelial cells by diffusion through the lipid bilayer of cell membranes (Yacobi et al., 2010). Considering their aerodynamic diameter, particles measuring 10 µm or larger primarily affect the membranes of the nasopharynx upon inhalation. Particles within the 5–10 µm range typically deposit on the airways and are generally cleared by the body's alveolar macrophages and lung lymphatic system. Particles ranging from 1 to 2.5 µm tend to navigate towards the terminal bronchioles, where they accumulate and can induce tissue damage, a characteristic feature observed in cases of centrilobular emphysema (Schraufnagel et al., 2019a).

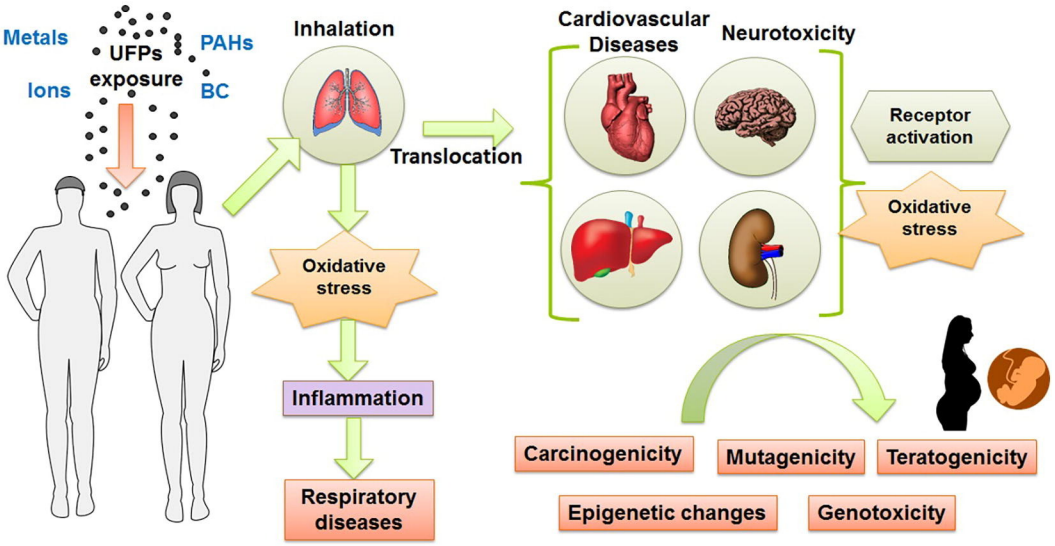


Figure 0-2 . Overview of toxicological effects resulting from exposure to ultrafine particles (UFPs, <0.1 µm in diameter).[adapted from ((Moreno-Ríos et al., 2022)]

Moreover, recent evidence has suggested that nanoparticles can breach the alveolar-blood barrier (ABB), entering the bloodstream and accumulating in extrapulmonary organs (Moller et al., 2008). Nanoparticles smaller than 34 nm have been observed to transit to regional lymph nodes within 30 min, eventually finding their way into the bloodstream (Kreyling et al., 2010). Likewise, nanoparticles less than 6 nm in diameter swiftly translocate from the alveoli to the bloodstream, reaching various tissues and organs in the body, and are subsequently cleared by the kidneys (Choi et al., 2010a, Choi et al., 2010b). A clear size-dependent effect from inhaling 15 nm versus 410 nm silver nanoparticles was observed in rats after nose-only exposure. Exposure to 15 nm silver nanoparticles increased the influx of neutrophils, cellular damage markers, pro-inflammatory cytokines, and total glutathione in the lungs. In contrast, exposure to 410 nm silver particles did not produce any effects (Braakhuis et al., 2014).

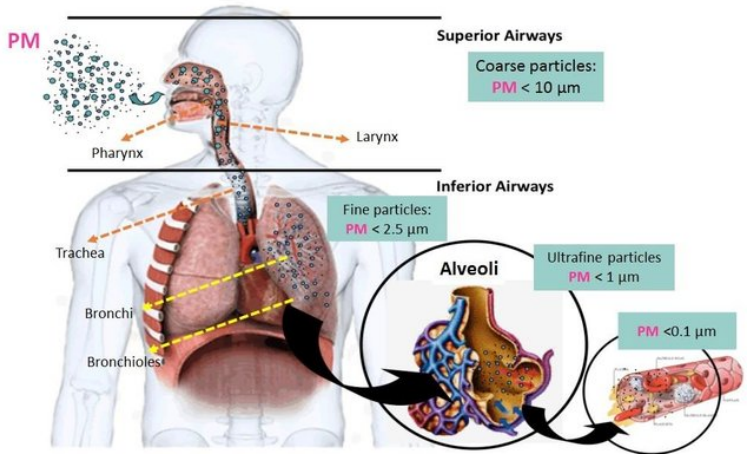


Figure 0-3. Respiratory tract and particle size classification [adapted from (Castelo, 2017)].

Several mechanisms have been proposed to explain the impact of nanoparticles on cardiovascular health. One such pathway involves an inflammatory response in the lungs triggered by the systemic release of cytokines (Seaton et al., 1995). For example, increased doses of cooking oil fumes elevated biomarkers of oxidative stress, pro-inflammation (TNF-α and IL-1β), and apoptosis (NF-κB and Caspase-3) in rats (Ma et al., 2021). Multiple investigations have demonstrated that nanoparticles migrate from the lungs into the circulatory system, directly impacting the cardiovascular system (Hoet et al., 2004, Oberdörster et al., 2005, Nemmar et al., 2001, Kreyling et al., 2002, Oberdörster et al., 2002, Takenaka et al., 2001). Oxidative stress, mitochondrial dysfunction, and endothelial dysfunction are among the mechanisms implicated in causing tissue damage in the cardiovascular system (Guo et al., 2021). Particles might activate sensory receptors on the alveolar surface, initiating signals that modulate autonomic nervous system activity. This altered autonomic function could induce changes in cardiovascular parameters, such as heart rate variability (HRV), blood pressure regulation, and vascular tone, ultimately contributing to cardiovascular dysfunction (Miller et al., 2012).

1.4. Toxicological evidence of UFPs translocation to the brain

Animal studies suggest that inhaled UFPs can reach the brain through two main pathways: axonal transport from the nasal olfactory mucosa to the olfactory bulb (1-4) (Oberdörster et al., 2009, Emad et al., 2021) and breaching the Blood Brain Barrier (BBB) following systemic absorption (Qi et al., 2022, Peters et al., 2006, Lucchini et

al., 2012, Heusinkveld et al., 2016). The BBB, comprised of tightly bound endothelial cells, regulates material transport between the bloodstream and the brain. Nanoparticles deposited on the nasal mucosa in the upper respiratory tract (URT) can travel to the brain's olfactory bulb via the olfactory route. Similarly, nanoparticles in the lower respiratory tract (LRT) can cross the Air Blood Barrier (ABB) into the bloodstream and subsequently enter the brain across the BBB (Kreyling, 2016). Additionally, inflammatory responses triggered by UFPs can generate cytokines in the blood, impacting the central nervous system (CNS) (Balasubramanian et al., 2013).

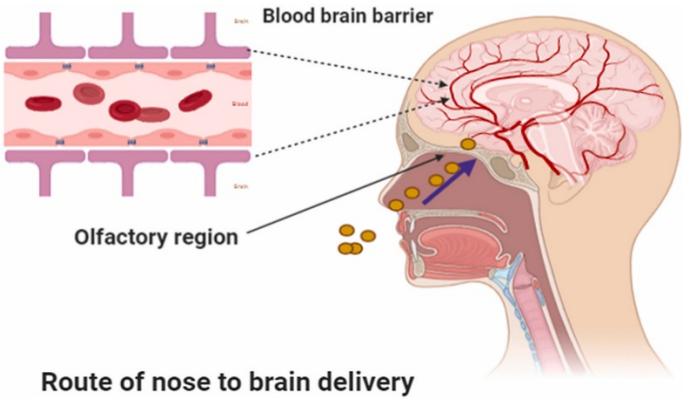


Figure 0-4 . Illustration of the translocation route of nanoparticles from the nasal cavity to the brain. [adapted from (Emad et al., 2021)].

Researchers conducting animal inhalation studies reported that as much as 20% of UFPs deposited on the olfactory mucosa could travel to the olfactory bulb. This movement suggests that these particles can navigate the olfactory system and reach deeper brain areas, raising concerns about potential neurotoxic effects and their implications for neurological health (Oberdörster et al., 2004). Additionally, particles migrating from the olfactory mucosa to the olfactory bulb represent a potential pathway to circumvent the BBB (Tian et al., 2019). The olfactory-brain translocation route has been identified as a possible pathway for zinc oxide (ZnO) nanoparticles to enter the brain following inhalation in rats (Kao et al., 2012). Researchers have show that the likelihood of ^{192}Ir nanoparticles (20 nm) accessing the brain through neuronal pathways from the nasal cavity is 45 times higher than from nanoparticles deposited in the lungs. Moreover, the proportion of ^{192}Ir nanoparticles in the brain is higher following nasal exposure than intratracheal inhalation (Kreyling, 2016).

Two hours after injection, the insulin-targeted PEG-coated gold nanoparticles (20 nm) concentration in mouse brains was more than five times higher than in the control group (Shilo et al., 2014). Similarly, adult rats exposed to polydisperse aerosols of uranium tetraoxide (UO_4) particles (4.5 μm) via a nose-only inhalation system demonstrated significantly elevated uranium concentrations, four hours after exposure, in all brain regions compared to controls. Elemental uranium was identified in distinct areas, including the dendrites of olfactory neurons, the paracellular junctions of neuroepithelial cells, and the olfactory nerve tracts (Ibanez et al., 2019). Additionally, increased levels of neurotransmitters (glutamate and glycine) and changes in IL-1β mRNA expressions in the olfactory bulb were observed 6 and 11 h after the instillation of carbon black nanoparticles (14 nm and 95 nm) in mice (Mitsushima et al., 2008). A six-hour exposure to ^{13}C UFPs led to a notable and prolonged accumulation of ^{13}C in the olfactory bulb along with a significant but variable increase in the cerebrum and cerebellum of rats, persisting for up to seven days after exposure (Oberdörster et al., 2004).

Researchers conducting further animal investigations have revealed various effects linked to exposure to UFPs, extending beyond neurodevelopmental impacts. Investigators in these studies noted changes in emotional behavior, impairments in learning abilities, disruptions in neurotransmission processes, alterations in spontaneous motor activity, and compromised performance in avoidance behaviors (Kumar et al., 2013b).

1.5. Objectives

The research presented in this thesis was designed to answer the fundamental question: What are the short-term cardiopulmonary and neurological effects of controlled exposure to cooking-generated UFPs and gases, and what are their individual contributions to these health outcomes? To address this, the key objectives of this thesis were to investigate the short-term cardiopulmonary and neurological responses to exposure to cooking aerosol using an improved study design. Some previous clinical studies that addressed short-term exposure to cooking aerosol and health outcomes lacked a proper study design (Table 1-1). In such studies, study participants were invited to the exposure chamber without eliminating their unwelcome exposures to different sources which could impact the exposure study as confounders. Such

unwelcome exposures could be exposures to UFPs from indoor sources, including cooking at participants' homes, and from outdoor sources, including traffic while commuting to the exposure facility, up to 24 hours prior to the start of the exposure study. Similar unwelcome exposures could take place during the exposure study between the post-exposure measurements, particularly when the study participants are asked to leave the facility and return after a certain period, such as 24 hours. Other limitations of such studies include the lack of exposure assessments for UFPs below 10 nm, the lack of measurements of multiple exposure factors, the lack of measurements of various neurological responses, and the lack of identifying the relative contribution of gas and UFPs. Additionally, exposure studies on cooking sources observed the synergistic effects of cooking particles or emission gases, without exploring their individual contributions (Soppa et al., 2014, Soppa et al., 2017, Fedak et al., 2019). In this thesis, we aimed to decouple the effects by specifically investigating the effects of gases by using P100 respirators and mitigating the particles' effect as part of our intervention method.

Table 0-1. Summary of clinical studies investigating the short-term (within 24 hours) effects of cooking-related air pollution exposure on cardiovascular and respiratory function.

Previous study	Exposure sources	Study design	Post exposure Period	Health outcome	Particle-number size distributions
Fedak et al. (2019)	LPG, gasifier, fan rocket, elbow rocket, three stone fires	William Square design/ Cross over	Remain in the facility till 3 h post exposure, leave the facility and come back after 24 h	Blood pressure, Lung function	10-500 nm

Soppa et al. (2014)	candles burning (CB), toasting bread (TB), frying sausages (FS)	Randomized Sham-Controlled Exposure	Participants left after exposure and returned after 24 h	Lung function	<100 nm
Soppa et al. (2017)				Blood pressure	

1.6. Hypothesis

The thesis research has several hypotheses, including.

1. Exposure to cooking-generated particles and gas emissions is hypothesized to affect blood pressure and heart rate.
2. Cooking-generated particles and gases cause lung inflammation and alter PEFR.
3. Inhalation of cooking-generated particles increases FeNO levels.
4. Acute exposure to cooking-generated gases has the potential to impair short-term cognitive performance.

Research on the impact of cooking aerosols on brain cognitive impairment is currently lacking. Existing epidemiological and cohort studies have focused on the association between solid fuel cooking and cognitive assessments. However, there is a notable absence of clinical studies investigating the effects of gas and electric stoves on cognitive impairment.

No 24 h controlled human-exposure study has been conducted on the effects on the heart, brain, and lungs due to cooking sources (gas and electric stoves), where participants remained in the controlled experimental environment for the entire post-exposure period. Understanding the relative contributions of UFPs and gases emitted during cooking to cardiopulmonary and neurological outcomes under controlled human-exposure studies remains unexplored.

1.7. Thesis overview

This doctoral dissertation is structured into seven chapters, outlined as follows:

Chapter 1 provides an overview of the background linking cooking-related pollution to health outcomes. Furthermore, it introduces the objectives and hypotheses of the study.

Chapter 2 delves deeply into the mechanisms by which particles enter the human body and the cardiovascular, respiratory, and neurological effects of ultrafine particles on humans, based on the latest epidemiological and clinical studies available in the literature

Chapter 3 describes the main methodology employed in analyzing health outcomes resulting from short-term and 24 h post-exposure to cooking aerosols.

Chapter 4 provides the results of the 30-minute post-exposure to cooking aerosols using electric stoves, the exposure assessment, and the health measurements.

Chapter 5 shows the main results of the 2 h post-exposure to cooking aerosols using electric stoves and gas stoves. The exposure assessment and the health measurement results are provided.

Chapter 6 shows the main results of the 24 h post-exposure to cooking aerosols using electric stoves and gas stoves. The exposure assessment and the health measurement results of the standard and intervention methods are provided.

Chapter 7 summarizes the significant insight of the performed thesis research work and offers valuable recommendations for future studies.

1.8. Author's Contribution

This thesis is based on a series of studies conducted between 2019 and 2024. The candidate (Motahareh Naseri) was the primary researcher responsible for the conceptualization, experimental design, data collection, data analysis, interpretation, and manuscript writing for all included studies. The contributions of co-authors were limited to supervision, statistical advice, data collection assistance, or manuscript editing, as indicated in the individual publications. Specific contributions are detailed below:

- **Study 1:** Acute Cardiovascular Responses to Frying Aerosol Exposure: A 24-Hour Randomized Controlled Exposure and Intervention Trial

(submitted to Neurotoxicology Journal, 2025): Study design, data collection, data analysis, interpretation, and manuscript writing

-
- **Study 2:** Human exposure to aerosol from indoor gas stove cooking and the resulting cardiovascular system responses (published in *Toxicology Reports*, 2024): Study design, data collection, data analysis, interpretation, and manuscript writing

- **Study 3:** Interaction of Cooking-Generated Aerosols on the Human Nervous System and the Impact of Caloric Restriction Post-Exposure (published in *Neutrients, 2024*): Study design, data collection, data analysis, interpretation, and manuscript writing

- **Study 4:** Human exposure to aerosol from indoor gas stove cooking and the resulting nervous system responses (published in *Indoor Air, 2021*): Study design, data collection, data analysis, interpretation, and manuscript writing
- **Study 5:** The impact on heart rate and blood pressure following exposure to ultrafine particles from cooking using an electric stove: (published in *Scinece of the Total Environment*, 2021): Data collection, data analysis, interpretation, and manuscript writing
- **Study 6**: The impact of frying aerosol on human brain activity (published in *Neourotoxicology, 2019*): Data collection, data analysis, interpretation, and manuscript writing

Chapter 2: Literature Review

2.1. Characteristics of cooking emissions

2.1.1. Cooking methods and particle emission

Emissions from cooking vary in their concentrations and compositions. Several factors influence these emissions, including the cooking methods, habits, oil type and temperature, heating source, condiments, and the pan utilized. Cooking habits refer to the individual preferences and methods a cook follows while preparing food. For instance, some individuals opt for slow cooking at lower temperatures over an extended period, whereas others prefer high-temperature, quick-cooking techniques. Additionally, the timing of ingredient additions varies; some cooks incorporate condiments at the start of the process, while others add them toward the end. A cooking method or recipe is characterized by the combination of various elements, including oils, food ingredients, vegetables, condiments (additives), sauces, and other components. Cooking methods, including boiling, steaming, stewing, stir-frying, pan-frying, deep-frying, grilling, broiling, baking, toasting, and microwaving, affect emissions. Research suggests that grilling and frying produce more particles than other cooking methods (Olson and Burke, 2006, Massey et al., 2012, Wan et al., 2011). For example, See & Balasubramanian (2008) in their study of five different types of common cooking methods investigated cooking plain tofu. The baseline mass concentration of $PM_{2.5}$ was determined to be 15.4 $\mu g/m^3$, which increased to 72.3 $\mu g/m^3$, 91.6 $\mu g/m^3$, 120 $\mu g/m^3$, 130 $\mu g/m^3$, and 209 $\mu g/m^3$ during steaming, boiling, stir-frying, pan-frying, and deep-frying, respectively. Additionally, the authors also showed that the mass concentration of the 16 PAHs had the same trend as the $PM_{2.5}$ concentration (See and Balasubramanian, 2008).

$PM_{2.5}$ concentrations were observed to be 4670 $\mu g/m^3$, 237 $\mu g/m^3$, 1136 $\mu g/m^3$, and 1592 $\mu g/m^3$ during fish broiling, meat frying, egg frying, and meat broiling, respectively (Kang et al., 2019). Comparatively, Li et al. (2015) the investigated chemical compositions of particles collected from meat roasting, a frying chimney outlet in a cafeteria, fish roasting, snack-street boiling, and a boiling chimney outlet in a cafeteria in China. Meat roasting and frying produced the highest $PM_{2.5}$ particles, 1107 $\mu g/m^3$ and 992 $\mu g/m^3$, respectively. A more recent study in the kitchen areas of five student studio apartments, the researchers found that the ranges (and medians)

of mean concentrations of $PM_{2.5}$ were 6–37 (30) μg/m^3 for deep-frying, 2–38 (7) μg/m^3 for stir-frying, 2–11 (4) μg/m^3 for boiling, and 2–7 (3) μg/m^3 for steaming (Tang and Pfrang, 2023).

Research has also indicated that frying generates higher levels of UFPs and $PM_{2.5}$ compared to other cooking methods, such as grilling, especially when foods like onions, eggplant, potato chips, pork meat, bacon, and cheese are prepared (Buonanno et al., 2011, Nasir and Colbeck, 2013, He et al., 2004). Grilling ground beef produced a total PM concentration of approximately 13.85 mg/m³ under poor ventilation conditions (Amouei Torkmahalleh et al., 2017b). Among the various cooking methods, deep-frying led to the highest rise in particle number concentration, reaching 6.0×10^5 particles.cm^{-3}. Pan-frying followed by an increase to 1.1×10^5 particles.cm^{-3}. Stir-frying resulted in a concentration of 9.3×10^4 particles.m^{-3} , while boiling produced 6.9×10^4 particles.cm^{-3}. The lowest increase was observed during steaming, which reached 5.4×10^4 particle.cm^{-3} (See and Balasubramanian, 2006). Production of particles during frying is a dynamic and complex process. During the frying process, cooking oils undergo a complex and dynamic chemical transformation influenced by high temperatures, air, and moisture (Dangal et al., 2024, Sébédio and Juaneda, 2007). Vegetable oils are predominantly composed of fatty acids with aliphatic carboxylic acid chains (Orsavova et al., 2015), featuring reactive carboxyl and unsaturated groups that contribute to the intricate chemistry of frying. Three main chemical reactions, hydrolysis, oxidation, and thermal alteration, cause various compounds during the heating oils (Erickson et al., 2023, Shaker et al., 2022). Triglycerides are the main components of cooking oils, with triolein and trilinolein being the predominant forms. These triglycerides break down during frying at high temperatures, leading to the production of common cooking emissions of fatty acids such as palmitic acid, linoleic acid, and oleic acid (Wang et al., 2021, Mahmud et al., 2023, Ojha et al., 2024). In the presence of moisture, fats undergo hydrolysis reactions where ester bonds are broken, producing diacylglycerides and free fatty acids. Further hydrolysis of diacylglycerides produces polar and lighter compounds such as monoglycerol, additional free fatty acids, and glycerol. These fatty acids generated through hydrolysis are reactive and contribute to forming PM and UFPs (Menalla et al., 2024, Perkins, 2007, Erickson, 2015). In this thesis we focus on frying, a popular cooking method, because of its

substantial contribution to the production of UFPs and $PM_{2.5}$ (See and Balasubramanian, 2006, Alves et al., 2021, Zhou et al., 2014).

2.1.2. Factors influencing cooking emissions

Numerous controlled studies have explored the impact of cooking oil type and temperature on particle emissions. Oil smoke temperature is a key factor influencing these emissions (Amouei Torkmahalleh et al., 2017b, Gao et al., 2013). For example, six frequently used commercial cooking oils (olive oil, soybean oil, rapeseed oil, peanut oil, corn oil, and sunflower seed oil) were heated and the results revealed that average particle number concentration decreased inversely with their smoke points: olive oil > peanut oil ≈ rapeseed oil > canola oil > corn oil > sunflower seed oil (Zhao et al., 2019). However, smoke temperature becomes important when particle number and mass concentrations emitted at a temperature higher than the oil's smoke temperature are compared with emissions at a temperature below its smoke point. Comparing the emissions of two or more different cooking oils based on their smoke point might not result in a consistent association between emissions and smoke temperature of the oils, as it depends on the heating temperature, which could be below or above the smoke temperature of all oils or some of the oils. This point has not been given attention in the literature when the findings were interpreted.

The authors of the study by Peng et al. (2017) suggested that using different cooking methods (stir-frying, pan-frying, and deep-frying) and oils (palm, rapeseed, sunflower, and soybean) can also affect the production of aldehydes, and their particle concentration during cooking. The findings revealed that deep frying resulted in the highest total aldehyde emissions, followed by pan-frying and stir-frying. Among the oils, sunflower oil produced the most aldehydes, regardless of the cooking method or food type, while rapeseed and palm oils had comparatively lower emissions. In another study, PM emissions were observed to be highest for olive oil, followed by mustard, saffola, and peanut oil, with the lowest emissions observed for soybean oil. Also, the highest average daily dose for both males and females was with olive oil, followed by mustard, saffola, and peanut oil, with the lowest average daily dose found in soybean oil (Saini and Sharma, 2023).

Research has consistently demonstrated that higher cooking temperatures lead to increased numbers and mass concentrations of particles (Lachowicz et al., 2022, Buonanno et al., 2009, Kumar et al., 2013a, Amouei Torkmahalleh et al., 2013, Zhang

et al., 2010b). However, this finding might depend on the heating temperature if it is above or below the smoke temperature of all the studied oils or some of them which need further attention in the literature. The increases in the particle emission with temperature also depend on the type of oil used. "*Low-emitting oils"* showed considerably less particle concentration increase compared to those with higher emissions, particularly as oil temperature increased from 130°C to 197°C (Amouei Torkmahalleh, 2022). Suggesting that the associations between the heating temperature and oil emissions might vary when the heating temperature is below or above the smoke temperature of the heated oil as the chemistry of the oil changes at the smoke temperature. Notably, changes in $PM_{2.5}$ emissions due to temperature were observed consistently across all oils only when temperatures exceeded 150 °C (Amouei Torkmahalleh et al., 2012). Buonanno et al. (2009) found that grilling 50 grams of bacon on a gas stove resulted in a 70% increase in particle number concentration (PNC) and an increase in particle mode diameter from 22 nm to 57 nm as the grilling temperature rose from 82 °C to 114 °C. The particle number concentration during heating different oils up to 265 °C were as follows: soybean (7.8×10^5 particles/cm^3), corn (7.5×10^5 particles/cm^3), rapeseed (6.8×10^5 particles/cm^3), and sunflower (5.7×10^5 particles/cm^3) (Zhao et al., 2019).

Extensive literature indicates that cooking using a gas stove leads to higher levels of UFPs than cooking with an electric stove (Buonanno et al., 2009, Dennekamp et al., 2001, Jørgensen et al., 2013). Patel et al. (2020), for example, studied particle size distributions down to 1 nm emitted during stir-frying using a gas (propane) stove and an electric hot plate. They found that stir-frying on a gas stove resulted in a significantly higher particle concentration (10 times more) than cooking on a hot plate. Similarly, the number of particles emitted directly from the gas flame was higher (7 times more) than those from the hot plate for sizes larger than 4 nm. Wallace et al. (2008) previously demonstrated that gas flames emit UFPs at a higher emission rate (average of 10.6×10^{12} particles/min) compared to electric stoves (average of 5.4×10^{12} particles/min) across the size range of 2 to 64 nm. Several studies have consistently shown that gas flames also tend to generate particles with larger mean diameters than electric stoves (Dennekamp et al., 2001, Jørgensen et al., 2013, Wallace et al., 2008). Wallace et al. (2008) reported an average mean diameter of particles emitted from gas flames ranged from 4.3 nm to 24.0 nm, while for electric stove, the mean diameter ranged from 5.2 nm to 30 nm.

Amouei Torkmahalleh et al. (2018), further, showed that the cooking pan's material affects the emission of UFPs larger than 10 nm. They found that ceramic pans emitted the highest levels of UFPs, followed by granite, aluminum, and Teflon pans. However, their study was limited to particles larger than 10 nm and the utility of a diffusion charger device rather than a Scanning Mobility Particle Sizer (SMPS) or a Condensation Particle Counter (CPC).

2.1.3. Cooking generated particles' morphology

Understanding the structural characteristics of cooking-generated particles is crucial for assessing their transport behavior and potential toxicity. However, limited research has been conducted on the morphology of PM emitted from cooking. Available evidence indicates that cooking particles predominantly exhibit an aggregated, branched-chain-like morphology when frying on gas and electric stoves, although individual nanoscale spherical particles have also been observed (Buonanno et al., 2009). Furthermore, the combustion of solid fuels and gas stoves in the absence of food primarily generates nanosized spherules, which subsequently form submicron soot particles (Shen et al., 2017).

Li et al. (2019) examined the morphology of $PM_{2.5}$ and PM_{10} particles collected from four Chinese restaurants employing distinct cooking techniques. The cooking oil used across all restaurants was a blend comprising 94% soybean oil and 6% sunflower seed oil. In their study, cooking-derived particles were classified into six distinct morphological categories: rectangular, flocculent, flat, irregular, spheroidal, and spherical. For PM_{10}, the most prevalent morphology was the agglomerated rectangular shape, constituting approximately 55% of the total particles. Flocculent-shaped particles accounted for around 30%, whereas flat and irregular particles each represented about 5%. Spheroidal particles made up less than 2% of PM_{10}, while regular spherical particles were the least common, at approximately 1.5%. Similarly, $PM_{2.5}$ was primarily composed of rectangular particles (roughly 75%), followed by flocculent particles (close to 20%). Li et al. (2017) further reported that the cooking method influenced particle morphology. Specifically, water-based cooking predominantly generated rectangular particles, whereas oil-based cooking and roasting processes resulted in a higher abundance of spherical and spheroidal particles, respectively.

2.1.4. Particulate polycyclic aromatic hydrocarbon (PAH) emission

During cooking, the partial combustion or thermal breakdown of hydrogen- and carbon-based organic compounds can result in the formation of polycyclic aromatic hydrocarbons (PAHs)8. UFPs, with their high surface area to mass ratio, have an increased capacity to adsorb PAHs (Geng and Bai, 2024, Bozek et al., 2016). Oil-based cooking methods and foods with higher fat content produce more PAHs. Consequently, frying typically results in significantly higher levels of PAHs as a result of the consumption of large amounts of oils and the involvement of more water than roasting or char-broiling meats (Li et al., 2018). The total concentrations of PAHs during deep frying chicken with rapeseed oil using a gas stove were measured to be 650 ng/m^3 (Geng and Bai, 2024). Evidence from various experimental studies with different types of oils has indicated that among the several PAHs, naphthalene was most abundant in sunflower oil, anthracene in corn oil, and phenanthrene in soybean oil, mustard oil, peanut oil, and blend oil (Zhang et al., 2009). The total PAH emission factors for chicken, pork, and fish were lower during oil-free cooking compared to cooking with oil (Huang et al., 2023).

2.1.5. Particulate and gases volatile organic compound and secondary organic aerosol emissions

Numerous experimental studies have highlighted cooking as the primary source of gas and particle matter VOCs, and SOA. Aldehydes were identified as the predominant VOC during heating of corn oil (Zhang et al., 2023). Researchers investigating typical Chinese cooking methods found that concentrations of emitted VOCs for different cooking methods were the following: stir-frying (3.809 mg/m³) > quick-frying (2.724 mg/m³) > deep-frying (2.465 mg/m³) > boiling (1.161 mg/m³) ≈ stewing (1.149 mg/m³) > steaming (0.440 mg/m³). The predominant constituents were aldehydes, alkanes, unsaturated aldehydes, alcohols, and alkenes (Lu et al., 2021).

Stir-frying with common Chinese additives such as garlic, ginger, myrcia, and pepper generated significant amounts of methyl pyrrole (Arı et al., 2020). The SOA production efficiency was found to be 3.82×10^{-15}, 3.31×10^{-15}, 2.68×10^{-15}, 2.55×10^{-15}, and 1.7×10^{-15} μg $molecules^{-1}$ s^{-1} for sunflower, corn, canola, olive, and peanut oil, respectively. Multivariate linear regression results demonstrated a strong correlation (R^2= 0.97) between the SOA production rate and monounsaturated fat and omega-6 fatty acids content (Liu et al., 2017). The finding by Klein et al. (2019), based on more

than 100 cooking experiments, confirmed that cooking could result in high emission rates of unsaturated aldehydes, primarily driven by the frying process. Frying vegetables (49%) and meat (37%) produced the highest levels of primary organic compounds. Lu et al. (2024) provided evidence that cooking, particularly through stir-frying and deep frying, has the potential to be a major contributor to PB-ROS formation (Particle-Bound Reactive Oxygen Species) in kitchen environments. Among these methods, stir-frying was found to emit the highest levels of PB-ROS. The emissions discussed, particularly PM, PAHs, VOCs, and SOAs, are crucial to study because they pose significant health risks and contribute to indoor air pollution, especially in cooking environments where exposure is common and potentially hazardous.

2.2. Ultrafine particle (Nanoparticle) translocation in the human body

Inhalable particles (smaller than 100 μm) can penetrate the human body and be deposited in various organs (Wang, 2005). Based on the size, these particles might enter organs via systemic circulation (Allen et al., 2017, Bhargava et al., 2018, Clifford et al., 2018) after alveolar deposition (Guo et al., 2020), or they might reach the brain, bypassing the blood-brain barrier (BBB) or axonal transport along the olfactory nerve (Simko and Mattsson, 2014, Kreyling, 2016, Oberdörster et al., 2009). These particles exhibit a high surface area-to-mass ratio due to their small size, significantly enhancing their potential for translocation and tissue interaction. The extent and speed of this translocation depend on factors like particle size (smaller particles translocate more easily) and surface properties (e.g., charge, coatings with proteins, lipids, and functional groups). This interaction induces oxidative stress and inflammation in extrapulmonary organs (Particles, 2013, Stone et al., 2017). The effects on secondary organs might result from direct particle impact, mediators released at the entry portal and into the bloodstream, a combination of both, or neuronal signals (Heusinkveld et al., 2016) (Figure 2-1).

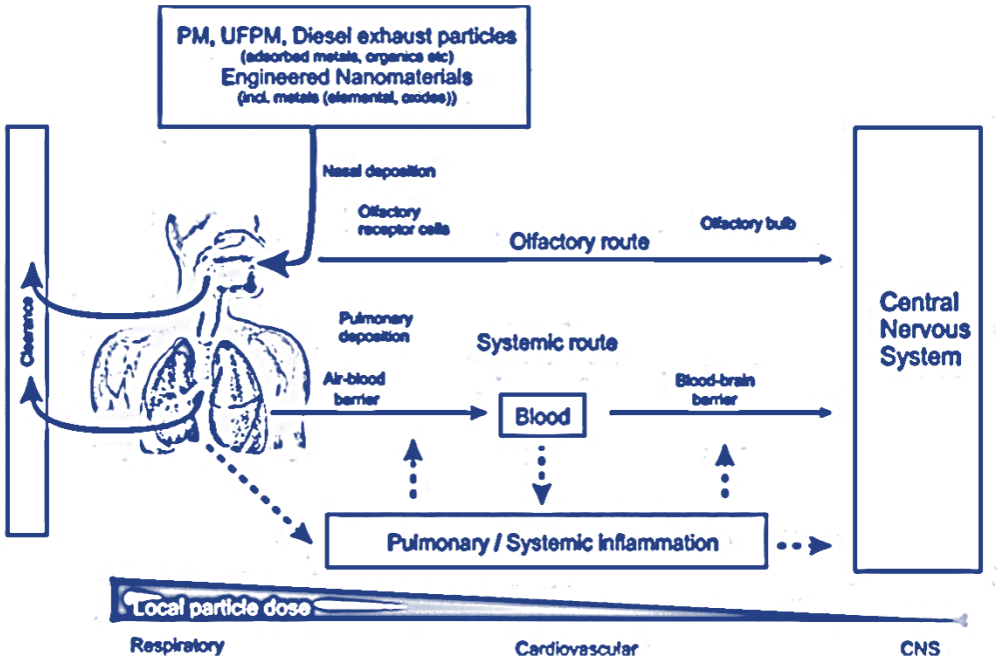


Figure 0-1. Schematic representation of nanoparticles' physiological pathway in the human body. Schematic representation of nanoparticles' physiological pathway in the human body [adapted from (Heusinkveld et al., 2016)].

Research on the toxicity of PM and UFPs in humans is scarce. Researchers investigated indoor air pollution in women using modeling techniques, showing that among five different sizes of PM ($PM_{1.0\ -\ 2.5}$, $PM_{0.50\ -\ 1.0}$, $PM_{0.25\ -\ 0.50}$, $PM_{<\ 0.25}$, and $PM_{2.5}$), the $PM_{2.5}$ fraction showed the highest deposition in the body. The highest amount of $PM_{2.5}$ (55.9%) was deposited in the head, 37.2% in the pulmonary region, and the most minor (6.7% in the tracheobronchial area (Dwivedi et al., 2023). Miller et al. (2017) exposed healthy volunteers to gold nanoparticles (3.8 nm) through acute inhalation. Gold nanoparticles were identified in blood and urine as early as 15 minutes (86%) to 24 hours after exposure and remained detectable for up to three months (Miller et al., 2017).

Researchers using computational fluid dynamics (CFD) estimated that less than 1% of inhaled UFPs reach the human olfactory region. The fraction of UFPs deposited in the olfactory region decreases as the particle size increases (Garcia et al., 2015). Researchers utilizing gamma camera imaging and ultrafine Technetium-99m (^{99m}Tc) labeled carbon particles showed that UFPs entered the human bloodstream within one minute of exposure. The concentration of UFPs peaked between 10 and 20 min and remained elevated for up to 60 min (Nemmar et al., 2002). The results also showed an

increase in UFP levels in the bladder within 45 min of exposure, while UFP levels in the liver stabilized rapidly within 5 min post-exposure. On the contrary, Brown et al. (2002) investigated the deposition and elimination of ultrafine ^{99m}Tc-labeled carbon particles in 10 patients with chronic obstructive pulmonary disease (COPD) and 9 healthy individuals. By measuring particle retention at 2 and 24 hours post-inhalation, they found no evidence of UFP accumulation near the liver and observed no rapid translocation of insoluble carbon UFPs from the lungs into the bloodstream. Similarly, other researchers reported that nanoparticles persisted within the lungs for up to 6 hours following inhalation. These results do not support the hypothesis that carbon nanoparticles (such as Technegas) translocate into the bloodstream after inhalation in healthy individuals. Instead, the small amount of radioactivity detected in the blood was attributed to the leaching of free 99mTc, rather than the particles themselves. This suggests that most inhaled ultrafine particles remain localized within the lungs, and that direct translocation into the bloodstream may be limited or influenced by factors such as particle type, surface chemistry, or lung condition (Mills et al., 2006).

As a result, the inflammatory effects on the human brain and heart during or shortly after exposure to cooking UFPs may vary depending on the size, concentration, dose, and even morphology of the particles. The olfactory function of young adult residents of Mexico City living in extreme air pollution was compared to that of controls from a relatively clean environment. UFPs were found to be deposited in the endothelial cytoplasm and basement membranes of the olfactory bulb (Calderón-Garcidueñas et al., 2010).

2.3. Health Impacts: Epidemiological and Clinical Evidence

2.3.1. Epidemiological studies on cardiopulmonary outcomes

Extensive epidemiological evidence has linked cardiopulmonary health to PM and UFP exposure. UFPs and nitrogen oxides were observed to be significant factors in pediatric asthma cases (Andersen et al., 2008). In another cohort study of Dutch residents, researchers discovered a link between long-term UFP exposure and myocardial infarction, heart failure, and all incidents of CVD (Downward et al., 2018). Similarly, a cohort study among California teachers found statistically significant associations between ischemic heart disease (IHD) and UFPs exposure from vehicle sources, meat cooking, and high-sulfur fuel combustion (Ostro et al., 2015). Lung

cancer has also been associated with $PM_{2.5}$ and black carbon concentration in a city with low pollution levels (Lim et al., 2023). In a cross-sectional study, the investigators explored the relationship between exposure to fine and UFPs in indoor home environments and blood pressure measurements and found that an increase in $PM_{2.5}$ was associated with a 3.2 mmHg increase in DBP (Gibey et al 2023). In another recent investigation, short-term exposures to UFPs were linked to notable increases in blood pressure and arterial stiffness within 24 hours post-exposure. Specifically, each interquartile range increase in UFP concentrations was associated with a 2.10% rise in SBP (95% CI: 0.63% to 3.59%) (Jiang et al., 2024).

A large number of epidemiological studies have firmly established cooking as a risk factor for mortality and cardiorespiratory diseases (Shi et al., 2024, Yu et al., 2023, Ji et al., 2022, Yu et al., 2020, Juntarawijit and Juntarawijit, 2020, Hystad et al., 2019, Alam et al., 2012). Research in rural and urban regions of China examined participants' fuel usage: 44% used clean fuel (gas or electricity), 47% had switched from solid fuel (biomass or coal), and 9% were still using solid fuel. Persistent solid fuel users had a high risk of cardiovascular and respiratory mortality (Yu et al., 2020). However, those who stopped using solid fuel had their excess risk drop by over 60% after five years.

During a follow-up period of 9.4 years, a population-based cohort study was conducted among households in China, India, South Africa, and Tanzania to evaluate the health impacts of different cooking fuels: kerosene, clean fuel, or solid fuel. The results showed that individuals utilizing kerosene as their primary cooking fuel exhibited decreased lung function, as indicated by reductions in Forced Expiratory Volume in one second (FEV1) and Forced Vital Capacity (FVC). Furthermore, kerosene users exhibited a 34% higher prevalence of respiratory symptoms, a 44% increase in dyspnea during regular activities, and a 72% higher incidence of chronic cough or sputum production compared to clean fuel users (Arku et al., 2020). Additionally, an investigation of the relationship between time spent in the kitchen and lung function levels in boys and girls showed a statistically significant decrease in forced expiratory flow at the 75% level among girls compared to boys (Corbo et al., 2001). Meta-analyses indicate that adopting clean cooking fuels reduces the prevalence of hypertension by approximately 16% among rural women. Moreover, systematic reviews indicate that the use of improved cookstoves is associated with significant improvements in both SBP and DBP levels among women (Onakomaiya et

al., 2019). The findings from women who had been using biogas or firewood for at least ten years showed that those using biogas had an SBP 9.8 mmHg lower and a DBP 6.5 mmHg lower (95% CI) compared to those using firewood (Neupane et al., 2015).

Interestingly, while exposure to UFPs was associated with higher blood pressure and impaired microvascular function, no similar associations were observed with $PM_{2.5}$ and PM_{10} (Olsen et al., 2014, Pieters et al., 2015). Likewise, a stronger correlation was observed between mortality rates from IHD and UFPs as compared to $PM_{2.5}$ (Ostro et al., 2015). Specifically, UFPs contributed to over 7% of emergency department visits related to cardiovascular problems, with the most immediate effects seen within two days for particles ranging from 30 to 100 nm (Liu et al., 2013).

Several epidemiological studies and health risk assessments have shown the association between lung cancer and cooking fumes. A cross-sectional analysis explored the link between long-term exposure to UFPs from airports and lung cancer incidence found that UFPs could induce inflammation and oxidative damage, potentially contributing to lung squamous cell carcinoma (HazRat, 1.08; 95% CI, 1.00–1.17) (Bookstein et al., 2024). A randomized controlled trial with asthma patients exposed to cooking particles and filtered air also found that cooking activities could affect airway biomarkers. Specifically, after exposure to cooking fumes, blood concentrations of lipids and lipoproteins increased, and there was evidence of oxidative DNA damage (Laursen et al., 2023).

Studies in Alabama have reported a connection between $PM_{2.5}$ exposure and lower peak expiratory flow rate (PEFR) in high school students (Cage et al., 2023). Research on the impact of various cooking fuels revealed that abnormal PEFR was observed in 43.4% of women using biofuels, 20.5% using kerosene, 23.4% using LPG, and 21.4% using a combination of fuels (Neelam et al., 2013). Additionally, two groups of asthma patients were categorized based on their frequency of grilling: the first group grilled less than once a week, and the second group grilled at least once a week. The results indicated a significantly lower median PEFR of 345.5 L/min in the second group compared to the first group (median, 375.1 L/min) (Lee et al., 2020).

2.3.2. Epidemiological studies on neurological outcomes

Exposure to PM or UFPs and other air pollutant gases has been linked to long-term neurological effects, such as cognitive impairment, a higher likelihood of neurodegenerative diseases, and an increased incidence of brain tumors. A multiethnic

cohort study in Los Angeles aimed to estimate the long-term effects of air pollutant exposure on chronic brain inflammation and microglial cell activation. The results showed that the risk of brain cancer in men increased with exposure to benzene (HazRat = 3.52, 95% CI = 1.55 to 7.55) and PM_{10} (HazRat = 1.80, 95% CI = 1.00 to 3.23), while no such association was found in women (Wu et al., 2020). There is a noted association between exposure to $PM_{2.5}$ and elevated incidence of Parkinson's disease and dementia (Chen et al., 2017b, Chen et al., 2017a, Shin et al., 2018). A large-scale population results revealed a correlation between primary carbonaceous particle emissions and the risk of malignant brain tumors, with the strongest associations observed for tumors linked to a combination of black and organic carbon emissions (Poulsen et al., 2020).

An epidemiological investigation into cancer incidence across 92 counties of Indiana found a correlation between VOC emissions in each county and the occurrence of certain brain and nervous system cancers (Boeglin et al., 2006). Additionally, data from six European cohorts suggested a link between exposure to NO_2, $PM_{2.5}$, and black carbon and the development of CNS tumors (Hvidtfeldt et al., 2023). However, findings from other cohort studies did not strongly support an association between air pollutants and the risk of brain tumors (McKean-Cowdin et al., 2009, Poulsen et al., 2016, Jørgensen et al., 2016). For instance, researchers in a recent study found no link between the spatial distribution of $PM_{2.5}$ magnetite nanoparticles within Toronto and the incidence of brain cancer (Ripley et al., 2024).

Very limited studies exist in the literature investigating the associations between exposure to UFPs and neurological impact. Researchers from Toronto have earlier highlighted the connections between prolonged exposure to UFPs from traffic and aviation sources and malignant brain cancer (Poulsen et al., 2020, Weichenthal et al., 2020). Building on this evidence, Weichental et al. conducted a cohort study across Montreal and Toronto, Canada, involving 1.9 million adults and followed the cohort members for malignant brain tumors. Over the follow-up period, 1400 incidents of brain tumors were identified. The results revealed a positive association between each 10,000/cm^3 increase in UFPs and brain tumor incidence (HazRat = 1.112, 95% CI = 1.042, 1.188) (Weichenthal et al., 2020). Furthermore, a correlation was observed between UFPs originating from airports and the incidence of malignant brain cancer over a 16.4-year monitoring period. The risk of malignant brain cancer among all subjects collectively increased by 12% [95% CI, 0.98–1.27] per interquartile range of

exposure to airport-related UFPs (approximately 6,700 particles/cm^3) (Wu et al., 2021).

2.3.3. Clinical studies on cardiopulmonary outcomes

Acute exposure to indoor emissions resulted in significant short-term impacts on respiratory and cardiovascular health. Biomarkers such as fractional exhaled nitric oxide (FeNO), blood pressure (BP), HR, and PERF are critical tools for assessing these impacts (Zhang et al., 2024). Elevated FeNO levels reflect airway inflammation, which is particularly sensitive to PM exposure, while transient changes in BP indicate cardiovascular stress (Kocot et al., 2020). This section discusses key studies that explore the relationship between indoor emissions, especially cooking aerosols, and these health parameters, emphasizing how emission characteristics influence the severity and nature of the health outcomes.

The two non-invasive biomarkers (PEFR and FeNO) have gained significant attention for evaluating airway inflammation. A peak flow meter measures the speed at which air is expelled from the lungs during forceful exhalation, indicating the clearance of airways (bronchi) in the lungs. FeNO refers to the Nitric Oxide (NO) concentration, measured in parts per billion in exhaled breath. When the airways are inflamed, NO concentration is increased in the lungs to help fight inflammation. Elevated NO levels are commonly associated with inflammatory conditions such as asthma, making FeNO measurement a valuable tool for detecting and managing these conditions (Saito et al., 2014).

Changes in FeNO levels have been widely used as markers of lung inflammation and responsiveness to acute particulate exposure. For example, Strak et al. (2012) observed that an increase in particle number concentration corresponds to an 11% rise in FeNO immediately after exposure and a 12% increase 2 h post-exposure. A 7% increase in FeNO remained until the next morning (Strak et al., 2012). Similarly, cooking-related studies revealed respiratory effects by affecting the FeNO level. Results from a study on 43 women revealed that cooking with electric burners decreased exhaled NO, indicating reduced lung inflammation during acute exposure (Stabile et al., 2015). On the other hand, participants who exclusively used LPG for home cooking exhibited 5.75% higher FeNO levels than those using natural gas (Hou et al., 2015). These findings suggest that emission characteristics significantly

influence respiratory outcomes, aligning with the broader concern of optimizing cooking environments to minimize health risks.

Blood pressure serves as a well-established indicator of cardiovascular health, and has shown transient but measurable increases following exposure to particulate matter emitted from indoor sources (Vasan et al., 2001; Lewington et al., 2003;). For instance, exposure to diesel particles and increased $PM_{2.5}$ has been shown to cause a transient increase in blood pressure, typically by at least 2 mmHg, shortly after exposure to $PM_{2.5}$ with concentrations ranging from 100 to 200 $\mu g/m^3$. However, this elevation in blood pressure generally normalizes within minutes to an hour after the cessation of exposure (Urch et al., 2005, Brook et al., 2018, Brook and Rajagopalan, 2009).

Several clinical studies have also investigated the cooking aerosol effect on blood pressure. For instance, forty-eight participants were exposed to pollutants from five different cook stoves (LPG, gasifier, three-stone fire, forced-draft rocket elbow, and natural-draft rocket elbow) for 2 h in a chamber with air-filtered conditions serving as a control. Their blood pressure was measured immediately, three hours, and 24 hours post-exposure. However, for the 24-hour post-exposure measurements, the participants left the facility and returned for a follow-up. The results indicated no observable differences in diastolic pressure post-exposure. However, 24 h after exposure, mean systolic blood pressure was significantly higher (by up to 3 mm Hg) for all (except for the rocket elbow stove) stoves compared to the control (Fedak et al., 2019). Evan et al. investigated the immediate effects on SBP, 10 minutes post-exposure, following 20-minute sessions of exposure to cooking oil fumes with a targeted peak concentration of 350 $\mu g/m^3$. Compared to a control group exposed to water vapor, all particulate exposures resulted in an increased low-frequency spectral power of SBP (exposure stage × chemical species, $p < 0.001$) (Evans et al., 2015). Likewise, Soppa's research group performed two separate studies (Soppa et al., 2014, Soppa et al., 2017), wherein the healthy participants were exposed to indoor pollution sources (candles burning, toasting bread, frying sausages) for two hours in a chamber. Lung function using FEV1 (forced expiratory volume in 1 second), FVC (forced vital capacity), and MEF 25%–75% (mid-expiratory flow) and blood pressure were monitored prior to exposure, as well as 24 h afterwards. Soppa et al. (2017) found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ during and after exposure (up to 24 hours) corresponded to a 2.2 mmHg rise in SBP, with the largest changes occurring one hour post-exposure.

However, PM generated from frying sausages and candle burning did not consistently affect blood pressure (Soppa et al., 2017). In another earlier study Soppa et al. (2014) linked elevated indoor fine particles from candle burning, toasting bread, and frying sausages with minor decreases in lung function among healthy adults (Soppa et al., 2014). However, the study was subject to uncertainties associated with the study design, such that the participants left the exposure facility after 4 h and came back after 24 h for second measurements, which could potentially cause additional exposure to other sources such as traffic.

Long-term interventions also highlight the potential for mitigating cardiovascular effects. Blood pressure measurements before and one year after implementing an improved cookstove showed a significant drop in average SBP from 114.5 ± 13.0 mmHg to 109.0 ± 10.4 mmHg (p=0.01). These findings collectively underscore the critical need to better understand the short-term effects of cooking aerosols on blood pressure by a controlled study to reduce acute and long-term health risks.

2.3.4. Clinical studies on neurological outcomes

The research examining the clinical effects of UFPs exposure on the human brain is notably scarce, highlighting a significant gap in our understanding of the neurological impacts of such tiny particles. Some clinical studies have demonstrated decreased brain connectivity, as detected by Functional Magnetic Resonance Imaging (fMRI), and an increase in beta band relative power (RP) using Electroencephalogram (EEG) following short-term exposure to diesel UFPs (Gawryluk et al., 2023). These changes in brain connectivity have been associated with reduced working memory (Crüts et al., 2008, Marfella et al., 2024). Recent studies integrates MRI brain analysis and environmental surveys in 141 healthy adults, revealing a positive association between a favorable environment and increased gray matter volume (GMV) and fractional anisotropy (FA) in key brain regions (Kokubun et al., 2025).

While literature on the neurological effects of cooking-generated aerosols is limited, studies on other sources, such as tobacco smoking and diesel exhaust, offer useful insights. For example, changes in fast-wave brain activity (beta2 band) as measured by EEG have been observed following exposure to diesel exhaust compared to sham experiments. This observation suggests that diesel particles may translocate to the brain via the olfactory bulb (Crüts et al., 2008). The primary physiological

effects of tobacco smoking include an increase in alpha (α) and beta (β) frequency bands and a reduction in delta (δ) frequency. In a study, tobacco smokers, following overnight abstinence, smoked their own cigarette, whereas non-smokers performed sham smoking using a placebo cigarette. Following this, α1, δ, and θ frequency amplitudes decreased, whereas α2 and β frequency amplitudes increased. These findings suggest that smokers experience higher arousal levels in brain activity and alertness after smoking compared to partial abstinence (Domino, 2003)[Domino, 2003 #295]. Another research indicated that tobacco smoking also leads to widespread bilateral neocortical increases in dominant alpha EEG frequencies, consistent with nicotine's stimulant effects on the brainstem reticular activating system (Domino et al., 2009).

These findings show the importance of understanding the effects of environmental exposures, such as cooking aerosols, on brain activity. Given the similarities in how UFPs from various sources, including diesel exhaust and tobacco smoke, affect brain function, it is critical to expand research on cooking-related aerosol exposure. Our work aims to fill this gap by investigating how cooking aerosols impact brain health and cognition, providing new insights into the neurological consequences of UFPs exposure from everyday indoor activities.

2.4. Cognitive impairment

To understand the broader implications of cooking emissions on human health, it is essential to examine existing studies on PM exposure and its impacts on cognitive and neurological function. The initial evidence supporting the detrimental neurobehavioral effects of ambient air pollutants in adults has emerged through epidemiological data, signaling a pressing need to understand these impacts more comprehensively (Chen and Schwartz, 2009). Data from the Dutch Lifelines cohort examined the relationship between PM exposure and cognitive decline using linear structural equation modeling. Findings indicated that higher PM exposure was associated with reduced cognitive function (Aretz et al., 2021). The relationship between solid fuel use for cooking and cognitive decline has also been widely studied. Research indicates that switching from solid fuels to cleaner alternatives can reduce cognitive impairment risk (Peng et al., 2024, Yang et al., 2024).

In a study using the Chinese Mini-Mental State Examination (MMSE), participants using biomass fuels had higher cognitive impairment risks, with a hazard

ratio of 1.19 (95% CI: 1.04, 1.37). International studies have shown similar results. Usage of biomass-based cooking fuel and lack of ventilation during cooking also correlated with a higher risk of mild cognitive impairment (MCI) in several countries, including China, Ghana, India, Mexico, Russia, and South Africa (Saenz et al., 2021, Smith et al., 2022).

Despite these findings, clinical studies on cognitive decline and impairment have been poorly investigated. For instance, investigators from a recent study examined the effects of short-term exposure to PM emitted from candle burning under normal and restricted nasal inhalation conditions in 26 adults. Participants were exposed to either high PM levels or clean air, with cognitive assessments conducted before and four hours after exposure. Short-term exposure to candle burning PM has been linked to delayed cognitive effects, particularly in selective attention and emotion recognition (Faherty et al., 2025).

Similarly, Shehab and Pope (2019) assessed cognitive function using various tests after exposure to candle burning and traffic pollutants. Three tests were used to evaluate cognitive performance after exposure to PM from candle burning, including MMSE, Stroop Color and Word, and Ruff 2 & 7. The tests were administered before and one hour after exposure to candle flames in a room, and before and approximately 30 minutes after exposure to traffic pollutants by walking, cycling, or commuting next to a major road. A statistically significant decline in cognitive function was observed in the MMSE test results following candle burning and outdoor commuting exposure. The outdoor commuting experiment also showed a statistically significant decrease in automatic detection speed on the Ruff 2 & 7 test, indicating short-term cognitive decline. However, the Stroop Color and Word test did not show significant differences in either experiment. The consistency in outcomes suggests that PM exposure is likely responsible for the observed short-term cognitive decline (Shehab and Pope, 2019).

The reviewed literature highlights the significant health risks associated with solid-fuel cooking and candle burning. While controlled exposure studies provide vital insights into the pathways of nanoparticle translocation, there is a growing body of toxicological, epidemiological, and clinical evidence linking cooking emissions to cardiopulmonary and neurological health effects. Moreover, existing studies on cognitive impairment related to cooking emissions primarily focus on long-term exposure and solid fuels, with no clinical research specifically addressing the short-term effects of cooking emissions from electric or gas stoves on cognitive function.

This gap in the literature underscores the need for more focused research on the immediate neurological impacts of cooking with electric stoves. No studies have yet explored this specific aspect. Hence, our research aims to fill this gap by investigating the short-term cognitive effects of cooking emissions from electric stoves, contributing to a more comprehensive understanding of the health risks associated with cooking environments.

Chapter 3: Methodology

3.1. Study participants

Study participants were recruited by a recruitment protocol approved by the Nazarbayev University Institutional Research Ethics Committee (IREC) with ethics codes 115/12022019 and 672/19012023. This protocol utilizes advertising through posters and emails on the Nazarbayev University campus. All interested participants received detailed information about the study's goals. They were given a questionnaire on interview day to collect age, height, weight, health conditions, and lifestyle data. The interviews were conducted face-to-face with a team member, making the procedure confidential but not anonymous. Age, sex, race/ethnicity, smoking habits, and medication use were self-reported. Participants excluded from the study included professional chefs, pregnant women, former smokers, individuals with a history of drug addiction, those suffering from cardiovascular, neurodegenerative, or respiratory diseases (such as asthma, bronchitis, or chronic obstructive pulmonary disease), metabolic disorders (including diabetes and thyroid conditions), individuals with known allergies or sensitivities to cooking oils. Additionally, individuals who had undergone recent surgeries or major medical procedures that could impact their cardiovascular or respiratory systems were also excluded.

Individuals were given a comprehensive tour of the study facility and a detailed overview of the study's process, requirements, and expectations. This initiative aimed to familiarize potential participants with the exposure facility, procedures, and researchers to mitigate stress-related reactions during the study. During the tour, the researcher outlined the schedule and length of the exposure sessions, described the types of data to be collected, and emphasized the importance of maintaining participant privacy and confidentiality throughout the study. This strategy aimed to build trust, alleviate anxiety, and create a supportive and cooperative environment, which is vital for the study's success. By addressing potential concerns in advance and providing clear, detailed information, the study team hoped to minimize any hesitations or fears that could lead to participant dropout, thereby ensuring a smooth and efficient study process.

Individuals who appeared to meet the criteria based on the questionnaire were asked to sign the consent form, which explained to them that they were entirely free to leave the interview, discard the form, or withdraw from the research at any stage. A

P100 respirator capable of removing %100 particles 0.02 to 2.9 μm in diameter and beyond was delivered to them, and they were asked to wear it from the home to the experiment's apartment while commuting to avoid exposure to other sources, such as traffic. Each study participant was assigned a code, and test results were labeled with the same code as the interview form, ensuring that the research team had no information regarding the study participants' identities throughout the experimental campaign. They were compensated for their time and travel expenses, which the ethics committee approved. Study participants were asked to refrain from taking unnecessary drugs or dietary supplements that could interfere with the results. Study participants were instructed to avoid attending any events or engaging in activities that could cause stress, anxiety, sleep deprivation, or exposure to air pollution at least one day prior to their scheduled experiment.

3.2. Study protocol

The experimental design of this work was authorized by the Nazarbayev University's Institutional Research Ethics Committee (IREC) with the ethics code 115/12022019 and 672/19012023. The study was divided into two phases: Phase 1, which focused on short-term post-exposure assessments, including 30 minutes post-exposure after cooking (Phase 1A), 2 hours post-exposure following cooking with a gas stove (Phase 1B), and 2 hours post-exposure after cooking with an electric stove (Phase 1C). Phase 2 involved a 24-hour post-exposure phase, which included both standard and intervention conditions. The standard condition was conducted without wearing a respirator, while the intervention condition involved wearing a respirator during both control and cooking days (Table 3-1).

The experiments were conducted in a fully furnished apartment with controlled conditions. All windows and doors were kept closed throughout the experimental study to prevent infiltration of outdoor contaminants into the houses. The study participants were comfortably seated in a living room during the post-cooking period and were encouraged to engage in conversation or reading to avoid monotony. Other indoor sources of PM and VOCs, including vacuuming, detergents, food additives, perfumes, and cosmetic materials, were strictly prohibited during the experiments. Food and beverages were also provided to the participants. There was no ventilation during the cooking for all experiments. During the control studies, participants engaged in the same activities, and health parameters were measured, but no cooking

was performed. In the exposure experiments, cooking was carried out. We aimed to make the conditions as similar as possible between the control and cooking days by scheduling both groups at the same time of day to avoid diurnal effects.

In Phase 2, a randomized controlled crossover design was used to assess the short-term effects of cooking exposure on health outcomes over cycles of two consecutive 24-hour periods. Each cycle consisted of an initial 24-hour period (Day 1), followed by the second (Day 2) where participants were randomly assigned to control or exposure experiments, and the third (Day 3), during which participants left at 9:00 a.m.

Table 0-1. Overview of the experimental phases conducted in this thesis

	Experiments number	**Post-exp time**	**Stove type**	**PM**	**PNC**	**IAQ**	**Oil temp**	**Health outcome**
Phase I	Phase IA	30 min	electric	Yes	Yes	Yes	Yes	BP, HR
	Phase IB	2 h	gas	No	Yes	Yes	Yes	BP, HR
	Phase IC	2 h	electric	Yes	Yes	Yes	Yes	BP, HR
Phase II	Standard	24 h	gas	Yes	Yes	Yes	Yes	BP, HR, ECG, SpO_2, PEFR, FeNo
	Intervention	24 h	electric	Yes	Yes	Yes	Yes	BP, HR, ECG, SpO_2, PEFR, Cognitive test

3.3. Cardiopulmonary outcome measurements

Blood pressure (SBP, DBP) and heart rate (HR) were measured using a blood pressure monitoring device at the brachial artery, approximately 1–2 cm above the medial side of the elbow joint on the upper left arm. Measurements were taken after 5 min of relaxation in a sitting position, with the patient's back and left arm supported and feet flat on the floor. Study participants' arms were positioned at a 90-degree angle during the measurements. All measurements took place under quiet conditions with controlled temperature and humidity. Researchers conducted the measurements following the American Heart Association's "Recommendations for Blood Pressure Measurement in Humans and Experimental Animals" (Pickering et al., 2005).

According to the manufacturer, the blood pressure accuracy of the device was ±3 mmHg or 2% of the reading, and the HR accuracy was ± 5% of the display reading. SBP was classified into the following categories: normal (SBP < 120 mmHg), elevated (120 mmHg ≤ SBP < 130 mmHg), stage 1 high blood pressure (130 mmHg ≤ SBP < 140 mmHg), and stage 2 high blood pressure (SBP ≥ 140 mmHg). DBP was categorized as normal (DBP < 80 mmHg), stage 1 high blood pressure (80 mmHg ≤ DBP < 89 mmHg), and stage 2 high blood pressure (DBP ≥ 90 mmHg) (Whelton, 2017).

During phase II experiments (24 h-post-exposure) study participants' HR was continuously monitored for 48 hours by attaching a portable ECG device (ARES, AthenaDiaX®) to their chests. Lung function was assessed using a peak flow meter (personal best® and Athma MD®) with an accuracy of ± 10% or ± 10 L/min, covering a range of 60-800 L/min. The device measured the maximum airflow during a single flow exhalation, adhering to the guidelines of the American Thoracic Society/European Respiratory Society (Miller et al., 2005). Blood oxygen saturation (SpO_2) levels were measured with a pulse oximeter.

During phase II standard study (with no respirator 24 h-post-exposure) FeNO monitor (NObreath® Bedfont) measures nitric oxide concentrations in the range of 5 – 300 ppb (parts per billion), with an accuracy of ± 5 ppb / ± 10% of the measured value. The breath test duration is 12 seconds. Subjects were instructed not to hold their breath before exhalation; instead, they were advised to take a deep breath and exhale slowly and steadily, as indicated in the equipment usage manual.

For the phase II intervention study, P100 respirators (3M™® particulate respirators, 8293, P100) were used to prevent particle exposure. These respirators are approved for a minimum filtration efficiency of 99.97% against solid and liquid

aerosols, including those containing oil. P100 respirators can effectively filter particles 0.02 to 2.9 μm in diameter and beyond (Eshbaugh et al., 2008).

3.4. Neurocognitive measurements

The cognitive assessment in this study utilized two key tools: the Hopkins Verbal Learning Test-Revised (HVLT-R) and the WAIS-IV Processing Speed Index (PSI). The Hopkins Verbal Learning Test—Revised (Brandt, 2001) is an extensively utilized tool for assessing verbal and visual memory in both clinical and research contexts. HVLT-R is a verbal memory assessment comprising a 12-word list presented over three learning trials. After each trial, they attempted to recall as many words as possible. Participants then complete delayed recall and recognition trials, where original words are mixed with distractors. A delayed recall score is computed by dividing the delayed free recall score by the higher raw score on either learning trial 2 or 3. The Recognition Discrimination Index (RDI) score is calculated by subtracting false positives from hits on recognition trials.

The WAIS-IV's Processing Speed Index (PSI) (Wechsler, 2008b) is a standard score derived from a participant's performance on coding and symbol search subtests. While the Cancellation subtest could also contribute to PSI calculation, only the Coding and Symbol Search subtests will be administered in this study. According to the WAIS-IV Technical and Interpretive Manual (Wechsler, 2008a), PSI measures an individual's ability to quickly and accurately process simple visual information. In the Coding subtest, participants are required to copy symbols from a key that pairs numbers with symbols, completing the task as quickly and accurately as possible within 120 seconds. Similarly, in the Symbol Search subtest, participants must identify symbols that match a given target within an array, again aiming to work as quickly and accurately as possible within the same 120-second time limit. The HVLT-R and WAIS-IV tests were conducted four times, including the 11:30 control day, 9:00, 10:30, and 9:00 last day. The practice, retest, or learning effect describes the tendency for individuals to perform better on a neuropsychological test when reassessed at a later time, even without any intervention (Jutten et al., 2020, Lim et al., 2021, Samaroo et al., 2020). To assess significant shifts in cognitive function while accounting for learning effects from repeated assessments, clinically validated, reliable change indices (Hill, 2019) will be utilized. Additionally, established test-retest reliability

metrics will be applied to minimize the likelihood of Type I errors (Franklin, 2003, Stein et al., 2010).

3.5. Exposure assessment

A condensation particle counter (CPC) (TSI®, 3007) was utilized to measure particle number concentrations. The CPC can detect particles as small as 10 nm and has a maximum measurable concentration of 10^5 particles/cm³. It recorded data at 1-second intervals. Additionally, NanoTracer (Philips Aerasense®, Netherlands) was used to study ultrafine particle concentrations, with a detection limit starting at 20 nm and a maximum concentration limit of 10^6 particles/cm³ as specified by the manufacturer. Particle mass concentrations were measured using the DustTrak (TSI®, DRX 8533) and a low-cost PM sensor equipped with Plantower sensor, which can detect particles down to 100 and 300 nm and can measure concentrations up to 150 mg/m³, logging data every second. Two indoor air quality meters (IAQ) models, including IAQ (TSI®, 7545) and IAQ (Smart Meter®, AZ-7755), were used to monitor the indoor temperature, RH, and CO_2 concentrations at 1-minute logging intervals. Oil and meat (pan kebabs) temperatures were continuously measured at one-minute intervals using a digital thermometer (Fluke®, Everett 54IIB) equipped with a K-type thermocouple probe (ThermoWorks®, THS-103-020).

In addition, during the intervention study, a new Scanning Mobility Particle Sizer (SMPS) (TSI®, 8533) was available and was used to investigate particle number and surface size distributions. The SMPS consisted of a classifier, a nano enhancer, and a condensation particle counter. It was equipped with a nano (short) differential mobility analyzer (DMA) to scan particles ranging from 1 nm to 30 nm. The flow settings for the Short DMA were: Sheath Flow = 20 L/min and Aerosol Flow = 2 L/min. The SMPS recorded data at 90-second intervals, comprising 60 seconds of scan time and 30 seconds of purge time. According to the instrument manual, the maximum total particle concentration detectable by the SMPS was 10^7 particles/cm³ for each size bin in the form of dN/dlogDp. Recordings exceeding this limit were excluded from the experiments. If internal contamination was observed, such as a sharp peak during the installation of a HEPA filter at the start of the experiment (quality control), the SMPS operation was halted, and the SMPS underwent a cleaning procedure. This cleaning effectively reduced internal contamination events and prevented the SMPS from becoming loaded with oily particles. The SMPS was installed in the bedroom, to

prevent distraction to the volunteer, and a connected tube was placed in the living room near where the participants were sitting.

Table 3-2 shows the instruments used in the different parts of phase I and phase II. In the short-term 2 h post-exposure experiments using an electric stove (phase IC), CPC and Nanotracer were used during the experiments. CPC was located next to the stove; however, the Nanotracer was placed in the living room, where participants spent their post-cooking time.

Table 0-2. Instruments used across different experimental phases

Particle characteristics	Phase IA	Phase IB	Phase IC	Phase II-Standard study	Phase II intervention study
Particle number concentrations	CPC	Nanotracer	CPC/ Nanotracer	Nanotracer	CPC/ SMPS
Particle mass concentrations	DustTrak	-	Rizgard	Rizgard	DustTrak
Indoor air quality	IAQ (TSI)	IAQ (TSI)	IAQ (Smart Meter)	IAQ (Smart Meter)	IAQ (TSI)

3.6. Statistical analysis

3.6.1. Phase I Experiments (short-term post-exposure)

The Friedman test is a non-parametric statistical method used to assess differences when the same characteristic is measured on each subject at different times or under various conditions. It is an alternative to the one-way ANOVA with repeated measures and is useful when the normality assumption is violated.

The test considers a null hypothesis (H_0) that the populations represented by the multiple conditions have identical distributions of scores. The Friedman test identifies any overall discrepancies across related means. The alternative hypothesis asserts that the distribution of scores in at least one related population differs from the others. The null hypotheses are as follows:

H_0: The means of the measurements of a condition at all time points remain unchanged and are equal.

H_1: At least the one-time point mean is statistically significantly different.

The associated groups are the subjects before, during, and after cooking, with μ representing the population means.

The Friedman test was necessary because the data were observed to be not normally distributed. In case the Friedman test indicated a statistically significant difference between the populations, the Wilcoxon test was used as a post hoc test to identify the source of these variations.

3.6.2. Phase II Experiments (24 h-post-exposure)

We employed a two-way repeated measures ANOVA to determine the significance of changes between each time point for both exposure and control conditions. This statistical test assesses whether there is a significant interaction effect between two within-subject factors on a continuous dependent variable (Muhammad, 2023, Park et al., 2009). In our study, these within-subject factors are time and exposure. We measured the effects of these factors on various dependent variables, such as blood pressure, HR, SpO_2, and PEFR.

Given that our data did not follow a normal distribution, we utilized the Aligned Rank Transform (ART) method to adapt the two-way repeated measures ANOVA for non-parametric data. The ART method involves transforming the data so that traditional ANOVA techniques can be applied, ensuring the validity and interpretability of the analysis despite non-normality and heteroscedasticity.

Null Hypothesis:

1. Main Effect of Time (Factor A): Time has no significant main effect on the dependent variable. This means that the dependent variable's means are equal across all time points.
2. Main Effect of Exposure (Factor B): No significant main effect of exposure on the dependent variable exists. This means that the dependent variable's means are equal across both exposure and control conditions.
3. Interaction Effect (Time × Exposure): There is no significant interaction effect between time and exposure on the dependent variable. This means that the effect of one factor is consistent across the levels of the other factor.

Procedure for Aligned Rank Transform (ART):

1. Alignment: The data are first aligned to adjust for the main effects and interactions, isolating the effects of interest.
2. Ranking: The aligned data are then ranked.
3. ANOVA: The ranked data are analyzed using the ANOVA.ART function to determine the significance of the main effects and interactions.

If the ANOVA.V indicates significant main effects or interactions, we perform follow-up tests to pinpoint where the differences lie. Specifically, we use the Wilcoxon signed-rank test to compare the exposure and control conditions at each time point (Wobbrock et al., 2011). We applied a **Bonferroni correction** to adjust for multiple comparisons and minimize the risk of Type I errors

3.7. Exposure assessment measurements

To estimate time-integrated exposure over the cooking and decay periods, concentration-time data were analyzed. The total exposure was approximated by dividing the data into discrete time intervals and summing the weighted contributions of each segment. This approach ensures an accurate representation of variations in concentration over time.

$$E = \sum_{i=1}^{n-1} \frac{(C_i + C_{i+1})}{2} \times (t_{i+1} - t_i)$$

Here, E represents the estimated exposure, n is the total number of data points, C_i is the concentration at time t_i, and $t_{i+1} - t_i$ is the time interval between consecutive measurements.

Chapter 4: Health and cooking aerosols: A 30-minute post-exposure study (Phase IA)

4.1. Introduction

Recent epidemiological evidence underscores the relationship between cooking practices and increased cardiovascular risks, particularly hypertension. Studies have demonstrated a dose-response association between hypertension risk and the frequency and duration of cooking, as well as an elevated cooking index, indicating that greater exposure to cooking environments significantly escalates the risk. Additionally, increased hypertension risk is linked to cooking predominantly with either electricity or natural gas (Liu et al., 2022). Similarly, findings from a home indoor monitoring study indicated a positive association between indoor particle number concentration and $PM_{2.5}$ levels with elevated blood pressure (Olsen et al., 2014). The type of fuel used during cooking appears to play a critical role in these health outcomes. Individuals cooking with solid fuels, such as wood or biomass, have been shown to exhibit significantly higher blood pressure levels compared to those using cleaner fuels, like electricity or gas. For instance, SBP was elevated by 1.87 mmHg, and DBP by 0.09 mmHg among solid fuel users (Yu and Zuo, 2022). Furthermore, individuals with the longest exposure to solid fuel experienced a 1.63% increase in SBP and a 1.31% increase in DBP compared to non-users (Yan et al., 2016).

These findings highlight the substantial burden of solid fuel emissions on cardiovascular health, especially in populations with prolonged exposure to cooking aerosols. Despite the wealth of epidemiological studies linking cooking emissions, particularly from solid fuels to chronic blood pressure changes, there remains a significant gap in understanding the short-term physiological effects of cooking aerosols. Specifically, limited research has explored how acute exposure to cooking emissions impacts cardiovascular outcomes such as blood pressure and heart rate. This chapter assesses the immediate cardiovascular effects of cooking aerosols. The objective was to investigate the impact of frying-generated particles on BP and HR following a controlled 30-minute exposure. By examining short-term responses, this research contributes critical insights into the exposure-response relationships.

This chapter focuses on investigating the impact of exposure and post-exposure up to 30 minutes on heart rate and blood pressure. The experiment involved 17

participants, with a 30-minute exposure duration using an electric stove in a kitchen dimension of 21.77 m^3. Health parameters, specifically blood pressure (BP) and heart rate (HR), were measured during the experiment.

4.2. Methodology: Short-term post-exposure (Phase I)

The experiments were conducted in different phases, including short-term post-exposure periods of 30 minutes, with both gas and electric stoves. In the short-term post-exposure studies, study participants arrived at the exposure apartment at the same time of day. After entering, they were asked to remove their mask and rest in the living room for 30 minutes. Background measurements began 30 minutes after participants entered the experimental apartment to allow for adaptation. Then, a researcher conducted cooking for 20 minutes while participants were instructed to remain seated next to the stove. Subsequently, participants relocated to the living room after exposure for further monitoring. For the short-term 30-minute post-exposure study, HR and blood pressure were measured using a blood pressure monitoring device (Omron10®, BP786 N) before cooking, immediately after cooking, and 30 minutes post-cooking

Various parameters during all experiment parts, including PM mass concentrations, particle number concentrations, meat and cooking oil temperatures, indoor air temperature, and relative humidity, were measured. Instruments were strategically positioned, most located above the stove, to gauge participant exposure during the 20-minute cooking period. Three mixing fans operated within the apartment to maintain consistent conditions, ensuring uniform concentrations for all participants.

Seventeen (10 female and 7 male) healthy participants, with an average age of 29 years, participated in this 30-minute post-exposure experiment (Table 4-1). Blood pressure and heart rate of the participants were monitored before exposure to the cooking aerosol, exactly after exposure, and 30 minutes after exposure. In order to analyze the collected data, the Friedman test, a non-parametric statistical method, was employed to identify any differences across the three time points. In case the Friedman test revealed statistically significant results, the Wilcoxon post hoc test was conducted to pinpoint the specific differences.

Table 0-1 . Personal characteristics of 17 study participants involved in Phase IA of the controlled exposure study

Personal characteristic	Measure
Age [years], mean (SD)	29 (8.5)
Female, n (%)	58.8%
Weight [kg], mean (SD)	72.8 (11.1)
Height [cm], mean (SD)	170.1 (5.3)
BMI, mean (SD)	24.5 (3.5)
Baseline blood pressure	
Systolic, mean (SD)	104.5 (9.8)
Diastolic, mean (SD)	68.8 (6.7)
Heart rate, mean (SD)	73.9 (9.5)

4.2.1. Short-term exposure cooking procedure (Phase I)

Hundred g of ground beef was mixed with 20 g of shredded and squeezed onions and 1 g of salt, pepper, and turmeric. Three pan kebabs, each weighing about 40 grams, were formed. A PTFE-coated aluminum pan (25 cm in diameter) was heated on a stove for 2 min before adding oil. Approximately 21 ml of sunflower oil was poured into a pan and heated. Eight minutes after heating, the pan kebabs were added to the pan. A wooden spatula was used to flip the kebab at the 11, 14, and 17-min. The stove was turned off at 20 minutes, but the measurements continued. Clean gloves were worn to prepare the cooking materials before each experiment, ensuring hygienic conditions and no contamination.

4.3. Results: Indoor air quality and health impact

4.3.1. Indoor temperature and relative humidity

Figure 4-1 shows average indoor air temperature and RH variations with time above the pan, along with the shaded area indicating standard deviations. At the start of the experiment, the room temperature was 27 °C. The indoor air temperature displayed three distinct phases throughout the cooking process. During the first 8 minutes, before the pan-kebabs were added, the temperature increased sharply. As the frying started, the temperature rose gradually, peaking around minute 20 when the stove was turned off. Thereafter, temperature plateaued and later showed a slight decline towards the end of the experiment.

The relative humidity followed a slightly different pattern. At the beginning of the experiment, RH was about 32%. Over the first 8 minutes, it steadily dropped to 28.8%, likely due to the increasing air temperature. However, when the pan-kebabs were added at minute 8, the RH rose slightly despite the continued rise in temperature. This increase could be attributed to moisture released from the meat. RH fluctuated during the cooking process, particularly around minute 17 when the meat was flipped for the third time, reaching approximately 31%. By minute 22, the RH had decreased to around 27.5%, corresponding to the ongoing temperature rise. In the final phase, as the stove was turned off and the oil began to cool, the RH started to rise again, reflecting the drop in room temperature.

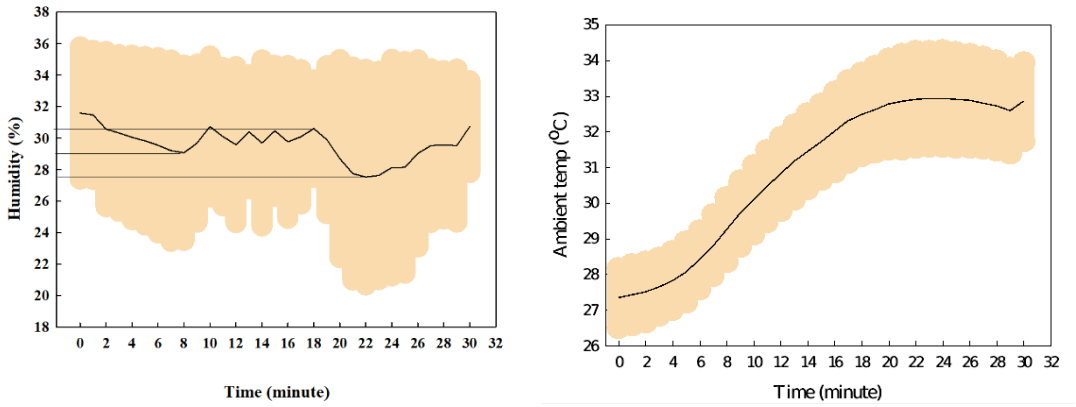


Figure 0-1. Average indoor air temperature and humidity during phase IA. The black line are the average values with the shaded area indicating the range among different experiments.

Figure 4-2 shows the changes in CO_2 concentrations during frying. The measurements began at a background level of approximately 720 ppm. As the cooking process started, the CO_2 levels rose steadily increased, reaching approximately 820 ppm by the time the stove was turned off. This upward trend persisted even after cooking concluded, with the concentration peaking at about 880 ppm toward the end of the measurement period. These observed CO_2 levels align closely with those reported by Amouei Torkmahalleh et al. (2018) during ground beef grilling experiments. Notably, the act of adding or turning the meat during frying did not appear to have a significant effect on CO_2 concentrations. It is worth mentioning that, beyond the frying itself, the presence of people in the room likely contributed to the gradual accumulation of CO_2 throughout the experiment.

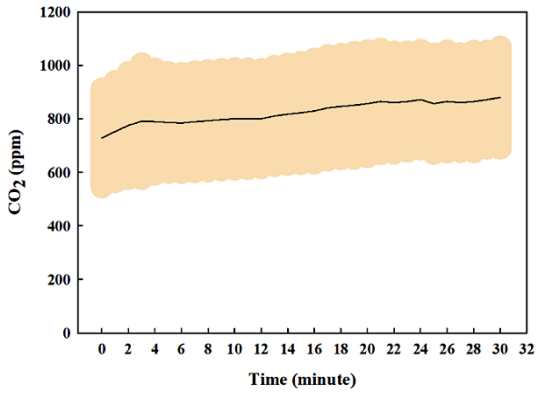


Figure 0-2. Average indoor carbon dioxide (CO_2) concentration measured during Phase IA of the exposure study.

Figure 4-3 shows the average oil and meat temperature changes over time while frying pan-kebabs. The starting point on the graph represents the placing of pan on the stove and turned it on. Once the oil was added, the temperature quickly reached 70 °C as the pan had been preheated for two minutes. The oil's temperature then rose steadily at two different rates. In the first seven minutes, the temperature increased from 70 °C to 140°C. At the minute 8, three pieces of pan-kebabs were added, having been kept at room temperature. This caused a slight dip in the oil temperature and changed the rate of heating. By minute 20, the oil temperature had reached approximately 160 °C when the stove was turned off. The temperature then fell back to around 70°C within 10 minutes.

At minute 8, when the meat was placed in the pan, its surface temperature rose from room temperature (25 °C) to about 52 °C by minute 11, while the oil temperature remained nearly stable. The pan-kebabs were then flipped for the first time, and by minute 14, its temperature had increased to approximately 62 °C. After flipping the pan-kebabs a second time, it continued to heat, reaching around 65 °C by minute 17. The meat was flipped a third time, after which its temperature started to drop, reaching about 45 °C by minute 20, when the stove was turned off. By this time, the pan-kebabs was well-cooked, having absorbed sufficient heat from the oil, achieving thermal equilibrium. However, as the room temperature above the pan was around 30 °C after 16 minutes, the pan-kebabs began to release heat to the surrounding air due to the temperature difference (69 °C vs. 30 °C). This observation aligns with the increase in indoor air temperature above the pan from minute 16 to minute 20. Essentially, the heat transfer from the pan-kebabs to the indoor air caused the meat's temperature to

drop while the air temperature above the pan continued to rise. This significant temperature difference facilitated convective heat transfer from the meat surface to the air above the pan, reducing the meat temperature to about 45 °C within three minutes.

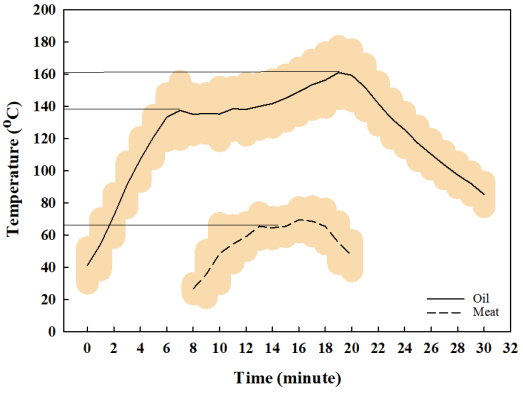


Figure 0-3. Average oil and meat temperatures recorded during cooking activities in Phase IA

4.3.2. Exposure Assessment

Figure 4-4 shows the variations in average $PM_{2.5}$ concentration over time. During the first two minutes of heating the empty pan, there was no increase in PM concentration, aligning with findings by Amouei Torkmahalleh et al. (2018) and Wallace et al. (2004), which suggest that an empty heated pan contributes to particle number concentration but not to particle mass concentration. The PM concentration remained stable until the oil temperature reached 140 °C at minute 6. Between minutes 6 and 8, the $PM_{2.5}$ concentration rose from 0.018 to 0.048 mg/m³. However, the addition of the meat at minute 8 led to decrease in the $PM_{2.5}$ concentration, and it continued to decrease until minute 12, reaching around 0.038 mg/m³. During this time, several factors could have influenced PM concentration, including oil temperature, meat temperature, meat moisture content affecting indoor RH, and the surface area of the oil and meat (Amouei Torkmahalleh et al., 2017a). Typically, PM concentration increases with higher oil and meat temperatures. Despite this, the observed PM concentration decreased between minutes 8 and 12, contrary to expectations given the constant oil temperature and rising meat temperature (Figure 4-3). This discrepancy could be explained by the meat's placement reducing the oil surface area available for

evaporation, as supported by data from (Buonanno et al., 2011) and (Amouei Torkmahalleh et al., 2017a), indicating that oil emits more particles than meat.

From minute 12 to 14, when the meat was flipped for the second time, PM concentration increased. This could be due to absorbed oil by the meat during flipping prevented further PM reductions. Additionally, rising temperatures of the oil and meat contributed to increased particle emissions, with temperatures between 130°C to 139°C for oil and 52°C to 62°C for meat, suggesting no remaining water for evaporation or bubble bursts. The stable RH during this period supports this observation.

During the third flipping (minute 14 to 17) and until the end of the experiment (minute 20), $PM_{2.5}$ concentration steadily decreased from approximately 0.055 mg/m³ to 0.035 mg/m³. By 30 minutes, the PM concentration nearly returned to the background level (0.025 mg/m³). This decline likely resulted from the continuous reduction in oil quantity and surface area as the oil either evaporated or was absorbed by the meat. Additionally, the well-cooked meat surface contributed less to particle emissions. The PM exposure during cooking and decay period was determined to be 1.02 mg/m^3.min.

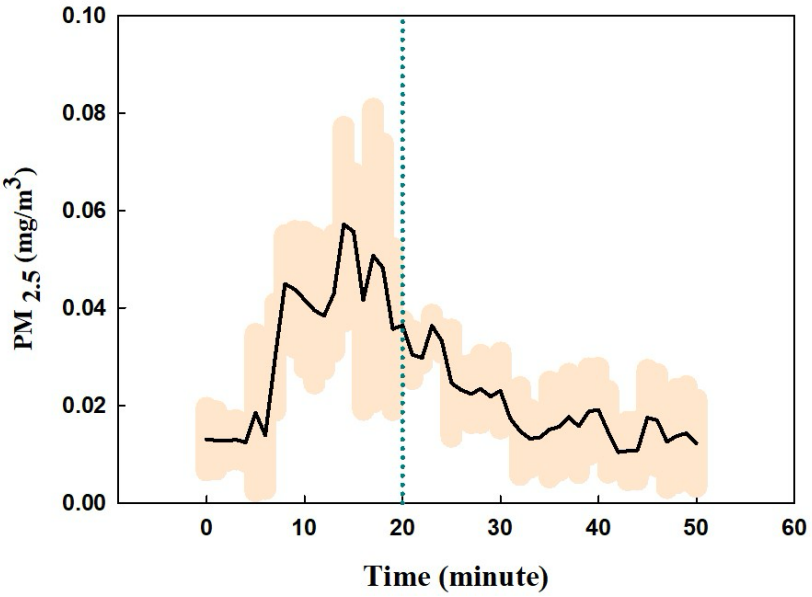


Figure 0-4. Average $PM_{2.5}$ concentration measured during Phase IA of the study

Figure 4-5 shows the average UFPs concentrations over time during the frying of pan-kebabs on an electric stove. The first 20 minutes represent cooking time, while the remaining 30 minutes reflect post-exposure time. Initially, the background UFPs concentration was about 9.0×10^3 particles/cm³. A peak concentration of 7.0×10^4 particles/cm³, occurred two minutes after the end of cooking, i.e., when the stove was turned off. This delayed peak can be attributed to the coagulation of particles smaller than 10 nm (below the detection limit of the CPC), which then grew to larger than 10 nm and become detectable. Background levels were re-established around 50 minutes after cooking. Additionally, the cumulative PNC exposure was determined to be 4.28 $\times$ 10^5 particles/cm³·min.

Literature reviewed by (Abdullahi et al., 2013) shows a wide range of PM and UFPs concentrations, varying from 8×10^{-3} to 1.40 mg/m³ and from 5.7×10^3 to 8.9×10^6 particles/cm³, respectively. Compared to these values, frying pan-kebabs using an electric stove in the current study resulted in relatively low exposure. Variability in concentration values reported in the literature can be influenced by factors such as PM source type, ventilation conditions, house volume, sampling port location, outdoor particle infiltration, and environmental conditions like surface materials, indoor temperature, and humidity.

Previous studies have also shown that frying meat can produce particle number concentrations in the range of 10^5 particles/cm³ (Zhang et al., 2010b, Jørgensen et al., 2013, Dennekamp et al., 2001). For instance, Zhang et al. (2010b) conducted a controlled study to assess the impact of stove type on $PM_{2.5}$ and particle number concentrations during chicken frying. They found that without ventilation and with medium heat, frying chicken on an electric stove resulted in particle number and mass concentrations of 0.35×10^5 particles/cm³ and 10 µg/m³, respectively, over a 28-minute heating duration. Buonanno et al. (2011) reported particle concentrations of 2.8×10^5 particles/cm³ from frying bacon and 2.7×10^4 particles/cm³ from frying pork meat.

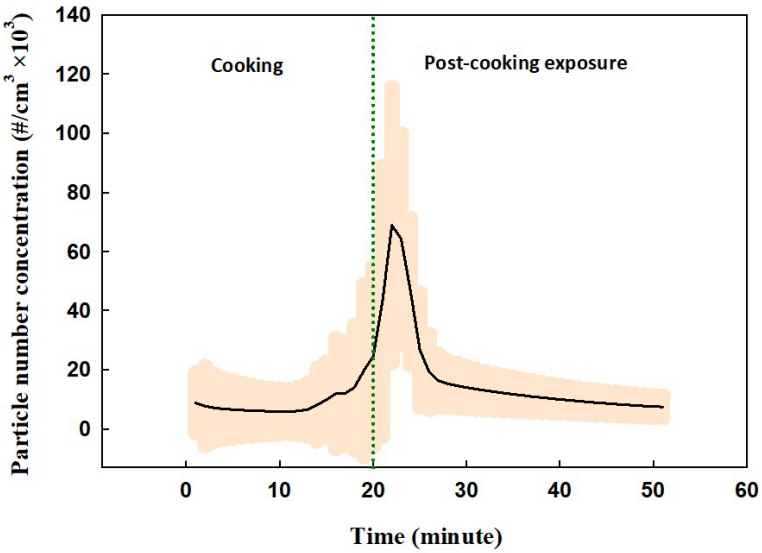


Figure 0-5. Average particle number concentration (measured by CPC) during phase IA

4.3.3. Health outcomes

The mean SBP values before cooking, immediately after cooking and 30 minutes after cooking were 100.2 ± 9.7 mm Hg, 98.2 ± 2.1 mm Hg and 100.0 ± 9.7 mm Hg, respectively. Friedman test analysis showed no statistically significant changes ($p < 0.05$) in the participants' SBP ($p = 0.23$, 95% CI) during any of the three measurement steps. Mean DBP values slowly decreased insignificantly ($p = 0.33$, 95% CI) from 68.8 ± 1.6 mm Hg before cooking to 66.2 ± 1.5 mm Hg 30 minutes after cooking (Figure 4-6). HR showed insignificant increasing trend ($p = 0.15$, 95% CI) from 68.15 ± 6.4 bpm before cooking to 71.3 ± 5.7 bpm exactly after cooking and 73.75 ± 7.2 bpm 30 minutes after cooking.

In conclusion, during our study we did not observe any significant changes in SBP, DBP, or heart rate following 30 minutes of exposure to frying aerosol. This result contrasts with some previous studies that found significant acute effects of particulate matter exposure on blood pressure, particularly in studies where specific sources of emissions, such as toasting bread, were linked to increases in blood pressure (Soppa et al., 2017). A potential reason for the different findings between this study and other studies could be the differences in the particle composition and concentration in the exposed particles emitted from different sources. For example, while Soppa et al. (2017) observed effects for toasting bread emissions, they did not observe any changes for frying sausage. Additionally, differences in findings may be attributed to

limitations in our study, including the small sample size and the lack of a control experiment, which may have affected our ability to detect potential effects.

Toxicological studies, such as those by Unfried et al. (2007), suggest that toxicity increases with decreasing particle size and higher surface area. Additionally, the absence of a response in blood pressure and HR may suggest that the 30-minute exposure duration was insufficient to trigger a noticeable physiological change. It is possible that a longer exposure duration or repeated exposure over several hours to a few days may be necessary to observe significant changes, as seen in epidemiological studies linking cumulative exposure to increased hypertension risk.

Hence, while we did not find significant changes in BP and HR following short-term exposure to frying aerosols, the results are consistent with the complexity observed in the literature regarding the acute effects of particulate exposure. The differences in findings across studies highlight the importance of factors such as particle composition, exposure duration, and individual variability in determining the cardiovascular effects of cooking aerosols. Further research, including studies with longer exposure durations is needed to better understand these acute effects and their potential health implications.

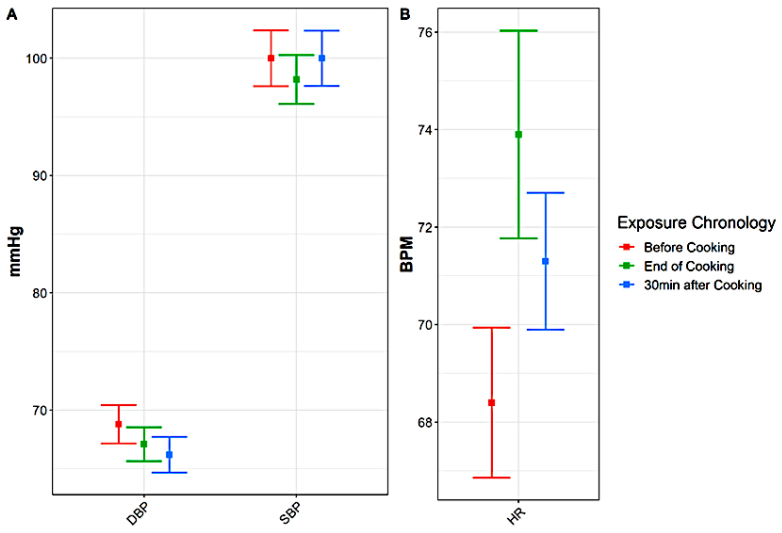


Figure 0-6. Participants' BP and HR during - phase IA (The bars in the figure represent the maximum and minimum values)

4.4. Concluding remarks

This chapter presented analyses of exposure assessments and health impacts during a 30-minute post-exposure period to cooking aerosols generated by an electric stove. The $PM_{2.5}$ concentrations exhibited a dynamic trend, that increased significantly during the initial frying stages and decreased after cooking was completed. This pattern corresponded to variations in oil temperature, interactions with meat, and oil evaporation dynamics, suggesting that oil was a more significant contributor to particle emissions than the meat itself. Additionally, UFP concentrations peaked after cooking, likely due to delayed particle coagulation, which contributed to post-exposure spikes. Blood pressure and heart rate were measured before exposure, immediately after, and 30 minutes post-exposure. The results showed no statistically significant changes in SBP, DBP, or HR, suggesting limited acute cardiovascular effects from short-term exposure to cooking aerosols. Because of the lack of control experiments, the diurnal effects which cause changes in baseline (without exposure) SBP, DBP or HR have not been taken into account in this analysis, which could, in fact, be a confounder. This effect will be separate from exposure effect in health outcome analysis during phase II of this thesis.

In conclusion, while the cooking process using an electric stove caused significant fluctuations in indoor air quality, particularly in $PM_{2.5}$ and UFP concentrations, the simultaneous effects of time (diurnal effect) and exposure on cardiovascular parameters were not assessed in phase IA. These findings highlight the need for further research to investigate the long-term health effects and explore strategies to reduce indoor exposure to cooking emissions.

Based on these observations, it was decided to extend the post-exposure period to 2 hours and incorporate a control group in subsequent experiments. This approach aims to assess the prolonged effects of cooking aerosol exposure on cardiovascular and respiratory health and provide a more comprehensive understanding of the long-term impacts. The addition of a control group will help isolate the effects of the exposure and offer a clearer comparison between exposed and non-exposed participants. Additionally, future experiments will include cooking with both gas and electric stoves to compare the effects of different cooking stoves on indoor air quality and health outcomes.

Chapter 5: Health and cooking aerosol: 2-hour post-exposure study (Phase IB and IC)

5.1. Introduction

Numerous studies have shown that exposure to $PM_{2.5}$ and gases like sulfur dioxide (SO_2) and nitrogen dioxide (NO_2) can lead to increases in blood pressure, potentially elevating the risk of cardiovascular disease. Studies on both human and animal subjects have consistently linked exposure to these pollutants with increased BP, underlining the cardiovascular risks associated with air pollution (Yan et al., 2016, Brook et al., 2010, Urch et al., 2005). For example, research by Brook et al. (2011) demonstrated that even short-term exposure to $PM_{2.5}$ concentrations in non-smoking individuals resulted in a significant increase in SBP, such that a 10 $\mu g/m^3$ rise in $PM_{2.5}$ was associated with a 1.41 mm Hg increase in SBP.

While much of the literature on particulate exposure and blood pressure focuses on outdoor air pollution, indoor cooking emissions, especially from biofuels, have also been shown to contribute to increases in BP. For example, rural Guatemalan women cooking with open wood fires exhibited significant increases in BP and pulse rate (Mann et al., 2013). Similar findings were reported in rural Chinese women exposed to $PM_{2.5}$ from biomass burning, highlighting the cardiovascular impact of cooking-related indoor pollutants (Huang et al., 2011).

To better understand the kinetics of the cardiovascular effects of the exposure we extended the post-exposure period to 120 minutes and introduced a control setup to the study to assess any potential changes in health parameters that could occur independently of cooking aerosol exposure such as diurnal effects. In the control group, participants were exposed to the same environment without cooking aerosols to account for any baseline variations. Furthermore, to explore the effects of electric versus gas stoves on indoor air quality and health, experiments were conducted with both stove types.

Despite the growing body of research, most studies on the effects of cooking fumes have primarily focused on biofuel combustion. Fewer studies have explored the cardiovascular impact of UFPs emitted from cooking with other sources, such as gas and electric stoves. To address this gap, the current work aims to investigate the cardiovascular effects of UFPs from frying, specifically focusing on the variations in blood pressure and HR up to 2 hours post-exposure. While several clinical studies have

monitored BP and HR during and after exposure to cooking fumes (Rizza et al., 2019, Du et al., 2017, Soppa et al., 2017), a significant challenge in previous studies was the uncontrolled exposure to other sources of particulate matter during the post-exposure period, such as traffic emissions or cooking at home. To eliminate this potential confounding factors, our study protocol ensured that participants were not exposed to external sources of PM during the study period, providing more accurate and reliable results. By controlling for these external factors, our study aims to contribute to the growing understanding of how short-term exposure to cooking aerosols impacts cardiovascular responses. This investigation underscores the importance of early detection and intervention to mitigate the long-term health risks associated with exposure to cooking-generated ultrafine particles, with particular emphasis on hypertension.

5.2. Methodology

This chapter focuses on a 2 h post-exposure study (phase IB and IC), which includes both control and exposure experiments involving cooking aerosols (Table 5-1). Upon arrival, participants rested for 30 minutes for adaptation and then stayed in the apartment for a total of 3 hours. Participants were instructed to sit by the stove during cooking. Throughout the post exposure time, study participants remained seated on a sofa in the living room, where they could read or engage in conversation to help maintain their focus and prevent boredom.

Table 0-1 . Overview of the characteristics of the short-term 2-hour post-exposure experiment

Exp.Parts	Study type	Post-exposure time	participants number	Stove type	Kitchen dimension (m^3)	Health parameter measurements
Part I B	Exposure	2h	33	gas	18.75	BP, HR
	Control	2h	10	gas	18.75	BP, HR
Part IC	Exposure	2h	30	electric	21.77	BP, HR
	Control	2h	16	electric	21.77	BP, HR

In the control experiments, participants engaged in the same activities as in the exposure experiments, except the stove was turned off to prevent any emissions. They sat on a sofa in the living room and were allowed to talk or read to avoid monotony.

They were required to sit next to the stove while it was off, as no actual cooking was performed during the control experiments. The purpose of the control group was to establish a baseline for comparison, allowing us to assess any changes in health parameters (e.g., blood pressure, heart rate) without the presence of cooking aerosols. On the other hand, the exposure group was exposed to the cooking aerosols generated by the electric or gas stoves, depending on the experiment, which allowed us to observe the specific health impacts related to cooking emissions. By comparing the two groups, we could isolate the effects of the cooking aerosols from any other environmental factors. However, the control study was conducted by a fraction of the sample used for the exposure study. A more accurate control study could have been taking place when each participant had served as its own control. This type of control study was established in Phase II of the study. The cooking procedure involved frying pan-kebabs were explained in detail in part 4.2.1. No ventilation was used, and the doors and windows remained closed throughout the experiment. Figure 5-1 shows the test room setup for electric stove study.

5.2.1. Methodology of gas stove study (phase IB)

A total of 33 individual healthy adults, aged 18 to 65 years, participated in the cooking study (Table 5-2). Of these, 10 volunteers took part in the control study, while all 33 were involved in the exposure study. For both the control and cooking experiments, SBP, DBP, and HR were measured at six specific time points: prior to cooking, immediately after cooking, and at 30 minutes, 1 hour, 1.5 hours, and 2 hours post-exposure. These measurements were obtained using a blood pressure monitoring device (Omron10®, BP786 N). To measure UFPs concentrations, a NanoTracer (Philips Aerasense®, Netherlands) was employed.

Table 0-2 . Demographic and health-related information of 33 participants enrolled in Phase IB of the study.

Personal characteristic	Measure
Age [years], mean (SD)	38 (14.8)
Female, n (%)	66%
Weight [kg], mean (SD)	68.9 (13.1)
Height [cm], mean (SD)	165.3 (8.0)
BMI, mean (SD)	25.4 (4.5)
Baseline blood pressure	

Systolic, mean (SD)	97.9 (13.5)
Diastolic, mean (SD)	70.8 (9.5)
Heart rate, mean (SD)	81 (6.5)

5.2.2. Methodology of electric stove study (phase IC)

A total of thirty healthy participants, comprising ten men and twenty women with an average age of 27, were selected based on specific criteria for the cooking experiments (Table 5-3). Among them, 16 participants also took part in the control study. Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were measured at five time points: prior to cooking, immediately after cooking, and at 30 minutes, 1.5 hours, and 2 hours post-exposure in both the exposure and control studies. Particle number concentrations near the gas stove were measured using a condensation particle counter (CPC) (TSI®, 3007). Additionally, a NanoTracer (Philips Aerasense®, Netherlands) was used to measure UFP concentrations near the location where the participants were seated.

Table 0-3 .Demographic and health-related information of 30 participants enrolled in Phase IC of the study.

Personal characteristic	**Measure**
Age [years], mean (SD)	27 (8.7)
Female, n (%)	63 %
Weight [kg], mean (SD)	63.6 (11.4)
Height [cm], mean (SD)	168.3 (5.0)
BMI, mean (SD)	25.1(3.5)
Baseline blood pressure	
Systolic, mean (SD)	99.9 (9.1)
Diastolic, mean (SD)	73.1 (7.8)
Heart rate, mean (SD)	76 (4.5)

Figure 0-1. Test Room Setup During Phase IC

5.3. Results: Indoor air quality and health impact – phase IB

5.3.1. Indoor temperature and relative humidity

Figure 5-2 shows the average indoor air temperature and humidity recorded over the 2 h of the experiments. The temperature ranged between 26°C and 28°C, showing a noticeable increase during the cooking period after the stove was turned on, followed by a decrease once cooking was completed. The initial part is similar to those observed in the previous experiments for 30 minutes. Humidity levels remained relatively stable throughout the experiments, fluctuating between 40% and 50%. The CO_2 concentration demonstrated an upward trend, with levels continuing to rise even after the end of the cooking period (Figure 5-3).

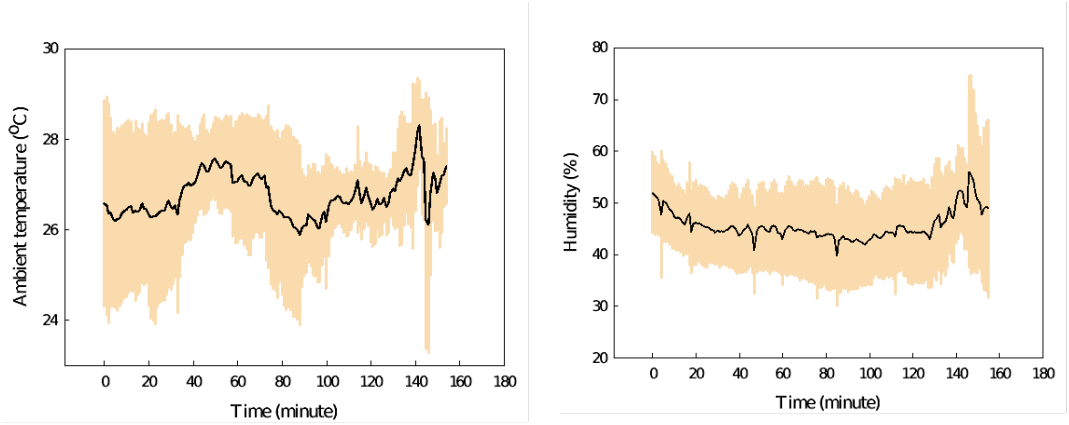


Figure 0-2. Average indoor air temperature and humidity recorded continuously throughout Phase IB.

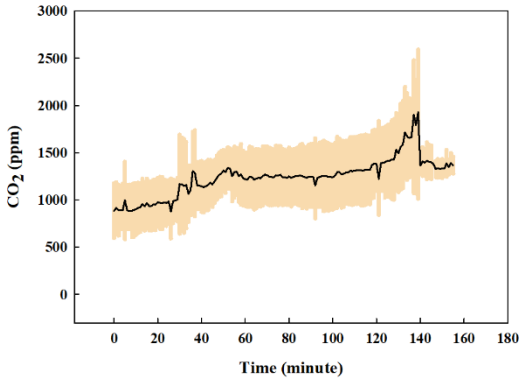


Figure 0-3. Average indoor carbon dioxide (CO_2) concentration (ppm) measured continuously throughout Phase IB

Figure 5-4 shows the average oil and meat temperatures changes over time during cooking. Time zero represents the moment when the pan was placed on the stove, and the gas stove was turned on. Two minutes later, oil was added to the pan. The oil temperature rapidly increased from 40°C to 145 °C within the first eight minutes. However, when the meat was placed in the pan at minute 8, the oil temperature dropped to 110°C by minute 14. This decrease occurred because the meat, being cooler than the oil, absorbed heat from the oil.

A second peak in the oil temperature, reaching 140°C, was recorded at 20 minute due to continuous heating, after which the stove was turned off, leading to a gradual reduction in oil temperature. Meanwhile, the meat's temperature rose to 65°C within three minutes of being added. As the meat was flipped at 11, 14, and 17 minutes, temporary drops in its temperature were observed.

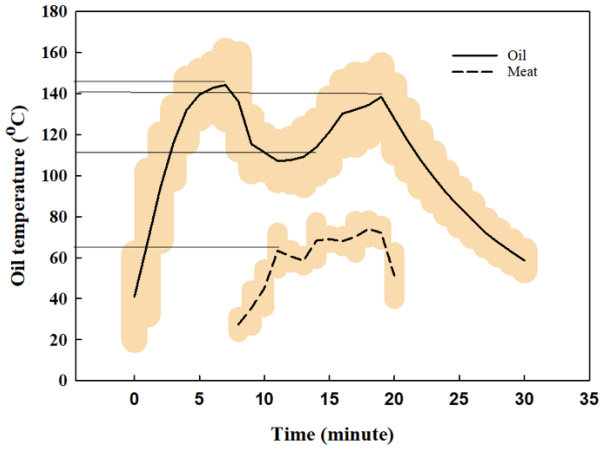


Figure 0-4. Average Oil and Meat Temperature During Frying Using Gas Stove in Phase IB

5.3.2. Exposure Assessment

Figure 5-5 shows the average particle number concentrations over time measured by Nanotracer, highlighting that cooking pan-kebabs generated a substantial amount of UFPs. The initial background particle concentration measured in the study was approximately 1.1×10^4 particles/cm³. The first noticeable peak occurred during cooking at 45 minutes, with a particle concentration of 8.6×10^4 particles/cm³. It further increased, reaching a maximum of 1.15×10^5 particles/cm³ eight minutes after cooking, likely due to particle coagulation. The total PNC exposure was measured at 4.29×10^6 particles/cm³·min. In comparaison, Zhang et al. (2010a) reported an average particle concentration of 6.04×10^5 particles/cm³ at high temperatures while frying chicken on a gas stove. At medium temperatures, the particle concentration dropped to 4.65×10^5 particles/cm³. Similarly, Buonanno et al. (2011) found maximum particle concentrations of 2.7×10^5 particles/cm³ during pork frying and 2.3×10^5 particles/cm³ while frying chips, which aligns with the concentrations observed in the current study.

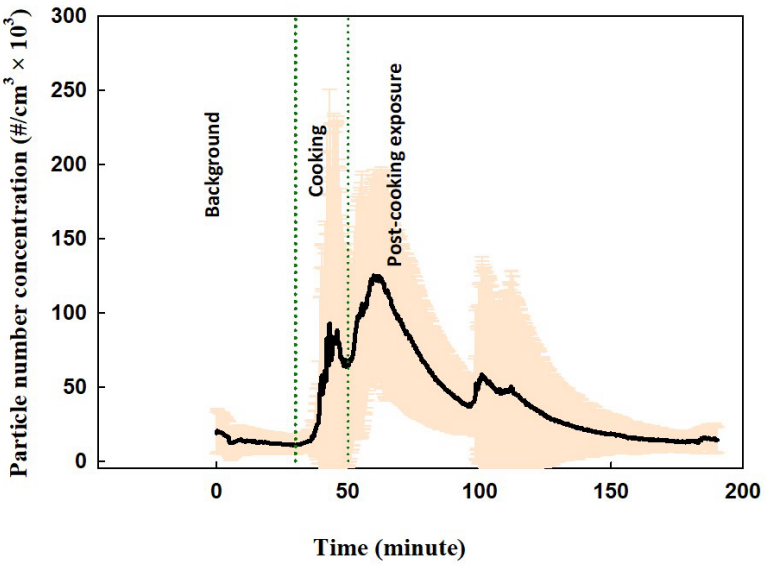


Figure 0-5. Average particle number concentration during phase IB

5.3.3. Health Outcomes

5.3.3.1. Control study

A total of ten participants were invited to take part in the control study. Figure 5-6 shows the average values of SBP, DBP, and HR across the six experimental steps for the control group. During the experiments, mean SBP values were recorded as follows: 108.8 ± 11.82 mm Hg, 115.7 ± 13.13 mm Hg, 114.9 ± 13.73 mm Hg, 121.0 ± 9.79 mm Hg, 112.3 ± 11.88 mm Hg, and 117.3 ± 9.63 mm Hg across the six measurement points. Notably, a statistically significant rise in SBP was detected between before exposure and 60 minutes post-exposure (P = 0.001, 95 % CI), followed by a significant drop between 60 to 90 minutes post-exposure (P = 0.049, 95 % CI). These significant fluctuations suggest a diurnal effect influencing SBP regulation post-exposure. The DBP showed insignificantly increasing trend after 2 h post exposure, with values of 72.0 ± 5.46 mm Hg, 74.8 ± 8.82 mm Hg, 72.4 ± 7.71 mm Hg, 74.4 ± 8.76 mm Hg, 72.3 ± 10.50 mm Hg, and 75.1 ± 6.35 mm Hg at the six BP measurement points, respectively. Heart rate showed an insignificant decreasing trend, with mean values of 49.52 ± 49.61 bpm, 53.11 ± 51.47 bpm, 52.18 ± 50.99 bpm, 53.49 ± 54.48 bpm, 51.74 ± 49.60 bpm, and 52.09 ± 53.78 bpm across the same six measurement points. These findings suggest that diurnal patterns do not influence HR and DBP.

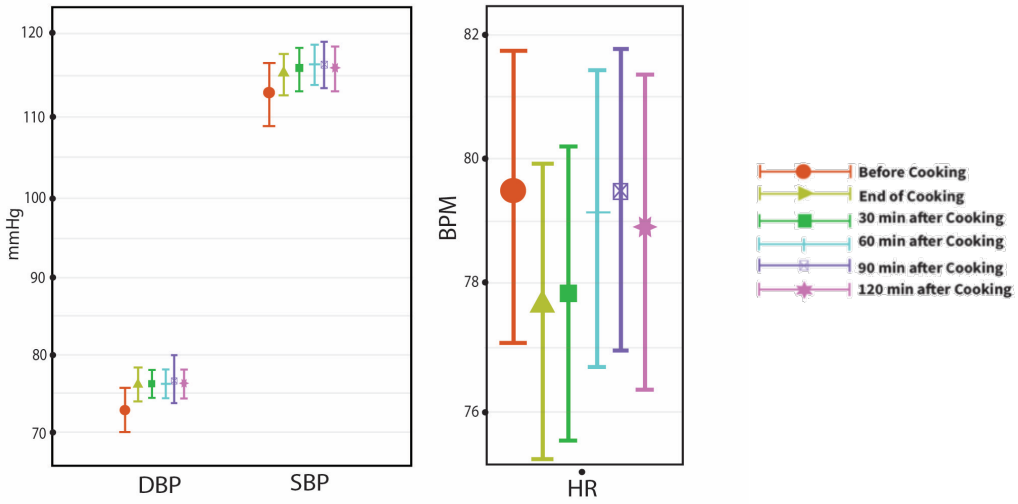


Figure 0-6. Mean values of SBP, DBP, and HR across the six steps of the control experiments in Phase IB, with error bars representing standard deviation

Figure 5-7 shows the average SBP, DBP, and HR for the six stages of the cooking experiments. The SBP measurements were recorded at six time points during the experiments, with mean values of 98.42 ± 25.71 mm Hg, 105.15 ± 14.72 mm Hg, 106.67 ± 15.27 mm Hg, 109.00 ± 13.41 mm Hg, 104.64 ± 23.56 mm Hg, and 108.58 ± 13.28 mm Hg. Statistically significant increases in SBP were noted between steps 1 and 4 ($p = 0.009$, 95% CI), steps 1 and 6 ($p = 0.012$, 95% CI), steps 2 and 4 ($p = 0.018$, 95% CI), and steps 2 and 6 ($p = 0.018$, 95% CI). Comparison with the control study before cooking and 60 minutes after cooking showed that exposure to cooking aerosols had a mitigating effect on SBP after one hour. The significant changes observed immediately after cooking, and at 60 and 120 minutes, as well as before cooking and two hours post-cooking, could be attributed to the effects of particles and gases, as no diurnal influence was detected.

DBP measurements were recorded across six time points, showing an insignificant increasing trend ($p = 0.838$, 95% CI) in mean values of 72.00 ± 9.86 mm Hg, 74.00 ± 7.27 mm Hg, 72.00 ± 9.50 mm Hg, 71.00 ± 9.42 mm Hg, 72.00 ± 11.92 mm Hg, and 81.00 ± 10.07 mm Hg. In contrast, HR showed a statistically insignificant decline ($p = 0.482$, 95% CI) during the post-exposure phase compared to pre-exposure levels (before cooking). Heart rate measurements were recorded at six time points,

with mean values of 83.48 ± 8.99 bpm, 77.61 ± 8.53 bpm, 78.85 ± 8.69 bpm, 77.11 ± 8.53 bpm, 79.10 ± 8.50 bpm, and 74.06 ± 14.94 bpm.

These findings are consistent with previous research reported in the literature. For instance, Cosselman et al. (2012) demonstrated that exposure to diesel exhaust particles led to a significant increase in SBP 2 h post-exposure, while no notable effects on DBP were observed up to 24 h after exposure. However, our assumption is that the diurnal effect is consistent across different populations. Since our control group represented only a fraction of the total exposure group, we did not compare each participant's changes with their own control condition. This assumption may not be entirely accurate, highlighting the need for a more robust experimental design to achieve a better understanding.

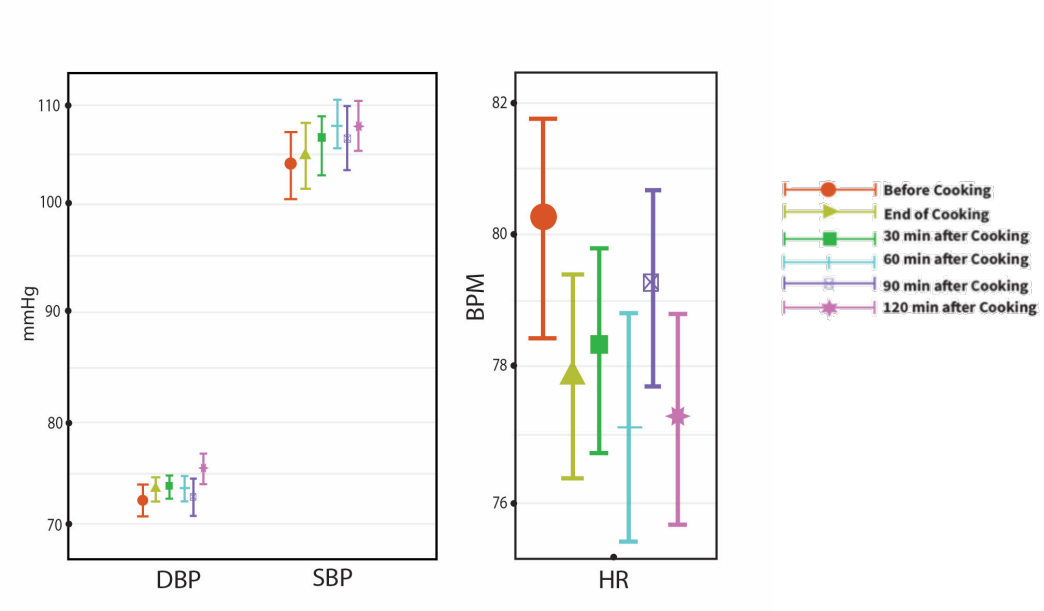


Figure 0-7. Mean values of SBP, DBP, and HR across the six steps of the exposure experiments (Phase IB), with error bars representing standard deviation Exposure study

5.4. Results: Indoor air quality and health impact - phase IC

5.4.1. Indoor temperature and relative humidity

Figure 5-8 shows the changes in average indoor air temperature and relative humidity above the pan during the experiments with electric stove. The starting temperature of the indoor air was approximately 28°C. A rapid increase in temperature was observed during the first 8 minutes before the kebabs were placed in the pan. After that, the temperature continued to rise more gradually throughout the cooking process until the stove was turned off at minute 20. Following this, the temperature began to stabilize, showing a slight decrease towards the end of the experiment. As for the relative humidity, it began at around 28% and increased slightly, reaching approximately 30% by the time the cooking process concluded.

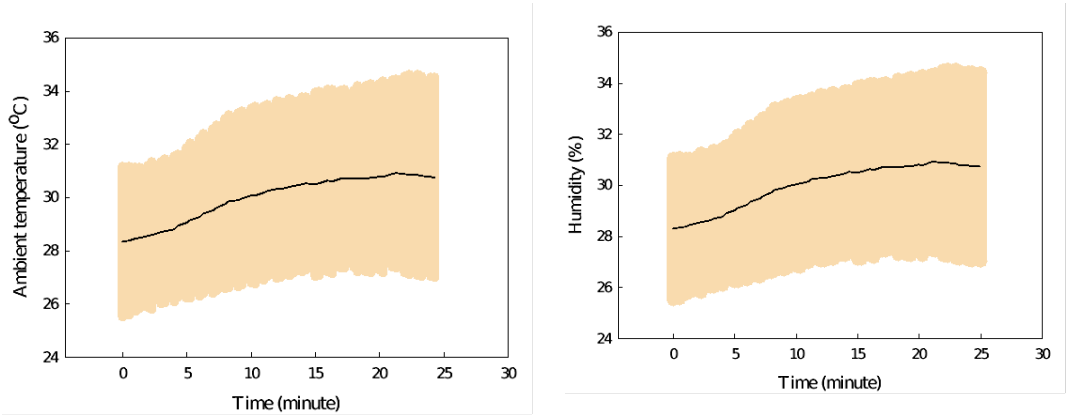


Figure 0-8. Average indoor air temperature and humidity during phase IC

Figure 5-9 shows the average CO_2 concentrations recorded during cooking with an electric stove. Starting at approximately 900 ppm, CO_2 levels gradually increased, peaking at 980 ppm when the stove was turned off. After the stove was switched off, CO_2 concentrations decreased and returned to background levels. The data indicates that the actions of flipping or adding meat had little impact on CO_2 levels. Comparing the CO_2 concentrations across different studies indicates that gas stoves resulted in higher indoor CO_2 concentrations compared to electric stoves. In the 2-hour gas stove cooking session, CO_2 levels peaked at 1,306 ppm after cooking, which is significantly higher than the peaks observed with electric stoves (820 ppm for 30 minutes post-exposure and 980 ppm for 2 hours post-exposure). This suggests that gas stoves emit more CO_2 during cooking, potentially due to the combustion of natural gas.

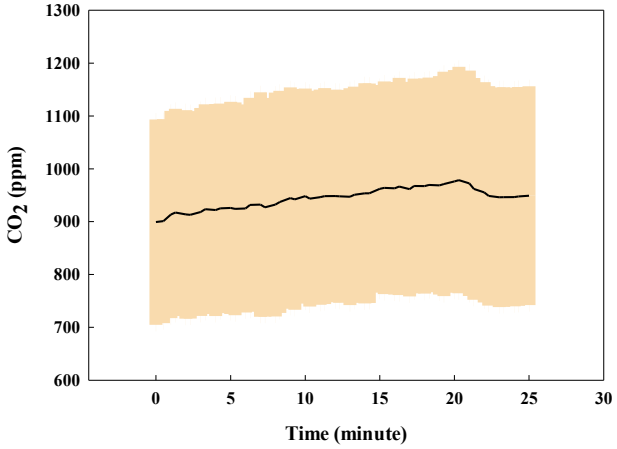


Figure 0-9. Average CO_2 concentration over time during phase IC

Figure 5-10 shows the average oil and meat temperature during the frying the pan kebabs. The first two minutes show the temperature of the empty pan. One minute after adding oil to the preheated pan, the temperature of the oil quickly rose to 70°C. Throughout the cooking process, the oil temperature followed an upward trend, reaching a peak at 145°C when the stove was turned off at minute 20. A brief dip in the oil temperature occurred at minute 8, which coincided with the addition of the pan kebabs, as they had been kept at room temperature prior to cooking. Once the stove was switched off, the oil temperature rapidly decreased. The meat was added to the pan at minute 8, and its temperature rose from 27°C to 67.5°C by minute 17. The temperature drops observed at minutes 11, 15, and 17 correspond to the times when the meat was flipped in the pan.

Both electric and gas stoves exhibited similar patterns in the oil and meat temperature changes, with both showing an initial increase in oil temperature followed by a temporary dip upon adding the meat. This dip occurred as the cooler meat absorbed heat from the oil, temporarily lowering its temperature. After this, the oil temperature steadily increased, reaching its peak towards the end of the cooking time. Similarly, the meat temperature rose gradually, with brief dips observed when the meat was flipped. Despite these fluctuations, the meat was well-cooked in both cooking methods by the end of the process. When designing the cooking process for a study involving gas and electric stoves, it is crucial to set the heating levels in such a way that the oil temperature remains within the ideal cooking range, avoiding the risk of

exceeding the smoke point. This approach ensures both safety and optimal flavor and nutritional quality of the food.

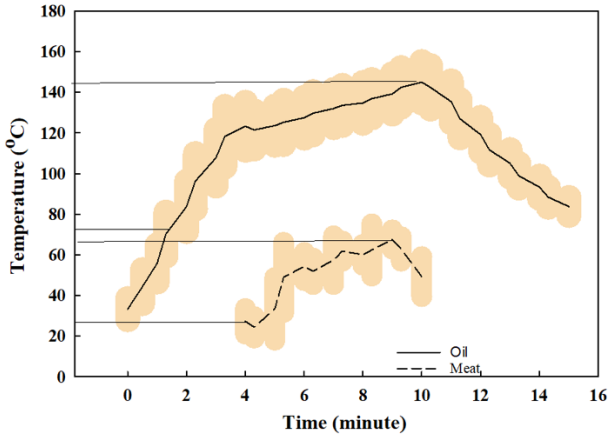


Figure 0-10. Average oil and meat temperature over time during phase IC

5.4.2. Exposure Assessment

Figure 5-11 shows the average $PM_{2.5}$ concentrations recorded during the frying pan kebabs. During the initial two minutes of heating the empty pan, there were no changes in $PM_{2.5}$ levels, aligning with findings from (Amouei Torkmahalleh et al., 2018) and (Wallace et al., 2015). The concentration of $PM_{2.5}$ rose from a background level of approximately 0.013 mg/m³ to a peak of 0.025 mg/m³ by minute 20. The observed fluctuations and decreases in PM levels after minutes 11, 14, and 17 may be associated with the flipping the pan kebabs. Studies by (Buonanno et al., 2011) and (Amouei Torkmahalleh et al., 2017a) indicated that heating cooking oil generates more particles than heating meat. Thus, the fluctuations in PM concentration during the flipping could result from the oil surface being replaced by the meat surface. The stove was turned off at minute 20, leading to a decline in PM concentration back to background levels approximately 10 minutes after cooking. The total PM exposure during the cooking and decay period was estimated to be 0.33 mg/m³·min.

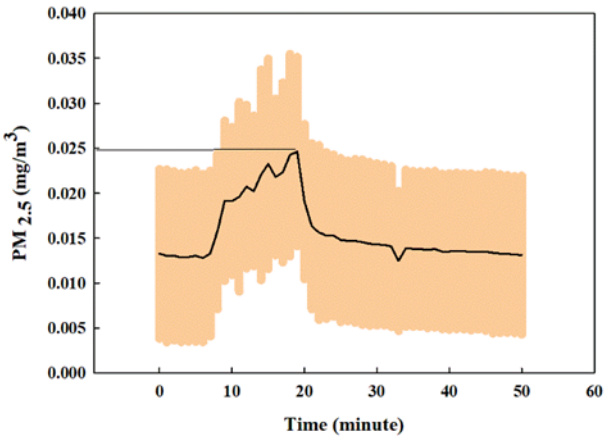


Figure 0-11. Average $PM_{2.5}$ concentration over time during phase IC

Figure 5-12 shows the average particle number concentration measured adjacent to the electric stove and the study participants. During cooking, the particle number concentration measured by CPC rose to a peak of 83 × 10^3 particles/cm³ by minute 20, subsequently declining once the stove was turned off. The total PNC exposure during the cooking and decay period was estimated to be 6.69 × 10^5 particles/cm³·min. The highest particle number concentration recorded by Nanotracer near the participants at the end of cooking was approximately 25.6 × 10^3 particles/cm³. After the cooking period, the average particle number concentration near the participants was about 13.5×10^3 particles/cm³.

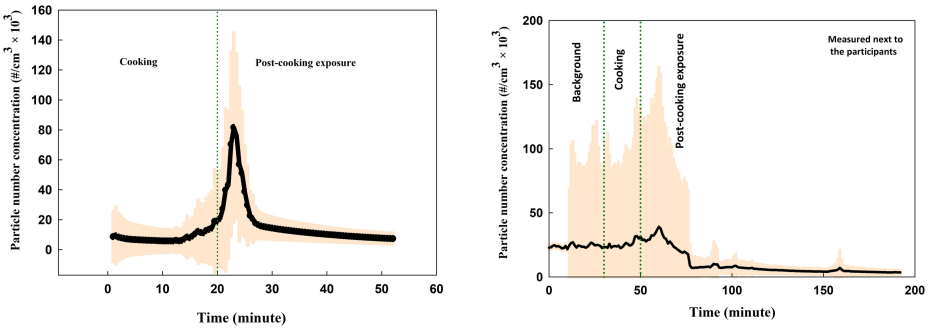


Figure 0-12. Average PNC variation over time during frying, measurements were taken near the stove using a CPC (Left) and during the post-frying, data were collected near the participant with a NanoTracer (Right).

5.4.3. Health Outcomes

5.4.3.1. Control study

Sixteen participants were selected to simulate the cooking and post-cooking procedures: they stood next to an off stove (no emissions) during the cooking period and sat in the living room during the post-cooking period. Statistically insignificant increasing trends were observed for SBP (p = 0.12, 95% CI) and DBP (p = 0.22, 95% CI) after 2 h post exposure. Mean SBP values were 99.9 ± 8.0 mm Hg before cooking, 98.0 ± 5.2 mm Hg immediately after cooking, 101.6 ± 7.5 mm Hg at 30 minutes post-exposure, 100.4 ± 7.8 mm Hg at 60 minutes post-exposure, and 102.5 ± 8.7 mm Hg at 90 minutes post-exposure. The corresponding mean DBP values were 67.6 ± 7.2 mm Hg before cooking, 71.2 ± 6.7 mm Hg immediately after cooking, 66.3 ± 7.3 mm Hg at 30 minutes post-exposure, 69.3 ± 7.7 mm Hg at 60 minutes post-exposure, and 69.5 ± 7.8 mm Hg at 90 minutes post-exposure.

Additionally, no statistically significant differences in HR were observed (p = 0.33, 95% CI), with mean HR values of 74.2 ± 7.2 bpm before cooking, 78.2 ± 8.6 bpm immediately after cooking, 72.1 ± 10.2 bpm at 30 minutes post-exposure, 75.4 ± 11.2 bpm at 60 minutes post-exposure, and 74.3 ± 10.2 bpm at 90 minutes post-exposure (Figure 5-13). These insignificant changes show no diurnal effect on HR, SBP, and DBP.

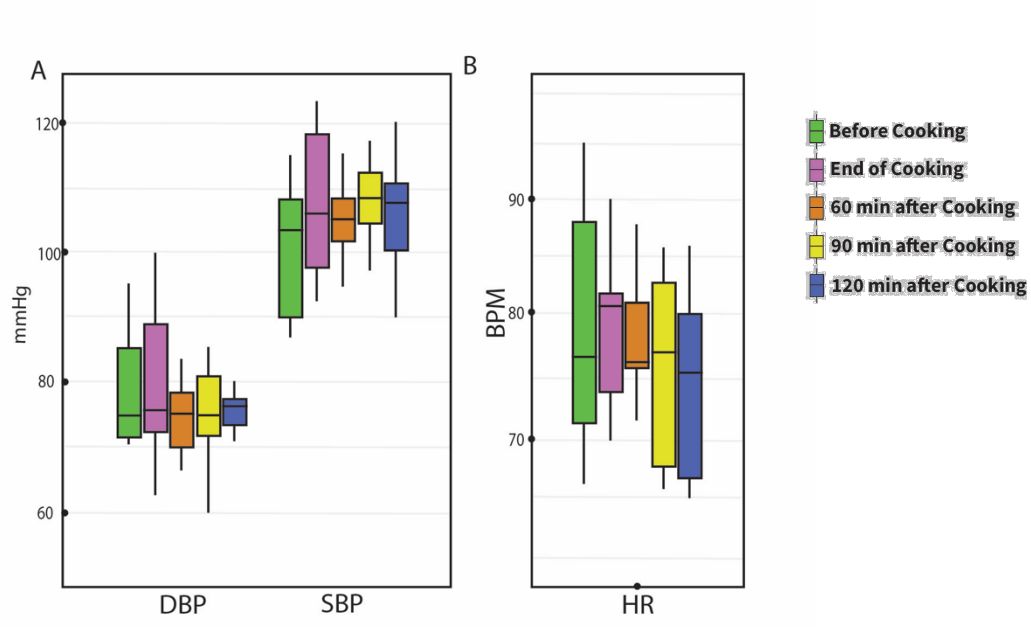


Figure 0-13. Mean BP and HR rate data from 16 subjects in the control experiment, with bars representing the observed range (minimum to maximum values)

5.4.3.2. Exposure study

The outcomes of the cooking experiments showed insignificant changes in mean SBP values (p = 104, 95% CI). The SBP was 105.0 ± 3.2 mm Hg before cooking and increased to 108.0 ± 3.8 mm Hg by the end of the cooking period. One hour after cooking, the SBP dropped to 107.0 ± 3.1 mm Hg, then rose again to 108.0 ± 1.8 mm Hg two hours after cooking. Similar to SBP, DBP changes were also statistically insignificant (p = 0.166, 95% CI). There was an increase in DBP from 77.7 ± 2.7 mm Hg before cooking to 78.8 ± 3.5 mm Hg immediately after cooking. DBP then decreased and stabilized over the subsequent measurements, recorded as 71.8 ± 7.2 mm Hg at step 3 and 71.8 ± 6.5 mm Hg at step 4. The mean DBP value measured two hours after cooking was 78.1 ± 3.7 mm Hg.

The overall statistical analyses revealed significant reductions in HR during the post-cooking period compared to the pre-cooking period. HR values were recorded as 79.7 ± 9.31 bpm at step 1, 79.3 ± 6.20 bpm at step 2, 77.7 ± 6.43 bpm at step 3, 75.5 ± 7.59 bpm at step 4, and 74.5 ± 8.0 bpm at step 5. These reductions became significant at 90 and 120 minutes post-cooking. Significant decreases were observed between the step 1 and step 4 (p = 0.047, 95% CI), step 1 and step 5 (p = 0.003, 95% CI), step 2 and step 5 (p = 0.007, 95% CI), and step 3 and step 5 (p = 0.017, 95% CI) measurements (Figure 5-14).

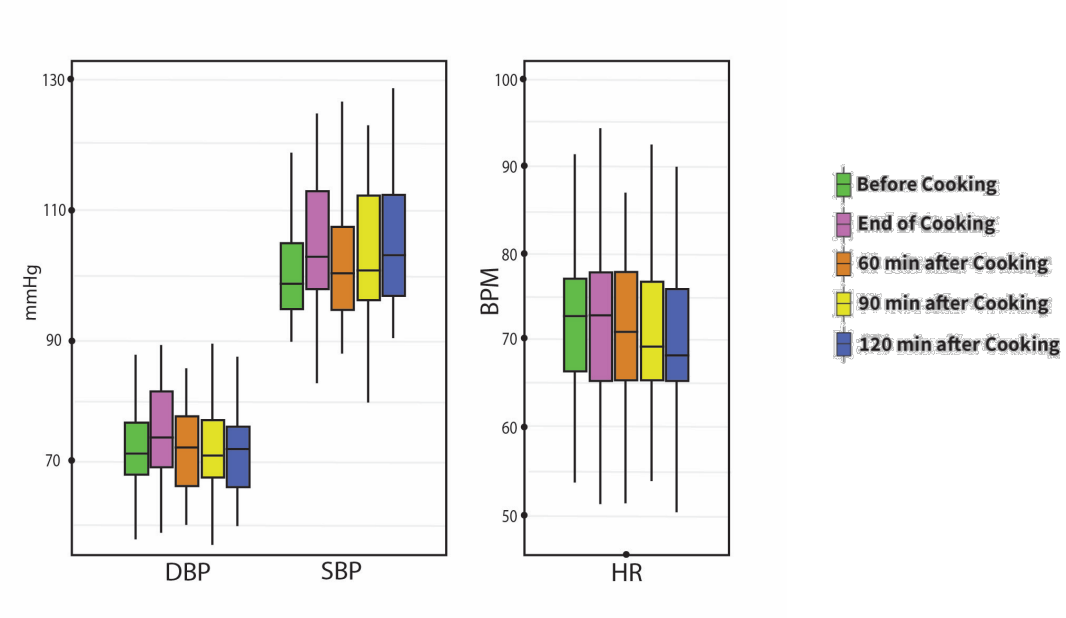


Figure 0-14. Mean BP and HR readings of participants taken before cooking, at the end of cooking, and at 60, 90, and 120 minutes post-cooking – Phase IC.

Cooking involves multiple factors, including exposure to particles, initial anxiety, and heat stress from the stove. Participants might have experienced elevated HR due to anxiety about the unfamiliar experimental conditions, which could persist for 90 to 120 minutes before dropping significantly as they adapted to the environment. Rizza et al. (2019) noted a reduction in heart rate (HR) while sitting compared to standing, though the difference was not statistically significant. This suggests that HR declines during the post-cooking phase, when participants were seated, in contrast to the cooking phase, when they remained standing near the stove. In the control group, where cooking was simulated with the stove turned off, participants likely experienced lower anxiety levels due to their prior familiarity with the experimental setup. In contrast to (Rizza et al., 2019), who observed HR increases following UFP exposure, the present study found HR reductions. These changes may be influenced by factors such as stress, anxiety, and thermal comfort associated with cooking. Additionally, diurnal variations could have played a minor role, though HR changes in the control group were not statistically significant.

5.5. Discussion

In the initial phase of our study, we measured the health effects of frying aerosols by observing participants 30 minutes after cooking. At this time point, no significant changes were detected. However, our study had some limitations, including the lack of a control group and a small sample size, which may have affected the ability to detect subtle health effects. To gain deeper insights, we extended the observation period to 2 hours and included comparative experiments using both gas and electric stoves, along with a control group. This adjustment aimed to capture delayed health responses and assess potential differences in the impacts of aerosols generated by these two cooking methods. All three cooking sessions led to changes in SBP, DBP, and HR, but the magnitude and significance of these changes varied. Phase IB (2-hour gas stove study) and phase IC (2-hour electric stove study) showed noticeable changes in SBP and HR, with the gas stove showing increased SBP during the cooking period and the electric stove showing a decrease in HR after cooking.

The exact mechanisms responsible for the observed cardiovascular effects are not yet fully understood. While systemic inflammation has been suggested as a potential factor, multiple clinical studies have found no detectable signs of systemic inflammation in human blood samples following exposure to diesel emissions (Cliff et al., 2016) or cooking fumes (Svedahl et al., 2013, Pedata et al., 2016). Similarly, research on particle translocation during short-term exposure (Brown et al., 2002, Mills et al., 2006, Wiebert et al., 2006) has found no evidence of particles entering the bloodstream. Although animal studies (Husain et al., 2015) have shown that nanoparticles can move into circulation, direct proof of this process occurring in humans during short-term exposure remains unavailable.

Chronic exposure to particulate matter has been associated with particle presence on the fetal side of the human placenta (Bové et al., 2019) and in the urine of healthy children (Saenen et al., 2017). However, there is no evidence that cooking-related particles enter the bloodstream. This could be attributed to their aggregated structures or rectangular morphologies, as observed in prior studies (Buonanno et al., 2009, Li et al., 2019). Predominant structures, such as aggregated branched chain-like forms from frying on gas and electric stoves (Buonanno et al., 2009), may impact their ability to penetrate the bloodstream during short-term, low-level exposure.

Cardiovascular changes may also result from systemic inflammation and activation of the autonomic nervous system (Brook and Rajagopalan, 2009, Langrish et al., 2009). Several clinical investigations into the effects of cooking fumes (Widdicombe and Lee, 2001, Baumgartner et al., 2018) and diesel engine particles (Zheng et al., 2012) reported no evidence of systemic inflammation in blood samples from study participants. It is important to note that SBP and DBP are regulated by different pathways, which leads to their distinct responses to external stimuli (Pieters et al., 2015). Some research has indicated that SBP can undergo more significant fluctuations than DBP due to increased arterial stiffness following exposure to PM (Baumgartner et al., 2018, Fedak et al., 2019). SBP, which is the pressure generated when the heart contracts and pumps blood through arteries, is influenced by the sympathetic nervous system and stress responses (Du et al., 2017). In contrast, DBP reflects the pressure in the vessels when the heart is at rest (Du et al., 2017). Inhaled particles may reach the brain via olfactory pathways, stimulating the nervous system (Crüts et al., 2008, Dorman et al., 2004). Therefore, the changes in SBP may stem from sympathetic activity linked to nervous system stimulation (Langrish et al., 2009).

The observed rise in SBP may be influenced by both particulate matter and gaseous pollutants, potentially through olfactory pathways. Previous studies have indicated a link between sensory perception and blood pressure regulation. Notably, self-reported changes in taste and smell have been associated with significant SBP elevations over a two-year period, with an adjusted mean difference of 5.1 mmHg (95% CI: 0.1 to 10.0, $p = 0.04$) compared to individuals without sensory alterations. However, no significant association was found when taste and smell changes were analyzed separately (Liu et al., 2018). Thus, the effects may arise from both gases and particles. Blood pressure naturally fluctuates due to numerous internal and external factors, with behavioral influences significantly affecting daily variations. We meticulously controlled various factors, including food intake, sodium consumption, and habits like drinking, smoking, and caffeine consumption, throughout the experiments. The changes in SBP observed during both control and cooking scenarios could be attributed to cooking aerosols and diurnal effects, as blood pressure typically rises in the morning and decreases at night, although variations occur throughout the day and night (Kawano, 2011). One analysis showed that the time of day accounted for 33% of the observed blood pressure variability (Clark et al., 1987). These findings highlight the complexity of the cardiovascular effects of particulate exposure and the need for

further research to explore the influence of exposure duration, particle composition, and individual variability on blood pressure and heart.

5.6. Concluding remarks

This chapter provided a comprehensive analysis of exposure assessments and health impacts associated with a 2-hour post-exposure period to cooking aerosols generated by electric and gas stoves. Cardiovascular parameters, including blood pressure and heart rate, were monitored at multiple time points: before exposure, immediately after cooking, and 30, 60, 90, and 120 minutes post-exposure. The findings revealed statistically insignificant increasing trends in DBP for both electric and gas stove studies (phase IB and IC). Decreasing trends in HR and increasing trends in SBP were also observed across both experiments. Significant changes in HR during the electric stove study and in SBP during the gas stove study could be attributed to exposure effects, assuming that the diurnal effect is consistent across different populations. One of the key limitations of our study was that the control group did not include all participants from the exposure group and had a smaller sample size. As a result, we were unable to compare each participant's physiological responses with their own control day at the same time points, making it difficult to completely eliminate the influence of diurnal variation. In the Phase IB and IC experiments, we assumed that the diurnal effect would be consistent across different populations and sample sizes. However, to accurately assess the potential confounding impact of diurnal variation, a more robust experimental design is required. To address this limitation, we refined our study design in the next chapter and Phase II experiments to better account for diurnal influences and improve the reliability of our findings.

Chapter 6: Disentangling Cardiovascular Effects of Cooking Emissions: Particles or gases: 24 h post-exposure study

6.1. Introduction

Indoor air quality (IAQ) significantly impacts human health, as individuals spend an estimated 90% of their time indoors. Epidemiological studies have highlighted the toxicological impacts of UFPs on lung health, including oxidative stress, respiratory tract deposition, inflammation, and cell apoptosis. Cooking emissions, particularly from oil fumes, have been identified as a major indoor source of UFPs and other harmful pollutants, contributing to respiratory and cardiovascular conditions such as asthma, hypertension, and reduced lung function. Liu et al. (2022) investigated the relationship between cooking practices and hypertension in China, focusing on natural gas and electric stove use. Their findings revealed a significant association between increased cooking frequency and duration with a heightened risk of developing hypertension. Hypertension is widely recognized as a major contributor to morbidity and mortality related to cardiovascular diseases (Brook et al., 2010, Guo et al., 2010, Brook et al., 2011). Additionally, evidence from systematic reviews and meta-analyses highlights a strong link between $PM_{2.5}$ exposure and elevated blood pressure. For instance, a 10 μg/m³ increase in $PM_{2.5}$ concentration was found to raise SBP by 1.39 mmHg and DBP by 0.89 mmHg (Liang et al., 2014). Recent studies have corroborated these findings, reporting that the increase in $PM_{2.5}$ leads to a 1.76 mmHg rise in SBP and a 0.31 mmHg increase in DBP, with an associated 15% higher risk of hypertension (Niu et al., 2023). Research conducted in Austria showed that an interquartile range increase in $PM_{2.5}$ was linked to a 3.2 mmHg elevation in DBP (Gilbey et al., 2023). These studies collectively emphasize the adverse cardiovascular impacts of air pollution, particularly in indoor settings influenced by cooking activities.

Previous studies have primarily explored the epidemiological and short-term clinical impacts of cooking aerosols from solid fuels. However, research on subclinical cardiopulmonary parameters, such as HR and peak expiratory flow rate (PEFR), linked to indoor exposure to UFPs from gas and electric stoves remains limited. Most investigations have focused on solid fuels and PM, overlooking the effects of UFPs. Moreover, no controlled human exposure studies, to our knowledge, have restricted participants' exposure to other sources for 24 hours. We aimed to assess the short-term cardiopulmonary effects of indoor cooking emissions from gas and electric stoves up

to 24 hours post-exposure. It also evaluated the influence of particles and gases through intervention methods in a controlled human exposure setting, addressing an unexplored area.

6.2. Methodology

A randomized controlled crossover trial with two groups will be employed to assess the short-term effects of cooking exposure on health outcomes across two sequences of three consecutive 24-hour periods. The study is divided into two parts: standard and intervention methods. Table 6-1 outlines the characteristics of the 24-hour post-exposure experiments and the health parameters measured during both standard and intervention studies.

Table 0-1 .Experimental design characteristics of Phase II studies

Experiments part	Study participants number	Stove type	Kitchen dimension (m^3)	Wearing respirator	Health parameter measurements
Standard method	30	gas	18.75	No	BP, HR, ECG, Blood oxygen, Peak flow, FeNo
Intervention method	30	electric	21.77	Yes	BP, HR, ECG, Blood oxygen, Peak flow, Cognitive test

The power analysis was conducted by a biostatistician from the Biostatistics Core at the Center for Clinical and Translational Science (CCTS) at the University of Illinois at Chicago (UIC) to design the study and determine the appropriate sample size. The analysis recommended that 20 participants would provide at least 90% power to detect time-by-treatment interaction effects for brain experiments. However, for heart and lung measurements approximately 60 subjects. For cognitive testing, we conducted cognitive assessments with 30 volunteers. For cardiovascular measurements, while the initial plan was to recruit 69 participants, due to resource and time constraints, we modified the study design to include two groups—one for the standard condition and one for the intervention study—to ensure feasibility while maintaining statistical validity.

A total of 69 participants took part in the study, divided into two groups: the standard study group, which included 19 males and 20 females aged between 18 and 75; and the intervention study group, which consisted of 21 males and 9 females aged

between 18 and 45 (Table 6-2). Study participants stayed in the apartment for 48 consecutive hours for both standard and intervention methods. One of the 24-hour periods was a control day (without cooking), and the other was a cooking day. Participants entered the apartment at 08:00 a.m., and stayed in the living room for a one-hour adaptation. At 09:00 a.m., a portable electrocardiogram (ECG) was attached to each volunteer's chest to monitor heart activity. Health parameters were measured, including SBP, DBP, blood oxygen saturation (SpO_2), and PEFR, 21 times over 48 hours. FeNO was also measured during the standard test part. The first measurement was taken one hour after arrival (09:00 a.m.) and continued at intervals throughout the day (10:00, 10:30, 11:00, 11:30, 12:00, 14:00, 16:00, 18:00, and 20:00). The final measurement occurred at 09:00 a.m., on the third day, 49 hours after arrival. Participants remained in the living room throughout the test, including the cooking period. Like the earlier experiments, several parameters including PM mass concentrations, particle number concentrations, cooking oil temperature, indoor air temperature, and relative humidity were monitored throughout the duration. Instruments were placed in the living room near where study participants sat during the experiments. Three mixing fans operated within the apartment to maintain consistent conditions and ensure uniform concentrations for all participants.

Table 0-2 . Characteristics of participants involved in the standard and intervention parts of the Phase II study

participants Characteristics	**Standard study**	**Intervention study**
Number of participants	39	30
Male (%)	51%	73%
Age, y	43.3 (13.9)	21.5 (5.8)
BMI	23.11 (2.3)	22.28 (3.0)
SBP, mm Hg	110 (13.5)	102 (10.6)
DBP, mm Hg	71 (9.3)	67.5 (8.5)
HR, bpm	70 (6.3)	68.8 (10.5)
Exposure characteristic		
Kitchen dimension, m^3	18.75	21.77

6.2.1. Intervention experiments with masks

In order to deconvolute the effect of cooking emission gases from the particles, interventions were carried out. The same procedure was conducted for the intervention

method as the standard method. However, during the intervention method study, On both the cooking and control days, participants were required to wear a P100 respirator starting at 09:25 and continue wearing it until the particle concentration levels stabilized back to baseline, which generally occurred around 15:00. Figure 6-1 shows the cooking setup during the standard and intervention studies.

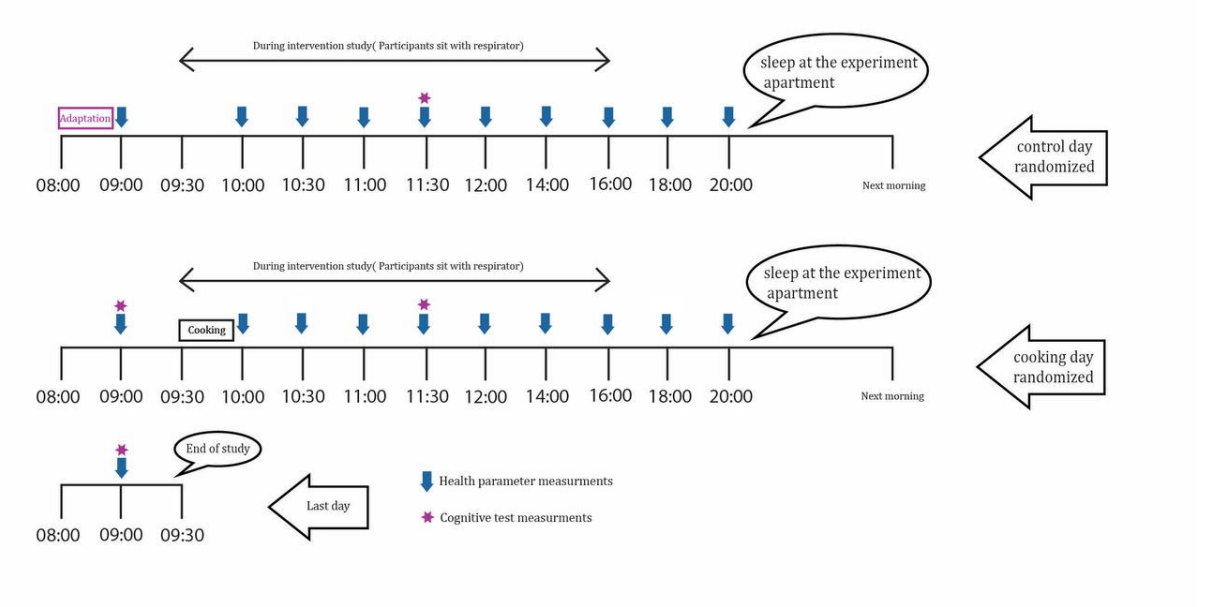


Figure 0-1. Study design of phase II experiments

6.2.2. Cooking Procedure

Cooking was conducted at 09:30 a.m., on the cooking day for 20 minutes. For the cooking, 45 g of chicken and 100 g of potatoes were fried in 30 ml and 50 ml of sunflower oil, respectively. Two PTFE-coated aluminum pans (25 cm in diameter) were heated on a stove for 2 minutes before adding the chicken and potatoes (Figure 6-2). After 10 minutes, the chicken was flipped, and the potatoes were stirred with a wooden spatula. Like earlier experiments, the stove was turned off after 20 minutes of cooking and doors and windows remained closed. A researcher conducted cooking with gas or electric stoves for standard and intervention studies, respectively.

Figure 0-2. Cooking set up during standard (left) and intervention (right) studies – Phase II

6.2.3. Heart rate variability measurement

Study participants' HR were continuously monitored for 48 hours using a portable three-lead Holter monitor (ECG, ARES, AthenaDiaX®) attached to their chests. To ensure optimal electrode contact, participants' skin was prepared by shaving if necessary, cleaning, and applying slight abrasion. The 24-hour ECG recordings were analyzed using Kubios HRV Scientific 4.1.2 software. This software includes noise detection features that automatically identify noisy segments from raw ECG data and interbeat interval data (RR or pulse-to-pulse intervals). The noise detection sensitivity was set to the default "Medium" level, which excludes all segments marked as noise from HRV analysis. Individual intermittent abnormal beat intervals (e.g., ectopic beats) were not typically classified as noise, as they can be reliably corrected through the software's automatic beat correction feature, performed after noise detection by default.

The time-domain analysis assessed variations in HR over time by evaluating the intervals between consecutive normal cardiac cycles. From the continuous ECG recordings, each QRS complex was identified, and the normal RR intervals (N-N intervals), representing sinus depolarization and instantaneous heart rate, were calculated. The time-domain metrics used to analyze HRV included mean ± standard deviation (SD) of the standard deviation of N-N intervals (SDNN), the square root of the mean of the sum of the squares of differences between adjacent N-N intervals (RMSSD), and the proportion of interval differences between successive N-N intervals exceeding 50 ms (pNN50).

Frequency-domain analysis employed power spectral density estimation, using autoregressive (AR) methods. For studies focusing on controlled exposures with long-term HRV analysis (e.g., 24-hour segments), AR methods offer improved reliability. Individual RR intervals were transformed into frequency bands with distinct spectral characteristics. Parameters derived from this analysis included low-frequency (LF) power (0.04 – 0.15 Hz), reflecting both sympathetic and parasympathetic modulation, and high-frequency (HF) power (0.15 – 0.40 Hz), representing parasympathetic activity.

6.2.4. Cognitive function assessment

The Hopkins Verbal Learning Test–Revised (HVLT-R) was utilized to evaluate verbal memory, focusing on immediate recall, delayed recall, and recognition. This assessment involves presenting participants with a list of 12 words, read aloud over three trials, with recall tested after each trial. The immediate recall score is calculated as the total number of words recalled across all three trials. Following a delay of approximately 20–25 minutes, participants were asked to recall the words again. The delayed recall score represents the number of correctly recalled words, with a maximum score of 12. During the recognition phase, participants were presented with a list of 24 words, comprising 12 target words from the original list and 12 distractor words. They were instructed to identify whether each word was part of the initial list. The recognition discrimination index (RDI) was calculated as the difference between the number of correctly identified target words (true positives) and the number of distractor words incorrectly identified as target words (false positives). This measure highlights participants' ability to distinguish between previously learned and unfamiliar words.

The Wechsler Adult Intelligence Scale (WAIS) is a standardized tool used to assess cognitive abilities across various domains, including verbal comprehension, working memory, perceptual reasoning, and processing speed. The WAIS includes several subtests such as Symbol Search and Coding. The Symbol Search subtest evaluates processing speed by requiring participants to scan a series of symbols and identify matches within a specified time frame. The raw score is determined by the number of correct matches minus the number of errors made within the time limit. The Coding subtest assesses processing speed, attention, and psychomotor coordination by requiring participants to pair symbols with numbers according to a provided key within

a time limit. The raw score represents the total number of correct responses within the given timeframe.

6.3. Results: Indoor air quality and health impact - Standard study

6.3.1. Monitoring changes of indoor environment

Figure 6-3 shows the average indoor air temperature, RH and CO_2 concentration during cooking time. The background CO_2 concentration was approximately 800 ppm and showed an increasing trend during the cooking period, reaching a maximum of 1300 ppm. The CO_2 concentration declined after the stove was turned off, stabilizing at around 1100 ppm 30 minutes after cooking. The temperature fluctuates between 25°C and 26 °C, maintaining a consistent environment conducive to comfort and health. Similarly, the relative humidity varies between 40% and 50%, falling within the recommended range for indoor air quality (Figure 6-3).

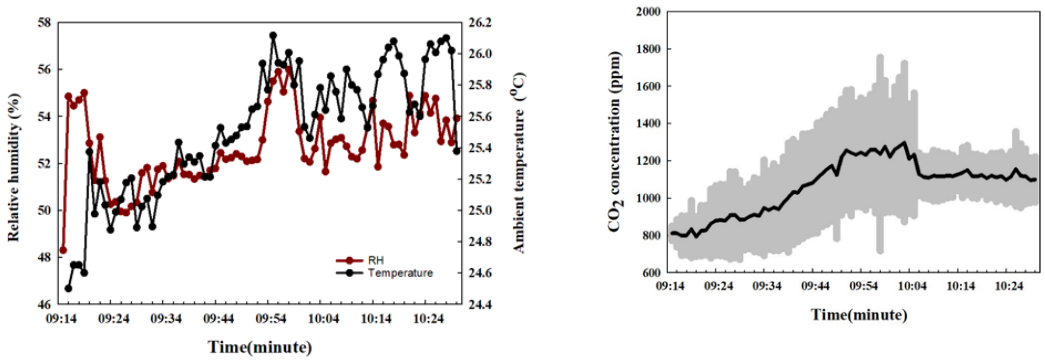


Figure 0-3. Average indoor air temperature, RH and CO_2 concentration during Phase II- standard study

6.3.2. Aerosol and particle measurements

Figure 6-4 illustrates the average $PM_{2.5}$ mass concentration over the 48-hour duration of the standard study. On the control and final days, the average $PM_{2.5}$ mass concentrations were 22.5 µg/m³ and 16.5 µg/m³, respectively. On the cooking day, the $PM_{2.5}$ mass concentration increased significantly to 193.45 µg/m³ from a background level of 30.8 µg/m³ immediately after the cooking period, before returning to near background levels approximately 8 hours post-cooking. During frying, the cooking oil temperatures peaked at 212.1°C for chicken and 161.3°C for fries (Figure 6-5).

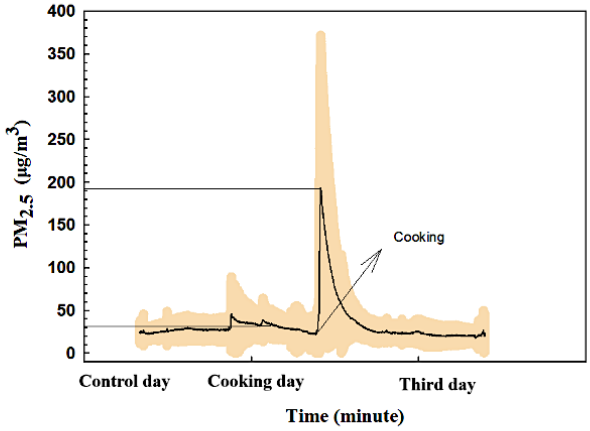


Figure 0-4. Average $PM_{2.5}$ concentration during phase II experiments - standard study

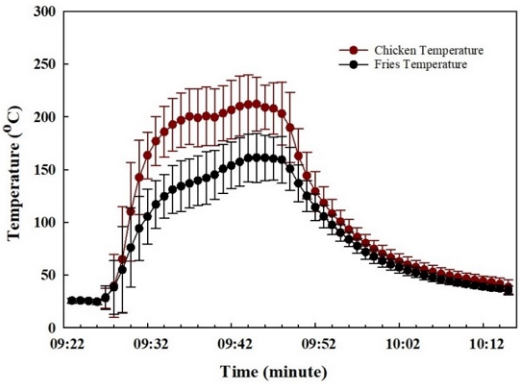


Figure 0-5. Average oil temperature during chicken and fries frying - standard study

The particle number concentration (PNC) peaked at 5.62×10^5 particles/cm³, rising from a background average of approximately 1.4×10^4 particles/cm³. By around 15:30, the PNC returned to background levels (Figure 6-6).

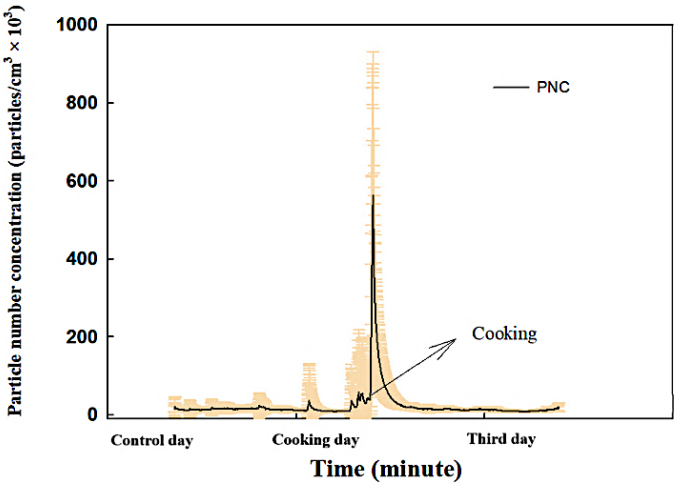


Figure 0-6. Average PNC during phase II experiments - standard study

6.4. Results: Indoor air quality and health impact - Intervention study

6.4.1. Monitoring changes of indoor environment

The figure 6-7 shows the diurnal variation in room temperature and RH during a controlled experiment, comparing trends between a cooking day and a control day. The room temperature begins at 22.3 °C in the morning, gradually increasing throughout the day to reach a peak of 26.8 °C by 19:00. After 19:00, the temperature decreases sharply to 24 °C. Relative humidity starts at 34% in the morning and decreases steadily until 19:00, coinciding with the temperature rise, as the air's moisture-holding capacity increases with higher temperatures. After 19:00, RH increases sharply back to 34%, correlating with the drop in temperature, which reduces the air's ability to retain moisture and results in a higher RH. The cooking day exhibits higher RH levels compared to the control day, reflecting the additional moisture introduced into the environment during frying.

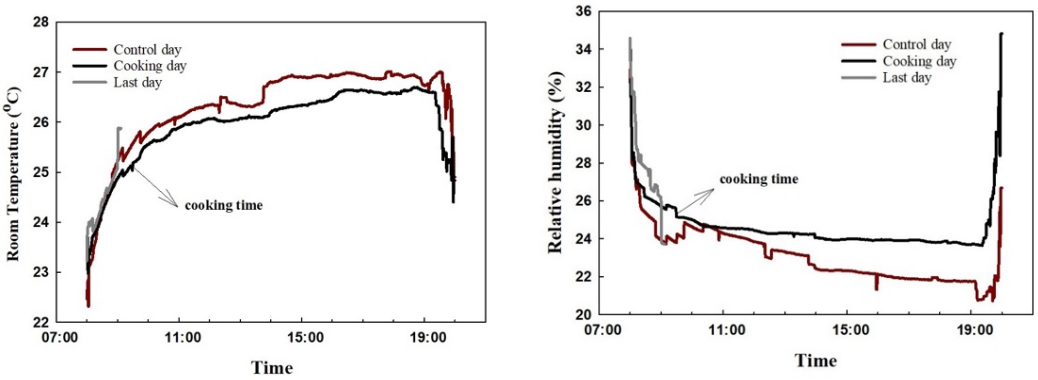


Figure 0-7. Average ambient temperature and RH during cooking, control and last day- intervention study

The figure 6-8 presents the average ambient concentrations of CO_2 and formaldehyde (CH_2O) during cooking and control days. On the control day, CO_2 levels start at 629 ppm in the morning and gradually rise to a peak of 670 ppm around noon, likely due to accumulated human activity. Following this peak, a drop in CO_2 concentration is observed during both cooking and control days, potentially reflecting a decrease in occupancy. CO_2 levels then rise again at approximately 14:00, indicating renewed activity. CO_2 and CH_2O concentrations are consistently higher on the cooking day compared to the control day, reflecting emissions from cooking activities. A distinct peak in CH_2O is observed immediately following the cooking period, suggesting that the release of formaldehyde is closely associated with frying process.

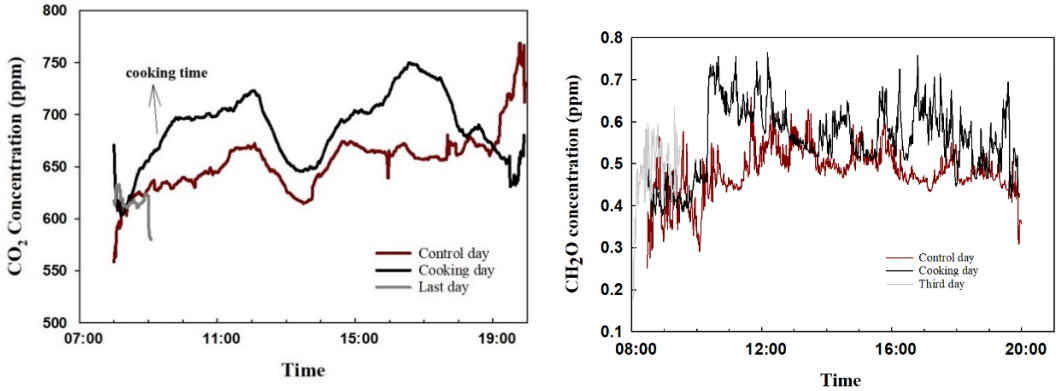


Figure 0-8. Average ambient CO_2 and CH_2O concentration during cooking, control and last day- intervention study

Figure 6-9 presents the average concentrations of CH_4 (methane) and NMHCs (non-methane hydrocarbons) during the cooking and control days. The data reveal that CH_4 levels were consistently higher on cooking days compared to control days,

suggesting that frying activities are a significant source of methane emissions. Similarly, NMHC concentrations were elevated during cooking, with notable peaks associated with the frying of chicken and fries. The first NMHC peak, observed immediately after cooking, reached 3300 ppm. This rise can be attributed to the release of hydrocarbons from cooking oil vapors and the thermal degradation of food components during high-temperature frying. The second peak, occurring at 14:00 and reaching 3600 ppm, is likely related to the persistence and accumulation of cooking-related emissions.

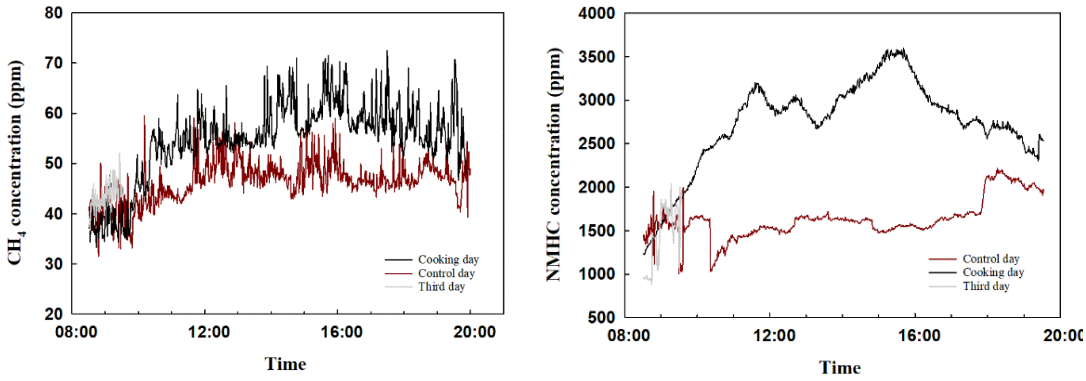


Figure 0-9. Average CH_4 and NHMC concentration during cooking control and last day - intervention study

Nitrogen dioxide (NO_2) concentrations also were higher on the cooking day compared to the control day, with a pronounced peak observed during the cooking period (Figure 6-10). This peak corresponds to the release of NO_2 from combustion-related activities, such as using stoves or heating oil at high temperatures, which are common during cooking. After the peak, a decreasing trend in NO_2 levels was observed, likely due to the dissipation of emissions over time and the influence of ventilation or air exchange. In contrast, the control day exhibited a gradual increasing trend in NO_2 concentrations throughout the day. This could be attributed to background sources, such as human activity, or emissions from external environments, accumulating indoors over time.

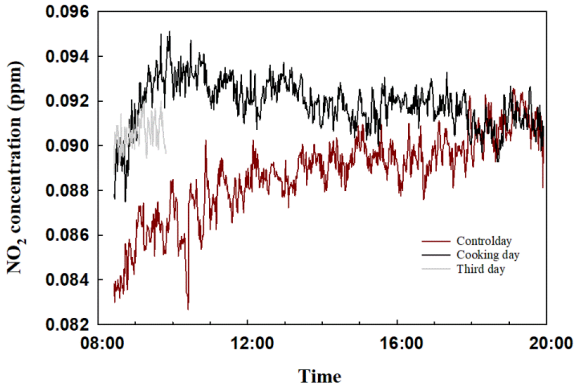


Figure 0-10. Average NO_2 concentration during cooking control and last day - intervention study

6.4.2. Aerosol and particle measurements

Figure 6-11 displays the average $PM_{2.5}$ concentration during the intervention experiments involving 30 study participants. On control days, the average daily concentration was 16.14 µg/m³. On the cooking day, the background concentration was 15.49 µg/m³, which peaked at 62.1 µg/m³ immediately following the cooking period. Approximately two hours post-cooking, the concentration returned to background levels around 13:00. During frying, the cooking oil temperature reached a maximum of 150.4°C for fries and 128.9°C for chicken. The slight decrease at 9:50 a.m., corresponds to the point when the chicken and fries were flipped after 10 minutes of cooking (Figure 6-12).

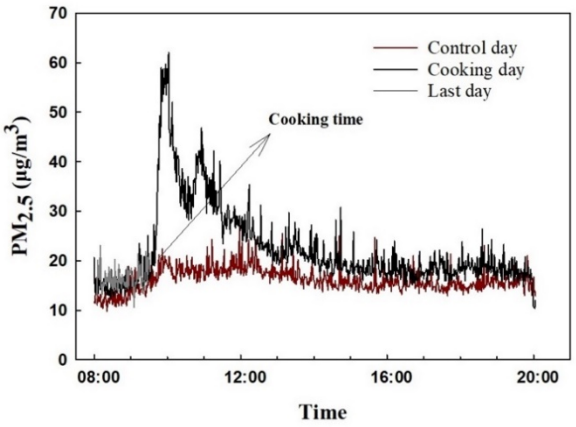


Figure 0-11. Average $PM_{2.5}$ concentration during cooking, control, and last days - intervention study

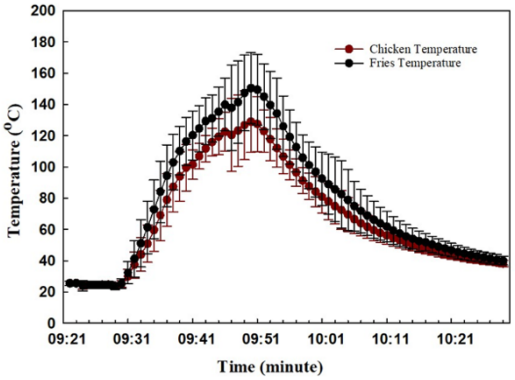


Figure 0-12. Oil temperature during chicken and fries frying -intervention study

The comparison of cooking with electric and gas revealed that $PM_{2.5}$ levels returned to near background concentrations about two hours after cooking, suggesting a relatively quick dissipation of particles. In contrast, the resultd from gas stove study showed a gradual return to background levels, taking around eight hours, indicating a longer-lasting presence of elevated $PM_{2.5}$ concentrations. This could partly be due to different types of particles originating from combustion of natural gas from the stove as compared to the particles from cooking emissions.

The average particle surface concentration (5–30 nm) displayed a dynamic trend, beginning at approximately 2.27×10^6 $\mu m^2/cm^3$, as the background level (Figure 6-13). A significant peak was observed during the cooking period, reaching 6.47×10^6 $\mu m^2/cm^3$. This increase is likely associated with the generation of UFPs during high-temperature cooking processes, such as frying, which release substantial quantities of VOCs and condensation nuclei. Following the cooking activity, the particle surface concentration gradually decreased, returning to background levels around 13:00. Apart from the cooking period, the concentration trends on both cooking and control days remained similar, indicating that cooking activities were the primary source of the observed spikes in UFPs concentrations. This highlights the importance of managing cooking emissions to mitigate short-term indoor air pollution and potential health risks. The average mode diameter of particles increased from a background size of 23.28 nm to 30.51 nm following cooking activities. This growth suggests the condensation and coagulation of ultrafine particles during cooking, as VOCs and semi-volatile compounds emitted from heated oils and food condensate onto particle surfaces, leading to particle growth. During both cooking and control days, the particle size remained relatively stable around 25 nm, indicating that the observed increase to

30 nm was directly associated with cooking emissions. This trend underscores the significant impact of cooking activities on the size distribution of indoor UFPs, emphasizing the need for adequate ventilation and exposure mitigation strategies during cooking.

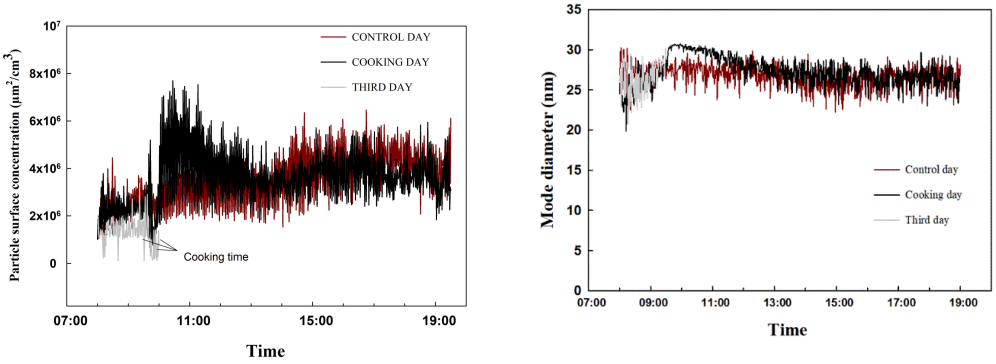


Figure 0-13. Average particle surface concentration and mode diameter (5-30 nm) measured by SMPS during cooking control and last-day - intervention study

The average particle number concentration measured by CPC during the control and final days for 30 study participants was 3.10 × 10^3 and 4.12 × 10^3 particles/cm^3, respectively. On the cooking day, the background PNC level was 3.06 × 10^3 particles/cm^3, which increased to a maximum of 1.0 × 10^5 particles/cm^3 immediately after the cooking period. The concentration returned to background levels around 13:00 on the cooking day (Figure 6-14). The PNC levels were comparable in both studies. However, the electric stove's PNC returned to baseline by 13:00 on the cooking day, while the gas stove's PNC remained elevated until 16:00, suggesting a longer duration of elevated particle exposure with the gas stove.

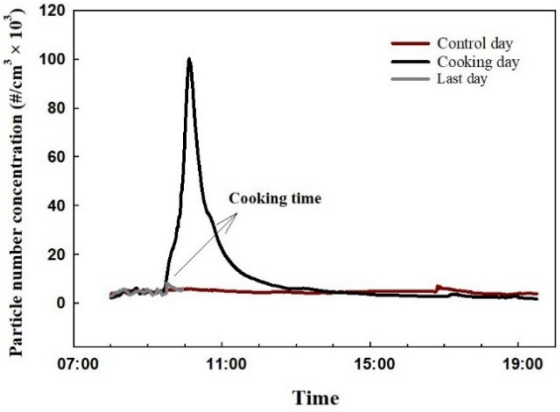
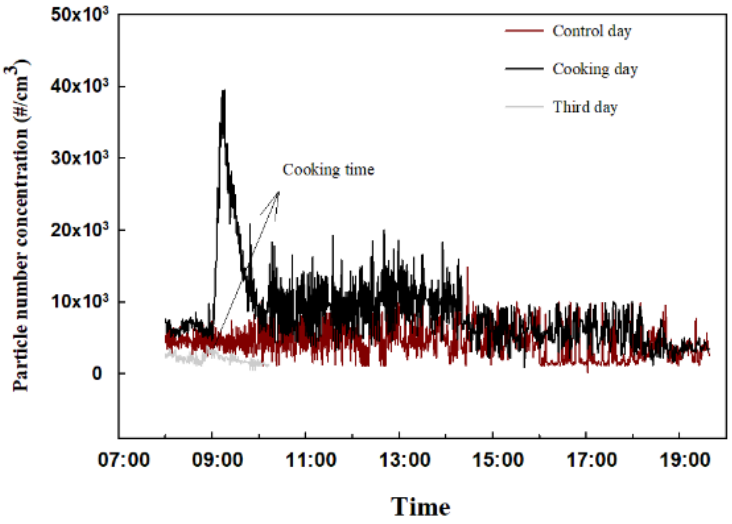


Figure 0-14. Average particle number concentration measured by CPC during cooking, control, and last days - intervention study

Figure 6-15 shows the particle number concentration measured during the intervention study on both control and cooking days, specifically for particles sized 5–30 nm, as measured by the SMPS. The background average concentration remained around 4 × 10^3 particles/cm^3 during both the control and cooking days. After the cooking period, the PNC peaked at 3.99 × 10^3 particles/cm^3 before gradually returning to background levels by 14:00. Figure 6-15 (right) shows that the mode diameter of particles, which was initially around 7.5 nm in the background, increased significantly to 29 nm during the cooking period. This increase is likely due to the formation and aggregation of ultrafine particles produced by the combustion process and the breakdown of oils during cooking, leading to the creation of larger particle clusters. Once the stove was turned off, the mode diameter gradually decreased, returning to background levels by 14:00. In contrast, on the control and third days, the average mode diameter remained relatively stable, fluctuating between 7 and 15 nm. This consistency on non-cooking days highlights that the observed particle growth during cooking is specific to cooking emissions, were heat and combustion processes influence particle size dynamics.



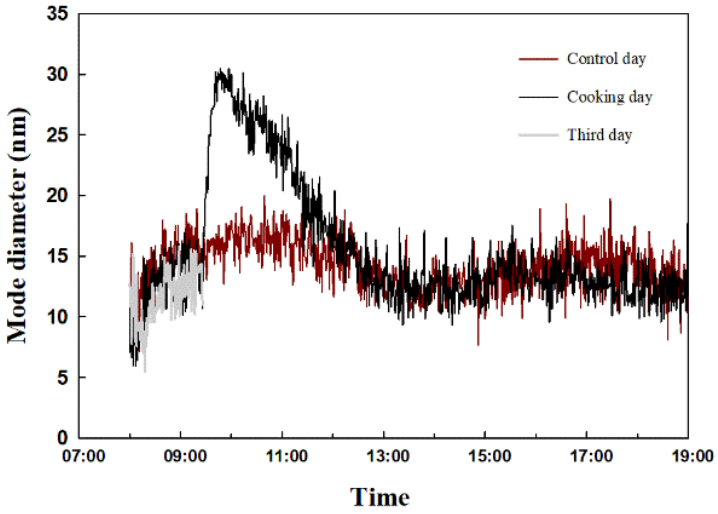


Figure 0-15. Average particle number concentration and mode diameter (5-30 nm) measured by SMPS during cooking control and last-day - intervention study

6.5. Health assessment results: Standard study (gas and particle effect)

6.5.1. Blood pressure and blood oxygen saturation

Figure 6-16 shows the effects of exposure on SBP, DBP and blood oxygen levels (SpO_2) during both cooking and control experiments. Blood pressure was measured using the Omron 10® Model BP786N device, while blood oxygen levels were assessed using the Pulse Oximeter (Model Oxywatch C20) by ChoiceMMed America Corp. On the control day, SBP and DBP increased from their baseline levels at 9:30 (111.28 ± 14.38 mm Hg and 73.71 ± 9.60 mm Hg, respectively) to peak values at 10:00 (113.13 ± 14.74 mm Hg and 77.07 ± 12.07 mm Hg, respectively). Following this peak, both SBP and DBP gradually declined, reaching their lowest levels at 16:00 (107.87 ± 15.21 mm Hg and 68.82 ± 8.99 mm Hg, respectively). Subsequently, both measures rose again, peaking around 20:00 (113.10 ± 16.82 mm Hg for SBP and 73.20 ± 9.52 mm Hg for DBP). SBP did not show statistically significant changes (95% [CI]; $p = 0.122$), indicating no observable diurnal pattern. In contrast, DBP exhibited significant fluctuations during the baseline period ($p = 4.83 \times 10^{-8}$, 95% CI) across multiple time points (Table 6-3), pointing to the presence of diurnal variation in DBP.

On the cooking day, an insignificant decrease in SBP and DBP was observed immediately after cooking at 10:00 (109.38 ± 15.80 mm Hg and 69.87 ± 8.24 mm Hg,

respectively), followed by an upward trend until 12:00 (112.28 ± 23.59 mm Hg for SBP and 75.30 ± 11.34 mm Hg for DBP). A sharp decline occurred at 14:00 (110.23 ± 15.74 mm Hg for SBP and 69.38 ± 11.34 mm Hg for DBP), after which both SBP and DBP increased again, peaking at 18:00 (111.69 ± 15.01 mm Hg for SBP and 74.12 ± 10.68 mm Hg for DBP). The values then decreased again at 20:00 (115.76 ± 17.67 mm Hg for SBP and 76.56 ± 11.51 mm Hg for DBP). The fluctuation in SBP during control days was not statistically significant and show no diurnal effect. However, DBP showed significant changes in several time intervals indicating diurnal effect (Table 6-3).

Table 0-3 . Statistically significant Bonferroni-Corrected Wilcoxon test results (*p*-values) indicating diurnal variation in DBP across control days in the Phase II standard study

Time interval	**10:00 - 14:00**	**10:00 - 16:00**	**10:30 - 16:00**	**11:00 - 16:00**	**11:30 - 16:00**	**12:00 - 16:00**
***p*-Values**	0.0152	0.0007	0.0042	0.0009	0.0336	0.0007

We failed to reject Hypotheses 2 and 3 for SBP and failed to identify any statistically significant main effect, of cooking aerosol exposure on SBP, nor any meaningful interaction between exposure and time. However, a different trend was observed for DBP. Specifically, Hypothesis 3 was rejected (95% CI; $\boldsymbol{p} = 5.6\times10^{-6}$) indicate that cooking aerosol exposure significantly influenced DBP and that this effect varied with time.A notable and immediate reduction in DBP was observed post-exposure, particularly at 10:00 a.m. (95% CI; $p = 8.2\times10^{-4}$). By 4:00, 6:00, and 8:00 p.m., the effect had diminished and even reversed slightly, with DBP showing non-significant increases during these later time points. Further analysis using Wilcoxon signed-rank tests revealed statistically significant interactions between exposure and time on DBP at three key intervals: between 10:00 and 16:00 (95% CI; $p = 0.0012$), between 10:00 and 18:00 (95% CI; $p = 0.0384$), and between 10:00 and 20:00 (95% CI; $p = 0.00028$). These results collectively suggest a time-dependent impact of cooking aerosol exposure on diastolic blood pressure, with the most pronounced effects occurring within the first 6 to 8 hours following exposure.

The SpO_2 concentration starts at 97.82 ± 1.03 % at 9:30 a.m., slightly fluctuates through the morning, and peaks at 98.15 ± 0.92 % around 11:30 a.m. Following this peak, it shows a small decline to 97.70 ± 1.60 % at 12:00, stabilizing in the afternoon, with minor variations ranging between 97.79 ± 1.14 % ppm and 97.97 ± 0.86 % by the evening. The cooking day SpO_2 levels start at 97.65 ± 1.01 %, showing a gradual increase and peaking at 98.03 ± 1.07 % around 12:00 p.m. Afterward, the concentration drops to 97.50 ppm ± 1.05 % at 16:00, followed by a minor rise to 98.24± 0.85 % at 20:00. Fluctuations in blood oxygen saturation (SpO_2) during the baseline period were minimal and lacked any statistically significant diurnal variation (95% confidence interval; P = 0.235). Furthermore, there was no significant interaction between time and exposure (95% CI; p = 0.474), nor a main effect of exposure (95% CI; p = 0.333), indicating that exposure to cooking-generated aerosols did not significantly affect SpO_2 saturation levels.

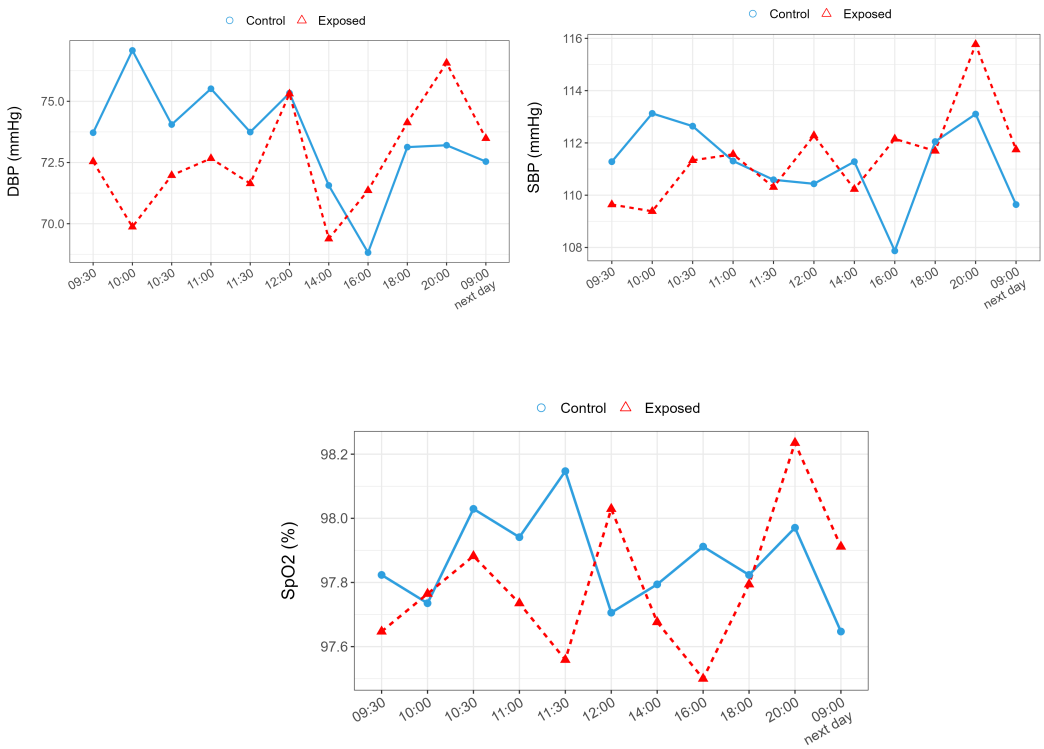


Figure 0-16 Mean changes in DBP, SBP and blood oxygen measured on the cooking (exposure) day and the control (non-exposure) day during Phase II of the standard study

6.5.2. Lung function

Lung function was assessed by measuring PEFR and fractional exhaled nitric oxide (FeNO) levels during the standard study. On the control day, FeNO levels remained relatively stable with minimal fluctuation, and no diurnal variation was observed (95% CI, P = 0.678). On the cooking day, the most significant effect occurred immediately after cooking (10:00), with FeNO levels showing a sharp increase compared to pre-exposure levels (15.7 ± 9.6 ppb on the control day and 27.2 ± 10.2 ppb on the cooking day). This was followed by a decreasing trend until 20:00, when FeNO levels returned to approximately pre-exposure levels (16.75 ± 8.25 ppb). Significant interaction effects were identified (95% CI; $p = 1.83 \times 10^{-7}$), indicating that the effect of cooking aerosol exposure on FeNO varied across different post-exposure time points. The interaction effect was particularly evident at 10:00 a.m., immediately following the cooking activity. In addition to these interaction effects, a significant main effect of exposure was observed, with increased FeNO levels immediately post-exposure (95% CI; p = 0.0093) and 30 minutes after exposure (95% CI; p = 0.0144) (Figure 6-17).

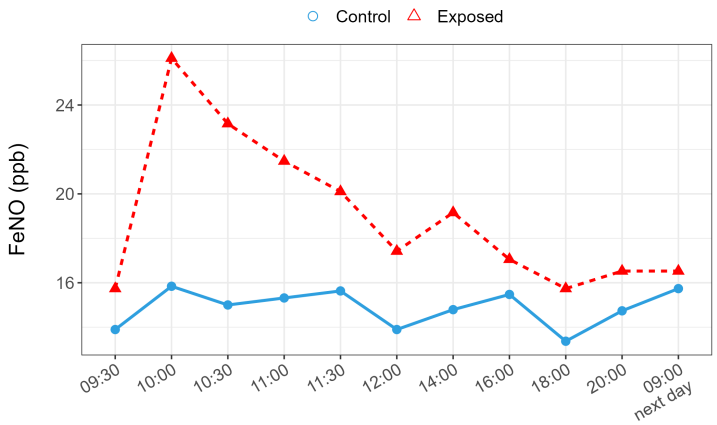


Figure 0-17. Mean change in FeNO level on the cooking (exposure) day and the control (non-exposure) day during Phase II of the standard study

On the control day, PEFR exhibited a sharp increase at 11:00, reaching 378.82 ± 140.6 l/min from the baseline level of 327.94 ± 117.8 l/min, followed by a decreasing trend that continued into the evening (Figure 6-18). However, observed changes were not significant between any measurement points during the control days for PEFR, indicating no diurnal effect. On the cooking day, PEFR showed an insignificant decreasing trend after cooking, remaining stable until 16:00, followed by fluctuations that led to a substantial decline on the third day. The lowest PEFR was observed at

18:00, measuring 370.29 ± 135.9 l/min. Statistical analysis failed to reject hypothesis 2 and 3 , indicating that no significant main effect or interaction effect of exposure was observed for PEFR.

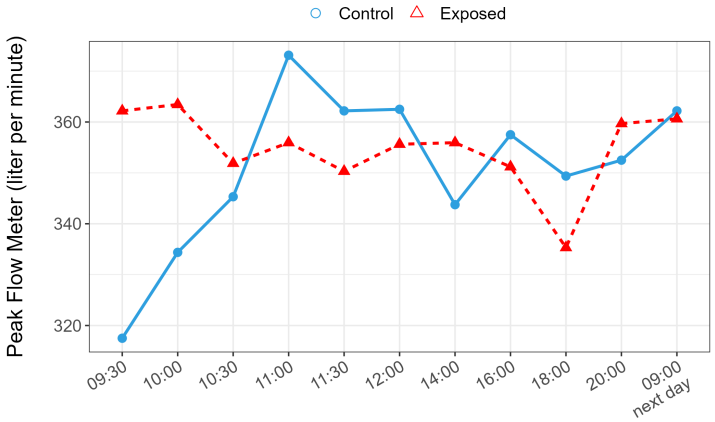


Figure 0-18. Mean change in PEFR on the cooking (exposure) day and the control (non-exposure) day during Phase II of the standard study

6.5.3. Heart rate

The heart rate (HR) data observed with the Omron 10 device for the control day exhibited a subtle diurnal trend with fluctuations throughout the observation period. Starting at 73.03 ± 7.68 bpm in the early morning, HR showed a slight decline, reaching its lowest value of 68.02 ± 9.86 bpm by 11:00. An increasing trend was then observed, with HR peaking at 75.66 ± 8.55 bpm at 14:00 before gradually decreasing to 71.71 ± 9.17 bpm by 20:00. The statistical analysis showed statistically significant differences between several time intervals during the control days, indicating the diurnal effect of HR (Table 6-4).

Table 0-4. Statistically significant Bonferroni-Corrected Wilcoxon test results (*p*-values) indicating diurnal variation in DBP across control days in the Phase II standard study

Time interval	**9:00 - 11:00**	**10:00 - 11:00**	**11:00 - 14:00**	**11:00 - 16:00**	**11:30 - 14:00**	**11:30 - 16:00**	**12:00 - 14:00**
***p*-Values**	0.0127	0.036	0.006	0.0212	0.0031	0.041	0.0413

On the cooking days, the HR began at 73.28 ± 8.66 bpm in the early morning and increased slightly to 73.82 ± 8.89 bpm immediately after cooking (Figure 6-19). This was followed by a gradual decline, with HR dropping to 71.02 ± 8.68 bpm at 11:30. Subsequently, a modest increase was observed, with HR rising to 72.00 ± 8.94 bpm by mid-afternoon. The evening period showed a peak of 75.33 ± 8.88 bpm at 16:00, followed by a decrease to 71.33 ± 8.66 bpm at 20:00. Neither the interaction (95% CI; p = 0.389), between exposure and time nor the main effect (95% CI; p = 0.223), of exposure reached statistical significance, suggesting that cooking-related particles and gases had no discernible impact on heart rate.

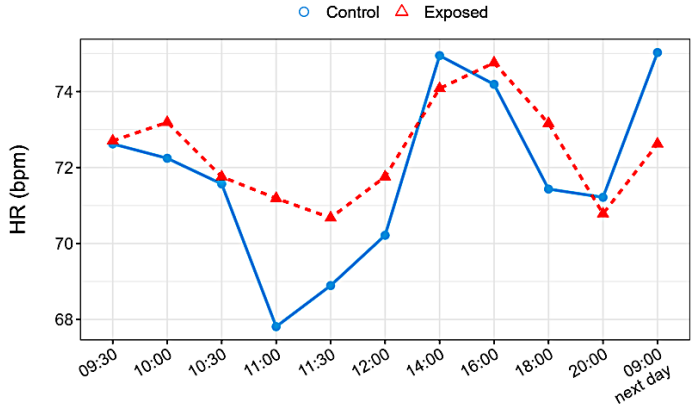


Figure 0-19. Mean changes in HR on the cooking (exposure) day and the control (non-exposure) day during Phase II of the standard study

6.5.4. Heart rate variability analysis

Cardiologist analysis on ECG showed no instances of atrial fibrillation, tachycardia salvos, bradycardia, or pauses with ectopic beats were observed within 24 hours post-cooking, in contrast to the control day. The heart rate variability (HRV) metrics, including SDNN, RMSSD, pNN50, LF and HF were analyzed to assess the autonomic nervous system's response during control and cooking days. The results showed no statistically significant differences between the cooking and control days (Table 6-5).

Table 0-5. HRV parameters recorded from participants in the standard study. Values represent mean ± standard deviation

HRV metrics	Control day	Cooking day	*p* value -t test
SDNN (ms)	37.09 ± 17.89	36.63 ± 18.07	0.472
RMSSD (ms)	35.88 ± 23.43	35.01 ± 23.55	0.142
PNN50 (%)	14.83 ± 14.82	14.09 ± 15.83	0.364
LF (ms^2)	834.05 ± 730.17	825.93 ± 707.76	0.767
HF (ms^2)	536.33 ± 674.88	565.27 ± 723.69	0.142

SDNN, representing the standard deviation of NN intervals and overall heart rate variability, remained consistent between the control and cooking days, indicating no significant (95% CI, $p = 0.472$) impact of cooking emissions on autonomic flexibility. Similarly, RMSSD, a metric sensitive to short-term changes in parasympathetic activity, showed stable values, suggesting that vagal tone and short-term cardiac autonomic regulation were not significantly altered (95% CI, $p = 0.142$). Additionally, pNN50, which measures the percentage of successive NN intervals differing by more than 50 ms and reflects parasympathetic activity, remained comparable across both days. These findings collectively indicate that exposure to cooking emissions did not lead to clinically meaningful changes in heart rate variability or parasympathetic control. Frequency-domain metrics, including LF power and HF power, showed no significant differences. LF power reflects both sympathetic and parasympathetic modulation, while HF power is primarily associated with parasympathetic activity. Clinically, reduced HF power can indicate lower parasympathetic activity, and changes in LF power can reflect altered autonomic balance. The stable LF and HF values suggest that cooking generated aerosol emissions had no significant impact on either branch of the autonomic nervous system.

6.6.Health assessment results: intervention study (gas effect)

6.6.1. Blood pressure and blood oxygen saturation

Figure 6-20 displays data on SBP, DBP and blood oxygen saturation (SpO_2) levels during the intervention study's cooking and control conditions. During control days, at 9:00, SBP started at 103.57 ± 11.37 mm Hg, while DBP was at 67.13 ± 8.71 mm Hg. Both values showed a slight increase at 10:00, with SBP reaching 103.4 ± 12.54

mm Hg and DBP at 68.90 ± 6.27 mm Hg. The trend continued with a slight decrease in SBP to 102.87 ± 10.78 mm Hg and DBP to 68.07 ± 7.03 mm Hg by 10:30. However, at 11:00, there was a more noticeable increase in both SBP (104.93 ± 10.60 mm Hg) and DBP (71.23 ± 6.67 mm Hg).

After reaching this peak, both SBP and DBP showed a gradual decline. By 11:30, SBP decreased to 103.37 ± 11.34 mm Hg and DBP to 70.23 ±5.87 mm Hg. At 12:00, SBP increased to 103.5 ± 10.81 mm Hg, and DBP rose to 71.10 ± 6.14 mm Hg, remaining steady through the early afternoon. From 14:00 onward, both SBP and DBP followed a declining trend, with SBP dropping to 105.8 ± 11.47 mm Hg and DBP to 67.70 ± 6.04 mm Hg at 14:00. By 16:00, SBP remained fairly stable at 104.77 ± 10.70 mm Hg, while DBP slightly increased to 68.23 ±8.07 mm Hg. In the evening, at 1800, SBP slightly decreased to 104.77 ± 10.70 mm Hg, and DBP returned to 67.13 ± 6.77 mm Hg. Finally, by 20:00, SBP reached its highest level of the day at 107.13 ± 12.70 mm Hg, while DBP increased to 69.47 ± 5.87 mm Hg. However, no diurnal effects on SBP or DBP were observed during the baseline periods in the intervention study.

During the cooking day, both SBP and DBP increased immediately after cooking (105.70 ± 11.37 mm Hg and 71.0 ± 7.61 mm Hg, respectively) compared to pre-exposure levels (103.41 ± 22.0 mm Hg for SBP and 68.3 ± 7.31 mm Hg for DBP). SBP showed a decline 30 minutes after cooking (104.07 ± 12.61 mm Hg) and stabilization around 104.0 ± 12.27 mm Hg by 11:00. A secondary rise occurred, peaking at 105.57 ± 11.35 mm Hg at 11:30 before decreasing to 103.7 ± 11.88 mm Hg at 12:00. In the afternoon, SBP increased again, reaching 106.97 ± 14.87 mm Hg at 16:00 and remained elevated at 105.67 ± 9.58 mm Hg at 18:00 and 106.57 ± 13.34 mm Hg at 20:00. However, DBP decreased one hour after cooking (71.16 ± 10.38 mm Hg) followed by a decrease to 69.37 ± 6.20 mm Hg at 11:00. DBP then rose again, reaching 71.3 ± 5.87 mm Hg at 12:00, before experiencing a fluctuating decline in the afternoon. It reached 68.73 ± 6.69 mm Hg at 14:00, peaked at 70.8 ± 15.13 mm Hg at 16:00, and finally decreased to 67.8 ± 8.06 mm Hg at 18:00. DBP returned to near control levels by 20:00 (70.36 ± 8.93 mm Hg).

When comparing the average SBP and DBP between the control and cooking days, both were generally higher on the cooking day, particularly in the hours immediately following cooking. However, no significant interaction (SBP: 95% CI; $p = 0.812$, DBP

95% CI; $p = 0.187$) or main effect interaction (SBP: 95% CI; $p = 0.827$, DBP 95% CI; $p = 0.747$) was observed for SBP or DBP, as Hypotheses 2 and 3 were not rejected.

Throughout the control day, blood oxygen saturation (SpO_2) remained relatively stable, fluctuating between 97.6% and 97.97%. The lowest value occurred at 14:00 (97.7 ± 0.70 %), while the highest was at 20:00 (97.97 ± 1.47%). As fluctuations during the control days for SpO_2 (95% CI, $p = 0.865$) was not statistically significant no diurnal effect was observed in this population. Blood oxygen saturation (SpO_2) remained relatively stable throughout the cooking day, with minimal fluctuations. SpO_2 began at 97.4 ± 1.19 % at 9:00, slightly increased to 97.53 ± 0.82 % at 10:00, and continued to rise, reaching 97.8 ± 1.09 % at 10:30. It decreased slightly to 97.63 ± 1.32 % by 11:30 and stabilized at 97.6 ± 1.04 % between 14:00 and 18:00. By 20:00, SpO_2 remained consistent at 97.57 ± 1.22 %. Statistical analyses revealed no significant main effects of exposure (95% CI, $p = 0.222$) or time-by-exposure interactions (95% CI, $p = 0.651$). These findings indicate that short-term exposure to cooking-related gases did not affect systemic oxygenation, with O_2 levels remaining stable throughout different times of the day (Figure 6-20).

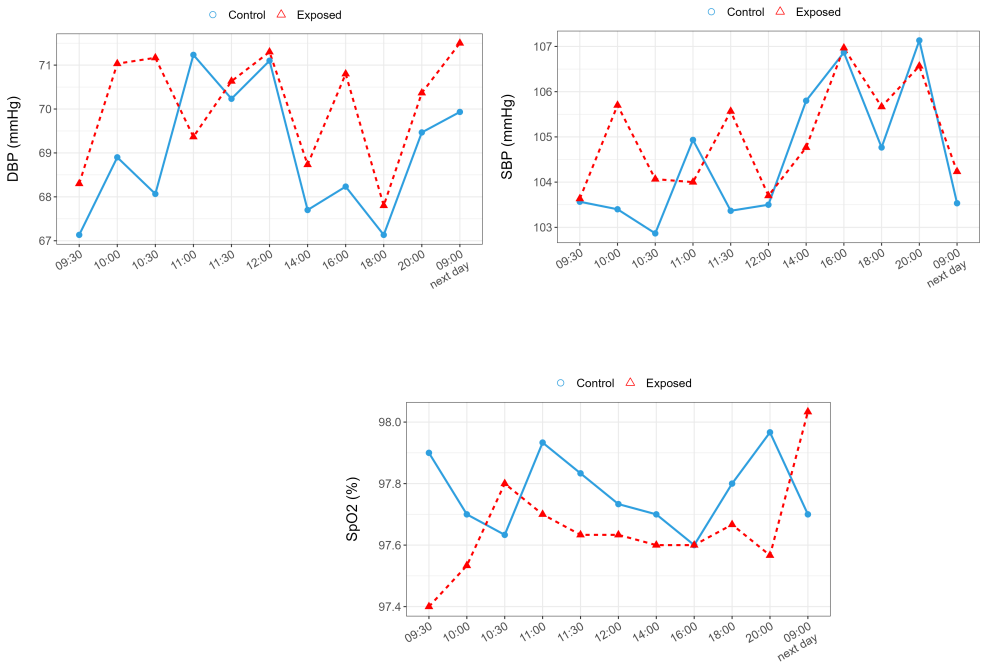


Figure 0-20. Mean changes in DBP, SBP and blood oxygen on the cooking (exposure) day and the control (non-exposure) day during Phase II of the intervention study

6.6.2. Lung function

The PEFR values began at 459 ± 134.07 L/min at 9:00, rising to 495 ± 194.7 L/min by 10:00. The lowest PEFR levels were recorded at 11:30 on the control day, while the highest levels, 550.67 ± 176.4 L/min, were observed at 18:00, before slightly decreasing to 520.67 ± 180.6 L/min at 20:00. These changes in the control day were statistically insignificant and showed no diurnal effect (95% CI, p = 0.653). On the cooking day, PEFR initially increased after cooking (446.3 ± 135.9 L/min) and remained elevated for approximately 30 minutes post-cooking (493.0 ± 177.4 L/min), followed by fluctuations. Notably, two sharp increases in PEFR occurred at 16:00 (523.3 ± 166.0 L/min) and 20:00 (524.3 ± 179.8 L/min). Overall, PEFR showed a downward trend compared to the control day, except at 10:30 and 11:30, when it exceeded control levels (Figure 6-21). We did not provide sufficient evidence to reject Hypotheses 2 and 3, indicating that neither the main effect of exposure (95% CI, P = 0.166) to cooking-generated gases nor the interaction (95% CI, P = 0.743) between exposure and time was statistically significant.

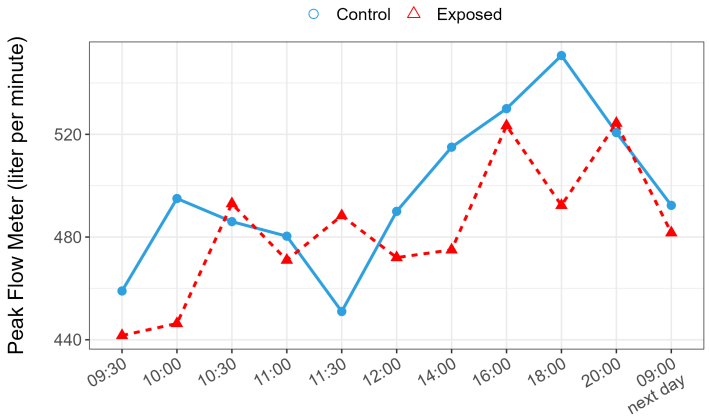


Figure 0-21. Mean changes in PEFR on the cooking (exposure) day and the control (non-exposure) day during Phase II of the intervention study

6.6.3. Heart rate

Dring control days, the heart rate (HR) remains relatively stable between 69.87 ± 10.9 bpm and 68.2 ± 10.68 bpm from early morning until noon, showing only minor fluctuations. At 14:00, there is a noticeable increase to 69.8 ± 11.26 bpm, followed by a substantial spike to 76.37 ± 11.87 bpm, indicating a sudden rise in HR. After reaching its maximum peak, the HR decreases steadily to 71.0 ± 11.51 bpm by 20:00. The

changes in HR during control days were statistically significant between 11:00 and 16:00 (95% CI; p = 0.0054), 11:30 and 16:00 (95% CI; p = 0.0098), and 12:00 and 16:00 (95% CI; p = 0.0239), providing evidence of diurnal effect. On the cooking day at 9:00, the HR was 69.87 ± 10.83 bpm, representing a stable baseline before cooking activities commenced. Cooking occurred between 9:30 to 10:00, during which the HR increased slightly to 70.4 ± 11.40 bpm by 10:00. Following the cooking activity, HR decreased marginally to 68.87 ± 10.76 bpm 30 minutes after cooking. However, an increasing trend was observed thereafter, with HR peaking at 74.80 ± 10.39 bpm by 16:00. Subsequently, a gradual decline was observed, with HR decreasing to 72.20 ± 9.95 bpm at 20:00. However, analyses showed no significant main effect of exposure (95% CI; p = 0.267) or time-by-exposure interaction (95% CI; p = 0.552), suggesting that short-term exposure to cooking-emitted gases did not notably influence heart rate patterns (Figure 6-22).

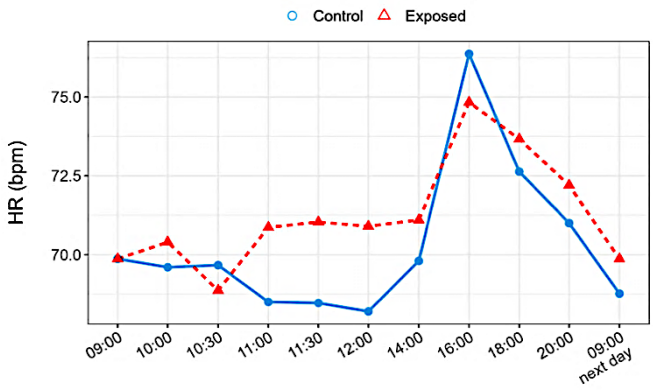


Figure 0-22. Mean changes in HR on the cooking (exposure) day and the control (non-exposure) day during Phase II of the intervention study

6.6.4. Heart rate variability analysis

No occurrences of atrial fibrillation, tachycardia salvos, bradycardia, or pauses with ectopic beats were detected up to 24 hours after cooking with an electric stove, similar to the observations made with gas stove cooking, when compared to the control day. The analysis (Paired t test results) of heart rate variability (HRV) metrics, including SDNN, RMSSD, pNN50, LF power and HF power, revealed no significant differences between the control and cooking days (Table 6-6). This suggests that short-

term exposure to cooking gases emissions had no measurable impact on autonomic nervous system regulation in this study's context.

SDNN, a measure of overall heart rate variability and autonomic flexibility, showed no significant change between the control day and cooking day. Clinically, reductions in SDNN are linked to poor cardiovascular outcomes, whereas the stable values observed here suggest preserved autonomic function during cooking exposures. Similarly, RMSSD, a key indicator of vagal tone, remained stable, reinforcing the conclusion that cooking emissions did not significantly impact parasympathetic activity. Furthermore, pNN50, which represents the percentage of successive NN intervals differing by more than 50 ms and reflects parasympathetic modulation, also showed consistent values between the control and cooking days. This overall stability across HRV parameters indicates that cooking emissions did not produce clinically meaningful changes in autonomic function.

Frequency-domain parameters also did not show significant differences. LF power, which reflects both sympathetic and parasympathetic influences, was 1076.88 ± 630.07 ms² on the control day and 1042.39 ± 423.32 ms² on the cooking day (95% CI; $p = 0.733$). Similarly, HF power, primarily representing parasympathetic activity, showed comparable values of 792.94 ± 688.04 ms² and 765.10 ± 498.53 ms² on the control and cooking days, respectively (95% CI; $p = 0.649$). These findings suggest that autonomic balance was maintained despite exposure to cooking emissions.

Table 0-6. HRV parameters recorded from participants in the intervention study. Values represent mean ± standard deviation

HRV metrics	**Control day**	**Cooking day**	***p*value -t test**
SDNN (ms)	45.60 ± 12.97	45.42 ± 10.33	0.873
RMSSD (ms)	44.12 ± 19.88	45.17 ± 17.19	0.699
PNN50 (%)	19.95 ± 14.06	19.68 ± 12.30	0.791
LF (ms^2)	1076.88 ± 630.07	1042.39 ± 423.32	0.733
HF (ms^2)	792.94 ± 688.04	765.10 ± 498.53	0.649

6.6.5. Results of cognitive tests

The results of the WAIS Symbol Search and Coding subtests at various time points during both the control day and the cooking days are summarized in Table 6-7. These metrics assess processing speed and mental control abilities. For the Coding subtest, the mean score on the control day at 11:30 a.m. was 80.68 ± 13.65. During the cooking day, participants showed minor fluctuations in their scores, with 78.44 ± 12.37 at 9:00 a.m., and 81.48 ± 14.11 at 11:30 a.m. By the third day, the score increased to 85.67 ± 18.43. However, in the WAIS-IV Coding task, no significant effects of exposure to cooking-generated gases were observed across time points.

For the Symbol Search test on the control day at 11:30 a.m., participants had a mean score of 41.38 ± 1.47. On the cooking day, scores initially decreased, with the score dropping to 39.03 ± 7.50 at 9:00 a.m. but then improved throughout the day and rising to 42.45 ± 6.97 at 11:30 a.m. On the third day at 9:00 a.m., participants showed an even higher mean score of 45.90 ± 6.95. For the WAIS-IV Symbol Search test, a significant effect of main effect was observed. Adjusted Wilcoxon tests further indicated a significant effect of exposure type (95% CI; $p = 0.0082$) at 11:30 a.m. However, the interaction between time and exposure type was not significant (95% CI; $p = 0.473$).

These significant changes could be the combined effect of cooking-generated gases and the practice effect. Several studies have reported improvements in WAIS test scores after repeated administration, typically after one week, three months, or six months (Estevis et al., 2012, Lee et al., 2023, Duff et al., 2012, Siders et al., 2006). Given that previous studies observed practice effects over longer intervals, it is reasonable to assume that the repetition of the test four times over two consecutive days may have contributed to the observed improvements. Our results indicate a general trend of improvement, particularly by the third day, suggesting that participants performed better over time. Theoretically, exposure to cooking-generated gases should have no effect or even a detrimental impact on cognitive performance. However, in this study, we were unable to conclusively separate the effects of gas exposure from the practice effect. The dominant practice effect likely overshadowed any potential impact of gas exposure, as evidenced by the significant improvement observed on the third day.

Table 0-7. Symbol Search and Coding subtest scores for control and cooking days (Mean ± SD)

WAIS metric	Control day 11:30	Cooking day 9:00	Cooking day 11:30	Third day 9:00
Symbol search correct score	41.38 ± 1.47	39.03 ± 7.50	42.45 ± 6.97	45.90 ± 6.95
Coding correct score	80.68 ± 13.65	78.44 ± 12.37	81.48 ± 14.11	85.67 ± 18.43

The results of the HVLT-R metrics across various time points for both control day and cooking day are summarized in Table 6-8. For the immediate recall score, on the control day at 11:30 a.m., participants had an average score of 28.24 ± 3.75, while the scores during the cooking day at different time points were slightly lower. At 9:00 a.m. on the cooking day, the score was 24.79 ± 4.38, and at 11:30 a.m., it further decreased to 25.14 ± 3.69. The HVLT Total Recall Score showed a significant significant interaction effect (95% CI; $p = 0.0019$), whereas Adjusted Wilcoxon tests confirmed the significance of the interaction at 11:30 a.m. (95% CI; $p = 0.041$).

Delayed recall scores generally followed a similar pattern, with scores on the control day at 11:30 a.m. to be higher (9.82 ± 1.70) than those observed during the cooking day. On the cooking day, the delayed recall scores showed a decline, with the score at 9:00 a.m. being 8.76 ± 2.17, and at 11:30 a.m., it further decreased to 8.03 ± 2.16. On the third day at 9:00 a.m., the delayed recall score increased to 9.27 ± 1.90, showing a recovery toward baseline levels. For the HVLT Delay score, a significant interaction effect was present (95% CI; $p = 0.011$), suggesting that the combined influence of time and exposure affected delayed recall. Adjusted Wilcoxon results partially supported this interaction at 11:30 a.m. (95% CI; $p = 0.032$).

The recognition performance, measured through the Recognition Discrimination Index (RDI), showed relatively stable results across all time points, both on the control and cooking days. The RDI remained consistent, with scores of 23.60 ± 0.77 at 11:30 a.m. on the control day, and 22.93 ± 1.36 at 9:00 a.m., 22.72 ± 1.89 at 11:30 a.m., on the cooking day and 23.13 ± 1.14 on the third-day. Finally, for the HVLT RDI (Recognition Discrimination Index), no significant effects were found for main effect, or interaction. These results indicate that while some cognitive tasks exhibited significant changes over time or due to exposure type, interaction effects were

generally limited to specific measures related to verbal memory recall, suggesting a potential time-dependent sensitivity to exposure.

Table 0-8 . Mean ± SD of HVLT-R metrics for control and cooking days

HVLT-R metrics	**Control day 11:30**	**Cooking day 9:00**	**Cooking day 11:30**	**Third day 9:00**
Immediate Recall	28.24 ± 3.75	24.79 ± 4.38	25.14 ± 3.69	26.83 ± 3.38
Delayed Recall	9.82 ± 1.70	8.76 ± 2.17	8.03 ± 2.16	9.27 ± 1.90
RDI	23.60 ± 0.77	22.93 ± 1.36	22.72 ± 1.89	23.13 ± 1.14

For the HVLT-R, there was no noticeable improvement in scores across repeated testing sessions. This could be attributed to the use of a new word list in each session, which prevented participants from benefiting from prior exposure and familiarity with the material. Since different words were presented each time, participants could not rely on memory from previous sessions to enhance their recall.

In contrast, for Symbol Search and Coding, the same test was administered in each session. This repetition likely contributed to improved performance over time due to practice effects (PE), as participants became more familiar with the task format and response patterns rather than experiencing actual cognitive improvements. If we assume that practice effects exist in HVLT-R, the exposure to cooking-generated gases may have mitigated these effects, preventing an increase in scores. In this case, the results would reflect the combined influence of both exposure and practice effects. However, if we assume there is no practice effect due to the changing word lists, then we can conclude that cooking-generated gases impaired initial learning and memory consolidation (IR & DR), while recognition ability (RDI) remained intact.

Further experiments are needed to disentangle the practice effect as a confounding variable from the potential effects of exposure. A well-controlled study design that isolates these variables could provide more definitive insights into the relationship between cooking emissions and cognitive performance.

6.7. Discussion

To the best of our knowledge, there is limited research on the cardiopulmonary effects of cooking-related air pollution. Fedak et al. (2019) conducted a study to assess acute blood pressure responses following exposure to emissions from various cookstove technologies, including the three-stone fire, rocket elbow, fan rocket elbow, gasifier, and liquefied petroleum gas stoves. Thirty minutes after exposure, SBP was significantly lower for the three-stone fire treatment (500 μg/m³ $PM_{2.5}$) compared to the control group (-2.3 mm Hg; 95% CI), with a similar trend observed for the gasifier stove (35 μg/m³ $PM_{2.5}$; -1.8 mm Hg; 95% CI). However, no significant differences were found at three hours post-exposure. By 24 hours, SBP was elevated by 2 to 3 mm Hg for all stove treatments compared to the control, with the exception of the rocket elbow stove. In another randomized, sham-controlled study, arterial blood pressure responses to fine particle and UFPs exposure from frying sausages and burning candles were not significantly different compared to sham exposures to room air. However, exposure to bread toasting was associated with an increase in SBP for each 10 μg/m³ rise in PM_{10} and $PM_{2.5}$ levels, with the most substantial changes observed 1 hour after exposure (Soppa et al., 2017).

Consistent with our findings, Fedak et al. (2019) also reported no significant differences in DBP 24 h post-exposure. Arterial blood pressure responses to brief exposure to fine particles and UFPs from frying sausages and burning candles were not statistically significant. However, exposure to bread toasting resulted in an increase in SBP for every 10 μg/m³ rise in PM_{10} and $PM_{2.5}$ levels, with the most substantial changes observed one hour after exposure (Soppa et al., 2017). Exposure to frying sausages led to a decrease in forced expiratory volume at one second (FEV1) at both 4 hours and 24 hours post-exposure, whereas exposure to candle burning was associated with an increase in FEV1 at those same time points. In the fully adjusted model, fine particles from candle burning and frying sausages were linked to minor negative changes in lung function. However, no such association was observed for bread toasting and lung function (Soppa et al., 2014).

The variations in blood pressure changes observed in these studies may be due to differences in exposure time, composition, concentrations, and protocols, which complicate physiological interpretation. For instance, these studies involved longer exposure durations (2 hours) compared to our study, which had a 30-minute exposure. Moreover, participants in those studies left the facility 3 to 4 hours after exposure and

returned 24 hours later, commuting by car, bike, or on foot without respiratory protection, potentially exposing them to other environmental factors such as traffic-related air pollution. In contrast, our participants remained in the apartment for the entire 48-hour period to minimize exposure to external sources of pollution.

Previous studies on controlled human exposure to air pollutants have yielded somewhat inconsistent results regarding cardiopulmonary outcomes. A wood-smoke chamber trial found no change in systolic blood pressure (SBP), diastolic blood pressure (DBP) or heart rate (HR) six hours post-exposure (Hunter et al., 2014), whereas a 2-h exposure to diesel exhaust at 200 μg m^{-3} produced persistent SBP elevations for up to 24 h, peaking 30–60 min after exposure, without affecting DBP or HR (Cosselman et al., 2012). Conversely, 2-h graphene-oxide exposures (200 μg m^{-3}) had no measurable impact on HR, SBP, DBP, peak expiratory flow rate (PEFR) or inflammatory markers during the ensuing 4 h (Andrews et al., 2024). Consistent with our findings, previous studies have also reported no significant changes in cardiovascular parameters, including blood pressure and HR (Törnqvist et al., 2007, NightingaleI et al., 2000), or lung function (FEV) (NightingaleI et al., 2000) following 24 hours post-exposure to diesel exhaust. Additionally, pulmonary function (FEV and FVC) remained stable in participants exposed to concentrated air particles (CAPs) and filtered air for two hours during intermittent exercise, 20 hours post-exposure.

Extending this evidence base, healthy non-smokers underwent five-hour semi-controlled sessions at Amsterdam Schiphol Airport, breathing a mean ultrafine-particle (UFP) concentration of $\approx 5.35 \times 10^4$ cm^{-3} while exercising intermittently inside a mobile laboratory ~300 m from the runways and ~500 m from two highways. Although routine endpoints (spirometry, HR, BP) again remained unchanged, breathomics and metabolomics captured clear biological responses. Multilevel partial least-squares linear discriminant analysis (PLS-LDA) of electronic-nose data discriminated post- from pre-exposure breath-prints with an AUROC of 0.83 overall and 0.98 on high-UFP days (Lammers et al., 2021). Untargeted ^1H-NMR of urine collected 24 h post-exposure revealed significant aviation-UFP-linked depletions in taurine (-0.26 AU), dimethylamine (-0.02 AU) and pyroglutamate (-0.005 AU), metabolites integral to glutathione recycling, redox buffering and nitric-oxide regulation (Selley et al., 2021). Together, these omics-based signals indicate that short-term aviation UFP exposures elicit acute oxidative-stress and endothelial responses that remain invisible to conventional cardiopulmonary metrics, highlighting both the

sensitivity of omics biomarkers and the need to assess whether repeated exposures culminate in long-term vascular injury.

Previous research highlights that short-term particle exposure could influence the autonomic regulation of the cardiovascular system, with some studies suggesting potential effects on both parasympathetic and sympathetic function (Li et al., 2021, Carandina et al., 2024). Heart rate variability (HRV) is a well-established indicator of autonomic nervous system function and balance. Reduced HRV is widely recognized as an unfavorable prognostic biomarker for cardiovascular disease (CVD), as it reflects diminished autonomic flexibility and an impaired ability to respond to physiological and environmental stressors (Bilchick et al., 2002). Our study measured HRV parameters during and control days, and the results indicated no significant changes between the two conditions. This finding aligns with the broader literature, which has reported inconsistent associations between PM exposure and alterations in HRV. A meta-analysis of animal studies demonstrated that short-term exposure to PM can lead to a significant reduction in SDNN, LF, and LF/HF in rodents (Huang et al., 2020). The previous crossover and controlled studies showing mixed and often not consistent results regarding the effect of PM exposure on HRV (Huang et al., 2021).

Frequency-domain parameters such as high-frequency (HF) HRV, often associated with parasympathetic activity, have shown mixed results. For example, some studies have reported a significant decrease in HF during or after 2-hour exposures to UFPs and PM, indicating a potential reduction in parasympathetic function and vagal tone (Devlin et al., 2014, Vora et al., 2014, Devlin et al., 2003, Brook Robert et al., 2014). However, contrasting findings have also been observed, with studies showing a statistically significant increase in HF following 2-hour exposure to UFPs and PM (Fakhri Asghar et al., 2009, Samet et al., 2009). Regarding sympathetic activity, low-frequency (LF) HRV have been used as markers of sympathetic function and sympathetic-vagal balance, respectively (Rowan et al., 2007). While some studies noted increases in LF and LF/HF following exposure to coarse or ultrafine and PM (Samet et al., 2009, Devlin et al., 2014), others reported no significant changes in these parameters (Byrd et al., 2016).

Time-domain metrics such as RMSSD and pNN50, also indicative of parasympathetic function (Rowan et al., 2007), have similarly yielded inconsistent outcomes. While a RMSSD (Zareba et al., 2009) reported an increased following exposure to ultrafine and PM, others reported a decrease in pNN50 post-exposure to

fine PM in elderly populations (Devlin et al., 2003). Similarly, SDNN, a composite HRV measure affected by both sympathetic and parasympathetic inputs, has demonstrated inconsistent findings across studies. For instance, Graff et al. (2009) observed a reduction of 14.4% from baseline in the SDNN for every 10 μg/m³ increase in CAP concentration, whereas Zareba et al. (2009) reported an increase in SDNN at at a UFP exposure level of 10 μg/m³, but not at 25 μg/m³. Graff et al. (2009) did not find any statistically significant changes in other HRV variables such as HF, LF, and pNN50.

However, several other studies examining HRV parameters following UFPs exposure report no significant changes in SDNN, RMSSD, pNN50, LF and HF (Heusser et al., 2019, Tobaldini et al., 2018, Byrd et al., 2016, Huang et al., 2012). These discrepancies highlight the complex and multifaceted nature of autonomic responses to PM exposure, which may vary depending on factors such as participant characteristics, particle size, exposure concentration, and study design. Variations in PM concentration and exposure duration may contribute to these disparities, with some studies suggesting that lower PM concentrations might elicit different responses than higher levels. In our study, the lack of significant changes in HRV parameters between cooking and control days may reflect the relatively short duration of PM exposure during cooking or the influence of individual variability in autonomic responses. Additionally, factors such as age, baseline health, and genetic predispositions (e.g., antioxidant defenses) may play a role in modulating HRV responses. The inconsistencies observed across studies, including ours, underscore the complexity of autonomic regulation in response to PM exposure and highlight the need for further research with standardized methodologies, extended exposure durations, and larger participant cohorts to better elucidate the relationship between PM and HRV.

P-100 respirators are known to filter 100% of both oily and non-oily UFPs larger than 185 nm (CDC, 2009, Rengasamy and Eimer, 2012). They have been shown to be highly effective against UFPs as small as 20 nm (Rengasamy et al., 2008), making them an efficient tool for minimizing UFP exposure. However, their use has been linked to increased CO_2, temperature, and humidity levels in the breathing zone (Acuti Martellucci et al., 2022, Gładyszewska-Fiedoruk and Teleszewski, 2022, Sofronova et al., 2023). The effects of CO_2 buildup on cardiopulmonary function when wearing respirators or surgical masks for extended periods are not consistently reported. Some studies have found that wearing N95 masks can reduce respiratory rate

and oxygen saturation within an hour, while increasing heart rate until the mask is removed (Bao et al., 2023, Tornero-Aguilera and Clemente-Suárez, 2021). Furthermore, wearing face masks has been linked to an increase in pulmonary artery systolic blood pressure (SBP) in both healthy children and those with congenital heart disease (Ahmadi et al., 2024).

Studies have observed that face masks can influence ventilation, oxygen uptake, and heart rate (Lässing et al., 2020)., although other research found no significant effects on blood pressure, lactate, perceived exertion, heart rate, or oxygen saturation (Ahmadi et al., 2024, Lässing et al., 2020), and no differences in heart rate variability (Tornero-Aguilera and Clemente-Suárez, 2021). In one study, healthy volunteers walking in a city center with a facemask experienced lower systolic blood pressure (SBP) (114 ± 10 vs. 121 ± 11 mmHg, $P < 0.01$), though heart rate remained unchanged (91 ± 11 vs. 88 ± 11/min; $P > 0.05$). Over a 24-hour period, heart rate variability (HRV) improved with facemask use (SDNN: 65.6 ± 11.5 vs. 61.2 ± 11.4 ms, $P < 0.05$; LF-power: 919 ± 352 vs. 816 ± 340 ms², $P < 0.05$). These findings suggest that wearing a facemask may mitigate the adverse effects of air pollution on BP and HRV (Langrish et al., 2009).

However, masks can affect CO_2 levels, temperature, and humidity, which is a confounding factor in many studies. In our study, we ensured that participants wore masks during both control and cooking days to eliminate this variable. By comparing each participant's cooking data to their own control day data, we effectively accounted for these effects. P-100 respirators filter out UFPs but allow gases to pass through. Our comparison of the standard and intervention study results indicates that neither gases nor particles from frying had an impact on heart rate, blood pressure, SpO_2, or PEFR in healthy individuals after short-term exposure, even up to 24 hours post-exposure. Our study design improves upon previous research by utilizing more controlled conditions. With protocols enabling within-person comparisons and eliminating confounders such as diurnal exposure from commuting, we conducted a more efficient analysis. In contrast, earlier studies (Soppa et al., 2014; 2017; Fedak et al., 2019) allowed participants to leave the facility and return after 24 hours, potentially exposing them to additional sources or contaminants during their commute, without the use of masks.

Many studies have demonstrated that PM can impair cognitive function, with short-term cognitive decline often linked to environmental exposures. For example,

research on the effects of traffic-related air pollution (TRAP) on commuter health revealed significant cognitive deficits, including slower response times and reduced multitasking ability, associated with increased particulate exposure (Mallach et al., 2023). Satish et al. (2012) highlighted that air quality, particularly elevated CO_2 levels, can significantly impact cognitive performance, with a notable decline in decision-making abilities and other cognitive domains at higher CO_2 concentrations (2,500 ppm). Similarly, Satish et al. (2006) observed significant declines in decision-making and productivity under exposure to latex paint fumes. Shehab and Pope (2019) used the Mini-Mental State Examination (MMSE), the Stroop Color and Word test, and the Ruff 2 & 7 test to assess cognitive performance before and after exposure to candle burning and outdoor commuting. Their findings indicated significant declines in MMSE scores following both candle burning and commuting, as well as a significant reduction in automatic detection speed on the Ruff 2 & 7 test after outdoor commuting. However, no significant differences were observed in Stroop test results under either exposure scenario. While most studies agree on the adverse effects of indoor environmental pollutants on cognitive function, Herbig et al. (2018) found no significant cognitive effects from laser printer emissions, although participants with heightened sensitivity demonstrated increased psychological susceptibility to symptoms. This contrast with our findings may be attributed to differences in exposure types such as cooking-related ultrafine particles in our study versus laser printer emissions in Herbig et al.'s work and the differing sensitivities of the participants. The results demonstrated significant cognitive changes, particularly in recall tasks, due to cooking emissions. Both our findings and those of related studies emphasize the negative cognitive effects of particulate matter, especially ultrafine particles, which can impair recall ability and psychomotor performance.

In our study, short-term exposure to cooking-generated aerosol did not result in statistically significant changes in systolic blood pressure (SBP), heart rate (HR), heart rate variability (HRV), oxygen saturation (SpO_2), or peak expiratory flow rate (PEFR) up to 24 hours after cooking with a gas stove. However, it led to a significant reduction in diastolic blood pressure (DBP) immediately after cooking (~9.35%) and a significant increase in fractional exhaled nitric oxide (FeNO), observed both immediately after cooking (~73.24%) and 30 minutes later (~60.27%). Results from the intervention study indicated that cooking-generated gases alone (from an electric stove) had no significant impact on SBP, DBP, HR, HRV, SpO_2, or PEFR within 24

hours of exposure. However, cognitive testing revealed significant impairments: immediate recall decreased by 10.98% and delayed recall by 18.22%, both observed 90 minutes after cooking. These findings suggest that cooking-generated gases alone have minimal physiological effects on cardiopulmonary indicators but may transiently impair specific domains of memory and processing speed.

6.7.1. Demographic Modifiers: BMI, Age, and Sex

A priori power calculations by our collaborators at the University of Illinois Chicago estimated that a total sample of 60 participants would be required to achieve at least 90 % power to detect the mean treatment effect. However, financial and logistical constraints limited enrollment to 30 participants in each study section (standard and intervention). Because the study enrolled only 30 participants—half of the original target—a fully adjusted factorial analysis (BMI × age × sex × day × time) would have been grossly underpowered and prone to unstable coefficients. To preserve internal validity without inflating Type I or Type II error, we therefore adopted an exploratory, within-subject difference-in-differences (DiD) estimator. The resulting exposure effects—expressed as negative values for decreases and positive values for increases—were stratified by BMI (> 25 vs ≤ 25 $kg \cdot m^{-2}$), age (> 30 vs ≤ 30 years), and sex, and are summarized in Table 6-3 for the standard study (without a P100 mask) and 6-4 for intervention study.

Table 0-9: Percent change (Δ %) in cardiopulmonary outcomes after exposure in standard study (no respirator), stratified by BMI, age and sex—difference-in-differences estimates

Modifier	DBP Δ %	SBP Δ %	HR Δ %	PEFR Δ %	SpO_2 Δ %
BMI > 25 kg m^{-2}	−7.10	−0.77	+4.99	+9.48	+0.39
BMI ≤ 25 kg m^{-2}	−7.98	−2.16	+0.13	−7.38	+0.16
Age > 30 y	−7.54	−1.55	+2.10	−1.06	+0.20
Age ≤ 30 y	−8.93	−3.43	−3.12	−17.90	+0.25
Female	−7.71	−0.82	−0.98	+0.86	−0.21
Male	−8.94	−4.07	+4.08	−5.48	+0.70

In the standard study, diastolic blood pressure (DBP) declined uniformly across all six demographic strata (−7 % to −9 %), indicating that the exposure effect is robust to variation in BMI, age, and sex. By contrast, the systolic blood-pressure (SBP) response was modest, falling by only 0.8 %–4 %. Lean participants (BMI ≤ 25 kg·m^{-2}) experienced nearly a three-fold larger SBP decrease than participants with BMI > 25 (−2.16 % vs. −0.77 %). Younger adults (≤ 30 y) showed a two-percentage-point greater SBP decline than older adults (−3.43 % vs. −1.55 %). Men registered the greatest proportional fall in SBP (−4.07 %), approximately five times that observed in women (−0.82 %). These descriptive results suggest that younger, leaner, and male individuals may experience slightly greater systolic-pressure suppression during acute exposure to cooking aerosols. Heart-rate (HR) responses differed by demographic group. Participants with higher BMI and those over 30 years exhibited clear HR increases after exposure, whereas lean, younger participants remained virtually unchanged. Men showed a 4 % rise in HR, while women exhibited a ~1 % decrease. Peak expiratory flow rate (PEFR) declined most in lean, younger, and male participants, whereas peripheral oxygen saturation (SpO_2) remained essentially stable across all categories.

Table 0-10: Percent change (Δ %) in cardiopulmonary outcomes after exposure in intervention (with respirator), stratified by BMI, age and sex—difference-in-differences estimates

Modifier	DBP Δ %	SBP Δ %	HR Δ %	PEFR Δ %	SpO_2 Δ %
BMI > 25 kg m^{-2}	+8.7	+0.8	+6.8	+3.0	−0.8
BMI ≤ 25 kg m^{-2}	+3.3	+2.2	+0.8	−8.6	+0.5
Age > 30 y	−2.5	+7.2	+2.0	−1.7	+0.4
Age ≤ 30 y	+2.1	+1.4	+1.7	−6.4	+0.3
Female	+2.2	+11.0	−0.9	−4.6	+0.6
Male	+0.1	−0.4	+1.9	−6.7	+0.3

In the intervention study—conducted while participants exposed to fried food on an electric stove wearing a P100 respirator—obese participants exhibited a pronounced pressor response. their DBP rose to more than twice the magnitude observed in lean subjects. DBP increases were similar for adults ≤ 30 years and > 30 years, while women showed a modest rise (+2 %) and men virtually none (+0.1 %). Sex emerged as the dominant modifier of the SBP response. Women experienced a

robust 11 % jump in SBP, whereas men showed no change. Age played a secondary role: participants > 30 years recorded a 7 % SBP rise—roughly five times that of younger adults—whereas BMI had only a minor influence on SBP. HR increased by nearly 7 % in obese participants but remained largely unchanged in lean individuals. Older and younger adults exhibited similarly small HR increases (~2 %), yet men showed a larger HR rise than women. PEFR climbed by ~3 % in heavier, younger, and male participants, while it declined in all other groups. SpO_2 stayed essentially constant across BMI, age, and sex categories, indicating that acute cardiovascular and airway endpoints—rather than blood-oxygen saturation—dominate the physiological response under these masked-frying conditions.

Preliminary patterns point to lean, young, and male participants exhibited the steepest declines in PEFR**.** Conversely, obese cooks reported fewer respiratory symptoms yet still experienced appreciable increases in blood pressure and heart rate, while SpO_2 remains stable. In summary, lean, young, male cooks are most vulnerable in airway function and systolic-pressure metrics, whereas obese and older individuals carry the greater cardiac load. Because these effect-size differences are small and derived from descriptive data, a study powered for interaction testing (≥ 60 participants) is needed to confirm whether BMI, age or sex exerts a statistically significant modifying influence on the acute vasodepressor response to cooking aerosols.

6.8. Concluding remarks

In this controlled-exposure experiment, we investigated the emissions of ultrafine particles (UFPs) and gases generated during cooking, and their effects on blood pressure, heart rate (HR), lung function, oxygen saturation (SpO_2), and cognitive performance in healthy volunteers. Short-term exposure to aerosols (a combination of particles and gases) or gases released during frying chicken and fries—using either gas or electric stoves—did not result in statistically significant changes in systolic blood pressure (SBP), heart rate, heart rate variability (HRV), peak expiratory flow rate (PEFR), or SpO_2 up to 24 hours post-exposure. However, exposure to the combination of UFPs and gases from gas stove cooking led to a significant increase in fractional exhaled nitric oxide (FeNO) and a decrease in diastolic blood pressure (DBP), suggesting a potential acute respiratory response. Additionally, exposure to cooking-

related gases was associated with transient impairments in memory and attention, possibly due to neurocognitive disruption induced by cooking gas emissions.

The investigators highlighted the impact of stove type on indoor air quality, with gas stoves producing higher baseline and peak concentrations of $PM_{2.5}$ compared to electric stoves. This led to a prolonged exposure to elevated particle levels following gas stove cooking. One of the strengths of this study was its controlled design, which eliminated confounders such as diurnal exposure during commuting, and allowed for within-person comparisons. Participants wore masks during both the control and cooking days to minimize potential biases from mask-induced physiological changes.

Regarding cognitive function, HVLT-R and WAIS tests were administered to assess cognitive performance during control and cooking days. We observed a temporary significant reduction in immediate recall performance and delayed recall scores following exposure to cooking emissions. However, recovery was observed by the third day, suggesting these effects were transient. The Recognition Discrimination Index (RDI) showed no significant differences across the control and cooking days, indicating that cooking generated gases emissions did not impair the ability to recognize previously learned words or distinguish them from distractors. Additionally, performance on the WAIS subtests revealed mixed findings. Symbol Search scores showed a significant difference between the pre-cooking measurement on the cooking day and the third-day measurement, indicating an overall improvement in processing speed over time, suggesting that the effect of cooking-emitted gases and practice effects were both present.

Chapter 7: Conclusion

7.1.Summary and main findings

In this study impact of cooking emissions from gas and electric stoves on indoor air quality and short-term health outcomes in healthy individuals was examined. Gas stoves were found to generate significantly higher UFPs concentrations, approximately 1.2 times greater than electric stoves. This increase was linked to the combustion processes inherent to gas stoves, which release reactive gases and localized heat, accelerating oil degradation and increasing particle emissions. $PM_{2.5}$ concentrations followed a dynamic pattern, with levels peaking during frying and decreasing after cooking, while gas stoves caused prolonged exposure to elevated particle levels compared to electric stoves. These findings underscore the significant contribution of stove type to indoor air pollution.

We hypothesized that cooking-generated aerosols and gases could impact cardiopulmonary and cognitive function. Short-term exposure to cooking aerosols—comprising both gases and ultrafine particles (UFPs)—led to a statistically significant decrease in diastolic blood pressure (DBP) immediately after cooking. In contrast, exposure to cooking-generated gases alone for a similar duration did not result in any significant changes in DBP. However, due to limitations in the study design, it is not possible to attribute the DBP reduction solely to the presence of UFPs. These limitations include differences in participant characteristics—particularly age—

between the two exposure groups, as well as variations in emission levels from gas versus electric stoves. To address these issues, future studies should use repeated exposures on the same group of participants under both conditions and ensure consistent use of a single stove type to allow for more accurate comparisons. Beyond the immediate post-cooking period, no statistically significant differences in diastolic blood pressure (DBP) were observed at later time points—up to 24 hours—in either exposure scenario. Similarly, no significant effects were detected in other physiological markers, including heart rate (HR), heart rate variability (HRV), peak expiratory flow rate (PEFR), systolic blood pressure (SBP), or blood oxygen saturation (SpO_2) at any point during the 24-hour monitoring period. In contrast, fractional exhaled nitric oxide (FeNO) proved to be a highly sensitive marker of exposure to cooking emissions. Significantly elevated FeNO levels were observed both immediately and 30 minutes after exposure to emissions from gas stove cooking. Notably, significant time-by-exposure interaction effects were found immediately after cooking. These findings suggest a possible synergistic effect between the gaseous and particulate phases of cooking emissions on airway inflammation over time. The presence of strong interaction effects—despite limited main effects at isolated time points—points to the importance of temporal dynamics in exposure response. This could be attributed to limited statistical power or small effect sizes that make it difficult to detect significant changes at individual time points. Importantly, these time-dependent interaction effects align with toxicological and clinical evidence (e.g., (Pujalté et al., 2017, Andrews et al., 2024, Miller et al., 2017) suggesting that the timing of particle translocation into the bloodstream may influence cardiovascular outcomes.

Cognitive performance, assessed using the HVLT-R (Hopkins Verbal Learning Test–Revised) and WAIS-R (Wechsler Adult Intelligence Scale–Revised) tests, revealed several noteworthy findings. Notably, the symbol search subtest of the WAIS-R showed a significant improvement from the control day to the cooking day at 11:30 a.m. This improvement, along with an overall upward trend by the third day, suggests the presence of a learning effect or increased familiarity with the testing procedures over time.

In contrast, the HVLT-R results showed a significant decline in immediate and delayed recall scores before and 90 minutes after exposure to cooking emissions. The lack of improvement across repeated HVLT-R sessions is likely attributable to the use

of different word lists in each session, which limited the participants' ability to benefit from prior exposure or familiarity. Since each session introduced new verbal material, memory consolidation from earlier sessions could not enhance subsequent performance. If practice effects were present in the HVLT-R, the exposure to cooking-generated gases may have attenuated these effects, preventing performance improvements. Under this scenario, the findings would reflect a combination of both exposure-related cognitive disruption and dampened learning effects. Alternatively, if no practice effect occurred due to the rotating word lists, it could be inferred that cooking emissions had measurable impact on HVLT-R outcomes. Further research using a more tightly controlled design is necessary to distinguish between these possibilities and to conclusively determine the cognitive effects of exposure to cooking-related gases.

In Phase II of the study, we implemented a controlled exposure design that minimized confounding factors such as diurnal exposure during commuting and variability in post-exposure follow-up times, and allowed for within-subject comparisons. To reduce the influence of traffic-related air pollution, participants wore masks during commuting periods. While acute cardiovascular and pulmonary effects were minimal under these controlled conditions, the findings emphasize the critical role of stove type in determining indoor air quality. Notably, aerosols emitted from gas stove use may lead to subtle respiratory effects, as evidenced by elevated fractional exhaled nitric oxide (FeNO) levels.

It is important to recognize that long-term exposure to UFPs (ultrafine particles) occurs through repeated short-term exposures. These cumulative daily exposures may lead to an increased overall particle dose that promotes inflammation, oxidative **stress,** and cellular damage over time. Therefore, investigating the short-term exposure–response relationship provides a valuable foundation for understanding and hypothesizing about the long-term health effects of cooking-related UFPs.

In conclusion, results of the study demonstrate that gas stoves contribute more significantly to indoor air pollution than electric stoves and may induce subtle respiratory changes, as shown by increased FeNO levels. The observed cognitive findings further suggest a transient disruption in memory and attention, potentially due to neurocognitive distraction or mild impairment from exposure to cooking-generated gases. These findings underscore the need for continued research into the chronic

health impacts of repeated exposure to cooking emissions, especially in relation to cognitive and respiratory health.

Based on the results and analyses presented in this thesis, the following key conclusions can be drawn:

- A well-controlled design in Phase II minimized confounding factors such as commuting-related exposures, allowing clearer attribution of health effects to cooking emissions and enabling within-subject comparisons.
- Gas stoves emit higher levels of ultrafine particles (UFPs) compared to electric stoves due to combustion-related processes and higher localized heat, which intensify oil degradation and particle formation.
- Short-term exposure to cooking aerosols (UFPs + gases) was associated with a significant decrease in diastolic blood pressure (DBP) immediately after cooking, while exposure to gases alone did not produce significant cardiovascular effects.
- A significant time-by-exposure interaction effect was observed for diastolic blood pressure (DBP), with changes emerging at 6, 8, and 10 hours post-cooking, suggesting a delayed physiological response to cooking-generated aerosols (UFPs + gases).
- Fractional exhaled nitric oxide (FeNO) levels increased significantly immediately and 30 minutes after exposure to emissions from gas stove cooking, suggesting subtle airway inflammation and highlighting FeNO as a sensitive biomarker of exposure.
- No significant changes were observed in heart rate (HR), systolic blood pressure (SBP), peak expiratory flow rate (PEFR), or peripheral oxygen saturation (SpO_2) during the 24-hour post-exposure monitoring period following exposure to either cooking-generated aerosols or gases.
- Results of the study did not show any significant changes in heart rate variability (HRV) parameters—including high-frequency (HF), low-frequency (LF), standard deviation of NN intervals (SDNN), the proportion of successive NN intervals that differ by more than 50 ms (pNN50), and root mean square of successive differences (RMSSD)—following exposure to either cooking-generated aerosols or gases. This may suggest that short-term exposure to these

emissions does not elicit measurable autonomic nervous system responses under the controlled conditions of this study.

- Cognitive testing using HVLT-R revealed decreased immediate and delayed recall scores following exposure, suggesting potential impairment in memory consolidation or retrieval due to cooking generated gases emissions.

7.2.Answer to the Main Question

To answer the central question of this thesis—*What are the short-term cardiopulmonary and neurological effects of controlled exposure to cooking-generated UFPs and gases, and what are their individual contributions to these health outcomes?*—this work demonstrates that short-term exposure to cooking-generated aerosols, particularly ultrafine particles (UFPs), is associated with measurable acute changes in cardiopulmonary and neurological indicators. Controlled human exposure studies revealed that exposure to aerosols (UFPs + gases) led to a significant decrease in diastolic blood pressure (DBP) immediately after cooking, followed by a time-dependent interaction between exposure and physiological response observed at 6, 8, and 10 hours post-cooking.

Additionally, subtle increases in fractional exhaled nitric oxide (FeNO) were observed immediately and 30 minutes after exposure, indicating airway inflammation. In contrast, cooking-generated particles and gases did not significantly affect heart rate (HR), heart rate variability (HRV), systolic blood pressure (SBP), peak expiratory flow rate (PEFR), or peripheral oxygen saturation (SpO_2). Notably, exposure to cooking gases alone did not elicit these physiological changes. These findings suggest that UFPs play a critical role in mediating the observed responses. Therefore, the individual contribution of UFPs to short-term health effects appears to be more substantial than that of the gaseous components alone.

7.3.The limitations of the current study and future suggestion

Several limitations of this study should be considered. Firstly, a priori power calculations performed with colleagues at the University of Illinois Chicago indicated that a total of 60 participants would be needed to achieve ~90 % power for detecting the planned mean treatment effect. Financial and logistical constraints, however,

limited actual enrollment to 30 participants per study section (standard and intervention). The restricted sample size precluded the use of mixed-effects models with body-mass index (BMI), age, and sex specified as fixed factors and participant as a random effect—an approach that would better account for within-subject correlation and effect modification. Secondly, we also did not explore the chemical composition of the emitted particles and gases in detail, which could provide more specific insights into the mechanisms driving the observed health effects. Secondly, while cognitive tests and cardiopulmonary assessments were conducted, other physiological and biomolecular markers, such as oxidative stress or systemic inflammation, were not included, which could offer a more comprehensive understanding of the biological impacts.

Also, the Omron 10 blood pressure monitor used in this study is not a reference-grade instrument, resulting in higher uncertainties in the measurements compared to those obtained with reference devices. Gabdrashova et al. (2021) compared blood pressure data from 10 participants using an Omron BP monitor and a clinical-grade blood pressure cuff reported a 10% bias in systolic blood pressure (SBP) measurements with the Omron 10. Despite this bias, the observed differences did not impact the analyses in this study, which focused on relative changes in blood pressure (BP) and heart rate (HR) during and after cooking, rather than on absolute values. For future studies requiring precise BP measurements, reference-grade instruments such as Holter monitors or clinical BP cuffs are recommended. Participants were allowed to engage in conversation during the experimental sessions, which may have influenced BP readings (Zheng et al., 2012).

In this study, health parameters were measured before and immediately after the 20-minute cooking exposure, but not continuously during the cooking period itself. This approach may have missed capturing acute physiological responses occurring during exposure, especially in susceptible individuals who could experience immediate effects such as arrhythmias or cardiac events. For future work, it is recommended to incorporate real-time monitoring of cardiopulmonary and neurological parameters throughout the exposure period. Continuous measurement during cooking would provide a more comprehensive understanding of the timing and dynamics of physiological responses and could better identify any transient or rapid changes in vulnerable populations.

This analysis focused on short-term exposures at concentrations representative of typical cooking environments. However, repeated daily exposure may lead to either cumulative biological effects or physiological adaptation, particularly in healthy individuals who might maintain homeostasis within these exposure ranges. Thus, the observed changes may reflect normal physiological variability rather than adverse effects. For future studies, it is important to examine responses to higher concentrations of cooking-generated particles, ideally above typical environmental levels, to better understand the dose-response relationship and potential thresholds for adverse health effects. Conducting such studies under appropriate ethical and regulatory approvals (e.g., IREC) will help clarify whether adaptation occurs and identify vulnerable populations who might be at greater risk.

Although the design of the study allowed us to compare health outcomes between exposure and non-exposure days, the daily particle concentrations varied within each exposure condition. Because the sample size for each concentration stratum was small, we lacked statistical power to model a robust concentration-response relationship (e.g., dose-response curve across multiple concentration bins). Future studies with a larger number of exposure sessions—or a continuous exposure system that can precisely step concentrations—are needed to quantify how incremental changes in particle levels translate into physiological responses.

Future studies should address these limitations by including diverse participant groups, extending the duration of exposure, exploring chemical and molecular mechanisms to enhance the applicability of findings.

7.4.Proposed Future Work

Future research should focus on enhancing our understanding of the health impacts of cooking aerosols by addressing several key areas. One critical aspect is the comparative assessment of gas-phase pollutants and aerosol particles. A two-group, randomized controlled crossover trial would provide a more rigorous study design to investigate the short-term effects of cooking emissions on primary and secondary health outcomes. This trial could be structured into two experimental phases, each consisting of three consecutive 24-hour periods (adaption day, control and exposure days). Participants would complete a three-day session, followed by a washout period, before returning for another three-day session under standard and intervention

conditions. This approach would enhance within-person comparisons while minimizing the influence of external confounders. To quantify the influence of key confounders (age, sex and BMI) with adequate statistical precision, a substantially larger cohort will be required. Power projections indicate that a minimum of 60 participants is only sufficient for detecting the main

Additionally, to better assess the neurological effects of UFP exposure, advanced techniques such as fMRI and EEG could provide deeper insights into short-term cognitive responses. Investigating intervention strategies, such as respirators and air purifiers, over extended periods could also help refine mitigation measures and ultimately improve public health outcomes.

The role of olfactory stimuli, particularly cooking smells, warrants further investigation, as sensory perception may influence both physiological and cognitive responses. Experimental methods to control or mask olfactory stimuli will be crucial to isolating the specific effects of aerosol exposure. Future studies should also explore the dose-response relationship between UFP concentration and health outcomes, such as blood pressure variations. Identifying whether health effects correlate directly with exposure levels and determining thresholds for significant health impacts could inform new regulatory guidelines.

Finally, research on long-term and occupational exposure to cooking aerosols is essential. Prolonged exposure studies could help evaluate chronic health effects, while investigations in occupational settings—such as restaurant kitchens and food industry workplaces—could provide insights into cumulative health risks over time.

7.5.Recommendations for the General Public

These recommendations are intended to minimize exposure to potentially harmful cooking emissions and to promote healthier indoor air quality:

- Use kitchen ventilation systems, such as range hoods, during cooking—especially frying—to effectively reduce exposure to ultrafine particles (UFPs) and gaseous pollutants.
- Prefer electric stoves over gas stoves, when possible, as gas stove frying was associated with greater respiratory and cognitive effects.

- Reduce the frequency and duration of high-temperature frying, particularly indoors, to minimize peak exposure.
- Individuals with respiratory sensitivity or underlying cardiovascular conditions may benefit from additional precautions such as limiting time spent in the kitchen during frying or using personal protective measures (e.g., high-efficiency masks).
- Consider alternatives to frying—such as baking or steaming—as healthier cooking methods from both a nutritional and air quality perspective

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