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Human exposure to aerosol from indoor gas stove cooking and the resulting nervous system responses

Mehdi Amouei Torkmahalleh[1] | Motahareh Naseri[1] | Sholpan Nurzhan[2] | Raikhangul Gabdrashova[2] | Zhibek Bekezhankyzy[3] | Aidana Gimnkhan[3] | Milad Malekipirbazari[4] | Mojtaba Jouzizadeh[5] | Mahsa Tabesh[5] | Hamta Farrokhi[5] | Hossein Mehri-Dehnavi[5] | Reza Khanbabaie[5] | Sahar Sadeghi[1,6] | Ali Alizadeh khatir[7,8,9] | Sergei Sabanov[10] | Giorgio Buonanno[11] | Philip K. Hopke[12] | Flemming Cassee[13] | Byron Crape[14]

[1]Department of Chemical and Materials Engineering, School of Engineering and Digital Sciences, Nazarbayev University, Nur-Sultan, Kazakhstan

[2]Department of Biology, School of Sciences and Humanities, Nazarbayev University, Nur-Sultan, Kazakhstan

[3]Department of Chemistry, School of Sciences and Humanities, Nazarbayev University, Nur-Sultan, Kazakhstan

[4]Department of Industrial Engineering, Bilkent University, Bilkent, Ankara, Turkey

[5]Department of Physics, Neuroscience Laboratory, Babol Noshirvani University of Technology, Babol, Iran

[6]Biomedical Engineering Team, Haj Azizi Hospital, Mazandaran University of Medical Sciences, Sari, Iran

[7]Department of Neurology, School of Medicine, Babol University of Medical Sciences, Babol, Iran

[8]Mobility Impairment Research Center, Babol University of Medical Sciences, Babol, Iran

[9]Clinical Research Development Unite of Rouhani Hospital, Babol University of Medical Sciences, Babol, Iran

[10]Department of Mining, School of Mining and Geosciences, Nur-Sultan, Kazakhstan

[11]Department of Civil and Mechanical Engineering, University of Cassino and Southern Lazio, Cassino, Italy

[12]Department of Public Health Sciences, University of Rochester Medical Center, Rochester, New York, USA

[13]National Institute for Public Health and the Environment, Bilthoven, The Netherlands

[14]Department of Medicine, School of Medicine, Nazarbayev University, Nur-Sultan, Kazakhstan

Correspondence
Mehdi Amouei Torkmahalleh, Department of Chemical and Materials Engineering, School of Engineering and Digital Sciences, Nazarbayev University, Nur-Sultan 010000, Kazakhstan.
Email: mehdi.torkmahalleh@nu.edu.kz

Ali Alizadeh khatir, Department of Neurology, School of Medicine, Babol University of Medical Sciences, Babol, Iran.
Email: a.alizadeh@mubabol.ac.ir

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Abstract

Our knowledge of the effects of exposure to indoor ultrafine particles (sub-100 nm, #/cm^3) on human brain activity is very limited. The effects of cooking ultrafine particles (UFP) on healthy adults were assessed using an electroencephalograph (EEGs) for brain response. Peak ultrafine particle concentrations were approximately 3×10^5 particle/cm^3, and the average level was 1.64×10^5 particle/cm^3. The average particle number emission rate (S) and the average number decay rate (a+k) for chicken frying in brain experiments were calculated to be 2.82×10^{12} (SD = 1.83×10^{12}, $R^2 = 0.91$, $p = 0.0013$) particles/min, 0.47 (SD = 0.30, $R^2 = 0.90$, $p < 0.0001$) min^{-1}, respectively. EEGs were recorded before and during cooking (14 min) and 30 min after the cooking sessions. The brain fast-wave band (beta) decreased during exposure, similar to people with neurodegenerative diseases. It subsequently increased to its pre-exposure condition for 70% of the study participants after 30 min. The brain slow-wave band to fast-wave band ratio (theta/beta ratio) increased during and after exposure, similar

to observed behavior in early-stage Alzheimer's disease (AD) patients. The brain then tended to return to its normal condition within 30 min following the exposure. This study suggests that chronically exposed people to high concentrations of cooking aerosol might progress toward AD.

KEYWORDS
EEG, frying aerosol, neurodegenerative disease, ultrafine particles

1 | INTRODUCTION

Prior studies have reported that residential and commercial cooking significantly increased exposure to ultrafine particles (UFPs).[1,47] Studies regarding the indoor sources of particles have identified cooking as one of the most significant indoor sources of particles.[6,28] Exposure to particles from frying food on a gas or electric stove was demonstrated to be higher than from other indoor sources.[22,29,33,42,51,52] reported that frying produced about 10^{14} particles over 15 min, mostly in the ultrafine size range (<100 nm).[52] Their results showed that $PM_{2.5}$ concentration was three times higher during cooking compared to the non-cooking period. A previous study showed that cooking on gas or electric stoves was a major source of UFP, which resulted in personal exposure to ~10^5 particles.cm^{-3}.[51] In a review by[1] cooking UFP (refers to the food and fuels together) concentrations ranged from 5.7×10^3 to 8.9×10^6 particles.cm^{-3}.[1] Cooking fumes include large numbers of small particles with a high surface area that could enhance their toxicity.[34] Particle sizes emitted from cooking are generally within the ultrafine particle (UFP) range. [1,47,35] showed that cooking can produce particles smaller than10 nm.[35] The particle mode diameter values (the diameter representing the highest particle concentration) during cooking were reviewed by[1] and reported to range between 6–100 nm depending on appliance used and food being cooked.[1]

Thus, given the ability of ultrafine particles to induce adverse health outcomes,[34,38,16,53] studying the effects of cooking aerosol on human health is important, and given the limited literature on this subject, it requires further investigation.

Inhaled nanoparticles may translocate to the brain through two major pathways, including the blood circulation after alveolar deposition and axonal transport from the olfactory mucosa of the nasal into the olfactory bulb[3] that was demonstrated in animal studies.[34,9] The most likely pathways for crossing the blood-brain barrier (BBB) are adsorptive transcytosis and receptor-mediated transcytosis. Nevertheless, there is still a lack of evidence to conclude the transport of nanoparticles across the BBB.

Although animal studies provided new insights on the possible pathways of the translocation of nanoparticles to the brain.[10] Animal studies might not represent the UFP toxicity in humans[2] given differences in anatomy and the composition and morphology of the cooking UFPs compared to the nanoparticles in the animal studies.

An electroencephalograph (EEG) is a device that can detect and record the brain activity generated by the cortex. EEG measures the brain's electrical activity using electrodes placed on the scalp surface. Through these electrodes, the electrical signals are recorded and amplified for processing and monitoring. EEG can reflect changes in the brain due to certain diseases. Higher amplitudes of delta (brain wave with the frequency of 1–3.5 Hz) and theta (brain wave with the frequency of 4.5–7 Hz) were noted in AD patients, especially in the left temporal lobes.[39] Generally, working memory is related to the theta, alpha, and beta bands. [13,19,45] The primary changes in AD patients (mild stage of AD) were associated with increases in theta band power and decreased beta band power compared to a healthy population (HP). In the moderate disease stage, these changes are followed by decreases in alpha band power. The delta band power is the latest change, and it is known to increase during the later stages of the disease. [13,19] For the beta band power, beta 1 (13–18 Hz) showed more distinct changes than the broad beta band.[34] In general, power differences among individuals were most notable for the theta band, followed by the beta and alpha bands, while differences in the delta band were less prominent.[45]

In neurodegenerative diseases such as dementia, the EEG of patients is usually different from that of healthy people. Studies that focused on the brain have evaluated power changes in the temporal, frontal, and parietal regions. [33,42] Pathologic studies and clinical findings found that the hippocampus and medial temporal region start to degenerate during early phase Alzheimer's disease (AD). Other associated cortices degenerate later.[43] The differences in parieto-temporo-occipital regions can be distinguished between healthy and AD patients.[22] The temporal and parietal lobes are the first regions to be influenced in AD and strongly associated with memory.[34] Significant increases in the theta band and decreases in high-frequency power in the temporal regions were reported.[34]

The ratio between RP values from different frequency bands can also suggest AD, particularly at the early stages. Such ratios

Practical Implications

- Long-term exposure to frying aerosol may promote neurodegenerative diseases
- EEG is a reliable tool to assess the impact of cooking aerosol on human brain
- Employing respirator and/or sufficient ventilation during cooking reduces the risk of neurodegenerative diseases

included theta/alpha and theta/(alpha+beta) that fairly distinguish between the healthy group and AD patients. [4,17] The theta/(alpha +beta) and theta/alpha ratios for the AD patients compared to the HP were increased in the frontal, temporal, and posterior regions. Three groups were investigated, including healthy, mild cognitive impairment (MCI), and mild AD. In that study, the theta/alpha ratio of the healthy group was 0.66, while it was increased to 0.92 for the MCI group. In another study between healthy and mild AD that was done by the authors, this ratio was 0.34 for the healthy group, which was increased to 2.25 for mild AD.[8]

Toxicological studies have shown that nanoparticles can translocate to the animal brain upon exposure and may lead to neurodegenerative diseases such as autism spectrum disorder (ASD).[15,24,25] Epidemiological studies also reported associations between exposure to ultrafine particles, and brain cancer[54] as well as neurodegenerative diseases.[26] However, such toxicological and epidemiological observations have not yet been systematically investigated through direct human exposure studies on the progression toward neurodegenerative diseases occurring as a result of short-term and long-term exposures to UFPs. This knowledge gap requires investigation through studies of short- and long-term exposures to UFPs. Before performing the more difficult long-term studies, short-term studies can provide insights into the impacts of inhaled UFPs on the brain.

Studies investigating the short-term impacts of aerosol (gas and particles) on the human brain are limited to diesel engine exhaust[7,12] and smoking.[10,11,55] No study of exposures to indoor aerosols including cooking on the brain have been reported except our recent study.[32] We reported that brain activity in the EEG beta band initially decreased upon exposure to electric stove cooking aerosol with subsequent increases during the post-exposure period.[32] An increase in the beta band after exposure to UFPs in diesel engine aerosol was also observed by 7. Both studies reported changes to the frontal cortex of the brain over the post-exposure period. [10,12,36] However, the prior work does not cover a wide range of cooking aerosols particularly given that a gas stove is a known large source of ultrafine particles.[31]

The present study investigated the impact on brain activity from acute exposures to UFPs from cooking on a gas stove. The main objective of this study was to investigate whether the short-term exposures to UFPs from gas stove cooking resulted in changes in the brain wave patterns similar to the brain wave patterns of people with early-stage neurodegenerative diseases. Such a similarity in the brain waves with neurodegenerative diseases suggests that frequent contact with gas cooking particles can increase the risk of neurodegenerative diseases in the future.

2 | MATERIALS AND METHODS

2.1 | Study participants

Thirteen (eight men and five women) non-atopic, non-smoking, healthy (physically and mentally) adults with ages between 18 and 46 years participated in the experiment. The selection criteria are shown in the interview form (FS1) in the supplemental material file. The research team reviewed the interview forms and selected participants according to health criteria for the eligible volunteers. The physical and mental health conditions of the participants were determined based on their responses to the questionnaire. Detailed inclusion and exclusion criteria are specified in the interview form (FS1). Nonsmokers, non-alcohol users, who did not suffer from any known diseases and did not use any drugs or medications, were considered to be healthy. Non-healthy study participants who suffered from respiratory, cardiovascular, or nervous diseases, pregnant women, smokers, former smokers, drug addicts, study participants with a history of head trauma, and professional chefs were excluded from the study.

Cooking habits, daily life activities like physical activities, sleep quality, average time spend outdoors, and general health conditions of the study participants were recorded using a questionnaire (available in Supplementary Material). The study participants were provided with necessary information about the investigation and the cooking protocols before signing the consent form (available in Supplementary Material). The qualified study participants were provided with a code (participant identification number), the address, and the experiments' schedule. They signed a consent form on the day of the experiments. However, the study participants had the right to withdraw any information, including their health condition and test results, from the database at any stage of the process. The ethics committee of the Nazarbayev University approved the experimental procedure of this study under the approval code of 115/12022019.

2.2 | Environmental measurements

Indoor temperature, humidity, and CO_2 concentration were monitored by a Smart Meter model AZ-7755, with 1 min manual logging intervals. A DustTrak DRX Model 8533 (TSI, St. Paul) and a Model 3007 condensation particle counter (CPC) (TSI, St. Paul,) were employed to simultaneously monitor PM_1, $PM_{2.5}$, $PM_{4,}$ and PM_{10}, and particle number concentration (PNC), respectively, with 1-second logging intervals. PM_4 is commonly used in industrial hygiene studies as respirable particles. PM_1 and $PM_{2.5}$ () are particles emitted by higher temperature processes like combustion or are infiltrated from the ambient aerosol. PM10 in indoor air is typically from dust resuspension. Ultrafine particles larger than 10 nm were detected by the CPC that was limited to PNC values of 10^5 particles.cm^{-3}. Thus, they cannot be detected by the DustTrak. Measurement corrections for both systems are described in the supplemental material. The particle instruments were located above the stove at the participant's breathing height. Before each experiment, zero-check were done to ensure the reliability of the instruments.

A digital thermometer (Model 54IIB, Fluke,) equipped with a K type thermocouple probe (Model THS-103–020, ThermoWorks,) with 1 min logging intervals was employed to monitor the cooking temperature (oil and chicken).

All environmental measurements (Indoor temperature, humidity, CO_2 concentration, UFP concentration, and PM concentration) started before cooking (background) and continued until post-exposure time. Oil and chicken temperature were monitored during the cooking period.

The thermometer was a newly purchased instrument. All other equipment underwent calibration and maintenance by manufacture almost a year before the study.

2.3 | Effect assessments

Quantitative electroencephalography (QEEG) (Brain Master Discovery 24 amplifier,) was employed to record the brain wave pattern using an electro-cap with 19 electrodes according to the 10–20 international system (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, and Pz). The left and right ears were connected to two electrodes as a linked-ear reference,[40] and AFz was used as ground. The EEG was sampled at 256 Hz. EEG in eye-closed condition was recorded three times (before, during, and after the exposure).

Details of the setup and data processing are provided in the supplementary materials. The EEG data were processed to obtain the power in each of six frequency bands, including delta (1–3.5 Hz), theta (4.5–7 Hz), alpha (7.5–12 Hz), beta1 (12–15 Hz), beta2 (15–17.5 Hz), and beta3 (18–25 Hz). Relative power (RP) was defined as the percentage of power in any given band relative to the total power. Several electrodes were placed on different cranial positions to measure different lobes of the brain, including frontal (right and left), temporal (right and left), central, parietal (right and left), and occipital (right and left). Left occipital, right occipital, left parietal, right parietal, centro parietal, left central, right central, vertex, left mid temporal, left posterior temporal, right mid temporal, right posterior temporal, left mid frontal, left inferior frontal, left front polar, and right mid frontal, right inferior frontal, right front polar were defined using electrodes O1, O2, electrodes P3, P4, Pz, electrodes C3, C4, Cz, electrodes T3, T5, electrodes, electrodes T4, T6, electrodes F3, F7, Fp1, and electrodes F4, F8, and Fp2, respectively. More information about EEG frequency bands is available in the supplementary materials.

2.4 | Experimental protocol

The experiments were conducted in the kitchen of an apartment located in Babol city, Iran. The apartment was a one-bedroom apartment equipped with a gas stove (Figure S1 shows a photograph of the actual location). The kitchen volume was 18.75 m^3 ((H (2.5 m), L (3.0 m), and W (2.5 m)). Natural ventilation at a rate of 0.6 $h^{-1}$was the driving force for the air exchange. The apartment floor was fully covered by a carpet with a cement roof with no ceiling board. The gaps and cracks across the windows and doors were insignificant. The furnished apartment located away from the highway and traffic emissions. The apartment was located in a region with relatively low noise pollution. The experiment was conducted in August 2019.

The penetration of outdoor particles into the experimental house was limited during the experiments by closing the windows and doors. Windows and doors were closed without any obvious cracks or damages. However, they were opened between experiments to establish a lower initial concentration. Volunteers were allowed to talk, watch, read the news through their phones, and read books to avoid tedium. Factors including indoor temperature and relative humidity (RH), food temperature (oil and meat) were recorded continuously throughout the experiments.

The study exposed volunteers to cooking particles. Thus, we removed other potential indoor sources, such as resuspended dust. We asked the study participants to remove their shoes upon arriving at the apartment to minimize the dust resuspension. We removed sources of volatile organic compounds (VOCs) such as detergents, food additives, and cosmetic materials.

2.5 | Effect experiments

The participants' brain activities were recorded 20 min before the exposure (1) during the exposure, which took 14 min (2), and 30 min after the exposure (3) when background conditions were reestablished. The background $PM_{2.5}$ was 27 $\mu g.m^3$ and was reestablished 5 min after opening the doors and windows by reaching 30 $\mu g.m^3$.

The participants experienced a complete resting condition during the EEG tests that took 5 min per each test. Between EEG measurements, study participants rested without exercises and sat in an upright position at 2.5 m distances from the kitchen. Two experiments were done each day. The first volunteer came in the morning and the second one in the afternoon. Medium size chicken drumsticks were pre-cooked in boiling water for 30 min at low heat. Then, the stove was turned off to stop the boiling, resulting in reducing the chicken temperature to the indoor temperature. A PTFE-coated aluminum pan (22 cm diameter) containing 70 ml sunflower oil and 2 g salt was pre-heated on a gas stove at low heat until the oil temperature reached 120°C. Added salt was used to lower the overall PM emissions during frying.[47,48,50] Then, one chicken drumstick (approximately 160 g) was put into the pan. When the oil temperature reached 200°C, the chicken drumstick was flipped with a fork, and a flame distributor was placed under the pan to prevent overheating the chicken. Two minutes after flipping the drumstick, the peak UFP concentration was attained, and the second EEG recording was conducted for 5 min. The gas stove was turned off when the second EEG measurement was completed, and the windows were opened.

2.6 | Control experiments (simulated cooking)

Eight study participants were selected to simulate the cooking and post-cooking procedure. The study participants stood next to the stove during the cooking period while the stove was off (no

emissions and no exposure more than the background) and sat in a living room during the post-cooking period. This control experiment (providing controls for the study) resulted in no additional exposure to particles beyond the indoor background. The brain activity of the participants was monitored during control experiments. The background concentration during the control experiments was similar to the background concentration during the cooking experiments.

2.7 | Data analyses

Statistical analyses were performed in this study to compare the cooking and non-cooking periods with respect to their effect on the human brain. The effects of the cooking period on the brain could have resulted from different exposure variables, including UFPs, particle mass, and emitted gases. The relative power data failed the Shapiro-Wilk normality test. The non-parametric Friedman test[18] was applied given the same characteristics were measured for each subject at different times or under several different conditions. The Friedman test detects any overall differences among related means given a null hypothesis (H_0) that the populations represented by the multiple conditions have the same distribution of scores. The alternative hypothesis would state that at least one related population's distribution of scores is different from the others. If the Friedman test showed a statistically significant difference among the cases, the Wilcoxon test[56] was run like the post hoc test to identify where these differences occurred. Here are the null hypotheses: $H_0 = \mu_1 = \mu_2 = \mu_3$.

H_0 = mean RP/is the same at all time.

H_1 = mean RP is significantly different at one or more time points.

μ is the population mean of RP, and the related groups are the subjects either before, during, and after cooking.

2.8 | Air exchange rate calculation

The air exchange rate was calculated using the decay period of CO2 using the following Equation 1:

$$\ln\frac{(Cin.t - Cin,b)}{(Cin,max - Cin,b)} = -at \quad (1)$$

The negative slope of the graph $\ln\frac{(Cin.t - Cin,b)}{(Cin,max - Cin,b)}$ versus time is the air exchange rate.

$C_{in,t}$: CO2 concentration at time t.

$C_{in,b}$: CO2 background concentration.

$C_{in,max}$: Maximum concentration of CO2.

t: time of the experiment.

a: air exchange rate.

The particle number emission rate was calculated using the mass balance approach[49] shown in Equation 2:

$$\frac{dCin}{dt} = PaCout - (a+K)Cin + \frac{S}{V} \quad (2)$$

C_{in}: indoor number or mass concentration.

C_{out}: outdoor number or mass concentration.

P: penetration coefficient.

a: air exchange rate.

K: deposition rate.

S: emission rate.

V: volume of the kitchen.

The decay rate (a+k) was calculated from solving the equation in the decay period of particles when the stove had been off. By assuming the Cout to be constant, leading to: Equation 3

$$\ln\frac{(Cin.t - Cin,b)}{(Cin,max - Cin,b)} = -(a+K)t. \quad (3)$$

The negative slope of the graph $\ln\frac{(Cin.t - Cin,b)}{(Cin,max - Cin,b)}$ versus time is the decay rate, where Cin, t is indoor number or mass concentration (part/cm^3 or μg/m^3) at any time, Cin,b is background concentration (part.cm^{-3} or μg.m^{-3}), and *Cin*, *max* is maximum concentration (part/cm^3 or μg/m^3).

Wallace et al.[52] solved the mass balance differential equation for the contribution of cooking at any time *t* to the indoor concentration: Equation 4 and equation 5

$$\triangle C = \frac{S}{V(a+k)}\left[1 - \exp(-(a+k)t)\right] \quad (4)$$

$$Ln\triangle C = Ln\frac{S}{V(a+k)} + Ln\left[1 - \exp(-(a+k)t)\right] \quad (5)$$

Δ*C* is the difference between the measured indoor and background concentrations. The y-intercept of the graph *Ln*Δ*C* versus $Ln\left[1 - \exp(-(a+k)t)\right]$ is $Ln\frac{S}{V(a+k)}$. Given (*a*+*k*) and the volume of the hood, S was calculated.

3 | RESULTS

3.1 | Exposure assessment

Figure S7 shows the average UFP concentration variations (1s logging intervals) with time for the frying chicken using a gas stove. The first 14 min shows the cooking time, while the decay period commenced at minute 14 and ended at minute 16. The initial background concentration was approximately 12×10^3 particle/cm^3, while the peak concentration was 3.00×10^5 particle/cm^3. After 16 min (960 s), the background concentration (12×10^3 particle/cm^3) was established. From the 200 s of the experiment (approximately 3 min), an increase in particle number concentration was observed to 600 s (10 min). This time interval was when the chicken was put in the pan and flipped (minute 6) with the oil temperature above 180°C. Between 600 s and 900 s of frying, the particle number concentration remained stable even if cooking continued. Figure S8 presents the $PM_{2.5}$ concentrations in the breathing zone during the cooking.

The PM concentration did not change for the first 6 min (until the oil temperature reached 180°C). A slight increase in $PM_{2.5}$ concentration was observed between minutes 5 and 7 when $PM_{2.5}$ concentrations were approximately 32 μg/m^3 and 36 μg/m^3, respectively. This increase could be due to the sharp increase in chicken temperature from 33.4°C to 68.2°C over this period (Figure S3). The second increase in $PM_{2.5}$ concentration was observed between 8 and 10 min when a peak concentration of 48 μg/m^3 was observed. This increase in $PM_{2.5}$ concentration could be attributed to the increased oil temperature and the bubbles bursting at the surface of the cooking oil.[14]

3.2 | Brain response

3.2.1 | Electrode analysis

Table 1 shows the average relative power (RP) values across the whole brain among the 13 study participants for each band before cooking (Period 1), at the end of cooking (Period 2), and 30 min after the cooking (Period 3).

No statistically significant differences were found between the theta and beta1 bands measurements across all electrodes among the three measurement intervals (Table S1 and Table S2). The delta band results showed statistically significant reductions between periods 1–3 for Pz, between periods 1–2 for P3, between periods 1–2, and periods 1–3 for P4. The alpha band showed statistically significant increases between periods 1–2, and periods 1–3 for P3, P4, Pz, and between periods 1–3 and periods 2–3 for Cz, respectively. The beta2 band showed significant decreases between periods 2–3 for F8 and between periods 1–3 for Pz. The beta3 band showed statistically significant differences between periods 2–3 for FP1, between periods 1–2 and 2–3 for FP2, and between periods 1–2 for F7 (Table S3). Figures S9,S10 show the average RP of the alpha band, where 15.7% of the electrodes (3 out of 19) showed statistically significant differences between periods 1 and 2 (before cooking and at the end of the cooking). Figures S11–S13 present the average RP values for the delta, beta2, and beta3 bands, respectively. Overall, the brain wave patterns could significantly change shortly after the exposure to the inhaled particles (the maximum concentrations corresponded to 0.048 mg/m^3 and 3.06×10^5 particle/cm^3) compared to the background period (on average approximately 0.028 mg/m^3 and 12×10^3 particle/cm^3) (before the exposure).

TABLE 1 Average relative power (RP) values among 13 study participants over the whole brain

Band/exposure time	Period 1	Period2	Period3
Alpha	32.1 ± 12.0	34.8 ± 12.2	35.1 ± 11.4
Theta	18.2 ± 4.9	18.6 ± 5.4	18.4 ± 4.6
Delta	25.6 ± 6.2	24.4 ± 5.9	23.7 ± 5.7
Beta1	8.0 ± 3.1	8.8 ± 4.7	8.4 ± 4.6
Beta2	4.5 ± 1.5	4.2 ± 1.4	4.2 ± 1.1
Beta3	8.4 ± 4.1	7.4 ± 3.3	7.8 ± 3.2

3.2.2 | Lobe analysis

Table 2 summarizes the bands and lobes that experienced statistically significant changes and also the percentage of the population that experienced such changes. Table S2 presents the *p* values for all bands at all lobes during the experiments' three steps. Table S3 shows that delta, alpha, theta, and beta3 bands statistically significantly differed during the three periods of the experiments, while no statistically significant changes were observed for beta1 and beta2 bands. Figure 1 and Figure S14 show that the delta band decreased with time for most lobes. However, only the decrease at the parietal lobe was found to be statistically significant during exposure ($p_{\text{before-during}} = 0.002$) and after the exposure ($p_{\text{before-after}} = 0.027$), compared to the before exposure (Table 3 and Table S3). The recorded relative power (RP) at the parietal lobe was found to be 19.4 ± 6.3, 16.9 ± 6.5, and 16.4 ± 6.8, before exposure, during exposure, and 30 min after the exposure, respectively.

TABLE 2 Different types of brain wave patterns are recorded at different lobes

Number[2]	Lobe	Statistically significant Before-During 1–2	Statistically significant Before-After 1–3	Statistically significant During –After 2–3	Statistically increase or decrease
1	Parietal	Delta (83.33%[1])	Delta (75%)	–	Decrease
2	Parietal	–	theta	–	Decrease (91.67%)
5	Parietal	alpha (75%)	alpha (83.33%)	–	Increase
3	Left temporal	–	alpha	–	Increase (83.33%)
4	Central		alpha	–	Increase (83.33%)
6	Occipital	–	alpha	–	Increase (83.33%)
7	Frontal	beta3 (91.67%) decrease	–	beta3 (75%) increase	decrease – increase

[1] Percentage values refer to the percentage of the study participants who experienced statistically significant changes in the given frequency band and lobe.

[2] Numbers refer to the number of different scenarios where statically significant changes occurred on different brain lobes.

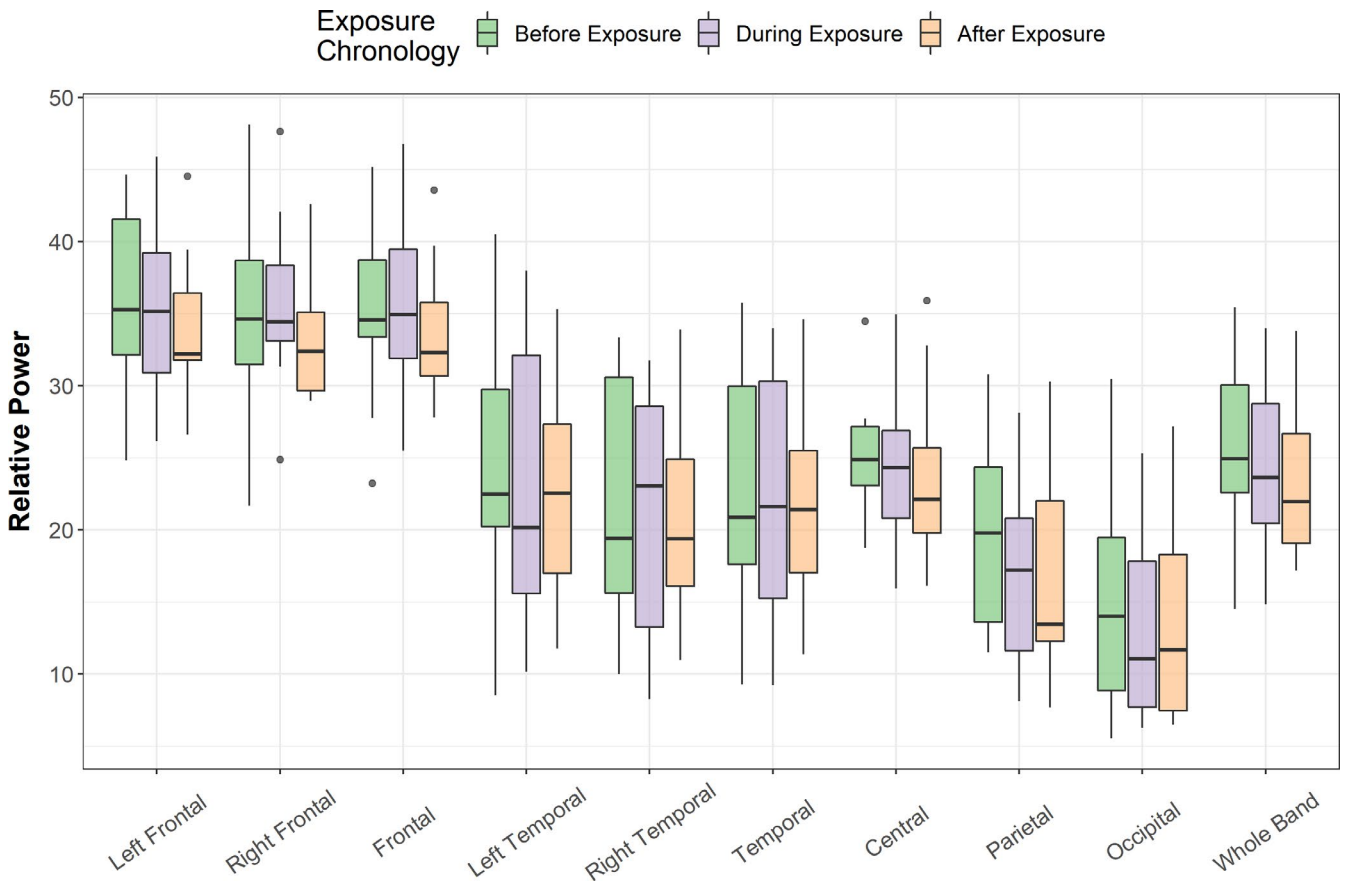

FIGURE 1 Box and whisker plot showing the variations of delta band for different exposure periods for different lobes of the brain-chicken frying (statistical significance was observed for the parietal lobe)

A reverse trend was observed for alpha band compared to the delta band such that the relative power of the alpha band increased with time at all lobes (Figure 2 and Figure S15). This increase in alpha band power was observed after the exposure and was statistically significant for the left temporal (RP_{before} = 31.7 ± 14.5; RP_{after} = 34.7 ± 13.8 with $p_{before\text{-}after}$ = 0.016), parietal (RP_{before} = 31.7 ± 14.5; RP_{after} = 34.7 ± 13.8 with $p_{before\text{-}after}$ = 0.009), central (RP_{before} = 31.7 ± 14.5; RP_{after} = 34.7 ± 13.8 with $p_{before\text{-}after}$ = 0.001), and occipital (RP_{before} = 31.7 ± 14.5; RP_{after} = 34.7 ± 13.8 with $p_{before\text{-}after}$ = 0.009) lobes (Table 3).

Similar increases in the alpha band were observed when exposure to particles from electric stove meat frying.[32] Figure 3 and Figure S16 show that the beta3 band decreased during the exposure compared to the before exposure period but then increased at 30 min after the exposure implying that the brain tended to return to its normal condition. A similar decreasing and increasing variation was observed when people were exposed to the aerosol from electric stove meat frying.[32] However, in the present study, we observed that the initial decrease during the exposure (compared to the before the exposure) with the subsequent increase after the exposure (compared to the end of the cooking), were both statistically significant at the left frontal (RP_{before} = 8.3 ± 2.5,RP_{during} = 7.2 ± 1.9,RP_{after} = 8.1 ± 2.3 with $p_{before\text{-}during}$ = 0.016; $p_{during\text{-}after}$ = 0.034) and frontal lobes (RP_{before} = 8.5±2.5; RP_{during} = 7.4±1.9; RP_{after} = 8.2±2.4 with $p_{before\text{-}during}$ = 0.05; $p_{during\text{-}after}$ = 0.016). Table 3 shows that more than 90 percent of the subjects experienced initial decreases in beta3 upon exposure to the cooking aerosol. However, 66 (left frontal) to 75% (frontal) of the population experienced increases in beta3, 30 min after the exposure, suggesting that the initial decrease in the beta3 was maintained over time in the remainder of the population.[32] observed a similar decreasing and increasing variation with time for the beta3 band. However, statistically, significant increases were observed only over the post-exposure period compared to the before exposure period, at the left frontal and frontal lobes.

The theta band experienced statistically significant decreases in the post-exposure period compared to the before exposure period, but only at the parietal lobe (RP_{before} = 17.2 ± 6.0; RP_{during} = 17.8 ± 7.9; RP_{after} = 16.1 ± 5.8; $p_{before\text{-}after}$ = 0.021; $p_{during\text{-}after}$ = 0.016). No statistically significant changes were observed for beta1 and beta2 at all lobes. Figures S17–S24 show the relative power variations for theta, beta1, and beta2 at all lobes of the brain, respectively. Figure 4 shows the human brain's images, which illustrate where statistically significant changes in bands occurred.

Table S4 presents the results of different studies on theta/alpha and theta/(alpha+beta) among HP and AD patients.

Since changes in early-stage AD patients are associated with increases in the theta power and decreases in the beta power, we investigated a new ratio as theta/beta combining the two frequency

TABLE 3 Relative power changes from step 1 to step 3 in each band power, and a different ratio of band powers obtained in this study

This study	Age	Delta	Theta	Alpha	Beta 1	Beta 2
Frontal	18–46 (Mean 22)	0.354 to 0.356 to 0.336	0.213 to 0.222 to 0.224	0.198 to 0.210 to 0.214	0.064 to 0.070 to 0.067	0.043 to 0.041 to 0.040
Temporal		0.233 to 0.223 to 0.219	0.167 to 0.171 to 0.170	0.325 to 0.354 to *0.350*	0.086 to 0.093 to 0.090	0.048 to 0.044 to 0.046
Parietal		0.194 to *0.169* to *0.168*	0.172 to *0.171* to **0.161**	0.416 to 0.459 to *0.472*	0.088 to 0.094 to 0.090	0.045 to 0.042 to 0.040
Central		0.251 to 0.239 to 0.237	0.214 to 0.221 to 0.210	0.279 to 0.302 to *0.312*	0.080 to 0.083 to 0.080	0.051 to 0.049 to 0.045
Occipital		0.156 to 0.129 to 0.133	0.134 to 0.128 to 0.131	0.495 to 0.535 to *0.536*	0.095 to 0.117 to 0.109	0.039 to 0.034 to 0.036
Whole Brain		0.256 to 0.244 to 0.237	0.182 to 0.186 to 0.184	0.348 to 0.348 to 0.352	0.079 to 0.088 to 0.083	0.044 to 0.041 to 0.041

[1] Italic numbers show significant changes in comparison with step 1.

[2] Bold numbers show statistically significant changes between steps 2 and 3.

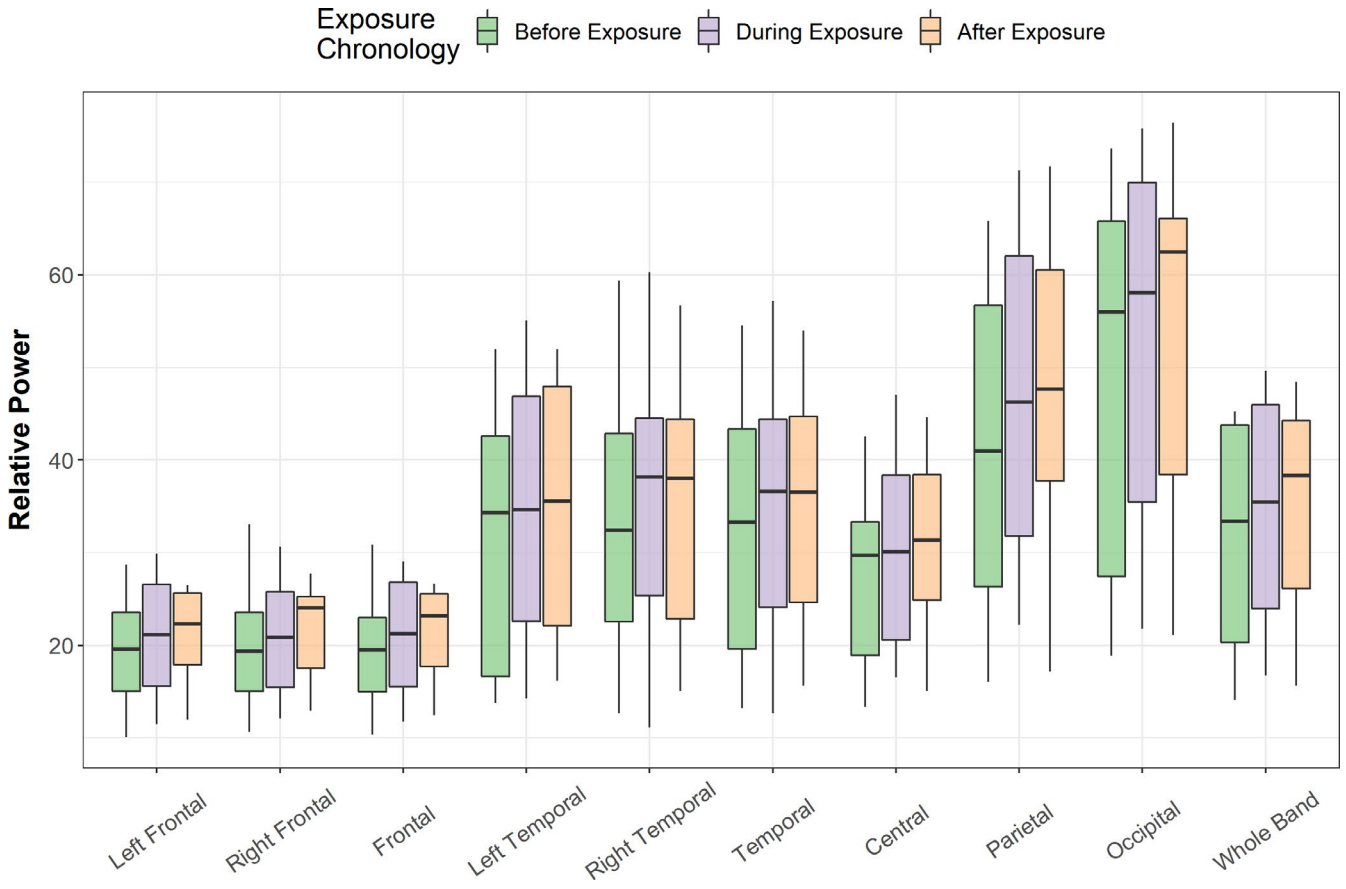

FIGURE 2 Box and whisker plot showing the variations of the alpha band for different exposure periods for different lobes of the brain-chicken frying (statistical significance was observed for the parietal, left temporal, central, and occipital lobes)

bands. To investigate the transient impact of cooking UFPs and gases on the brain and potential similarity between the brain behavior during and after the exposure with the early stage of the AD patients, we examined and compared the theta/beta and theta/beta3 ratios in different studies on AD with the current study (Table 3).

This increasing trend in theta/beta or theta/beta3 ratio upon exposure to cooking aerosol compared to before exposure was quite consistent with the trend observed between HP and early-stage AD patients. However, the increase was more pronounced for AD patients compared to being exposed to the cooking aerosol.

Beta 3	Theta/Alpha	Theta/ (Alpha+Beta)	Theta/Beta	Increase (step 1 to 2)	Theta/Beta 3	Increase (step 1 to 2)
0.085 to *0.074*[1] to **0.082**[2]	1.076 to 1.057 to 1.047	0.546 to 0.562 to 0.559	1.109 to 1.2 to 1.185	0.09	2.51 to *3* to 2.73	0.49
0.098 to 0.087 to 0.090	0.514 to 0.483 to 0.486	0.3 to 0.296 to 0.295	0.720 to 0.763 to 0.752	0.04	1.70 to 1.97 to 1.89	0.27
0.072 to 0.065 to 0.064	0.413 to 0.373 to *0.341*	0.277 to 0.259 to *0.242*	0.839 to 0.851 to 0.830	0.01	2.39 to 2.63 to 2.52	0.24
0.089 to 0.082 to 0.085	0.767 to 0.732 to *0.673*	0.429 to 0.428 to 0.402	0.973 to 1.033 to 1	0.06	2.40 to 2.70 to 2.47	0.3
0.095 to 0.117 to 0.108	0.271 to 0.239 to 0.244	0.185 to 0.159 to 0.166	0.585 to 0.478 to 0.518	0.1	1.41 to 1.09 to 1.21	0.32
0.084 to 0.074 to 0.078	0.523 to 0.534 to 0.523	0.327 to 0.338 to 0.332	0.879 to 0.916 to 0.911	0.04	2.17 to 2.51 to 2. 36	0.34

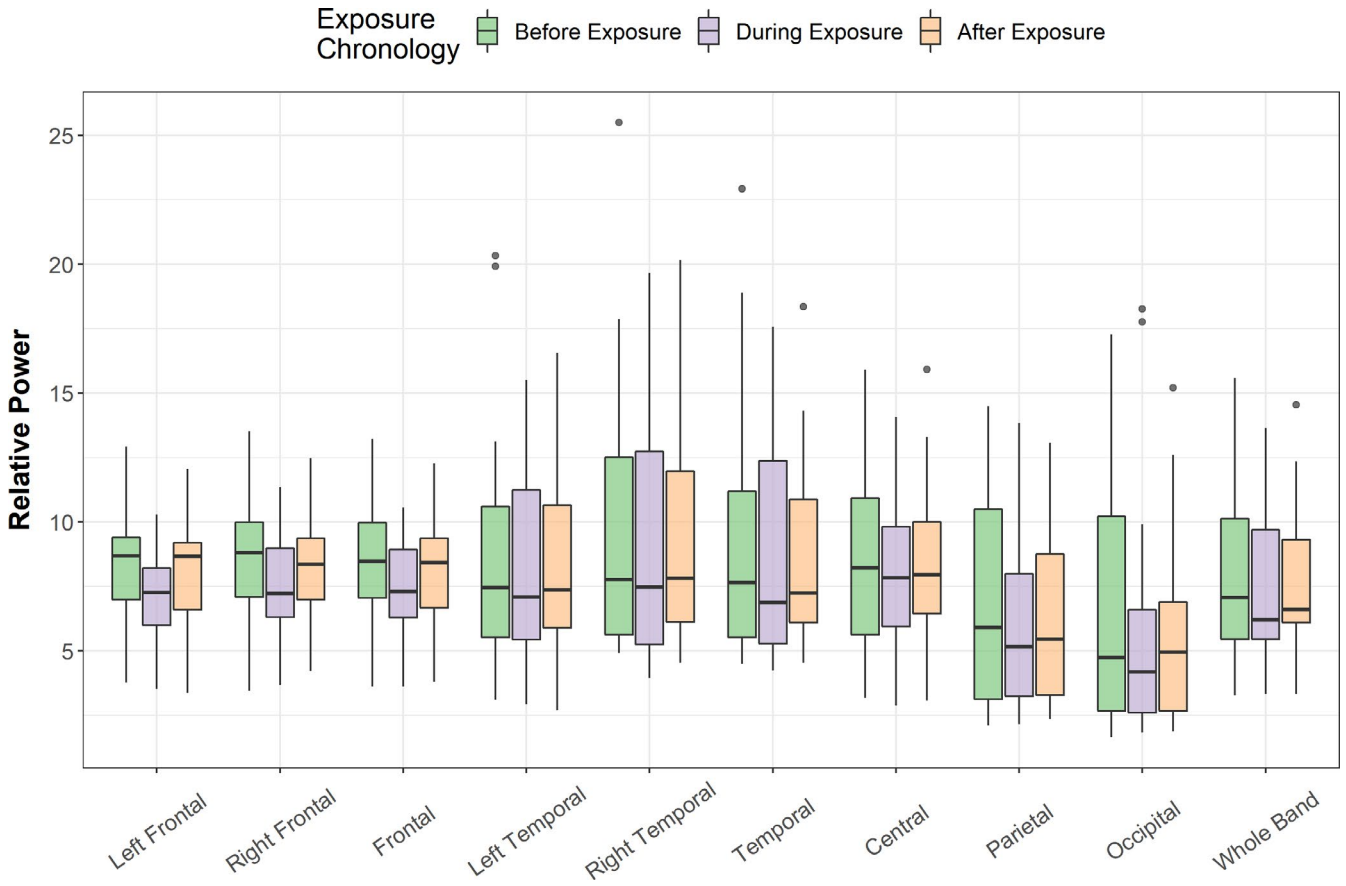

FIGURE 3 Box and whisker plot showing the variations of beta3 band for different steps of exposure at different lobes of the brain-chicken frying (statistical significance was observed for the left frontal, frontal, and parietal lobes)

4 | DISCUSSION

The cooking UFP concentrations were detected by CPC, and it is limited to evaluating the UFP number concentrations down to 10 nm. However, this limitation could not affect our conclusion because the study's aim is not on exposure assessment or dose estimation. These instruments do not measure comparable portions of the particles in indoor air. PNC is dominated by the smallest particle sizes that have little mass and are too small to scatter light effectively.

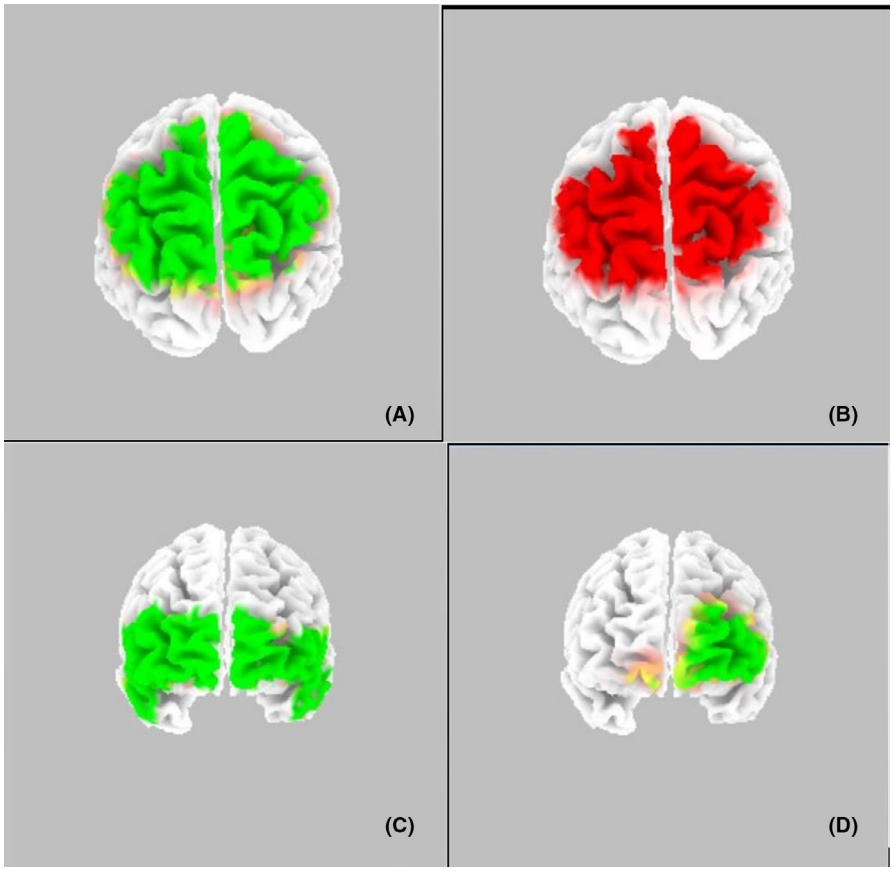

FIGURE 4 The images of the human brain: colors illustrate where statistically significant changes in bands occurred (figure a: decrease in parietal lobs for delta band), (figure b: increase in parietal lobe for the alpha band), (figure c: decrease in frontal lobe for beta3 band), and (figure d: decrease in left frontal lobe for beta3 band)

The average particle number emission rate (S) and the average number decay rate (a+k) for chicken frying in brain experiments were calculated to be 2.82×10^{12} (SD = 1.83 × 1012, R2 = 0.91, $p = 0.0013$) particles/min, 0.47 (SD = 0.30, R2 = 0.90, $p < 0.0001$) min^{-1}, respectively. The results are consistent with the He et al.[22] that found the emission rate to be in the range of $0.2–4 \times 10^{12}$ particles/min in the study of 15 homes during cooking in Australia. The rate of UFP emission during oil-based cooking was reported to be in the range of $0.259–14.83 \times 10^{12}$ particles/min in the literature.[20,46,57]

Our results showed that exposure to gas stove frying induced brain wave pattern (EEG). The brain responded to the inhaled aerosol during cooking and 30 min after. A similar decreasing pattern was observed when people were exposed to the cooking aerosol from meat frying on an electric stove.[32]

Some other studies compared relative power (RP) values between Healthy persons ((HP) and Alzheimer's disease (AD) patients in different frequency band powers. Table S4 presents the RP values for HP and AD subjects reported in other studies. For example, the RP for healthy groups compared with AD patients increased in the delta and theta bands. For high-frequency bands, it was decreased in the alpha, beta1, and beta2.[37] Similar patterns were observed in other studies, as cited in Table S3.

Tables S4,S3 in the main paper show the rp values and rp ratios for different frequency bands studied for hp and ad patients in the literature and this study, respectively. in this study, the eeg measurements before the exposure represent the measurement for healthy populations. thus, it can be compared with the measurements during and after the exposure to investigate possible acute progression toward neurodegenerative diseases (particularly at the very early stages) upon or after exposure to ufps. cooking using gas (as used in the present study) and electric stoves[32] led to reductions in the fast-wave activity beta3 band upon the exposure, similar to what was reported for ad patients (Table S4) and other neurodegenerative diseases (Table S5,Table S6).[21,23,27,30,44] particularly, such reductions in the beta activity were observed in the brain's frontal lobe again similar to the neurodegenerative diseases such as alzheimer's and parkinson's.[23,30,27,21,44] this reduction was statistically significant for gas stove cooking. Nevertheless, the decreases in the brain's slow-wave bands, such as delta and theta, and increases in the fast-wave alpha because of exposure to gas stove aerosol, showed no similarity to neurodegenerative diseases.

Table S5 compares the results of the current study with previous EEG studies performed on human subjects exposed to aerosol from smoking, diesel exhaust, electric stove frying, and EEG results of human subjects with neurodegenerative disorders such as Parkinson

and Dementia. Typically, such diseases showed reductions in the fast-wave bands' activities, including beta, and increases in the activities of the slow-wave bands such as delta, similar to AD. Exposure to combustion fumes such as smoking and diesel exhaust decreased the slow-wave bands' activity, including delta and theta.[7,10,11] Neither type of exposure (smoking and diesel) showed similarity with neurodegenerative disease in delta and beta frequency bands. Similarities in fast-wave bands (beta and alpha) were observed in the human subjects' brain responses exposed to aerosol from smoking, diesel exhaust, electric stove frying, and gas stove frying (the present study).

This study showed changes to the brain wave pattern during and shortly after the exposure that could be likely due to the translocation of the particles to the brain through olfactory pathway. This hypothesis is supported further by the observed changes mainly in the frontal lobes of the brain for beta frequency band. The contribution of the second pathway for the particle translocation that is the blood circulation is less likely given the short post-exposure period in this study.

Evidence exists in the literature, suggesting the translocation of ambient UFPs into the human blood due to chronic exposure. There have been recent reports of black carbon particles found on the fetal side of human placenta[5] and in the urine of healthy children.[41] However, no strong evidence of particle translocation into the human circulation during short-term exposure was reported in the literature. Furthermore, our understanding of the morphology of the cooking particles is poor particularly we do not know whether morphology of the cooking particles facilitate the passage of the particles through lung-blood barrier or not. The presence of the aggregates of particles with chain-like structure was confirmed by Buonanno et al.[6] during grilling using a gas stove. This aggregate structure of the particles might be an obstacle for particles to enter the blood circulation. However, the formation of individual nano-sized spheroid particles was also reported.[6]

5 | LIMITATION OF THIS STUDY

[35] reported that most of the UFPs from frying are smaller than 10 nm. Thus, using a CPC with the lowest cutoff of 10 nm, the concentration of the UFPs is significantly underestimated. However, since this study's focus is not on exposure assessment or dose estimation, this limitation does not impact the present study's conclusions.

The results of our study are based on one-point measurement as reference (before exposure). Other words, all exposure and post-exposure data were compared with a single-point measurement of before exposure representing the normal status of the study participants. This limitation needs to be improved in the future studies by conducting control experiments where the study participants will be available in the experimental apartment without any cooking activities for the same duration as the exposure experiments.

Brain waves may undergo daily variations. Thus, the time of day of the measurements may add to the uncertainties as we examined the study participants in the morning and afternoon. However, we compared each study participant with itself before exposure data in the same part of the day.

Brain EEG could be affected by length or mode of commute, suggesting that the health status of the study participants might have been impacted during the cooking experiments not only due to the exposure to the cooking particles but also due to the exposure to the commuting and traffic particles that may impose health effects during the post-exposure period. We have corrected this issue in our ongoing work by offering N-99 respirators to the study participants during commuting to the experimental house to avoid potential health impacts resulting from the inhalation of the commuting and traffic particles.

We do not know the seen effect is related to the gas or particle or both of them. So, we use the term of aerosol to consider the effect of gas and particle simultaneously.

The emission rate is calculated in this study because it could be helpful to consider two control strategies to reduce the exposure and health effect in future studies by reducing the emission rate and ventilation. Thus, we need to investigate the effect of particles on the brain in different emission rates in future work.

Neurodegenerative diseases are chronic and perennial diseases, and their effects on the brain develop over a long period. However, our study addresses the short-term effects of cooking particles on the brain. Studies investigating the long-term effect of cooking particles on the brain are required.

6 | CONCLUSION

This study is among the first to report the impact on the human brain activity of the exposure to an aerosol produced during frying. The fast-wave beta band changes show similarities with those patterns observed in people diagnosed with Parkinson's and Alzheimer's diseases but revert to normal patterns upon the termination of the exposure. Likewise, the theta/beta ratio increased upon and after exposure to cooking aerosol, similar to the brain behavior of early-stage AD patients, but then the brain tends to revert to the normal condition (before exposure), attaining a normal status as healthy people. This similarity suggests that people who are chronically exposed to cooking aerosol might progress toward AD. This study was conducted with a small number of study participants over short periods of exposure and post-exposure. Although the results of the current study were statistically significant, we cannot draw a conclusion on the significance of the results from biological and clinical points and associations with AD. The current study shows that the brain of the healthy persons behaves like a AD brain immediately during

exposure; then, it reverts to the normal status shortly after the exposure suggesting an oscillatory behavior. However, the results of the current study warrant a hypothesis stating this oscillatory response persists and becomes more severe during the long-term exposure (eg, over 10 years) to cooking aerosol on a successive daily basis such that the brain response may deteriorate with time and after a sufficiently long-term exposure it never reverts to the normal status leading to a status similar to the neurodegenerative disease. For our future work, we plan to conduct a study on the commercial chef and household women who are exposed to cooking fumes on a daily basis.

The changes in EEG during gas stove cooking could be due to both gases and particles, and a definitive conclusion on the relative importance of the gases and particles requires further investigation. New studies should focus on the subjects who are chronically exposed to cooking fumes, such as professional cooks or women who cook at home with poor ventilation conditions, particularly using solid fuels.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Mehdi Amouei Torkmahalleh involved in conceptualization, supervision, writing-original draft. Motahareh Naseri, Aidana Gimnkhan, Raikhangul Gabdrashova, Zhibek Bekezhankyzy, and Sholpan Nurzhan contributed to investigation. Milad Malekipirbazari and Mojtaba Jouzizadeh involved in formal analysis. Mahsa Tabesh and Hamta Farrokhi involved in investigation and formal analysis. Hossein Mehri-Dehnavi, Philip K. Hopke, Flemming Cassee, and Ali Alizadeh khatir (MD) involved in writing-review and editing. Reza Khanbabaie and Giorgio Buonanno involved in resources, and writing-review and editing. Sahar Sadeghi involved in formal analysis, and writing-review and editing. Sergei Sabanov involved in funding acquisition, project administration, and writing-review and editing. Byron Crape involved in writing-review and editing.

ORCID

Mehdi Amouei Torkmahalleh https://orcid.org/0000-0003-0092-1558
Philip K. Hopke https://orcid.org/0000-0003-2367-9661

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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