Level and determinants of serum perfluoroalkyl acids (PFAAs) in a population in Klang Valley, Malaysia

Mohd Redzuan Ramli1,2,∗, Minoru Yoneda2, Mustafa Ali Mohd1, Didi Erwandi Mohamad Haron1, Emmy Dayana Ahmad2

1. Introduction

In recent years, considerable attention has been paid to perfluoroalkyl acids (PFAAs), such as perfluoroalkyl carboxylic acids (PFCAs) and perfluoroalkyl sulfonic acids (PFSA’s). PFAAs are a group of synthetic fluorinated organic chemicals that have been commonly used for industrial and commercial purposes, such as in fire-fighting foams, grease-proof coating for food packaging, shampoos, stain repellents, and surface coatings for carpets and outdoor apparel. Due to their widespread use and unique physicochemical properties, such as bioaccumulation, high stability, and resistance to degradation, PFAAs are now persistently used and occurring on a global scale. Previous studies have documented that PFAAs were detected in air, water, soil, plants, sediments, biota, and food (Domingo and Nadal, 2017; Xiao, 2017). In addition, PFAAs were also found in human blood and urine (Sunderland et al., 2019; Worley et al., 2017).

Many studies have reported the adverse effects of PFAAs on human health, including liver disease (Bassier et al., 2019; Nian et al., 2019); endocrine disorders, such as altered fertility and puberty (Joensen et al., 2009; Lopez-Espinosa et al., 2011); increased total cholesterol (Jain and Ducatman, 2018; Nelson et al., 2010); and immune suppression (DeWitt et al., 2009; Dong et al., 2013). Concerns over the ubiquitous environmental contamination with PFAAs and their effects on human health have led to the restrictions and banning of some PFAAs. For example, perfluorooctane sulfonate (PFOS) and its salts, such as perfluorooctane sulfonyl fluoride (PFOSF) are listed in Annex B of Persistent Organic Pollutants by the Stockholm Convention, and perfluorooctanoic acid (PFOA) and its related precursors were proposed...
as persistent organic pollutants (UNEPA, 2016). Furthermore, perfluorohexanesulfonic acid (PFHxS) and several long-chain PFCAs, such as C8-C14, were included in the REACH candidate list of substances that were of great concern (European Chemicals Agency, 2017). Due to this restriction, shorter chain PFAAs, such as perfluorobutyric acid (PFBA), perfluorooxanoic acid (PFHxA), and perfluoropentanoic acid (PFPeA), are being widely manufactured as alternatives that may have less toxicity, compared with the toxicity of long-chain PFAAs (Buhrke et al., 2013; Luz et al., 2019). Consequently, the presence of these short-chain PFAAs in the environment and biota, including humans, has been increasing (Ji et al., 2012; M. Wu et al., 2017).

Human exposure to PFAAs comes from various routes and sources. Multiple studies have reported dietary intake (Domingo and Nadal, 2017; Ji et al., 2012), indoor dust and air inhalation (Cho et al., 2015; X. Liu et al., 2015), and dermal contact (Bjerregaard-Olesen et al., 2016) as potential routes of PFAA exposure in humans, but these findings were often inconsistent. For example, consumption of fish and seafood has been associated with an increased level of plasma PFAAs in South Korea and Norway (Averina et al., 2018; Kim et al., 2014), but no such association was found in the study by Eriksen et al. (2011) on Danish men. These differences can be attributed to the geographical differences in sources, pollution, and exposure.

Most of the previous studies on the PFAA level in humans focused on populations from developed countries (Eriksen et al., 2011; Gleason et al., 2015; Kärrman et al., 2009) or from nearby areas that were contaminated with PFAAs (Ingelido et al., 2018; Steenland et al., 2013; Worley et al., 2017). Studies on populations from poor and developing countries, particularly in Southeast Asia, are limited. Furthermore, the contributions of specific food types and lifestyle to the PFAA level in human vary by geographical region and population characteristic (Muhammad et al., 2017). In 2004, Kannan et al. analyzed the level of PFAAs in human blood from populations of several countries and detected PFOS and PFHxS in the samples collected from a population in Malaysia. However, this study was limited in sample size and did not explore the exposure route of PFAAs to humans. Therefore, more studies need to be carried out in order to determine the sources of exposure in such a population. The present study was initiated for this purpose.

Specifically, the aim of this study was to assess the levels of nine PFAAs (seven PFCAs and two PFBSs) in the serum samples of the residents of Klang Valley, Malaysia. Klang Valley, which is strategically located at the heart of Selangor State and Kuala Lumpur, is the most densely populated area in Malaysia and has various industries and a high level of traffic. Based on the responses of the participants to a questionnaire, we aimed to investigate the association of PFAA concentrations and the possible significant determinants, such as dietary habits and lifestyle. The results of this study can provide further information on the contamination of polyfluoroalkyl substances (PFAS), especially in developing countries, and subsequently assist the stakeholders in improving the control of these chemicals.

2. Materials and methods

2.1. Collection of serum samples

In May 2016, a total of 219 blood samples were collected from the donors in Klang Valley. The demographic data of this population, as well as the number of cases per category of determinants, are shown in Table 1. A certified medical doctor drew approximately 8 mL of blood from the median cubital vein and placed this blood in a 10-mL red cap Vacutainer (Becton Dickinson, New Jersey, USA) without any additive. The blood was allowed to clot at room temperature and was centrifuged at 3000 rpm for 15 min before being transferred into a 2-mL Nalgene cryovial (Thermo Fisher Scientific, New York, USA) for storage at −80 °C until extraction. The serum samples were extracted within three months after collection. Ethics approval was obtained from the Medical Research Ethics Committee of the University of Malaya Medical Centre.

<table>
<thead>
<tr>
<th>BMI</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 18.5</td>
<td>13 (6%)</td>
</tr>
<tr>
<td>18.5 – 25</td>
<td>103 (47%)</td>
</tr>
<tr>
<td>&gt; 25</td>
<td>58 (26.5%)</td>
</tr>
</tbody>
</table>

2.2. Chemical and reagents

A mixture of native PFCAs and PFBSs (PFAC–MXB), together with a solution of mass-labeled PFCAs and PFBSs (PFAC–MXA), were purchased from Wellington Laboratories (Guelph, Ontario, Canada). All standard and mass-labeled stock solutions were prepared in methanol hypergrade for LC-MS (Merck, Darmstadt, Germany) and were stored at −20 °C. High performance liquid chromatography grade formic acid, methanol, and ammonium acetate were obtained from Fisher Scientific (Hampton, NH, USA). A solution with ≥25% of ammonium hydroxide was purchased from Merck. Ultrapure (Type 1) water was prepared using Milli-Q Direct (MilliporeSigma, Burlington, USA). All reagents were used as received.

2.3. Questionnaire data

On the day of blood collection, a one-on-one interview was conducted by a trained investigator to gather sociodemographic (e.g., sex, weight, height, and age) and lifestyle (e.g., use of consumer products and smoking) information. Further information on dietary habits, such as frequency of food intake, and housing characteristics, such as the use of products that may contain PFAAs, were asked. All donors were given a brief explanation of the study and were asked to sign a consent form prior to the interview session.

2.4. Analysis of PFAAs

The procedures for serum extraction and analyses were slightly modified from a method that was previously reported elsewhere (Kärrman et al., 2005) and are provided as Supplementary Materials. The LODs for all compounds ranged from 0.08 to 0.22 ng/mL. The internal standard recoveries of the nine PFAAs ranged from 71% to 102%. More details on the LODs, LOQs, and recoveries are summarized in the Supplementary Materials (Table S1).

2.5. Statistical analysis

All statistical analyses were performed with SPSS Version 23.0 (IBM, New York, USA). For statistical analysis, the concentrations that were less than the LOD were replaced with the LOD divided by the square root of 2 (LOD/SQR2(2)). Only the PFAAs that were detected in more than 80% of the samples were used for statistical analysis. Natural
log-transformation was performed on the PFAA levels in order to approximate a normal distribution. Spearman’s rank correlation was performed to assess the relationship among PFAA levels in serum. The frequency of food intake was converted from a categorical format into a numerical value of times of serving per week. For example, “Never,” “1–3 times per week,” “4–6 times per week,” and “Once per day” were converted into values of 0, 2, 5, and 7, respectively.

To identify the potential determinants of serum PFAA levels, the unadjusted associations of each category (i.e., dietary habits and lifestyle) with the natural log-transformed serum PFAA levels were examined using multiple linear regression with backward elimination technique. The category of dietary habits included consumption of food from different food style categories (i.e., dietary habits and lifestyle) with the natural log-transformed serum PFAA levels were examined using multiple linear regression with backward elimination technique. The category of dietary habits included consumption of food from different food style categories (i.e., dietary habits and lifestyle) with the natural log-transformed serum PFAA levels were examined using multiple linear regression with backward elimination technique.

All the significant determinants (p < 0.05) in the unadjusted model analyses were examined in a mutually adjusted multiple regression model. Based on previous literature, the other possible determinants that were included in the mutually adjusted models were sex, body mass index [(BMI): weight in kilograms divided by height in meters squared], and age in years. For all the statistical analyses conducted in this study, a p-value below 0.05 was used to indicate statistical significance. Using the equation \[ \left( e^{\beta_i} - 1 \right) \times 100 \], the beta coefficients were back-transformed to observe the percent change in the serum PFAA levels for each determinant.

### 3. Results

#### 3.1. Concentration of PFAAs in the serum samples

The median and interquartile range of the PFAAs in the serum samples are summarized in Table 2. Among the nine PFAAs analyzed, only seven compounds were detected in more than 86.6% of the serum samples: PFBA and perfluorododecanoic acid (PFDoDA) were detected in only 12.8% and 8.2% of the samples, respectively. Moreover, the concentration of PFOA in most of the samples was very low and was close to the LOD for PFHxS.

In addition, the concentration of PFAAs increased with older age (Fig. 1). Similarly, the concentration of PFAAs, except perfluorodecanoic acid (PFDA), slightly increased with increasing BMI.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Median Concentration (ng/mL)</th>
<th>Interquartile Range (ng/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>2.31 (1.92–3.88)</td>
<td>1.42 (0.93–2.00)</td>
</tr>
<tr>
<td>Female</td>
<td>1.93 (1.48–3.36)</td>
<td>1.02 (0.57–1.65)</td>
</tr>
<tr>
<td>Age group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 20</td>
<td>2.46 (2.00–3.20)</td>
<td>1.67 (1.20–2.30)</td>
</tr>
<tr>
<td>21–30</td>
<td>2.93 (2.49–4.15)</td>
<td>1.53 (1.08–2.10)</td>
</tr>
<tr>
<td>31–40</td>
<td>3.04 (2.69–4.17)</td>
<td>1.88 (1.33–2.60)</td>
</tr>
<tr>
<td>&gt; 41</td>
<td>4.16 (3.84–6.42)</td>
<td>2.46 (1.88–3.70)</td>
</tr>
</tbody>
</table>

#### 3.2. Correlation among the PFAAs

The Spearman’s rank correlation coefficients among the PFAAs in all samples are listed in Table S2. PFOA and PFOS were positively correlated (p < 0.05) with most of the PFAAs (r = 0.142 to 0.653). Furthermore, a highly significant positive correlation (p < 0.01) was found among long-chain PFAAs such as PFOA, PFOS, PFHxS, and PFDA (r = 0.193 to 0.653); the strongest correlation was between PFOA and PFHxS (r = 0.653), followed by that between PFOA and PFOS (r = 0.528). The high correlation among these compounds suggested common exposure sources, particularly for PFOS and PFHxS (r = 0.453), both of which were believed to be the endstage products of PFOS degradation.

**Table 2:** PFAAs concentrations (ng/mL) in the serum of the general population of Klang Valley.

<table>
<thead>
<tr>
<th>Age group</th>
<th>Median Concentration (ng/mL)</th>
<th>Interquartile Range (ng/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 20</td>
<td>2.46 (2.00–3.20)</td>
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<tr>
<td>&gt; 41</td>
<td>4.16 (3.84–6.42)</td>
<td>2.46 (1.88–3.70)</td>
</tr>
</tbody>
</table>

**LOD:** Limit of Detection

Interruption range was not calculated for samples with LOD below 25%.
3.3. Univariate analysis of the determinants of serum PFAA exposure levels

In this study population, the consumption of some food was identified as a significant determinant for serum PFAAs (Table S3). Spearman’s correlations between food items indicate that all food variables in this study were not highly correlated between each other (Table S4). Subjects with high consumption of beef tended to show high serum levels of PFDA (p = 0.047) and PFUnDA (p = 0.025), whereas high consumption of lamb/mutton was associated with low serum levels of PFUnDA (p = 0.002) and high consumption of chicken eggs was associated with low serum levels of PFDA (p = 0.032). Interestingly, consumption of chicken, fish, seafood, and pork was not associated with the serum PFAA levels in this study population. Note that food serving and portion size were not considered in this study and that all dietary habits (serving/week) were not significantly associated with serum PFAA levels.

### Table 3
Mutually adjusted regressions analysis of determinants on natural log-transformed serum PFAAs concentration of Klang Valley general population.

<table>
<thead>
<tr>
<th>Effect</th>
<th>PFOA</th>
<th>PFOS</th>
<th>PFHxS</th>
<th>PFNA</th>
<th>PFDA</th>
<th>PFUnDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>0.5 (-0.4, 1.4)</td>
<td>1.5 (0.4, 2.6)</td>
<td>2.0 (0.5, 3.6)</td>
<td>0.9 (-1.2, 3.0)</td>
<td>1.5 (0.2, 2.8)</td>
<td>1.9 (0.2, 3.6)</td>
</tr>
<tr>
<td>Gender (ref = male)</td>
<td>-39.3 (-50.9, -25.1)</td>
<td>-27.0 (-43.2, -6.4)</td>
<td>-60.6 (-71.9, -44.9)</td>
<td>-17.9 (-47.4, 28.1)</td>
<td>0.2 (-26.7, 36.9)</td>
<td>36.4 (-5.2, 96.2)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>0.4 (-1.3, 2.0)</td>
<td>-0.4 (-2.3, 1.5)</td>
<td>0.2 (-2.5, 3.1)</td>
<td>-0.1 (-3.7, 3.6)</td>
<td>0.2 (-2.1, 2.6)</td>
<td>2.8 (-0.3, 5.9)</td>
</tr>
<tr>
<td>Dietary Habits (serving/week)</td>
<td></td>
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</tr>
<tr>
<td>Lamb</td>
<td></td>
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<tr>
<td>Beef</td>
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<tr>
<td>Chicken Eggs</td>
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</tr>
<tr>
<td>Consumer Product Uses (Yes/No)</td>
<td></td>
<td></td>
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<tr>
<td>Non-stick Pan</td>
<td>25.9 (-0.9, 59.9)</td>
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<tr>
<td>Camping Tents</td>
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<tr>
<td>Outdoor Cloth</td>
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<tr>
<td>Leather Sofa</td>
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<tr>
<td>Dental Floss</td>
<td>-17.9 (-31.9, -1.1)</td>
<td></td>
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<tr>
<td>Cosmetic</td>
<td></td>
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</tr>
<tr>
<td>Smoking (Yes/No)</td>
<td>9.0 (-30.0, 69.8)</td>
<td>37.8 (-18.7, 133.7)</td>
<td>-0.6 (-53.2, 111.3)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>R-Squared</td>
<td>0.136</td>
<td>0.101</td>
<td>0.159</td>
<td>0.050</td>
<td>0.015</td>
<td>0.136</td>
</tr>
<tr>
<td>p-value</td>
<td>&lt; 0.001</td>
<td>&lt; 0.001</td>
<td>&lt; 0.001</td>
<td>&lt; 0.001</td>
<td>0.003</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

The bold text indicated a statistically significant result (p < 0.05).

a Reference group is “no” or “never”.
b Beta coefficients were back-transformed by (e^β - 1)*100.
c 95% Confidence Interval.
participants answered tap water as their source of drinking water.

Of the lifestyle determinants evaluated in this study (Table S5), use of nonstick cookware was significantly associated with high PFNA concentration, and use of outdoor clothes and leather sofa were significantly associated with high PFUnDa concentration. In contrast, participants who used camping tents showed significantly low concentrations of PFNA and PFDA, whereas those who used dental floss and cosmetics had a high concentration of PFDA. Smoking was found to be significantly associated with increased serum levels of some PFAAs. Compared with nonsmokers, smokers had higher concentrations of PFOA, PFOS, and PFHxS by 61.4%, 83.5%, and 112.9%, respectively. However, it is important to note that the small number of participants who smoked may have contributed to the large variances with the associations.

3.4. Determinants of serum PFAA exposure levels in the mutually adjusted regression models

In the mutually adjusted regression models for the associations of the significant determinants with the PFAAs in each category (Table 3), the R² for the different PFAAs ranged from 0.050 to 0.159, with all p-values < 0.05. Age was significantly associated with all the serum PFAA levels assessed, except PFDA and PFNA. In addition, PFOA, PFOS, and PFHxS were significantly associated with sex; in particular, the serum levels of those PFAAs were higher in men than in women. Meanwhile, BMI was not associated with any of the PFAAs assessed.

The following were the results after correcting for the other determinants in these models. The associations of nonstick cookware with PFNA, dental floss/cosmetics with PFDA, and leather sofa with PFUnDa remained significant. Compared with participants who did not use nonstick cookware, those who used nonstick cookware had higher concentrations of PFNA (89.6%). Moreover, the PFDA concentration was higher in participants who used dental floss (39.4%) and cosmetics (35.9%) than in those who did not. The concentration of PFUnDa was 44.1% higher in participants who had a leather sofa than in those who did not. For dietary intake, consumption of beef was associated with 29.3% higher levels of PFUnDa, but it was no longer significantly associated with PFDA level. We also found that smoking was no longer significantly associated with all the PFAAs; this suggested that smoking did not account for the high level of PFAAs in this study population.

4. Discussion

The current levels of PFAA exposure in this study population were comparable with those reported in the studies in other countries, such as China (Li et al., 2011), Taiwan (Hsu et al., 2013), Korea (Cho et al., 2015), and USA (Worley et al., 2017; X. Wu et al., 2015), but were higher than those reported in the studies in other Asian countries, such as Singapore (Y. Liu et al., 2017), Vietnam (K. H. Harada et al., 2010), and China (Fu et al., 2014). However, other studies reported much higher serum concentrations of PFAAs (Kärman et al., 2009; Lindh et al., 2012). Compared with the current study, the previous study by Kannan et al. (2004) on a Malaysian population (N = 23) showed a higher median serum concentration of PFOS in both men (13.1 ng/mL) and women (12.7 ng/mL). In addition, contrary to our findings, their results showed a higher concentration of PFHxS in women (2.3 ng/mL) than in men (1.4 ng/mL). However, PFOA was not detected in their study, probably due to a higher LOQ (10 ng/mL), compared with that in our study (0.21 ng/mL).

Considering the fact that the PFAAs were not manufactured in Malaysia, the reason for the higher PFAA concentrations in Malaysia than in the other countries is unclear. The presence of PFAAs in this study population might be attributed to the exposure to products that contained PFAAs or to the consumption of food and water contaminated with PFAAs. Unfortunately, literature regarding the PFAA concentrations in Malaysian food products and water is scarce. One study on fish collected from a Malaysian river detected PFAAs in snakehead (Murakami et al., 2011). In addition, in 2009, a study by Zainuddin et al. (2012) on the presence of PFAAs in Langat River, Malaysia detected PFOS and PFOA concentrations of up to 43.5 and 5.94 ng/mL, respectively. The Langat River is one of the main sources of tap water supply for over 27% of the residents in Klang Valley. Considering the fact that the conventional drinking water treatment system around the world was reported to be inefficient in removing PFAAs (Boiteux et al., 2017; Kucharzyk et al., 2017), some of the untreated PFOA and PFOSO may end up being incorporated in the tap water supply and, subsequently, ingested by humans. Further studies that investigate the sources of PFAA exposure in a Malaysian population are needed.

In this study, the serum levels of PFAAs were found to be significantly associated with age. Similar observations were reported in other studies (Góralczyk et al., 2015; J. H. Lee et al., 2017; Sochorová et al., 2017). For example, a study on PFAAs in an adult population from South Korea showed a pattern of increasing concentrations of PFOS, PFOA, PFNA, PFDA, and PFHxS with increasing age (J. H. Lee et al., 2017). The same pattern for PFOS and PFOA was found in Japan and California (K. H. Harada et al., 2010; X. Wu et al., 2015). The relatively high PFAA levels in older people have been attributed to the long biological half-life of PFAAs. White et al. (2011) and Olsen et al. (2007) found that PFAAs are resistant to human metabolism and have long half-lives of 2.3–12 years; resulting in very slow excretion and increasing concentrations of PFAAs over time in humans. In addition, sustained and widespread lifetime exposure to PFAAs was reported to contribute to the increasing PFAA levels in humans (Calafat et al., 2006).

Similar to previous studies (Cho et al., 2015; Fu et al., 2014; Góralczyk et al., 2015), this present study showed an association between high PFAA levels and sex; in particular, the concentration of PFAAs was higher in men than in women. For example, in South Korea, the concentrations of PFOA, PFNA, PFHxS, and PFOS in whole blood were reported to be higher in men than in women (Cho et al., 2015). A similar observation was found in a population from Henan, China, where the median concentrations of PFOA, PFNA, PFDA, and PFOS were higher in men than in women (Fu et al., 2014). K. Harada et al. (2005) hypothesized menstrual bleeding as a route for elimination of PFAAs in women. That study was supported by the study of J. Lee et al. (2008), who showed that PFAAs were detected in breast milk, maternal blood, and cord blood. These observations suggested that women could effectively eliminate PFAAs by lactation, menstruation, or during pregnancy, thereby resulting in lower serum concentrations of PFAAs in women than in men.

On the other hand, BMI was not significantly associated with any PFAAs in this study; a similar relationship was observed in a residential community in Alabama, USA (Worley et al., 2017). This observation was expected, based on a previous report, which showed that PFAAs were bound mainly to plasma proteins and not to adipose tissues (Sanchez-Garcia et al., 2018).

Various studies suggested dietary intake as one of the major exposure pathways for contamination with several PFAAs in humans (Averina et al., 2018; Papadopoulou et al., 2017). For example, Y. Liu et al. (2017) reported that fish, shellfish, red meat, and poultry were important food contributors to PFAA intake. In this study population, multiple linear regression analysis of the relationship between dietary habits and PFAA concentrations consistently identified consumption of beef as a significant determinant of PFUnDa level. Likewise, Halldorsson et al. (2008) observed an association between serum PFAA concentrations and consumption of beef in a Danish population, although they classified beef as red meat, together with pork and lamb. In addition, PFAAs, including PFUnDa, have been detected in beef samples collected from Sweden and Taiwan (Chen et al., 2018; Vestergren et al., 2012).

Aside from dietary intake, another source of PFAA exposure might be associated with consumer products. Kang et al. (2018) and X. Wu
et al. (2015) reported outdoor equipment, such as waterproof clothes and camping tents, as significant positive predictors of the presence of PFAAs. In addition, similar to our study, the report by Fraser et al. (2012) showed significant associations between PFAA concentrations and some household and office items, such as leather sofas and stain-resistant carpets. The nonpolymer perfluoralkyl chain and polar end-group characteristics account for the excellent water- and stain-resistant properties of PFAAs in leather and carpet products. Indeed, PFAAs have been normally applied during the manufacturing process or to the finished products of carpet and leather; consequently, PFAAs have been frequently found in both these products (Herzke et al., 2012; Kotthoff et al., 2015). This association of PFAA level with the use of leather sofa implied that indoor air can be an important source of PFAA exposure (Fraser et al., 2013).

Intake of food prepared with nonstick cookware was associated with relatively high levels of PFNA; this finding was strongly supported by other studies (Kang et al., 2018; J. H. Lee et al., 2017). PFAAs are used as emulsifiers in the production of polytetrafluoroethylene, which is commonly used as a coating for nonstick cookware (Ellis et al., 2001). An analysis by Herzke et al. (2012) showed that the nonstick layer removed from a nonstick pan contained several PFAAs. During cooking, these PFAAs could seep into food products or evaporate to indoor air and may subsequently end up in the bloodstream of humans.

The associations of the serum levels of PFAAs with cosmetic products and dental floss had been scarcely reported in previous literature. Our findings on the significantly increased serum level of PFDA with the use of dental floss were, to some extent, similar to the results of a study in the USA (Boronow et al., 2019). Begley et al. (2005) found the presence of fluorinated compounds in dental floss. Schultes et al. (2018) reported the presence of perfluoralkyl and PFAS in various cosmetic products in Sweden. The Danish Environmental Protection Agency (EPA) found a total amount of PFAAs of up to 10,700 ng/g in concealers and that almost half of the cosmetic samples contained PFAAs above the 25 ng/g limit (Danish Environmental Protection Agency, 2018). Contact with these products may cause migration and exposure to PFAAs in humans. However, we could not find any previous literature on a significant association between PFAAs and cosmetics to support our finding.

There were several limitations in this study. First, this study had a small number of participants (n = 219) and all the participants were from an urban area in Klang Valley. Therefore, the results may not be generalizable to the populations in the other regions in Malaysia. In addition, the very small ratios of smokers to nonsmokers and of users to nonusers of camping tents may have resulted in the broad 95% confidence interval estimates for these variables. Therefore, caution should be taken when comparing our results with those of other studies. Furthermore, our study used self-reported questionnaires. Therefore, the amount of food consumption may not have been accurately quantified because the information bias (i.e., over and under-reporting) may have underestimated or overestimated the true associations. In addition, the lifestyle variables included in this study were limited. Because PFAAs are frequently detected in various kinds of media, further investigations on drinking water, cleaning products, food packaging, and cooking procedures, in addition to food groups, are also important.

5. Conclusions

In this study on a population of residents in Klang Valley, Malaysia, the levels of PFAAs in serum were associated with age, sex, and use of nonstick cookware, dental floss, cosmetics, and leather sofa but not with BMI and smoking. Moreover, the serum levels of some PFAAs were directly associated with the consumption of beef but inversely associated with the consumption of lamb and chicken eggs. The presence of PFAAs in dietary samples and environmental sources, such as air and water, needs to be further investigated to elucidate the exposure to PFAAs in the Malaysian population.

Conflicts of interest

The authors declare no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijheh.2019.09.005.

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