Toxicology Letters 255 (2016) 36–42

Contents lists available at ScienceDirect

Toxicology Letters

journal homepage: www.elsevier.com/locate/toxlet

Comparative study of oxidative stress biomarkers in urine of cooks exposed to three types of cooking-related particles

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HIGHLIGHTS

- Higher UFP, PM2.5 and PAHs exposure in cookers.
- Urinary 1-OHP, MDA and 8-OHdG reflect COFs exposure.
- RFO may cause increased oxidative DNA damage.
- RWO may cause increased lipid peroxidation.

ARTICLE INFO

Article history:
Received 19 April 2016
Received in revised form 13 May 2016
Accepted 17 May 2016
Available online 18 May 2016

Keywords:
Repeated frying oils
Restaurant waste oils
Fine particulates
Polycyclic aromatic hydrocarbon
Malondialdehyde
8-Hydroxy-2′-deoxyguanosine

ABSTRACT

Objectives: To evaluate how exposure to deep-frying oils, repeated frying oil (RFO) and restaurant waste oil (RWO) affects emission of polycyclic aromatic hydrocarbons (PAHs) and oxidative stress in male restaurant workers.

Methods: The study participants included 236 male restaurant workers in 12 restaurants in Shenzhen. Airborne particulate PAHs were measured over 12 h on each of two consecutive work days. Urinary 1-hydroxypyrene (1-OHP) measurements were used to indicate cooking oil fumes (COF) exposure, and urinary malondialdehyde (MDA) and 8-hydroxy-2′-deoxyguanosine (8-OHdG) were adopted as oxidative stress markers.

Results: The production and emission rates of ultrafine particles (UFPs) and $PM_{2.5}$ were higher in the exposed groups than in the control group. The concentrations of summed PAHs were in the order of RFO-frying group > RWO-frying group > deep-frying group > unexposed control group. Urinary 1-OHP was found to be a significant predictor of elevated urinary MDA and 8-OHdG concentrations (all, $P < 0.05$). UFPs were a significant predictor of elevated urinary 8-OHdG for restaurant workers ($P < 0.05$). The RFO- and RWO-frying groups had higher mean urinary concentrations of 1-OHP, MDA and 8-OHdG than the control group ($P < 0.05$). RFO exposure was found to be a significant risk factor for elevated urinary 8-OHdG and RWO exposure was found to be a significant risk factor for elevated urinary MDA (both, $P < 0.001$).

Conclusions: Concentrations of urinary 1-OHP, MDA and 8-OHdG reflect occupational exposure to PAHs from COFs and oxidative stress in restaurants workers. Exposure to RFO may cause increased oxidative DNA damage, and exposure to RWO might cause increased lipid peroxidation.

1. Introduction

Cooking is an important source of human exposure to particles less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) and ultrafine particles (UFPs) less than 100 nm in diameter (Torkmahalleh et al., 2012). Wallace and Ott (2011) conducted a 3-year study in a variety

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http://dx.doi.org/10.1016/j.toxlet.2016.05.017
0378-4274/

of microenvironments, including 3 homes, 2 cars and 22 restaurants. They found that cooking was one of the greatest sources of UFP exposure. In that regard, frying has been found to be associated with the highest particle emission rates. Most people are exposed to cooking oil fumes (COFs) on occasion, but such exposure is a significant occupational hazard for cooks (Ke et al., 2009; Lai et al., 2013).

Deep-frying is a cooking method in which food is immersed in hot oil. The correct deep-frying technique requires the temperature of the oil to be maintained at between 163 °C and 200 °C. Repeated frying oil (RFO) results from the repeated use of oil heated to high temperatures, such as that reused multiple times for deep frying (Flores-Alvarez et al., 2012). Restaurant waste oil (RWO) is refined oil recycled from restaurant grease-trap waste. Also known as hogwash oil, trench oil or gutter oil, RWO can be used to generate fuel oil and methane (Kobayashi et al., 2014), but cannot be reused in restaurants. However, it can be used illegally or inadvertently, and if so it has the potential to cause specific problems related to oil fumes.

The repeated use of deep-frying oil has been observed in fast-food restaurants, and Chinese-style cooking involves stir-frying and deep-frying food in oil or preheating oil before adding food. Thus, cooks may inhale high concentrations of COFs in these situations (Flores-Alvarez et al., 2012; Ke et al., 2009; Lai et al., 2013).

In previous studies, Chinese-style cooking was found to contribute approximately 30% to the indoor concentration of particles sized from 0.5 to 5 μm (Liao et al., 2006; Zhang et al., 2010). Cooking activities can involve the emission of gaseous pollutants and particulate matter (PM), both of which affect health (Abdullahi et al., 2013; Chiang et al., 1999). For example, polycyclic aromatic hydrocarbons (PAHs) and aldehydes emitted during cooking activities have been shown to have potential carcinogenic effects on both humans and animals (Abdullahi et al., 2013; Katragadda et al., 2010; Wei et al., 2010; Zhu and Wang, 2003). PM emitted from COFs has been associated with respiratory problems, lung cancer and cardiopulmonary death (Li et al., 2003; Martins et al., 2013; Mu et al., 2013). Due to their smaller size and larger surface area, UFPs have been shown to be more toxic to laboratory animals and humans than larger particles (See and Balasubramanian, 2008; Wan et al., 2011).

The pyrene metabolite 1-hydroxypyrene (1-OHP) in urine can be used as a biological marker for exposure to PAHs (Liu et al., 2006; Mukherjee et al., 2002). The metabolism of PAHs through the covalent binding of the metabolite to DNA and through the generation of reactive oxygen species from one-electron redox cycling may cause oxidative damage to DNA (Liu et al., 2006; Mukherjee et al., 2002).

Malondialdehyde (MDA) is a biological marker of lipid peroxidation resulting from oxidative stress. Urinary MDA can reflect global oxidative status in the human body (Pan et al., 2008a, b). 8-Hydroxy-2′-deoxyguanosine (8-OHdG) in urine is a biological marker of oxidative stress on DNA (Bowerman 2005; David et al., 2007; Ke et al., 2010; Kim et al., 2004).

Previous studies have provided substantial data on cooking emissions; however, The association among particulate and PAH exposure and the effects on urinary 1-OHP and 8-OHdG levels resulting from COF exposure is still poorly understood (Ke et al., 2009; Lai et al., 2013; Mukherjee et al., 2014).

Although it is difficult to assess the adverse effects of RFO and RWO on human health, we can evaluate how these oils affect PAH emissions in restaurants and the oxidative status of the workers exposed to them. To the best of our knowledge, this is the first investigation of an association between PAH exposure and oxidative stress and carcinogenic potencies in cooks using RFO and RWO.

2. Methods

2.1. Study subjects and sample collection

The experimental protocol conformed to the standards set by the Declaration of Helsinki, and the procedures were approved by the Ethics Committee of Shenzhen Center for Disease Control and Prevention. Informed consent was obtained from all subjects before their participation in the study.

Three hundred twenty-eight male restaurant workers aged 22–28 years who were non-smokers and employed for at least 6 months in 12 restaurants in Shenzhen were invited to participate. Of these workers, 236 who completed both a questionnaire and a health check-up were then recruited (response rate 72%). Ninety-two workers were excluded: 65 could not attend health check-ups, and 27 could not provide eligible urine samples.

The questionnaire was used to elicit the participants' health status, body weight and height, occupational history, smoking status, alcohol and fruit consumption and exposure to possible sources of PAHs during the previous 24 h. The cooks and waiters participating in the study had to be present at work for at least 4 days each week.

The COF exposure classification was based on the self-reported exposure to COFs produced by the types of cooking occurring in the restaurants. The cooks' exposure to PAHs was categorized according to food hygiene inspections and the participants' responses to the questionnaire. The categories were service staff from the same restaurants with hardly any COF exposure (the control group), deep-frying cooks with high levels of COF exposure, RFO-frying cooks identified by food hygiene inspections in fast-food restaurants who were later confirmed to have been exposed to COFs from RFO, and RWO-frying cooks identified by food hygiene inspections in hot-pot restaurants who were later confirmed to have been exposed to COFs from RWO. Upon discovery, further use of the RFO and RWO was prohibited. All of the restaurants had exhaust hoods in operation and used similar stove types, cooking temperatures and kitchen exhaust fans.

Pre-shift urine samples were collected on the first day of the work week from both the exposed and control groups. Immediately following the last shift of each work week, the participants were asked about COF exposure during the 24 h before the morning urine sample of 20 mL was collected in a polypropylene tube. Aliquots of these samples (2 × 10 mL) were stored frozen at −20 °C until analysis. Each sample was coded and analyzed without the knowledge of exposure status.

2.2. Exposure measurement

Daily area monitoring for particulate PAHs in cooking area was conducted over 12 h on each of two consecutive work days in all 12 restaurants. Following air sampling, the filters had surrogate standards added and were preserved in brown glassware at −4 °C. The filters were extracted by a series of three 15-min sonications with of a hexane:methylene chloride mixture (volume ratio 1:4). The extracts were concentrated to about 0.5 mL under a gentle stream of nitrogen, cleaned with silica gel and concentrated again to about 200 μL for analysis.

The PAHs and PM were measured using different pieces of equipment. PAH analysis was performed on an API 3000 atmospheric pressure photoionization-tandem mass spectrometer (APPI-MS/MS) from Applied Biosystems/MDS SCIEX (Toronto, Canada), connected to a Waters 2695 high-performance liquid chromatography (HPLC) system (Waters, Milford, MA). For comparison with previous studies of COF exposure, the concentrations of five PAH species in the filter were determined: pyrene (Pyr), benzo(a)anthracene (BaA), benzo(k)fluoranthene (BkF),