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Human cancer risk estimation for 1,3-butadiene: An assessment of personal exposure and different microenvironments

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Abstract

This study estimated the lifetime cancer risk (LCR) attributable to 1,3-butadiene (BD) personal exposure and to other microenvironments, including residential home, outdoor, in-office, in-vehicle, and dining. Detailed life expectancy by country (WHO), inhalation rate and body weight by gender reported by USEPA were used for the calculation, focusing on adult population ($25 \leq$ Age <65). LCR estimation of the adult population due to personal exposure exceeded the USEPA benchmark of 1×10^{-6} in many cities. For outdoor BD exposure, LCR estimations in 45 out of 175 cities/sites (sharing 26%) exceeded the USEPA benchmark. Out of the top 20 cities having high LCR estimations, developing countries contributed 19 cities, including 14, 3, 1, 1 cities in China, India, Chile, and Pakistan. One cities in the United States was in the list due to the nearby industrial facilities. The LCR calculations for BD levels found in residential home, in-vehicle and dining microenvironments also exceeded 1×10^{-6} in some cities, while LCR caused by in-office BD levels had the smallest risk. Four cities/regions were used for investigating source distributions to total LCR results because of their sufficient BD data. Home exposure contributed significantly to total LCR value (ranging 56% to 86%), followed by in-vehicle (4% to 38%) and dining (4 to 7%). Outdoor microenvironment shared highly in Tianjin with 6%, whereas in-office contributed from 2 – 3% for all cities. High LCR estimations found in developing countries highlighted the greater cancer risk caused by BD in other cities without available measurement data.

Keywords: 1,3-butadiene; Lifetime cancer risk; Personal exposure; Microenvironment

1. Introduction

1,3-butadiene (BD) is classified as a carcinogenic compound (Code C29790, National Cancer Institute) (NCI, 2016) or Group 1 carcinogen by International Agency for Research on Cancer (IARC, 2008). Even though BD is easily decomposed in the air, there are still noticeable levels in many urbanized cities, especially in dense traffic and industrial areas. Despite of its toxicity, BD is one of the main materials for the polymer industries, i.e. rubber, plastic, and other chemical industries, posing higher risk to the workers. Major sources of BD are listed as combustion activities, including mobile vehicle exhaust, industrial activities, forest fires, cigarette smokes, and leaks from ships and industrial facilities (USEPA, 2009).

Inhalation is considered as the major pathway of BD to the human body. Inconsiderable amount of this contaminant is exposed through dermal contact and digestion pathway, even though detected levels have been found in some plastic food containers (HHS, 2012). Acute exposure to BD cause eyes, nasal passage, lung, and throat irritations. It can cause headache, fatigue, decreased blood pressure and pulse rate, central nervous system damage and unconsciousness at high BD level exposure. Dermal exposure to BD also causes frostbite (OSHA, 2017).

The chronic effects due to BD exposure, i.e. cancer and cardiovascular system diseases, have been the controversial issues. Some factors such as cigarette smoke and other toxicants (i.e. benzene and styrene) may confound the true chronic effects caused by BD exposure (OSHA, 2017). However, BD is a carcinogenic agent, which was reported to cause DNA damage (Kennedy et al., 2009; Zhang et al., 2012). This toxicant metabolically activates the genotoxic epoxides, resulting in abnormality in DNA adducts and chromosomes (Zhao et al., 2000). One of the most important metabolites of BD is diepoxybutane (DEB) which causes DNA damage by producing ROS and 8-OHdG (Erexson and Tindall, 2000; Pagano et al., 2001).

BD is highly reactive and can be transformed to more toxic pollutants under the photochemical reactions (Angove et al., 2006).

Despite of its toxic properties, BD is commercially produced for polymer industries in the world. It is used as the raw materials in many industries, for example tires, car sealants, plastic bottles, and food wrap facilities (WHO, 2001). In 2013, the global BD market volume was 10,500 kilotonnes and is projected to be 14,180 kilotonnes by 2020 with the annual growth rate of 4.4% from 2014 to 2020 (https://www.grandviewresearch.com/press-release/global-butadiene-market).

Occupational health effect assessment due to BD has been documented in several literatures. People working at styrene-butadiene rubber factory were exposed to extremely high levels of BD (exceeding 100 ppmv for short-term exposure), leading to the increase in leukemia mortality (Cheng et al., 2007). Another study reported that the workers at a Polybutadiene Latex chemical industrial plant in China inhaled extremely high levels of BD, resulting in more DNA and chromosome damage than the non-exposed group or control group (i.e. teachers and students) (Wang et al., 2010). A study conducted by Arayasiri et al. (2010) also mentioned that the traffic policemen in Bangkok were exposed to considerably higher concentrations of BD (4.11 μg m^{-3}) than office policemen (0.37 μg m^{-3}), consequently having higher chance of cancer development.

Although BD contaminant is proved to pose cancer development, the cancer risk assessment has not been adequately studied. Several studies have conducted the experiments to quantify the concentrations of BD by personal exposure or in various microenvironments; however, few studies focus on assessing cancer risk by exposing to this contaminant. Therefore, this study aimed to collect the available levels of BD measured in cities worldwide from the literatures for cancer risk assessment and for comparison at city scale. Details of human body

characteristics of males and females such as inhalation rate and body weight of four age groups (i.e., 0 < Age <15, 15 ≤ Age < 25, 25 ≤ Age <65, and Age ≥ 65) from United States Environmental Protection Agency (USEPA) were employed in calculating the human cancer risk.

2. 1,3-butadiene concentrations

The criteria for selecting BD concentrations to estimate the cancer risk in each city are as follows: (1) data coverage in many cities over the country/region; (2) up-to-date measurement data. Desktop survey tools of Web of Science, PubMed, and Google Search were used to search for available BD concentrations in the literatures. This study focuses on BD levels due to personal exposure and in five different microenvironments, including residential home, outdoor, in-vehicle, in-office, and dining because people spend most of their time in these microenvironments, i.e. 68.7%, 7.6%, 5.5%, 5.4% and 1.8%, respectively (Klepeis et al., 2001). The spending time contributions reported by Klepeis et al. (2001) were used for calculations of all cities to ensure the consistency in calculating human cancer risk of all cities.

2.1 1,3-butadiene concentrations by personal exposure

Personal exposure to the air contaminants better reflects the impacts of daily human activities of both indoor and outdoor environments than exposure to separate microenvironment. There have been few studies that estimated the cancer risk caused by exposing to BD personally. A summary of BD concentrations associated with personal sampling is shown in Table 1. Most of studies have been conducted in Europe (Gustafson et al., 2007; Hagenbjork-Gustafsson et al., 2014; Harrison et al., 2009; Kim et al., 2002; Krusa et al., 2003; Yazar et al., 2011), China (Zhou et al., 2011), the United States (Kinney et al., 2002) and Mexico (Serrano-Trespalacios et al., 2004).

In Europe, the available studies associated with BD personal exposure were found in United Kingdom and Sweden. In United Kingdom, higher levels of BD personal inhalation were found during daytime (average 1.1 μg m^{-3}, max 26.3 μg m^{-3}) than nighttime (0.8 μg m^{-3}, max 7.9 μg m^{-3}) in Birmingham during 1999-2000 (Kim et al., 2002). Similarly, Harrison et al. (2009) measured the personal exposure to BD in four cities in United Kingdom, categorizing into urban (i.e. London, Birmingham), suburban (i.e. Birmingham), and rural (i.e. West Midlands, Wales). The high concentration of BD was found in urban Birmingham (0.56 μg m^{-3}, max 5.95 μg m^{-3}), followed by rural West Midlands (0.51 μg m^{-3}, max 4.03 μg m^{-3}), suburban Birmingham (0.37 μg m^{-3}, max 6.27 μg m^{-3}), rural Wales (0.24 μg m^{-3}, max 1.85 μg m^{-3}) and the lowest was observed in urban London (0.1 μg m^{-3}, max 0.76 μg m^{-3}). Due to the four-city coverage and up-to-date data, the results reported by Harrison et al. (2009) were used to estimate the cancer risk assessment in the current study. In Sweden, four studies were associated with the BD personal exposure. The first two studies, i.e. Krusa et al. (2003) and Yazar et al. (2011) were conducted in Stockholm in 2002 and 2009, respectively. There were no significantly different results between two studies (0.7 vs. 0.5 μg m^{-3} for average values and 3.1 vs. 2.3 μg m^{-3} for max values, respectively). Gustafson et al. (2007) compared the BD personal exposure levels in two different environments, i.e. homes with wood burners (0.33 μg m^{-3}) and without wood burners (0.14 μg m^{-3}) in Hagfors, Sweden. The study by Hagenbjork-Gustafsson et al. (2014) had the most comprehensive data because it was conducted in 5 cities of Sweden. The average concentrations of BD personal exposure in μg m^{-3} were 0.44, 0.45, 0.44, 0.54, 0.15, 0.54 and 0.51 in Umeå (2001), Stockholm (2002), Malmö (2003), Lindesberg (2005), Gothenburg (2006), Umeå (2007) and Malmö (2008), respectively. These values were obviously used for calculation in this study, excepting for the value observed in Stockholm which was replaced by study of Yazar et al. (2011) due to their up-to-date data in the city. The latest data in Umeå (2007) and Malmö (2008) were used

instead of the year 2001 and 2003. Whereas the results of Gustafson et al. (2007) were used for estimating cancer risk in Hagfors, Sweden. The average BD concentrations used in Tianjin (China) and Mexico City (Mexico) were 0.67 μg m^{-3} (Zhou et al., 2011) and 2.9 μg m^{-3} (max 8.3 μg m^{-3}) (Serrano-Trespalacios et al., 2004), respectively. Kinney et al. (2002) monitored the BD personal exposure in New York City during winter and summer season, i.e. 0.87 μg m^{-3} and 1.16 μg m^{-3}, respectively; hence, the average of these two values was utilized for cancer risk assessment in our calculation.

2.2 1,3-butadiene concentration in outdoor microenvironments

The measurements of ambient BD have been conducted in various sites, including urban, suburban, and rural backgrounds, roadside, parking lots, heavily trafficked roads, mountainous regions. Several countries have a nation-wide monitoring campaigns of BD such as the United States, China, Canada, and Australia (Table 2). Most of these countries have set up the monitoring stations to continuously measure the hazardous air pollutants, which are useful for comprehensive risk assessment studies. The sampling intervals were also varied amongst the monitored campaigns, which 24 h duration was the widest duration. The cancer risk caused by outdoor BD were estimated based on city level, depending on the available concentration data. Table 2 summarizes the available outdoor levels of BD measured in the cities around the world.

According to McCarthy et al. (2007), the annual average levels of BD recorded at the nation-wide air quality monitoring stations in the United States were 0.6, 0.4 and 0.3 μg m^{-3} in 1990, 1995 and 2000, respectively (Table 2). Knighton et al. (2009) measured the ambient concentrations of BD in Boston in 2007 by using Photo Transfer Reaction – Mass Spectrometry (PTR-MS) instrument, showing an average value of 0.077 μg m^{-3}. In Houston Texas, Hendler et al. (2010) collected the ambient levels of BD in 2008 at 30 monitoring sites

151 which were ranged from 0.07 to 2.36 μg m^{-3} whereas Reiss (2006) analyzed the BD levels at
152 14 monitoring stations around Texas state, showing an average value of 1.21 μg m^{-3} during
153 1997-2004. Another study conducted during 1999-2000 showed that the levels of BD outdoor
154 environments surrounding 41 homes in New York and Los Angeles were 0.1 and 0.105 μg m^{-3}
155 , respectively (Sax et al., 2004). According to databases of USEPA, the ambient
156 concentrations of BD from 2003 (found at 109 monitoring sites) to 2013 (found at 246
157 monitoring sites) had a decreasing trend. The annual average recording in 2013 of 85 cities in
158 the United States ranged from 0.001 to 2.094 μg m^{-3} (averaging 0.128 μg m^{-3}) (USEPA,
159 2015) and would be used in this study to estimate the cancer risk caused by the outdoor BD at
160 city level because of its up-to-date and wide-coverage data (Table 2). The detailed outdoor
161 BD concentrations in 85 cities (89 sites), United States collected in 2013 are showed in Table
162 S1, Supplementary Information (SI).

163 The Canadian Environmental Protection Service (EPS) reported on the air quality trend
164 analysis during 1990-2001 in 18 cities using the data collected at the air monitoring network.
165 The annual average levels of BD in these cities ranged from 0.008 to 0.4 μg m^{-3}, in which
166 higher BD concentrations were found in urban sites, i.e. Regina, Vancouver, and Calgary
167 than other less densely populated cities such as Saint John, Peterborough, and Kingston (EPS,
168 2004). Similarly, Curren et al. (2006) used more up-to-date and nation-wide VOCs
169 monitoring data from 1995-2003 at 20 urban and rural areas to analyze the ambient BD levels
170 in Canada. The annual average concentrations of BD at urban site were from 0.11 to 0.26 μg
171 m^{-3} while lower values were found at the rural sites (0.006 – 0.037 μg m^{-3}). Another study
172 conducted by Setton et al. (2013) used BD databases from Canadian National Air Pollution
173 Surveillance (NAPS) monitoring system in 2006 for estimating the outdoor cancer risk, with
174 a value of 0.096 μg m^{-3} but the authors did not report detailed levels of outdoor BD in each
175 city. Because of the up-to-date data availability in both urban and rural sites, the ambient BD

levels reported in Curren et al. (2006) were used for the outdoor cancer risk assessment in the current study (Table 2).

The volatile organic compounds (VOCs) samples were collected in 43 Chinese cities during the monitoring campaign in 2001 (Barletta et al., 2005). The outdoor BD levels ranged from 0.04 – 5.53 μg m^{-3}, averaging 1.12 μg m^{-3}; hence these data were applied for the outdoor cancer risk calculation in this study due to its massive sampling size covering 43 cities over mainland China (Table 2, detailed data in Table S2, SI). Another research conducted in Tianjin, China by Zhou et al. (2011) reported the outdoor average levels of BD (i.e. 0.36 μg m^{-3}) using passive sampling method. Similarly, Liu et al. (2016) used the on-line GC to collect and analyze 56 VOCs species in the atmosphere of Jinan from 2010 to 2012 and the authors reported BD level to be around 0.35 μg m^{-3}. Another monitoring program during 2006-2010 in Shanghai led by Cai et al. (2010) found similar results of BD levels (i.e. 0.35 μg m^{-3}) with the data reported in Tianjin (Zhou et al., 2011) and Jinan (Liu et al., 2016). In Hong Kong, Guo et al. (2004) used canister sampling method to collect 21 VOCs species at two air quality monitoring stations of Central & Western station and Tsuen Wan Station, and the average levels of BD were 0.27 and 0.44 μg m^{-3}, respectively in 2001. In the current study, the latest data reported by the Hong Kong Environmental Protection Department (EPD, 2014) at these two stations (0.05 and 0.08 μg m^{-3}, respectively) were used to estimate the outdoor cancer risk in Hong Kong.

In Japan, Laowagul and Yoshizumi (2009) monitored the ambient levels of BD at two sites in Tokyo, i.e. 0.49 μg m^{-3} for Shirogane and 0.91 μg m^{-3} for Hachimanyama. By using data available at monitoring station in Tokyo, Mita et al. (2006) mentioned that BD levels over the city were 0.064 μg m^{-3}. Another study conducted by Higashino et al. (2007) who used the modelling approach to estimate the annual average concentrations in Japan, categorizing into rural and mountainous regions (less than 0.125 μg m^{-3}), suburban (0.25 μg m^{-3}), and urban

(0.5 μg m^{-3}) in 2002. The data reported by Mita et al. (2006) were collected by monitoring methods at the air quality monitoring stations and were used for outdoor cancer risk estimations in Tokyo in this study (Table 2).

There have been scattered studies or researches focusing on VOC measurements in India. According to Sengupta (2011), the annual average BD levels measured in ambient air for 3 cities of Chennai, Delhi and Pune were 2.43, 1.72 and 2.65 μg m^{-3}, respectively; hence the data were used for the outdoor cancer risk calculation in this study (Table 2). In urban sites of Karachi (Pakistan), a VOC monitoring program conducted by Barletta et al. (2002) showed that the average concentration of BD was 1.77 μg m^{-3} (ranged from 0.07 to 7.51 μg m^{-3}). Chen et al. (2001) reported high levels of BD measured in Santiago city, Chile in 1996, i.e. 3.3 μg m^{-3}. These specific data were used for outdoor cancer risk estimation in each city. The VOCs monitoring campaign during 1996-2001 at 10 sites of Australia showed that the overall average concentrations of BD ranged from 0.22 to 0.88 μg m^{-3} (NSW, 2004) and were used for the outdoor cancer risk assessment in Australian cities.

In Europe, there have been several VOCs monitoring programs in some cities. For example, a measurement campaign of 21 oxygenated volatile organic compounds (OVOCs) using adsorbent sampling unit combined with gas chromatography – mass spectrometry (GC-MS) were conducted in Zurich, Switzerland covering four seasons during 2005-2006. The BD levels ranged from 0.09 to 0.2 μg m^{-3} (average 0.155 μg m^{-3}) in spring and summer seasons, from 0.07 to 0.29 μg m^{-3} (average 0.177 μg m^{-3}) in fall season and from 0.11 to 0.27 μg m^{-3} (average 0.199 μg m^{-3}) in winter season (Legreid et al., 2007). In United Kingdom, Dollard et al. (2001) reviewed the source contributions, national emissions, and concentration data at 13 monitoring sites, showing the annual average concentration of BD was 0.56 μg m^{-3}. At industrial sites, the levels of BD could reach to several thousand ppbv. In the current study, data in 2005-2007 reported by Delgado-Saborit et al. (2011) in three United Kingdom cities

(0.06, 0.01 and 0.03 μg m^{-3} for London Eltham, Harwell, and Cardiff, respectively) were used for the outdoor cancer risk estimations. In Sweden, Krusa et al. (2003) analyzed the outdoor BD levels at three sites in Stockholm for year 2002, averaging 0.08 μg m^{-3}, whereas Yazar et al. (2011) conducted the same experiment in 2009, reporting the result of 0.09 μg m^{-3}. Hence, the latter value would be used in this study due to its up-to-date data.

2.3 1,3-butadiene concentrations measured in residential home microenvironments

The levels of BD found in indoor environment were usually higher than those found in outdoor because of the contribution of outdoor source through air exchange and indoor source itself. People spend most of their time indoors, resulting in even higher exposure to BD. There were less studies about the human cancer risk and exposure associated with indoor BD levels than those outdoor environments. The residential home BD concentrations in several cities are summarized in Table 3. In the United States, Sax et al. (2004) measured the BD pollutant at residential homes in New York City, averaging 1.0 and 1.2 μg m^{-3} in winter and summer, respectively, and in Los Angeles, showing 0.5 and 0.2 μg m^{-3} in winter and fall, respectively. The BD levels measured for indoor air at some Canadian homes, averaging 0.12 μg m^{-3} (Setton et al., 2013). More studies have been documented in United Kingdom, for example 0.24 μg m^{-3} reported by Delgado-Saborit et al. (2011) for the home in 3 cities of London, West Midlands and rural South Wales, and 1.1 μg m^{-3} for Birmingham city by Kim et al. (2001). In Hagfors, Sweden, the levels of BD found for home microenvironment were 0.31 and 0.11 μg m^{-3} for wood-burning group and reference group, respectively (Gustafson et al., 2007), which the former was used for LCR estimations in the current study. In Asia, there was only one study reported the home BD levels, i.e. 0.54 μg m^{-3} in Tianjin, China (Zhou et al., 2011). The level of BD measured in Mexico homes was approximately 2.5 μg m^{-3} (Serrano-Trespalacios et al., 2004).

2.4 1,3-butadiene concentration in office, in-vehicle, and dining microenvironments

The BD levels found in offices have been studied in the United States (Loh et al., 2007), United Kingdom (Delgado-Saborit et al., 2011; Kim et al., 2001), China (Zhou et al., 2011) and Thailand (Arayasiri et al., 2010) (Table 4). The concentration of BD monitored in the United States (i.e. 0.2 μg m^{-3}) was considerably higher than values reported in United Kingdom reported by Delgado-Saborit et al. (2011), i.e. 0.08 μg m^{-3}, but still lower than levels of 0.3 μg m^{-3} observed in United Kingdom (Kim et al., 2001), level of 0.25 μg m^{-3} in China and 0.29 μg m^{-3} in Thailand. Loh et al. (2007) reported BD concentrations for all the studied sites, hence, we generally estimated cancer risk for the United States using their data. Similarly, Delgado-Saborit et al. (2011) combined the data of three cities (i.e. London, West Midlands, and South Wales) without reporting BD levels for separate city; hence, we used average value to report cancer risk estimations for these three cities. Whereas, Kim et al. (2001) reported BD levels measured in Birmingham city.

There have had limited studies related to BD levels measured in transportation means. Several studies (as summarized in Table 4) have been reported in the United States (Rodes et al., 1998), United Kingdom (Harrison et al., 2009; Kim et al., 2001), Sweden (Barrefors and Petersson, 1996), China (Zhou et al., 2011), and Ireland (McNabola et al., 2008; O'Donoghue et al., 2007). There was no significant difference in BD concentrations monitored in two United States cities of Sacramento and Los Angeles, averaging 2.25 and 2.97 μg m^{-3}, respectively (Rodes et al., 1998). In United Kingdom, Harrison et al. (2009) measured the in-vehicle BD levels in three cities of London, West Midlands, and South Wales, averaging 0.13 μg m^{-3}, but the authors did not report BD levels for individual city; hence, this value was used for estimating cancer risk for three cities. The BD samples were collected in Birmingham city, United Kingdom (Kim et al., 2001), showing an average of 3.53 μg m^{-3} which was considerably higher than those reported in Harrison et al. (2009). According to Barrefors and

Petersson (1996), commuters travelling in public trains and buses in Sweden were exposed to BD pollutant at 0.54 μg m^{-3} level. Zhou et al. (2011), however, reported slightly higher levels of BD measured in vehicles in Tianjin, China which could reach 0.62 μg m^{-3}. In Dublin of Ireland, there were two studies that showed the BD concentrations found in commuting means (McNabola et al., 2008; O'Donoghue et al., 2007). The higher value, 1.72 μg m^{-3} measured by O'Donoghue et al. (2007) would be used for cancer risk estimation in the current study.

A summary of BD levels monitored in dining locations is given in Table 4. The concentrations of BD measured in dining places such as restaurants, pubs and bars have been documented in some literatures. There have been reported in the United States (Loh et al., 2006), United Kingdom (Harrison et al., 2009; Kim et al., 2001), Ireland (McNabola et al., 2006), and Finland (Vainiotalo et al., 2008). In the United States, Loh et al. (2006) collected the BD samples in Boston city, showing an average of 1.05 μg m^{-3}. Harrison et al. (2009) reported the BD levels found in dining places in three sites (London, West Midlands, and South Wales), averaging 0.7 μg m^{-3}. This study used the average concentration for these three cities. However, the results from Kim et al. (2001) study in Birmingham showed significantly higher concentration (2.25 μg m^{-3}). Lower levels of BD monitored in Dublin, Ireland by McNabola et al. (2006) which was 0.22 μg m^{-3}. The differences in BD levels in restaurants with and without smoking activities were described in one Finns study (Vainiotalo et al., 2008) which were reported to be 2.7 and 0.52 μg m^{-3}, respectively. The average concentration (1.61 μg m^{-3}) was subsequently used for cancer estimations in Helsinki, Finland in this study.

2.5 1,3-butadiene concentrations in other microenvironments

Several studies have reported BD levels measured inside the stores in US (Loh et al., 2006) and UK (Kim et al., 2001). The latter authors also measured BD levels in other

299 microenvironments, i.e. perfume shop, library, lab, train station and coach station. However,
300 this study would not aim at these microenvironments because of the unavailability in people
301 spending time and the BD data of these microenvironments in many cities.

302 **3. Cancer risk estimation**

303 Inhalation is the major exposure pathway of BD. The cancer risks were estimated based on
304 the available literature data on BD levels of several cities. The chronic daily intake (CDI) for
305 a carcinogenic agent (USEPA, 1989), based on exposure duration, exposure frequency, body
306 weight, and lifetime, would be calculated by the following equation:

307 $$CDI=\frac{C_a \times IR \times EF \times ED}{BW \times AL} \quad (1)$$

308 where C_a is the concentration of BD (mg m^{-3}), IR is the rate of inhalation (m^3 day^{-1}), EF is the
309 frequency of exposure (day $year^{-1}$), ED is the duration of exposure (year), BW is the average
310 body weight (kg), and AL is the average lifetime (years). The lifetime ED were assumed to
311 be 7, 20, 45 and 65 years for group "0 < Age < 15", "15 ≤ Age < 25", "25 ≤ Age < 65", and
312 "Age ≥ 65", respectively (Table 5). The percentages of time spending in each
313 microenvironment was multiplied with lifetime ED to get the exposure duration in these
314 environments. The time spending in residential home, outdoor, in-vehicle, in-office, and
315 dining microenvironments were described above (Klepeis et al., 2001).

316 The standard body weight of males and females based on age groups were provided by
317 USEPA (2011b), as summarized in Table S3, SI. A 24 h exposure and seven days per week
318 were assumed for all countries. The average lifetime of human for each country were
319 extracted from WHO (2016). The absorption factor was assumed to be 90% (USEPA, 1985).
320 The average inhalation rate for males and females based on age group were reported by
321 USEPA (2011a) and shown in Table S4, SI. The cancer potency factor (PF) of 1,3-butadiene
322 is 0.6 mg kg^{-1} day^{-1} (Du et al., 2014).

323 The lifetime cancer risk (LCR), described as the probability to get cancer caused by toxicant
324 exposure, is the product of CDI and PF, as follows:

325 $LCR = CDI \times PF$ (2)

326 where LCR (in LCP) is the cancer risk associated with BD; CDI (mg kg^{-1} day^{-1}) is the chronic
327 daily intake of BD; and PF (mg kg^{-1} day^{-1}) represents the inhalation cancer potency factor of
328 BD.

329 **4. Results and Discussion**

330 *4.1 Lifetime cancer risk caused by 1,3-butadiene personal exposure*

331 The results of LCR estimations (one in million persons) caused by BD personal exposure of
332 adult population ($25 \leq$ Age <65) in some cities are presented in Table 6 and Fig. 1. The
333 average and maximum (available in some cities) BD levels were used to estimate the cancer
334 risk in this study. The average LCR values for male and female groups are presented in this
335 study. It is clearly seen that the LCR results of all available cities exceeded the USEPA of
336 one per million persons (1×10^{-6}).

337 The LCR estimations by personal exposure exceeded the USEPA for all the cities which may
338 be explained by high concentrations of BD. The BD levels measured by personal sampling
339 are higher compared to other microenvironment because it includes BD found in both
340 outdoor and indoor environments. The highest LCR estimation was found in Mexico City,
341 followed by New York City of all age groups. The adult group in Mexico City posed an
342 average LCR result of 1.67×10^{-4} (max 4.77×10^{-4}) which were 3 to 31 times higher than the
343 other mentioned cities. The second highest city, New York City, had the average LCR value
344 approximately 5.66×10^{-5}. High concentrations, i.e. 2.9 μg m^{-3} found in Mexico City
345 (Serrano-Trespalacios et al., 2004) and 1.01 μg m^{-3} measured in New York City (Kinney et
346 al., 2002) are one of the reasons leading to great LCR results in these cities. London (United

347 Kingdom) and Hagfors, Gothenburg (Sweden) are those cities having low LCR values, i.e.
348 5.45, 12.6 and 8.05 individuals in million persons. The other cities were found to have
349 moderate LCR estimations, ranging from 2.02×10^{-5} to 3.89×10^{-5}. The results of LCR
350 results for other age groups are presented in Table S5 (SI). The group "Age ≥ 65" of all cities
351 had the highest LCR values due to their long exposure duration to BD.

352 *4.2 Lifetime cancer risk caused by outdoor 1,3-butadiene*

353 Totally, there were 175 cities/sites in 12 countries having available outdoor BD
354 concentrations in the world (as summarized in Table 2), and were used for outdoor LCR
355 estimations in the current study. Comparing to personal exposure, the outdoor assessment had
356 lower LCR results because of less time spent even though several cities had the LCR values
357 exceeding the USEPA benchmark of 1×10^{-6}. Totally, 45 cities/sites out of 175 (sharing
358 about 26%) had LCR estimations greater than 1×10^{-6} benchmark for adult population ($25 \leq$
359 Age <65) with the range from 2.44×10^{-5} to 4.24×10^{-9}.

360 Noticeably for the adult population, there were 27 out of 43 (nearly 63%) cities in mainland
361 China experienced higher cancer risk with LCR values greater than the USEPA benchmark.
362 Meanwhile, the United States, Canada and Australia had smaller numbers of cities exceeding
363 the benchmark, i.e. 7 out of 89, 2 out of 20 and 2 out of 10 cities, respectively which shared
364 the corresponding percentages of 8%, 10% and 20%. The low BD concentrations recorded in
365 2013 in the United States is one of the reasons leading to low outdoor LCR results in most of
366 cities in this country, even though some hotspots were observed in some sites, i.e. Port
367 Neches (2.094 μg m^{-3}), suburban Houston (0.7 μg m^{-3}). Petrochemical industrial activity in
368 Port Neches, Texas is a probably reason causing high ambient BD in the region (TCEQ,
369 2009). Similarly, many industrial facilities in Houston release massive amount of BD to the
370 atmosphere, resulting in high observed BD levels there (Hendler et al., 2010). Three cities

Chennai, Pune, and Delhi (India), Karachi (Pakistan), Mexico City (Mexico), Santiago (Chile) had LCR estimations greater than 1×10^{-6}, while no cities in the United Kingdom, Tokyo (Japan), Zürich (Switzerland), Stockholm (Sweden), Hong Kong exceeded the benchmark.

The LCR results of 20 leading cities attributable to outdoor BD exposure of adult population are listed in Table 7 and other age groups in Table S6 (SI). Notably, China comprised of 14 out of 20 cities worldwide with the top 3 cities found in the list (Changsha, Kunming, and Hefei). Santiago shared the rank number 4 with LCR value of 1.38×10^{-5}, followed by another Chinese city (Beijing), i.e. 1.37×10^{-5}. All three Indian cities, including Pune, Chennai, and Delhi belonged to this top 20 with the corresponding ranks of 6, 7 and 13. There was one city in US, i.e. Port Neches in the list with a ranking of 12 (8.88×10^{-6}), which is behind Pakistan city (Karachi) with LCR estimation of 8.95×10^{-6}. The other top cities belong to China. It is clearly seen that most of leading cities having high LCR belongs to developing cities, except one site in the United States (Port Neches, Texas). The primary sources BD in Chinese cities is combustion activities (Barletta et al., 2005), which can be vehicle fuel combustion. Barletta et al. (2005) suggested an assessment of potential health risk in some cities based on the measured ambient BD in their study. In addition, the highly recorded outdoor BD concentration (Table 2, Table S1 & S2, SI) is one of the reason resulting in high LCR estimations. The differences in monitoring sites, year of sample collection and sampling intervals may cause variation of BD levels, subsequently leading to the discrepancy of LCR results. As expected, the group "Age $\geq$ 65" of all mentioned cities had the highest LCR results compared to other age groups because of their long exposure duration to BD (Table S6, SI).

The major source of BD found in outdoor environment is vehicular combustion which have been reported in some literatures (Delgado-Saborit et al., 2011; Kim et al., 2001; Laowagul

and Yoshizumi, 2009; Zhou et al., 2011). Besides, some specific industrial facilities release a certain amount of BD into the atmosphere, causing high BD detected in these area (Dollard et al., 2001; Zhou et al., 2011). An implementation of cleaner vehicle technologies, usage of public transportation means would help to reduce air pollution, including BD (Trang et al., 2015), subsequently reduces the potential health risk.

4.3 Lifetime cancer risk estimations in residential homes

The LCR estimations by indoor air pollutants are usually higher than those estimated by using outdoor levels. Fig. 2 illustrates LCR estimations due to home BD exposures in the United States cities (New York, Los Angeles), Canada, United Kingdom cities (London, West Midlands, South Wales, and Birmingham), Sweden (Hagfors), China (Tianjin) and Mexico (Mexico City). Because Setton et al. (2013) did not separate the indoor BD levels into different sites, "Canada" was used as a general term for this study. Similarly, Delgado-Saborit et al. (2011) did not report BD data for individual city (London, West Midlands, South Wales), hence; the average value found in the literature was used for all three cities. It is obviously seen that the estimated LCR values of all sites significantly exceeded the USEPA benchmark of 1×10^{-6}. The highest average LCR calculation for the adult group ($25 \leq$ Age <65) was observed in Mexico City, i.e. 9.87×10^{-5}, followed by New York City 4.22×10^{-5} and Birmingham 4.12×10^{-5}. It can be explained by the high levels of indoor BD measured in these cities (Table 3). Lowest LCR estimation was observed in Canada with a value of 4.44×10^{-6}. Other cities, including Los Angeles, UK cities, Hagfors, and Tianjin had LCR results ranging from 8.99×10^{-6} to 2.16×10^{-5}. By using the maximum BD concentrations for the calculation, the results of LCR estimations were different. The highest LCR value was observed in Birmingham city (4.04×10^{-4}), followed by New York City (3.41×10^{-4}) and Mexico City (3.28×10^{-4}) because of their high BD levels (Table 3). The results of LCR estimated by residential home BD for other age groups are shown in Table S7, SI.

Due to longer exposure time, the group "Age ≥ 65" had the highest LCR results compared to other younger age groups. The primary source of BD observed in residential homes are tobacco smoking (Carmella et al., 2009; Kim et al., 2001; Sax et al., 2004), painting products (Kim et al., 2001) and cooking fuels. In Mexico City, Serrano-Trespalacios et al. (2004) stated that the cooking fuel, i.e. liquefied petroleum gas (LPG) was one of the major sources of indoor BD contaminant. By investigating the major sources, detected levels and potential health risk of BD, we could have the overall risk assessment to get suitable solutions for residential homes, for example better ventilation system.

4.4 Lifetime cancer risk estimations in office, in-vehicle, and dining microenvironments

The estimations of LCR caused by in-office, in-vehicle, and dining exposures of adult population (25 ≤ Age <65) are illustrated in Fig. 3. The dominant LCR results are clearly seen in in-vehicle microenvironment, followed by dining and in-office places. The LCR estimations for these microenvironments for other age groups are presented in Table S8, SI.

For in-vehicle microenvironment, due to the high BD levels measured in Sacramento and Los Angeles (Rodes et al., 1998) and Birmingham (Kim et al., 2001), noticeably high LCR values were obviously found in these cities. In Dublin, Sweden, and China (Tianjin), the LCR estimations for the adult population ranged from 1.77×10^{-6} to 5.60×10^{-6}, exceeding the USEPA benchmark for all cities. The lowest LCR result was found for London, West Midlands, and South Wales, i.e. 4.32×10^{-6} which meets USEPA benchmark. As discussed above, vehicular combustion is one of major sources of BD; hence, the high concentrations of BD were obviously recorded in densely trafficked areas.

Low BD concentrations were measured in-office microenvironment (Table 4). For the adult groups, the LCR results estimated in our study did not exceed the USEPA benchmark for any city. The LCR ranged from 2.35×10^{-7} to 9.26×10^{-6}. However, there are two cities, i.e.

Birmingham (United Kingdom) and Bangkok (Thailand) had high values of LCR for group "Age ≥ 65" which were greater than 1×10^{-6} (Table S8, SI).

Similarly, slightly high LCR estimations due to dining BD exposure were observed in mentioned cities (Fig. 3) for the adult group. The LCR calculated in Birmingham city was dominant over other cities for all age groups (i.e. 2.45×10^{-6}), followed by Helsinki with a value 1.76×10^{-6}. The LCR estimations in Dublin (Ireland) and London, West Midlands, and South Wales (United Kingdom) did not reach beyond the benchmark. The high LCR value associated with dining BD concentrations is likely caused by cooking combustion and tobacco smoking found in several places.

4.5 Microenvironment distributions to the lifetime cancer risk

From the literatures, there were four case studies that had sufficient BD data for analyzing the source distributions to the LCR estimations, including Birmingham (Kim et al., 2001), London-West Midlands-South Wales (Delgado-Saborit et al., 2011; Harrison et al., 2009), Los Angeles (Rodes et al., 1998; Sax et al., 2004; USEPA, 2015) and Tianjin (Zhou et al., 2011). Even though BD measurements were conducted in different years for Los Angeles city, it is worthy to roughly examine the contribution of each emission sources in the city.

It is clearly seen that LCR value due to BD exposure at residential homes contributes considerably to the total LCR for all mentioned cities, ranging from 56% to 86% (Fig. 7). It is likely explained by the predominant time spending at homes as well as high BD concentrations. Exposure to in-vehicle was the second highest source distributions to total LCR results, excluding London, West Midlands and South Wales which dining microenvironment contributed 7% to total LCR estimations compared to in-vehicle (4%). In-vehicle microenvironment considerably contributed more than one third and nearly one fifth of total LCR estimations in Los Angeles and Birmingham, respectively. The LCR attributable

to in-office BD exposure had smaller shares from 2% to 3%. The outdoor BD exposure shared 6% of total LCR values which were two and six times higher than Los Angeles and London, West Midlands, and South Wales, respectively.

6. Conclusions

1,3-butadiene (BD) is a well-known carcinogenic compound, but the cancer risk assessment was not comprehensively studied for many cities. This study aimed to estimate the lifetime cancer risk (LCR) due to BD personal exposure and to five microenvironments, including the indoor, outdoor, in-office, in-vehicle, and dining at city levels worldwide. The LCR estimations were deeply investigated based on genders and age groups (i.e., $0 < \text{Age} < 15$, $15 \leq \text{Age} < 25$, $25 \leq \text{Age} < 65$, and $\text{Age} \geq 65$). The inhalation rate and body weight by age group reported by US Environment Protection Agency (US EPA) were used in the calculation of this study. The adult population ($25 \leq \text{Age} < 65$) was the focus of this study because of their large population and different microenvironments, even though other age groups were also mentioned in the text.

The personal exposure was involved in this study because it would be better reflected the BD levels found for both indoors and outdoors. All the reported cities or areas had the LCR estimations exceeding the USEPA benchmark of 1×10^{-6}. High LCR results were observed in many developing cities, which may associate with the serious air pollution status in these cities. The LCR values of adult population were found to be highest in Mexico City and New York City with value of 1.67×10^{-4} and 5.66×10^{-5}, respectively, which were 3 to 31 times higher than the other mentioned cities. It is likely caused by the high concentrations of personal exposure found in the literatures.

There have been many literatures reported the outdoor BD concentrations at city levels; hence, this study could capture a clear picture of LCR estimations worldwide (totally 175

cities/sites of 12 countries). Although LCR estimated using outdoor BD was lower than those estimated by personal exposure, many cities had LCR value exceeding the USEPA benchmark (45 out of 175 cities/sites, sharing 26%). The top 20 cities having high LCR results caused by outdoor BD were listed. Notably, 19 cities were belonged to developing countries, except one sites in the United States which may be caused by the densely industrial activities. There were 14 Chinese cities in the list with the top three were Changsha, Kunming, and Hefei. All three available cities of India were also in the list and the other cities were in Chile (Santiago) and Pakistan (Karachi). The major sources of outdoor BD are transportation and industrial activities

There have been few studies reporting the BD concentrations measured at residential homes which only cities or regions had sufficient BD data for LCR estimations. The results showed that all cities exceeded the USEPA benchmark of 1×10^{-6}, which is explained by high BD levels found in residential homes. In addition, dominant time spent at homes is also one of the reason leading to high LCR results. The high BD concentrations measured in in-vehicle microenvironment is one of the reasons causing high LCR estimations for most of the cities. However, low to medium LCR values were observed in other two microenvironments, including in-office and dining microenvironments.

The investigation of cancer risk in different microenvironments was used for analyzing the source contributions to total LCR results. Four cities or regions which had sufficient BD data were analyzed the source distribution of various microenvironments in this study, including Birmingham, London-West Midlands-South Wales, Los Angeles, and Tianjin. As expected, indoor exposure contributed significantly to the total exposure, ranging from 56% to 86% of total LCR results. In-vehicle microenvironment had second highest LCR shares for all cities, excepting for London-West Midlands-South Wales. The LCR estimated by outdoor BD only contributed significantly in Tianjin with 6%, while lower percentages were found for Los

Angeles and London-West Midlands-South Wales with 3% and 1%, respectively. Dining microenvironment contribute from 4% to 7%, while in-office LCR results shared very small, ranging from 2% to 3%.

The BD levels found in literatures were the major sources of uncertainty due to the unavailability of data in many cities. Also, the calculated parameter such as inhalation rate and body weight by age groups were adopted from USEPA, resulting in less representative for other regions. It has been the first time that the cancer risk due to BD exposure at city level worldwide in different microenvironments by gender and age groups is studied comprehensively. Further studies should investigate more available BD concentrations using local data sources to have comprehensive cancer risk assessment.

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Table captions

Table 1 Summary of personal exposure 1,3 – butadiene concentrations in some cities

Table 2 Summary of 1,3 – butadiene concentrations (μg m^{-3}) measured in outdoor microenvironments in some cities

Table 3 Summary of 1,3-butadiene concentrations (μg m^{-3}) measured at residential homes in some cities

Table 4 Summary of 1,3-butadiene concentrations (μg m^{-3}) measured in office, vehicle, and dining microenvironments in some cities

Table 5 Summary of human factors related to inhalation exposure pathway for different age groups

Table 6 The lifetime cancer risk estimation due to 1,3-butadiene personal exposure in some cities

Table 7 The lifetime cancer risk of 20 leading cities attributable to outdoor 1,3-butadiene exposure

Table 1 Summary of personal exposure 1,3 – butadiene concentrations in some cities

Country	City	1,3-butadiene concentration (µg m^{-3})					
		Range	Mean	Sampling method/size	Sampling duration	Sampling year	Source
United Kingdom	Birmingham	n.d – 26.3[1] n.d – 7.9[2]	1.1 ± 2.4[1] 0.8 ± 1.2[2]	Active/12	10 h daytime[1] 2 h nighttime[2]	1999 – 2000	Kim et al. (2002)
	City (average)	n.d – 6.31	0.4 ± 0.71	Active/100	24 h (5 days)	2005 – 2007	Harrison et al. (2009)
	Urban sites (average)	n.d – 5.95	0.44 ± 0.84				
	London	n.d – 0.76	0.1 ± 0.14				
	Birmingham	n.d – 5.95	0.56 ± 0.95				
	Suburban site *Birmingham*	n.d – 6.27	0.37 ± 0.62				
	Rural sites (average)	0.01 – 4.03	0.38 ± 0.62				
	West Midlands	0.01 – 4.03	0.51 ± 0.78				
	Wales	0.01 – 1.85	0.24 ± 0.35				
Sweden	Stockholm	0.08 – 2.3	0.5 ± 0.5	-/40	24 h (7 days)	2009	Yazar et al. (2011)
	Stockholm	0.2 – 3.1	0.7 ± 0.7	Active/40	24 h (7 days)	2002	Krusa et al. (2003)
	Hagfors	-	0.33[3] 0.14[4]	Active/24	24 h (1 day)	2003	Gustafson et al. (2007)
	Umeå	-	0.44	Passive/40	24 h (7 days)	2001	Hagenbjork-Gustafsson et al. (2014)
	Stockholm	-	0.45			2002	
	Malmö	-	0.44			2003	
	Lindesberg	-	0.54			2005	
	Gothenburg	-	0.15			2006	
	Umeå	-	0.54			2007	
	Malmö	-	0.51			2008	
China	Tianjin	-	0.67 ± 0.7	Passive/12	24 h (5 days)	2008	Zhou et al. (2011)
Mexico	Mexico City	- – 8.3	2.9 ± 2.4	Active/90	24 h	1998-1999	Serrano-Trespalacios et al. (2004)
United States	New York	-	0.87 ± 1.29[5] 1.16 ± 1.95[6]	Active/12	48 h	1999	Kinney et al. (2002)

Note: [1] Daytime (during the time between 07:00 – 22:00; 5 – 10 days); [2] Nighttime (00:00 – 02:00; 5 – 10 days); [3] with wood burners; [4] reference group (without wood burner); [5] winter sampling; [6] summer sampling

Table 2 Summary of 1,3 – butadiene concentrations (μg m^{-3}) measured in outdoor microenvironments in some cities

Country	City	1,3-butadiene concentration (μg m^{-3})					
		Range	Mean	Sampling method	Sampling duration	Sampling year	Source
United States	85 cities	0.001 – 2.094	0.128 ± 0.238	Monitoring station	24 h (annual average)	2013	USEPA (2015)
	Houston (14 sites)	0.12 – 3.2	0.57 ± 0.82	Monitoring station	24 h (annual average)	1997-2004	Reiss (2006)
	Houston (30 sites)[1]	0.07 – 2.36	0.41 ± 0.53	Monitoring station	24 h (annual average)	2008	Hendler et al. (2010)
	77 sites	-	0.3	Monitoring station	24 h (annual average)	2000	McCarthy et al. (2007)
	Boston	-	0.077	PTR-MS	-	2007	Knighton et al. (2009)
	New York city	n.d – 0.7[2] n.d – 2.0[3]	0.1 ± 0.2[2] 0.1 ± 0.4[3]	Thermal desorption – GC/MS	48 h	1999	Sax et al. (2004)
	Los Angeles	n.d – 1.7[2] n.d – 0.3[4]	0.2 ± 0.4[2] 0.01[4]			2000	
United Kingdom	London Eltham	0.04	0.06 ± 0.02	Monitoring station	24 h	2005	Delgado-Saborit et al. (2011)
	Harwell	n.d – 0.6	0.01 ± 0.06	Monitoring station	24 h	2005-2007	
	Cardiff	n.d – 0.1	0.03 ± 0.03	Monitoring station	24 h	2006-2007	
	9 cities	-	0.56 ± 0.18	Monitoring station	24 h (annual average)	1993	Dollard et al. (2001)
Switzerland	Zürich	0.09 – 0.20[6] 0.07 – 0.29[7] 0.11 – 0.27[8]	0.155[6] 0.177[7] 0.199[8]	Online GC-MS	50 min	2005-2006	Legreid et al. (2007)
Sweden	Stockholm	0.02 – 0.2	0.09 ± 0.04	Online GC-MS	24 h	2009	Yazar et al. (2011)
	Stockholm *Hornsgatan* *Rosenlundsgatan* *10km from city center*	 0.19 – 0.52 0.05 – 0.12 0.02 – 0.07	 0.39 0.08 0.04	Online GC-MS	24 h (7 days)	2002	Krusa et al. (2003)

Table 2 Summary of 1,3 – butadiene concentrations (μg m^{-3}) measured in outdoor microenvironments in some cities (cont'd)

Country	City	1,3-butadiene concentration (μg m^{-3})					
		Range	Mean	Sampling method	Sampling duration	Sampling year	Source
Canada	-	-	0.096	-	-	-	Setton et al. (2013)
	Urban sites *Halifax* *Montréal* *Ottawa* *Stouffville* *Toronto* *Hamilton* *Sarnia* *Windsor* *Winnipeg* *Edmonton* *Calgary* *Vancouver* Rural sites *Kejimkujik* *Point Lepreau* *Sutton* *Saint-Anicet* *L'Assomption* *Lemieux* *Simcoe* *Egbert*	-	0.26 0.23 0.17 0.05 0.12 0.14[5] 0.20[5] 0.11 0.15 0.18 0.26 0.17 0.002 0.02 0.006 0.016 0.037 0.009 0.012 0.011	Monitoring station	24 h (annual average) 4 h (annual average)	2003	Curren et al. (2006)
	18 cities	0.08 – 0.4	0.24	Monitoring station	24 h (annual average)	2001	EPS (2004)
Japan	Rural and mountainous regions Suburban Urban Average	-	< 0.125 0.25 0.50 0.22	Modeling approach	Annual average	2002	Higashino et al. (2007)

Table 2 Summary of 1,3 – butadiene concentrations (μg m^{-3}) measured in outdoor microenvironments in some cities (cont'd)

Country	City	1,3-butadiene concentration (μg m^{-3})					
		Range	Mean	Sampling method	Sampling duration	Sampling year	Source
Japan	Tokyo *Shirogane* *Hachimanyama*	- -	0.49 0.91	Online GC-MS	1 h	2001	Laowagul and Yoshizumi (2009)
	Tokyo	-	0.064	Monitoring station	-	-	Mita et al. (2006)
China	43 cities	0.04 – 5.53	1.12 ± 1.28	Canister sampling - GC	-	2001	Barletta et al. (2005)
	Jinan	-	0.35±1.196	Online GC-MS	30 min	2010-2012	Liu et al. (2016)
	Tianjin	-	0.36 ± 0.63	Thermal desorption – GC/MS	24 h (5 days)	2008	Zhou et al. (2011)
	Shanghai	n.d – 18.38	0.35 ± 0.99	Canister sampling – GC/MS	3 h	2006-2010	(Cai et al., 2010)
Hong Kong	Tsuen Wan Central/Western	-	0.08 0.05	Canister sampling – GC/MS	24 h (annual average)	2014	EPD (2014)
	Tsuen Wan Central/Western	-	0.44 ± 0.22 0.27 ± 0.16	Canister sampling – GC/MS	24 h	2001	Guo et al. (2004)
India	Chennai Delhi Pune	1.00 – 3.98 0.44 – 3.54 0.88 – 5.53	2.43 1.72 2.65	-	-	-	Sengupta (2011)

Table 2 Summary of 1,3 – butadiene concentrations (µg m^{-3}) measured in outdoor microenvironments in some cities (cont'd)

Country	City	1,3-butadiene concentration (µg m^{-3})					
		Range	Mean	Sampling method	Sampling duration	Sampling year	Source
Pakistan	Karachi	0.07 – 7.51	1.77 ± 1.77	Canister sampling – GC/MS	4 h	1998-1999	Barletta et al. (2002)
Mexico	Mexico City	- – 4.6	0.9 ± 0.9	Thermal desorption – GC/MS	24 h	1998 - 1999	Serrano-Trespalacios et al. (2004)
Chile	Santiago	-	3.3	Canister sampling – GC/FID	-	1996	Chen et al. (2001)
Australia	Sydney CBD	- – 3.09	0.88	Canister sampling - GC/MS	24 h	1996-2001	NSW (2004)
	Rozelle	- – 1.99s	0.44			1996-2001	
	St Marys	- – 0.66	0.22			1996-2001	
	Wollongong	- – 0.88	0.22			1997-2001	
	Newcastle	- – 1.99	0.22			1997-2001	
	Albion Park	- – 0.22	0.22			1997-2001	
	Kembla Grange	- – 0.44	0.22			1997-2001	
	Warrawong	- – 0.66	0.22			1997-2001	
	Beresfield	- – 0.88	0.22			1997-2001	
	Wallsend	- – 1.33	0.22			1997-2001	

* Note: n.d = not detected; [1] The reported unit (ppb) was converted to µg/m^3 using factor 2.21 (https://uk-air.defra.gov.uk/assets/documents/reports/cat06/0502160851_Conversion_Factors_Between_ppb_and.pdf); [2] winter season; [3] summer season; [4] fall season; [5] data in 2002; [6] spring and summer season; [7] fall season; [8] winter season

Table 3 Summary of 1,3-butadiene concentrations (μg m^{-3}) measured at residential homes in some cities

Country	City	1,3-butadiene concentration (μg m^{-3})					
		Range	Mean	Sampling method	Sampling duration	Sampling year	Source
United States	New York Los Angeles	n.d – 5.8[1] n.d – 12[2] n.d – 1.8[1] n.d – 1.5[3]	1.0 ± 1.4[1] 1.2 ± 2.6[2] 0.5 ± 0.6[1] 0.2 ± 0.3[3]	Thermal desorption – GC/MS	48 h	1999 2000	Sax et al. (2004)
Canada	-	-	0.12	-	-	-	Setton et al. (2013)
United Kingdom	London, West Midlands, and rural South Wale	n.d – 2.04	0.24 ± 0.31	Thermal desorption – GC/MS	12 h	2005-2007	Delgado-Saborit et al. (2011)
	Birmingham	n.d – 10.8	1.1 ± 1.90	Thermal desorption – GC/MS	3 times per day (12:00 – 14:00; 18:00 – 22:00, 24:00 – 22:00)	1999-2000	Kim et al. (2001)
Sweden	Hagfors	-	0.31[4] 0.11[5]	Thermal desorption – GC-FID	24 h	2003	Gustafson et al. (2007)
China	Tianjin	-	0.54 ± 0.30	Thermal desorption – GC/MS	24h (5 days)	2008	Zhou et al. (2011)
Mexico	Mexico City	- – 8.3	2.5 ± 2.1	Thermal desorption – GC/MS	24 h	1998 - 1999	Serrano-Trespalacios et al. (2004)

Note: n.d = not detected; [1] winter season; [2] summer season; [3] fall season; [4] wood-burning group; [5] reference group

Table 4 Summary of 1,3-butadiene concentrations (μg m^{-3}) measured in office, vehicle, and dining microenvironments in some cities

Country	City	Micro-environment	1,3-butadiene concentration (μg m^{-3})					
			Range	Mean	Sampling method	Sampling duration	Sampling year	Source
United States	-	In-office	-	0.2 ± 3.4[1]	-	-	-	Loh et al. (2007)
	Sacramento	In-vehicle	1.6 – 3.5[2]	2.25[2]	Canister sampling – GC/MS	2 h	1997	Rodes et al. (1998)
	Los Angeles	In-vehicle	2.3 – 3.7[2]	2.95[2]				
	Boston	Dining	- – 35.5[3]	1.05[3]	Thermal desorption – GC/MS	1.5 h	2004	Loh et al. (2006)
United Kingdom	London, West Midlands, and rural South Wales	In-office	0.01 – 0.39[1]	0.08 ± 0.08[1]	Thermal desorption – GC/MS	8 h	2005-2007	Delgado-Saborit et al. (2011)
		In-vehicle	-	0.13 ± 3.62[2]				Harrison et al. (2009)
		Dining	-	0.70 ± 12.18[3]				
	Birmingham	In-office	-	0.3 ± 0.2[1]	Thermal desorption – GC/MS	Twice per day (09:30 – 11:30; 14:00 – 16:00)	1999-2000	Kim et al. (2001)
		In-vehicle		3.53[2,4]		3 times per day (08:00 – 09:00; 12:00 – 14:00; 17:00 – 19:00)		
		Dining		2.25[3,5]		Once per pub or restaurant		
Sweden[6]	-	In-vehicle	0.3 – 0.9[2]	0.54[2]	Thermal desorption – GC/FID	-	1994	Barrefors and Petersson (1996)
China	Tianjin	In-office	-	0.25 ± 0.11[1]	Thermal desorption – GC/MS	24 h (5 days)	2008	Zhou et al. (2011)
		In-vehicle	-	0.62 ± 0.34[2]				

Table 4 Summary of 1,3-butadiene concentrations (μg m^{-3}) measured in office, vehicle, and dining microenvironments in some cities (cont'd)

Country	City	Micro-environment	1,3-butadiene concentration (μg m^{-3})					
			Range	Mean	Sampling method	Sampling duration	Sampling year	Source
Thailand	Bangkok	In-office	0.10 – 0.53[1]	0.29 ± 0.04[1]	Thermal desorption – GC/MS	8 h	2006	Arayasiri et al. (2010)
Ireland	Dublin	In-vehicle	-	1.47 ± 0.91[2,7]	Thermal desorption – GC/FID	-	2005-2006	McNabola et al. (2008)
		In-vehicle	0.82 – 3.29[2,8]	1.72 ± 0.75[2,8]	Tedlar Bag – GC/FID	-	2003	O'Donoghue et al. (2007)
		Dining	0.13 – 0.28[3]	0.22[3]	Thermal desorption – GC/FID	30 to 45 min	2004	McNabola et al. (2006)
Finland	Helsinki	Dining	0.26 – 10.1[3,9] 0.11 – 3.9[3,10]	2.7[3,9] 0.52[3,10]	Thermal desorption – GC/MS	5 h	2005-2006	Vainiotalo et al. (2008)

Note: [1] In-office data; [2] In-vehicle data; [3] Dining data; [4] Average BD concentration monitored in cars, buses, and train; [5] Average BD concentrations monitored in restaurants and pubs; [6] Average data of buses and trains; [7] Average data of cars and buses; [8] Data of buses; [9] Restaurants having smoking activities; [10] Restaurants without smoking activities

Table 5 Summary of human factors related to inhalation exposure pathway for different age groups

Parameter	Age groups							
	0 < Age < 15		15 ≤ Age < 25		25 ≤ Age < 65		Age ≥ 65	
	Male	Female	Male	Female	Male	Female	Male	Female
Inhalation rate (m^3 day^{-1})	8.7	7.6	17.2	13.3	16.7	12.9	13.4	10.3
Exposure duration (year)	7		20		45		65	
Body weight (kg)	17.4	18	77.3	65.9	88	75.3	83	70.6
Life expectancy (years)	Vary by countries (WHO, 2016)							

Table 6 The lifetime cancer risk estimation due to 1,3-butadiene personal exposure of adult population in some cities

Country/City	Lifetime cancer risk (in LCP)	
	Average	Max
United Kingdom		
London (urban)	5.45×10^{-6}	4.14×10^{-5}
Birmingham (Urban)	3.05×10^{-5}	3.24×10^{-4}
Birmingham (Suburban)	2.02×10^{-5}	3.42×10^{-4}
West Midlands (Rural)	2.78×10^{-5}	2.20×10^{-4}
Wales (Rural)	1.31×10^{-5}	1.01×10^{-4}
Sweden		
Hagfors	1.26×10^{-5}	-
Umeå	2.90×10^{-5}	-
Stockholm	2.68×10^{-5}	1.23×10^{-4}
Malmö	2.74×10^{-5}	-
Lindesberg	2.90×10^{-5}	-
Gothenburg	8.05×10^{-6}	-
China		
Tianjin	3.89×10^{-5}	-
Mexico		
Mexico City	1.67×10^{-4}	4.77×10^{-4}
United States		
New York City	5.66×10^{-5}	-

* Note: - data not available

Table 7 The lifetime cancer risk of 20 leading cities attributable to outdoor 1,3-butadiene exposure of adult population

No.	City	Country	Lifetime cancer risk (in LCP)
1	Changsha	China	2.44×10^{-5}
2	Kunming	China	2.05×10^{-5}
3	Hefei	China	1.85×10^{-5}
4	Santiago	Chile	1.38×10^{-5}
5	Beijing	China	1.37×10^{-5}
6	Pune	India	1.31×10^{-5}
7	Chennai	India	1.20×10^{-5}
8	Suizhou	China	1.07×10^{-5}
9	Wuhan	China	1.07×10^{-5}
10	Xiantao	China	9.76×10^{-6}
11	Karachi	Pakistan	8.95×10^{-6}
12	Port Neches	The United States	8.88×10^{-6}
13	Delhi	India	8.50×10^{-6}
14	Chongquing	China	7.81×10^{-6}
15	Guiyang	China	7.81×10^{-6}
16	Nanchang	China	7.81×10^{-6}
17	Changchun	China	6.83×10^{-6}
18	Taihu (Wuxi)	China	6.83×10^{-6}
19	Linchuan	China	5.86×10^{-6}
20	Wenzhou	China	5.86×10^{-6}

Figure captions

Figure 1 The lifetime cancer risk (one in million of population) of adult population (25 to 65) caused by 1,3-butadiene personal exposure. Error bars show the maximum lifetime cancer risk.

Figure 2 The lifetime cancer risk estimations by 1,3-butadiene levels in residential homes in some cities. Error bars show the maximum lifetime cancer risk.

Figure 3 The lifetime cancer risk estimations by 1,3-butadiene levels in in-vehicle, in-office and dining microenvironments in some cities. Error bars show the maximum lifetime cancer risk. LD, WM, SW are abbreviations of London, West Midlands and South Wales.

Figure 4 The lifetime cancer risk shares by different microenvironments in some cities

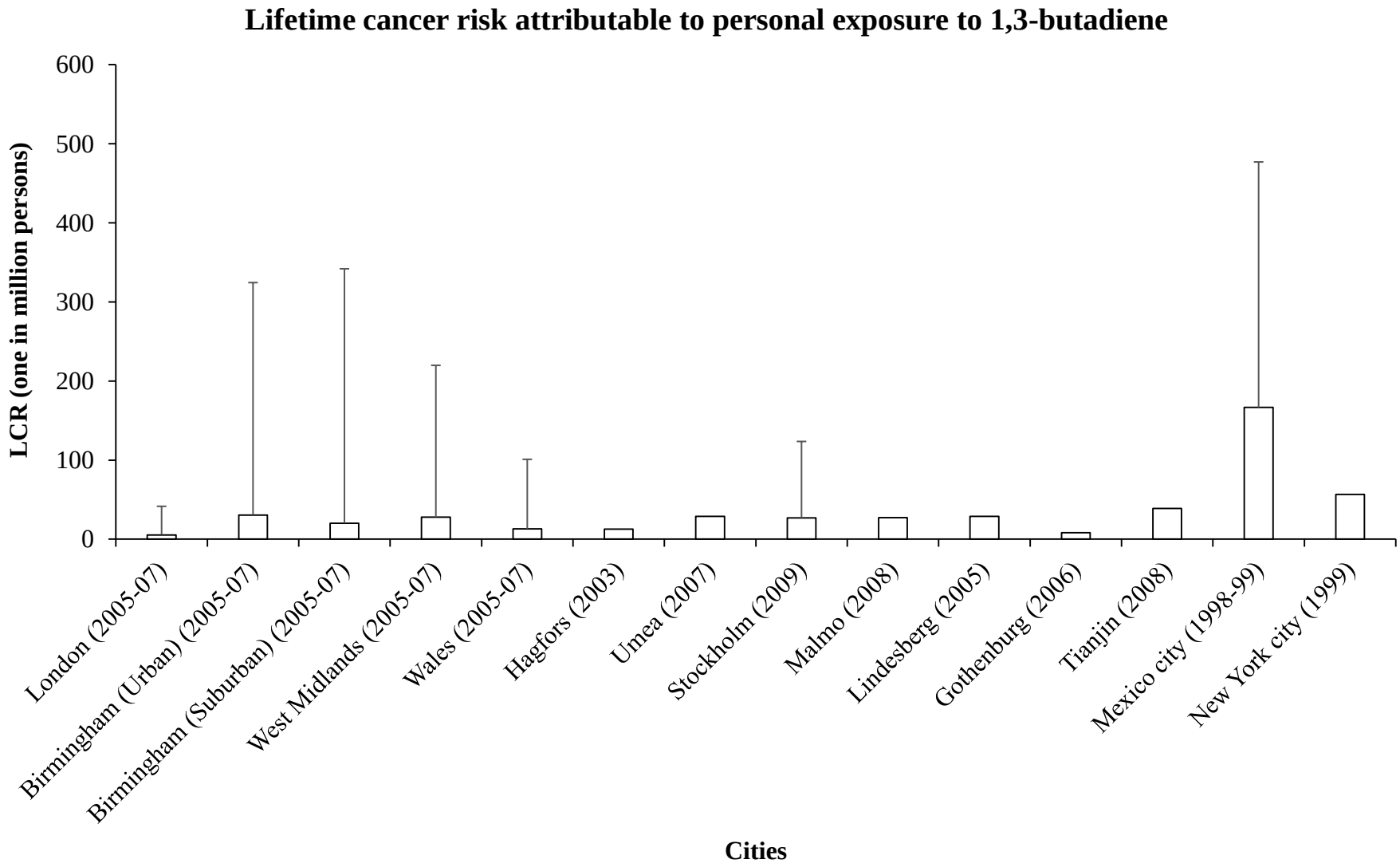

Figure 1 The lifetime cancer risk (one in million of persons) of adult population (25 to 65) caused by 1,3-butadiene personal exposure. Error bars show the maximum lifetime cancer risk.

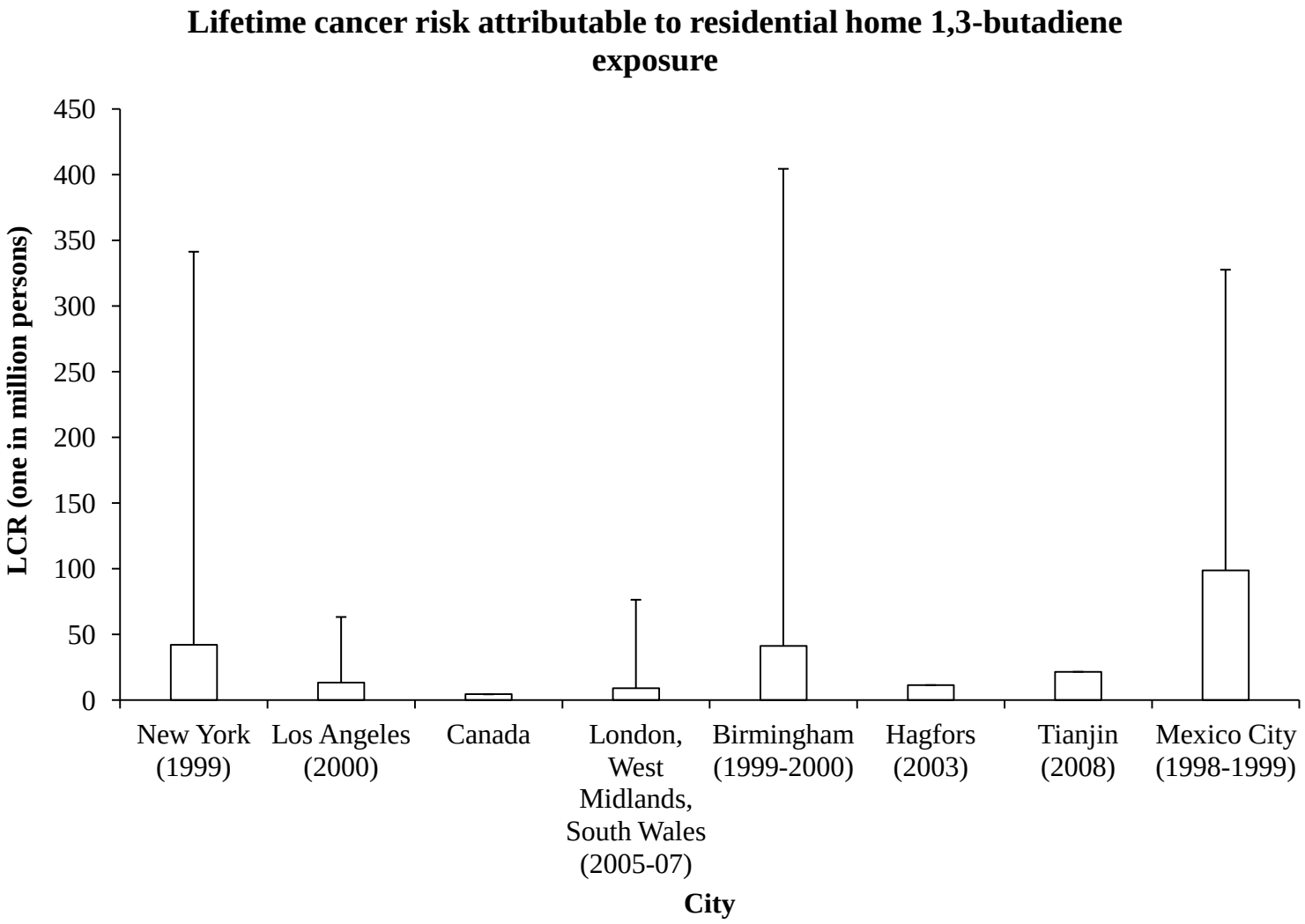

Figure 2 The lifetime cancer risk estimations by 1,3-butadiene levels in residential homes in some cities. Error bars show the maximum lifetime cancer risk.

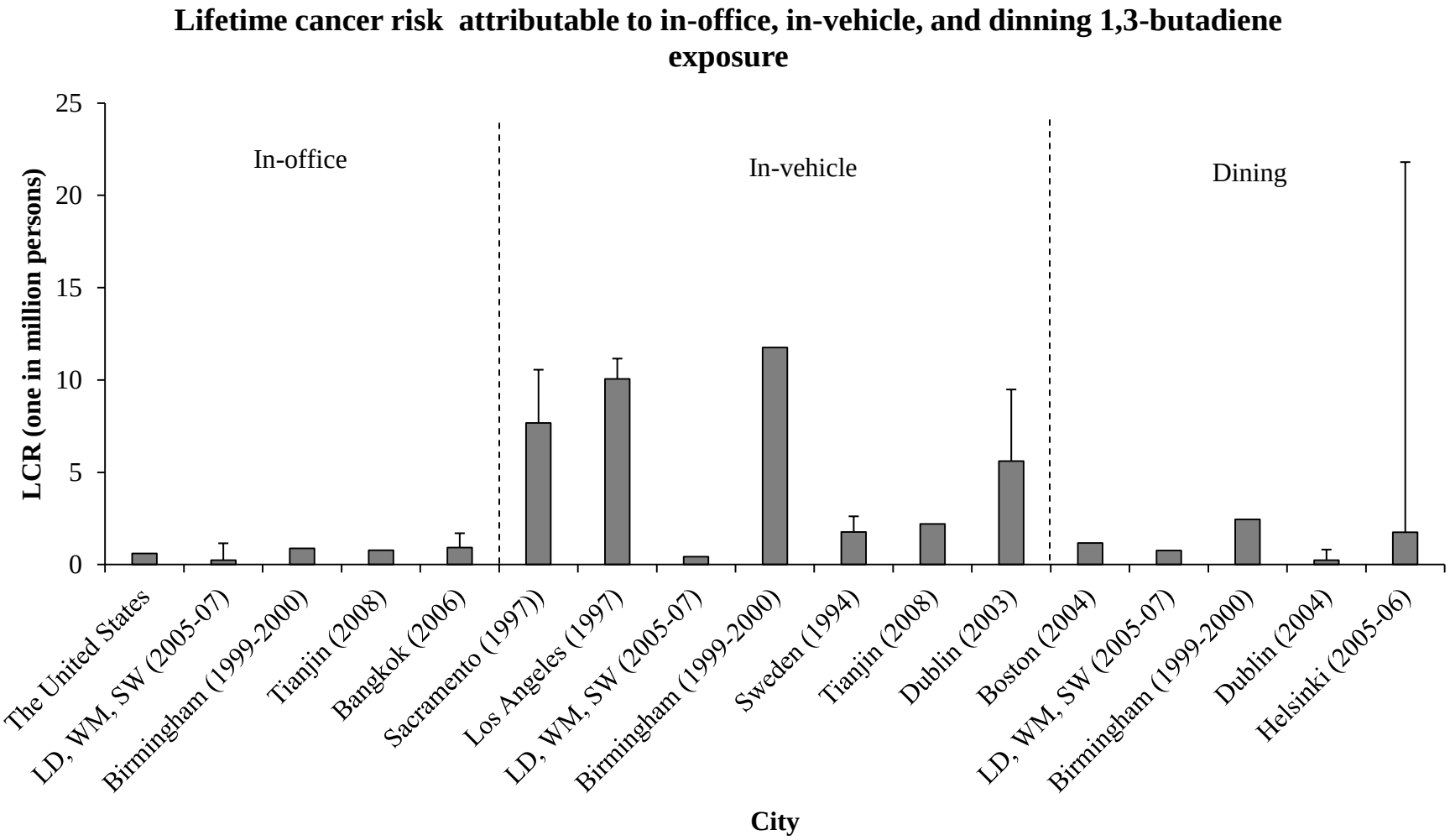

Figure 3 The lifetime cancer risk estimations by 1,3-butadiene levels in in-vehicle, in-office and dining microenvironments in some cities. Error bars show the maximum lifetime cancer risk. LD, WM, SW are abbreviations of London, West Midlands and South Wales.

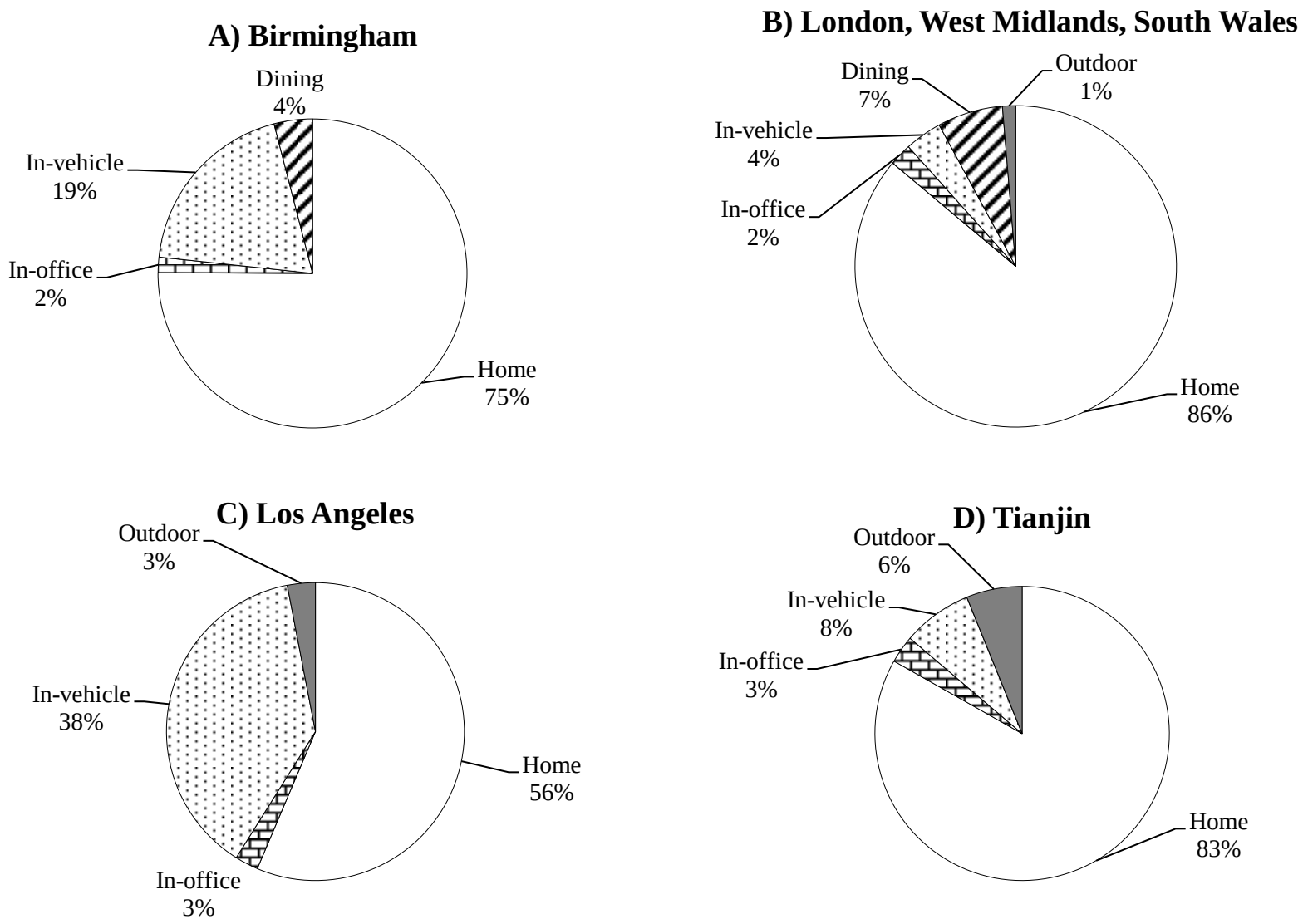

Figure 4 The lifetime cancer risk shares by different microenvironments in some cities

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