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# Review of Urban Bicyclists' Intake and Uptake of Traffic-Related Air Pollution

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ABSTRACT    *Bicycling as a mode of transportation is enjoying a boost in many urban areas around the world. Although there are clear health benefits of increased physical activity while bicycling, bicyclists may experience increased inhalation of traffic-related air pollutants. Bicyclists have two to five times higher respiration rates than travelers in motorized vehicles and this difference increases with bicycle travel speed and exertion level. The main goal of this work is to review the state of knowledge regarding urban bicyclists' intake and uptake of traffic-related air pollution and to identify key knowledge gaps. This review includes not only bicyclists' exposure to air pollution concentrations but also respiration rates, intake doses (the amount of pollutant that is inhaled), and uptake doses (the amount of pollutant that is incorporated into the body). Research gaps and opportunities for future research are discussed. This is the first review to specifically address bicyclists' health risks from traffic-related air pollution and to explicitly include intake and uptake doses in addition to exposure concentrations for travelers.*

## 1. Introduction

Bicycling as a mode of transportation is enjoying a boost in urban areas around the world through new bike-sharing systems, bicycle-specific roadway facilities, public outreach, and incentive programs (Pucher & Buehler, 2012). The push toward promoting bicycling is motivated by a range of environmental, economic, health, and social benefits. Although there are clear health benefits of increased physical activity, bicyclists may experience increased inhalation of traffic-related air pollutants (de Hartog, Boogaard, Nijland, & Hoek, 2010).

Human exposure to traffic-related air pollution has well-established negative health impacts for urban populations (Brook et al., 2010; Forastiere & Agabiti, 2013; Health Effects Institute, 2010; Nawrot et al., 2011). Air pollution exposure is particularly high for travelers because of proximity to mobile sources of pollution (Kaur, Nieuwenhuijsen, & Colvile, 2007), and air quality is a source of concern for urban bicyclists (Badland & Duncan, 2009). However, the health risks of air pollution exposure during travel are not easily characterized because of the numerous individual, environmental, and traffic factors involved.

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A conceptual diagram linking traffic-related pollution emissions and health effects is illustrated in Figure 1, adapted from Ott, Steinemann, and Wallace (2007). Motor vehicle emissions (a) degrade urban air quality (b) in accordance with atmospheric dispersive, chemical, and physical processes. Travelers' exposure concentrations (c) then depend on their travel trajectory. The inhalation of traffic-related air pollution (d) depends on travelers' breathing volume while exposed to a pollutant concentration. Uptake of the inhaled pollutants into the body (e) depends on processes in the respiratory tract and other body systems. Finally, the health effects (f) of air pollution uptake doses are a function of the toxicity of the pollutants and physiology of the individual. The processes between inhalation and uptake can be further demarcated as ($e_1$) intake dose (the amount of pollutant that crosses the body boundary at the mouth and nose), ($e_2$) absorbed dose (the amount of pollutant that is not exhaled but deposited or absorbed), ($e_3$) effective dose (the bioavailable amount of pollutant that reaches body tissue instead of being expelled from the respiratory tract lining by coughing, sneezing, etc.), and ($e_4$) uptake dose (the amount of pollutant that is incorporated into the body).

Past reviews of travelers' pollution exposure have been oriented by pollutant (Kaur et al., 2007; Knibbs, Cole-Hunter, & Morawska, 2011) and/or focused on in-vehicle exposures (El-Fadel & Abi-Esber, 2009). These reviews focused on exposure concentrations and provide little or no discussion of respiration or its effects on intake and uptake doses. The focus of this review is on bicyclists' exposure to, inhalation of, and uptake of traffic-related air pollution — i.e. steps (c) through (f) in Figure 1. This review is unique in focusing exclusively on bicyclists.

## 2.  Methodology

A systematic literature search for bicyclist exposure and dose measurements was performed through January 2014 using all 20 possible keyword combinations {A+B+C} utilizing the keyword sets A={bicycle, bicyclist, cyclist,
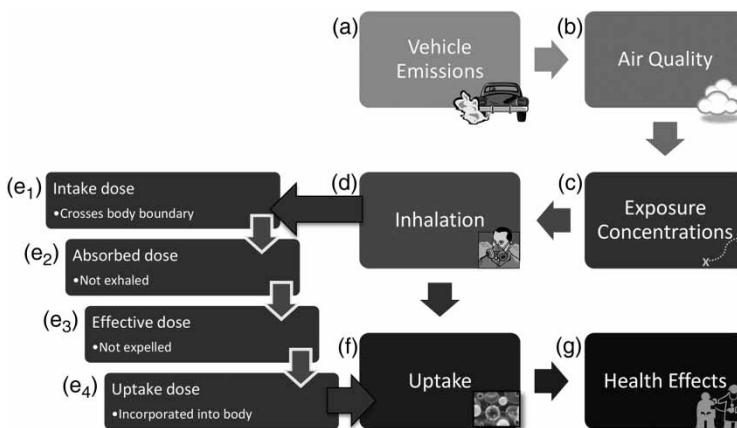


**Figure 1.**  Conceptual diagram of exposure pathway for traffic-related air pollution.
*Source*: Adapted from Ott, Stieneman, and Wallace (2007)

bike}, $B$={pollution} and $C$={exposure, intake, inhalation, uptake, dose}. An exhaustive search was performed using the WorldCat[TM] catalog. Additional references were found by reviewing cited reference lists and the Google Scholar[TM] search engine. There were 57 published papers describing original studies of on-road bicyclists and air pollution exposures with spatially explicit concentration data. There were 42 published papers with unique exposure concentration data measured on road by bicyclists. Details of the literature search method are presented in the Supplemental Material.

## 3.    Bicyclists' Air Pollution Exposure Concentrations

The main traffic-related air pollutants linked to health risks for road travelers and measured for bicyclists are carbon monoxide (CO), nitrogen oxides ($NO_x$) — including nitric oxide (NO) and nitrogen dioxide ($NO_2$), volatile organic compounds (VOC), and particulate matter (PM) of various sizes and composition: ultrafine particles (UFP), $PM_{2.5}$, $PM_{10}$, and elemental carbon (EC)/black carbon (BC). These pollutants are described in the Supplemental Material.

A traveler's exposure concentration is the concentration of pollutants in their breathing zone. Concentrations of traffic-related primary pollutants are particularly high near roadways — especially for shorter lived pollutants such as UFP and reactive VOC (Gordon et al., 2012; Karner, Eisinger, & Niemeier, 2010). Steep concentration gradients can be seen even on the scale of a few meters (Clifford, Clarke, & Riffat, 1997; McNabola, Broderick, & Gill, 2009b; Tiwary, Robins, Namdeo, & Bell, 2011). Exposure concentrations are sampled using a variety of pollutant-specific devices, each requiring specialized knowledge and careful sampling procedures (Vallero, 2008). Roadside studies of air pollution concentrations are more common than on-road data collections because on-road measurements are more difficult to execute (particularly for pedestrians and bicyclists). But the body of research on active travelers' pollution exposure concentrations has grown notably in recent years. On-road air quality sampling has become more precise and more portable because of improvements in measurement technology, power storage, and position tracking systems (Gulliver & Briggs, 2004; Steinle, Reis, & Sabel, 2013).

A literature search revealed 42 published studies reporting unique exposure concentration data collected with on-bicycle sampling devices. Summary information on all 42 studies is included in the Supplemental Material, allowing comparisons of methodologies and settings. Table 1 summarizes reported concentrations in all 42 studies, excluding results for 'rural' settings). Ranges of reported central value statistics and disaggregate (sample-level) values are presented, including the country where the low and high measurements were taken.

The mean on-road measurements in Table 1 are all well above typical urban background concentrations (see Supplemental Material). Table 1 shows that measured bicyclist exposure concentrations for most pollutants exhibit high variability among studies, with a standard deviation (SD) greater than 50% of the mean value for all pollutants except $PM_{10}$, and a SD greater than the mean for CO, benzene, and BC/EC. Bicyclists' average CO exposure concentrations have been measured in the range of 0.5–13 ppm, though all studies after 1995 report central value concentrations below 3 ppm.

**Table 1.**   Summary of the 42 studies directly measuring on-road bicyclists' exposure concentrations

| Pollutant | Units[a] | No. of studies (N)[b] | Years of studies | Reported central values[c] | | | | Reported disaggregate values | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Mean (SD) | Median | Low | High | Low | High |
| CO | ppm | 12 (16) | 1976–2013 | 2.8 (3.9) | 1.2 | 0.5 (New Zealand) | 13 (USA) | 0.1 (New Zealand) | 21 (USA) |
| $NO_2$ | µg/m$^3$ | 4 (5) | 1995–2006 | 55.8 (33.8) | 46.3 | 26 (Australia) | 114 (Netherlands) | 8 (Australia) | 262 (the Netherlands) |
| VOC[d] | | 9 | 1991–2011 | | | | | | |
| Benzene | µg/m$^3$ | 9 (12) | 1991–2011 | 17.2 (18.3) | 10.6 | 0.34 (Canada) | 56 (UK) | 0.1 (Canada) | 120 (UK) |
| Toluene | µg/m$^3$ | 6 (7) | 1991–2011 | 57.6 (51.6) | 50.5 | 1.07 (Canada) | 122 (UK) | 0.3 (Canada) | 1230 (Italy) |
| Xylenes | µg/m$^3$ | 5 (6) | 1991–2011 | 48.9 (45.2) | 44.3 | 0.6 (Canada) | 105 (Italy) | 0.15 (Canada) | 281 (Italy) |
| PM[e] | | | | | | | | | |
| UFP, PNC | pt/cc | 18 (31) | 2005–13 | 28450 (18 169) | 24 800 | 8734 (Belgium) | 93 968 (UK) | 1900 (USA) | 1 033 188 (USA) |
| $PM_{2.5}$ | µg/m$^3$ | 17 (29) | 2001–14 | 29.9 (22.8) | 23.5 | 4.88 (USA) | 88.1 (Ireland) | 0 (the Netherlands) | 130 (UK) |
| $PM_{10}$ | µg/m$^3$ | 10 (15) | 2001–14 | 50.2 (12.0) | 50.0 | 32.0 (New Zealand) | 72.7 (Belgium) | 8.2 (New Zealand) | 160 (Belgium) |
| BC, EC[f] | µg/m$^3$ | 9 (14) | 2002–13 | 6.85 (7.09) | 3.04 | 1.05 (Canada) | 21.0 (UK) | 0.09 (USA) | 63.83 (USA) |

[a]Conversion of reported values between µg/m$^3$ and ppb or ppm assumes molar gas volume of 24.45 L.
[b]Some studies report separate central value results by route. All routes are included except those designated as 'rural' settings. See Supplemental Material for details.
[c]When multiple central value statistics are reported in a study, a single value was selected as the arithmetic mean, geometric mean, or median, in that order.
[d]Various compounds are reported in the studies measuring VOC; only benzene, toluene, and (o-, m-, and p-) xylenes are reported in more than half of the VOC studies.
[e]In addition to BC, UFP, $PM_{2.5}$, and $PM_{10}$, six studies report PM of other sizes ($PM_1$ through $PM_5$) over the years 1991–2013.
[f]Excludes three additional studies that only report BC concentrations in units of absorbance.

### 3.1. *Modal Comparisons of Exposure Concentration*

A popular study design for traveler exposure studies is modal comparisons, in which exposure concentrations are compared for travelers using different transportation modes between the same origin and destination or along identical or parallel routes. Results from modal comparisons of exposure are inconsistent. Bicyclists sometimes have lower exposure concentrations than motorized modes, especially when they use facilities that are separated from traffic (Adams, Nieuwenhuijsen, & Colvile, 2001; Adams, Nieuwenhuijsen, Colvile, Older, & Kendall, 2002; Boogaard, Borgman, Kamminga, & Hoek, 2009; Chertok, Voukelatos, Sheppeard, & Rissel, 2004; De Nazelle et al., 2012; Dons, Int Panis, Van Poppel, Theunis, & Wets, 2012; Kaur et al., 2007; Kingham, Longley, Salmond, Pattinson, & Shrestha, 2013; Kingham, Meaton, Sheard, & Lawrenson, 1998; Knibbs et al., 2011; McNabola, Broderick, & Gill, 2008; van Wijnen, Verhoeff, Jans, & Bruggen, 1995). But modal comparison studies have also found insignificant differences in concentrations by mode, significantly higher bicyclist exposure concentrations than other modes, or inconsistent results by pollutant, location, or time of day (Boogaard et al., 2009; Chertok et al., 2004; De Nazelle et al., 2012; Int Panis et al., 2010; Kaur & Nieuwenhuijsen, 2009; Kingham et al., 2013; Nwokoro et al., 2012; Quiros, Lee, Wang, & Zhu, 2013; Ragettli et al., 2013; Waldman, Weiss, & Articola, 1977; Yu et al., 2012). Likely causes of inconsistent results across studies include differences in the proximity and intensity of motor vehicle traffic, varying availability and use of bicycle facilities, and instrumentation/sampling differences (see Supplemental Material for information on study methods).

Modal comparison exposure studies typically use the same routes or origins and destinations across modes and fix other travel characteristics (e.g. departure time). While potentially informative, these comparisons are not always realistic because pollution exposure is also affected by intrinsic modal travel differences. The more realistic modal comparisons allow self-selected routes or direct active travelers to use representative routes for their mode — but local transportation network characteristics may affect the results. Bicycle travel patterns are different from motorized ones because of distinct traveler characteristics, trip distances, and route preferences (Broach, Dill, & Gliebe, 2012; Plaut, 2005). Real-world bicycle trips tend to be shorter and in higher density parts of a city than trips using motorized modes. Bicycle trips are also highly seasonal (Nankervis, 1999), so a different distribution of meteorological conditions could be expected by mode, with a systematic influence on exposure concentrations. Most bicycle exposure studies occur during warmer months when a greater proportion of bicycling occurs (see the Supplemental Material), but the joint seasonality of mode splits and pollution levels should be considered when comparing travelers' exposures — especially for year-round bicyclists.

Although modal comparisons can be informative, they rarely provide practical insights into how to reduce exposure concentrations, other than mode shifts. Modal comparison studies rarely vary within mode factors (such as route choice), which can be the most important determinants of exposure concentrations during travel (Knibbs et al., 2011).

### 3.2. *Factors Affecting Bicyclists' Exposure Concentrations*

Multivariate analyses of travelers' exposure concentrations have shown that important factors include wind and weather, traffic and route, and the built environment around the roadway (Adams, Nieuwenhuijsen, & Colvile, 2001;

Berghmans et al., 2009; Boogaard et al., 2009; Hatzopoulou, Weichenthal, Dugum, et al., 2013; Kaur & Nieuwenhuijsen, 2009; Kaur et al., 2007; Knibbs et al., 2011; McNabola, Broderick, & Gill, 2009a; Quiros et al., 2013). But few studies have looked at bicyclist-specific factors that could influence exposure, such as lateral position in the road, proximity to exhaust pipes, breathing height, and the ability to 'dodge between' vehicles (Kaur et al., 2007).

Wind is consistently a significant factor for exposure, decreasing concentrations through dispersion (Adams, Nieuwenhuijsen, & Colvile, 2001; Hatzopoulou, Weichenthal, Dugum, et al., 2013; Hong & Bae, 2012; Jarjour et al., 2013; Kaur & Nieuwenhuijsen, 2009; Kaur et al., 2007; Kingham et al., 1998; Knibbs et al., 2011; McNabola et al., 2009a). Temperature is less consistently a significant factor, and effects can be difficult to distinguish from humidity because of a strong negative correlation (Adams, Nieuwenhuijsen, & Colvile, 2001; Hatzopoulou, Weichenthal, Dugum, et al., 2013; Kaur & Nieuwenhuijsen, 2009; Kaur et al., 2007; Kingham et al., 1998; Knibbs et al., 2011). Time of day is a factor that incorporates influencing effects of local weather and diurnal traffic patterns — particularly relevant for urban areas with diurnal temperature inversions that significantly affect pollutant levels.

After weather, the next most important factors for bicyclists' exposure concentrations can be combined into a single category: separation from motor vehicle traffic. These factors include the concentration-reducing effects of traveling on low-traffic routes (Hatzopoulou, Weichenthal, Dugum, et al., 2013; Hertel, Hvidberg, Ketzel, Storm, & Stausgaard, 2008), on separated bicycle facilities (Hatzopoulou, Weichenthal, Dugum, et al., 2013; Hong & Bae, 2012; Kendrick et al., 2011; Kingham et al., 2013, 1998), and during off-peak periods or weekends (Dons et al., 2013; Huang, Deng, Wu, & Guo, 2012; Kleiner & Spengler, 1976). Lacking more specific data, the influence of motor vehicle traffic on exposure concentrations is sometimes estimated using a proxy of facility type, time-of-day, or average daily traffic (ADT) estimates (Boogaard et al., 2009; Cole-Hunter, Morawska, Stewart, Jayaratne, & Solomon, 2012; Hong & Bae, 2012; Jarjour et al., 2013; Ragettli et al., 2013; Weichenthal et al., 2011).

The influence of motor vehicle traffic was measured in 14 different studies by comparing bicyclists' exposure concentrations on 'high-traffic' and 'low-traffic' routes or using a related dichotomy (inner-city/suburban, on-road/off-road, near-road/cycle path). The combined results are shown in Figure 2, with the median and range of reported percent increases on 'high-traffic' versus 'low-traffic' routes (see the Supplemental Material for sources). As expected, pollutants that are more dominated by motor vehicle sources in roadway environments (hydrocarbon VOC, UFP) show larger increases on high-traffic routes.

Explicit traffic variables such as motor vehicle volume or speed are often not included in bicyclist pollution exposure analysis because of a lack of concomitant data. When assessed, vehicle volumes, particularly truck or diesel vehicles, generally have a positive influence on pollutant exposure concentrations, though they are not always significant variables (Boogaard et al., 2009; Dons et al., 2013; Hatzopoulou, Weichenthal, Dugum, et al., 2013; Kaur & Nieuwenhuijsen, 2009; Knibbs et al., 2011; McNabola et al., 2009a; Quiros et al., 2013). Aggregate traffic variables such as ADT cannot reveal the potentially important influences of varying traffic volumes, speeds, queuing, and fleet composition over the data collection periods. Furthermore, highly aggregate traffic variables are often correlated with geometric roadway characteristics such as the number of lanes, which also influence pollutant concentrations through dispersion.
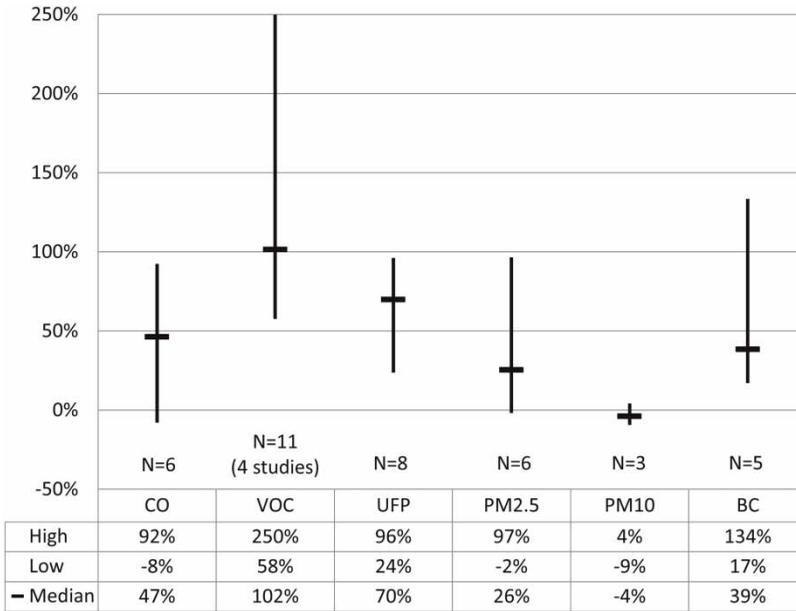
| | CO | VOC | UFP | PM2.5 | PM10 | BC |
|---|---|---|---|---|---|---|
| | N=6 | N=11 (4 studies) | N=8 | N=6 | N=3 | N=5 |
| High | 92% | 250% | 96% | 97% | 4% | 134% |
| Low | -8% | 58% | 24% | -2% | -9% | 17% |
| – Median | 47% | 102% | 70% | 26% | -4% | 39% |

**Figure 2.** Reported increases in bicyclists' exposure concentrations in 'high-traffic' versus 'low-traffic' routes and locations.

Notes: Urban/rural comparisons are excluded. Where multiple observations are reported per study (e.g. by city or time period), a weighted average by number of samples was used. For VOC, reported BTEX compounds (benzene, toluene, ethylbenzene, and xylenes) are included (11 comparisons for these compounds in different studies).

*Sources*: CO (Bevan, Proctor, Baker-Rogers, & Warren, 1991; Jarjour et al., 2013; Kingham et al., 2013; Kleiner and Spengler, 1976; Waldman et al., 1977; Weichenthal et al., 2011), VOC (Bevan et al., 1991; Kingham et al., 1998; McNabola et al., 2008; Weichenthal et al., 2011), UFP (Cole-Hunter et al., 2012, 2013; Jarjour et al., 2013; Kingham et al., 2013; Ragettli et al., 2013; Strak et al., 2010; Weichenthal et al., 2011; Zuurbier et al., 2010), PM2.5 (Adams, Nieuwenhuijsen, Colvile, McMullen, & Khandelwal, 2001; Jarjour et al., 2013; Kingham et al., 2013; McNabola et al., 2008; Weichenthal et al., 2011; Zuurbier et al., 2010), PM10 (Kingham et al., 2013; Strak et al., 2010; Zuurbier et al., 2010), BC (Jarjour et al., 2013; Kingham et al., 1998; Strak et al., 2010; Weichenthal et al., 2011; Zuurbier et al., 2010)

Traffic data used in bicycle exposure studies to date have been non-specific to the study period, limited in spatial and temporal coverage, and/or highly aggregated (in time and vehicle type). Of the 42 studies included in Table 1, only 4 report traffic data collected at the locations and time periods of air quality measurements. Kaur, Nieuwenhuijsen, and Colvile (2005)[1] and McNabola et al. (2008)[2] retrieved unclassified hourly vehicle volumes from traffic signal data at major intersections on the study routes. Hatzopoulou, Weichenthal, Dugum, et al. (2013) collected intermittent manual vehicle counts using five vehicle classes for 10–20 min periods sequentially at dozens of locations around the on-road measurement area. Quiros et al. (2013) performed intermittent manual vehicle counts for 5 min periods using nine vehicle classes (including bicycles and pedestrians) at a single location on the study corridor.

The next major factors for exposure concentrations, after weather and motor vehicle traffic, are the study setting and methodology. Comparing measured exposure concentrations across studies reveals wide ranges (Table 1), indicative of different study settings (time frame, city, and locational characteristics) and different experimental methods (instruments, sampling strategy, aggregation,

etc.). Potentially important differences among study settings include traffic patterns, weather conditions, vehicle fleets and fuels, urban form, and topography. Boogaard et al. (2009) compare bicyclists' on-road exposure concentrations in 11 Dutch cities over a three-month period (using a consistent methodology) and report coefficients of variability for UFP and $PM_{2.5}$ of 0.22 and 0.86 among cities. For comparison, the coefficients of variability for UFP and $PM_{2.5}$ among studies in Table 1 are 0.64 and 0.76.

## 4.    Bicyclists' Air Pollution Intake

The mass of air pollutants that cross the body boundary through the mouth and nose is the intake dose (Ott et al., 2007). Estimates of intake dose rates per unit time combine exposure concentrations with a respiration rate; intake dose rates per unit distance also take travel duration into account (as does total intake dose over a journey). Some studies consider only duration (not respiration) by estimating cumulative exposure, such as Nwokoro et al. (2012) and Ragettli et al. (2013). Measurement and analysis of bicyclists' pollutant intake facilitates a transition toward a dose-oriented estimation of health effects.

### 4.1.    *Respiration*

Respiration rate is commonly expressed as the minute respiratory volume (or minute ventilation, $V_E$) — which is the volume of air displaced per minute. Minute respiratory volume is the product of the tidal volume $V_T$ and the breathing frequency $f_r$ (breaths per minute). Tidal volume $V_T$ is the volume of air displaced in a single breath; typical ranges are 1.4–2.2 L for bicyclists and 0.6–0.8 L for persons at rest or in a car (Int Panis et al., 2010). Multiplying $V_E$ by the average exposure concentration yields the average pollutant inhalation rate in mass per unit time.

Table 2 summarizes published traveling bicyclists' respiration parameters (see Supplemental Material for a description of the measurement methods). Minute ventilation has been reported as 22–59 L/min for bicyclists: two to five times higher than for travelers in automobiles or at rest. Bernmark et al. (2006) found $V_E$ peaks for bicycle messengers of up to 97 L/min. The ranges of minute ventilations in Table 2 are related to the different average travel speeds and heart rates among the studies (included in Table 2), as well as potentially other experimental differences such as terrain, bicycle weight and condition, weather, and subject fitness. Greater exertion increases $V_E$ primarily by an increase in $V_T$ at lower levels of exercise and by an increase in $f_r$ at higher levels of exercise; $f_r$ is the dominant factor at 70–80% of peak exercise level (Weisman, 2003). Trained professional bicyclists can achieve a greater increase in $V_E$ through increases in $V_T$ than recreational bicyclists (Faria, Parker, & Faria, 2005b).

For active travelers such as bicyclists, $V_E$ will be a function of travel characteristics that determine power requirements. The major determinants of power output during bicycling are energy losses (resistance) and changes in kinetic and potential energy (acceleration and grades, respectively). The largest energy losses are typically aerodynamic drag followed by rolling resistance. Rolling resistance becomes a more important factor at lower speeds and in still air, when drag is less severe (Di Prampero, Cortili, Mognoni, & Saibene, 1979; Faria, Parker, & Faria, 2005a; Martin, Milliken, Cobb, McFadden, & Coggan, 1998; Olds, 2001; Whitt, 1971; Wilson, 2004). Nadeau, Truchon, Brochu, and Tardif

**Table 2.**   Respiration-related parameters measured for bicyclists

| Group | Minute ventilation, $V_E$ (L/min) | Tidal volume (L) | Breathing frequency (min$^{-1}$) | Heart rate (bpm) | Speed (kph) | Ratio of bicycle/ car, $V_E$[a] | Reference and method[b] |
|---|---|---|---|---|---|---|---|
| All | 23.5 | | | 100 | 12 | 2.0 | 1, estimated |
| | 28.7 | | | | 13.5 | 2.5 | 2, on-road |
| Male | 22 | | | 94 | 12 | 1.8 | 1, estimated |
| | 22.7 | | | | 14 | 1.9 | 3, on-road |
| | 25 | 1.25 | 20 | | 8 | 2.1 | 4, lab |
| | 28 | | | | | 2.3 | 5, lab |
| | 31 | | | 107 | | 2.6 | 6, estimated |
| | 31.4 | | | | 19.5 | 2.6 | 3, on-road |
| | 44.2 | | | 138 | 20 | 3.7 | 7, estimated |
| | 50 | 1.92 | 26 | | 19 | 4.2 | 4, lab |
| | 51.2 | | | | 24 | 4.3 | 3, on-road |
| | 59.1 | 2.2 | 27.9 | 129.6 | 20.5 | 4.9 | 8, on-road |
| Female | 22.6 | | | | 14 | 2.1 | 3, on-road |
| | 27.6 | | | 116 | 12 | 2.5 | 1, estimated |
| | 32.8 | | | | 19.5 | 3.0 | 3, on-road |
| | 46.2 | 1.4 | 32.7 | 140 | 19.5 | 4.2 | 8, on-road |
| | 51.8 | | | | 24 | 4.7 | 3, on-road |

Note: Blank cells are not reported. Methodologies are categorized as 'on-road' (direct on-road measurement of respiration using masks), 'lab' (laboratory ergometer-based respiration measurements), and 'estimated' (on-road measurement of heart rate and estimation of respiration using laboratory ergometer-based heart rate/ventilation relationships).
[a]Reference minute ventilation for car drivers of 12 L/min for males, 11 L/min for females, and 11.5 L/min for all, based on Adams (1993), Int Panis et al. (2010), O'Donoghue, Gill, McKevitt, and Broderick (2007), van Wijnen et al. (1995), Zuurbier, Hoek, Hazel, and Brunekreef (2009).
[b]References: 1 (Zuurbier et al., 2009), 2 (van Wijnen et al., 1995), 3 (Adams, 1993), 4 (McNabola, Broderick, & Gill, 2007), 5 (O'Donoghue et al., 2007), 6 (Bernmark, Wiktorin, Svartengren, Lewné, & Åberg, 2006), 7 (Cole-Hunter et al., 2012), and 8 (Int Panis et al., 2010).

(2006) measured $V_E$ of around 12, 23, and 35 L/min for bicycle ergometer work-loads of 0, 50, and 100 W, respectively, suggesting that the subjects in the studies in Table 2 experienced workloads ranging from around 50 W to well over 100 W of power.

Compilations of physical activity data often use metabolic equivalent of task (MET) to compare energy expenditure with a standardized unit; a MET is defined as $\text{MET} = \dot{e}/\text{RMR}$, where $\dot{e}$ is the rate of metabolic energy production and RMR is the resting metabolic rate (Ainsworth et al., 2011a, 2011b; U.S. Environmental Protection Agency, 2009). RMR is an individual-specific value (varying across individuals), often assumed to be 3.5 ml-$O_2$/min per kg body mass — i.e. $\text{MET} = \dot{e}/K \cdot m$, where $K$ is a constant and $m$ is body mass. Thus, MET values are directly proportional to energy expenditure for an individual and inversely proportional to an individual's body mass for a given energy expenditure.[3]

Resting activities are at an MET of 1, while 'general' bicycling is at an MET of 7.5 and bicycling 'to/from work, self-selected pace' is at MET 6.8 in the 'Compendium of Physical Activities' (Ainsworth et al., 2011a, 2011b). The Compendium lists 16 different types of bicycling as activities with energy expenditures ranging from 3.5 MET for 'leisure' bicycling at 5.5 mph to 16 MET for competitive mountain bicycle racing. Non-sport bicycling has been estimated to require 3.5–9

MET of energy expenditure, with power output of roughly 50–150 W, depending on the speed (Bernmark et al., 2006; De Geus, de Smet, Nijs, & Meeusen, 2007; Whitt, 1971). MET values have been employed to estimate bicyclists' respiration for pollution dose assessments using both reference MET values and MET values estimated from accelerometer measurements; average accelerometer-based MET for bicycling was estimated at 6.58 with a corresponding ventilation rate of 41 L/min (De Nazelle et al., 2012). Respiration was estimated from MET values using stochastic relationships between oxygen uptake rates and ventilation rates along with the individuals' body mass (De Nazelle, Rodríguez, & Crawford-Brown, 2009; Johnson, 2002).

### 4.2.  *Studies of Bicyclists' Pollution Intake*

Table 3 characterizes published studies of bicyclists' air pollution exposure, intake, uptake, or biomarkers that use spatially explicit exposure concentration data (modeled or measured). Studies are categorized according to how (and whether) they account for (1) respiration (i.e. intake), (2) uptake of gases or deposition of particles, and (3) health biomarkers. The last two dimensions are discussed in Sections 5 and 6, respectively. 'Constant' respiration refers to studies that apply fixed respiration rates by mode or individual; 'variable' respiration refers to studies that use varying respiration rates by trip or at a greater level of detail. The categorization in Table 3 proceeds roughly from least to most comprehensive (A–M) in terms of targeting farther along the exposure-health pathway, assessing linkages more directly (e.g. measuring versus assuming), and/or examining more intermediate steps between exposure and uptake or biomarkers.

Many studies consider only exposure concentrations and neglect the question of intake dose and the issue of varying respiration and energy expenditure by travel mode and condition (Type A). Similarly, some studies measure exposure concentrations and uptake doses or health biomarkers directly, but do not address the intermediate step of intake or respiration (Types J and K). Of the 19 studies in Table 3 that explicitly consider respiration, 16 use fixed values of $V_E$ for each travel mode or individual (Types B–E, G, H, and L). Type B studies (7 of the 19) apply an assumed $V_E$ for bicyclists based on other published research. Two studies (Types D and H) use bicycle ergometers in a laboratory to determine representative respiration values by mode. Of the eight studies that model respiration (Types C, F, G, L, and M), six use ergometers to develop individual subject functions to estimate on-road $V_E$ from field-measured heart rate (HR), one uses previously developed $V_E$-HR functions with field-measured HR, and one estimates respiration from accelerometer-based MET values — see Section 4.1. Only two of these eight studies (Types F and M) estimate intake using variable ventilation rates by trip (Cole-Hunter et al., 2012) or at 2 min aggregations (Nyhan, McNabola, & Misstear, 2014).

Two studies in Table 3 directly measure on-road bicyclists' minute ventilation in order to estimate intake dose (Types E and I). Van Wijnen et al. (1995) use fixed mode-specific respiration rates that are the averages of measured on-road minute ventilation for a set of test subjects traveling on the same test routes as the concentration measurements, but at different times. Int Panis et al. (2010) use simultaneously monitored on-road respiration and concentration data to estimate intake dose. Combining tidal volume and pollutant concentration measurements, Int Panis et al. calculate breath-by-breath mass intake and sum over trips, thus including both respiration and duration effects on total intake.

**Table 3.** Categorization of bicyclists' air pollution exposure, intake, uptake, and biomarker studies

| Study type | Number of studies[b] | (1) Respiration[a] | | | | | | (2) Uptake/deposition | | | (3) Biomarkers |
| | | Constant | | | | Variable | | | | | |
| | | Assumed | Modeled | Measured in-lab | Measured on-road | Modeled | Measured on-road | Assumed | Modeled | Measured | Measured |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 28 | | | | | | | | | | |
| B | 7 | • | | | | | | | | | |
| C | 3 | | • | | | | | | | | |
| D | 1 | | | • | | | | | | | |
| E | 1 | | | | • | | | | | | |
| F | 1 | | | | | • | | | | | |
| G | 1 | | • | | | | | • | | | |
| H | 1 | | | • | | | | | • | | |
| I | 1 | | | | | | • | | • | | |
| J | 2 | | | | | | | | | • | |
| K | 8 | | | | | | | | | | • |
| L | 2 | | • | | | | | | | | • |
| M | 1 | | | | | • | | | • | | • |
| Totals: | 57 | 7 | 6 | 2 | 1 | 2 | 1 | 1 | 3 | 2 | 11 |

Note: Grey cells mean that dimension was not assessed (respiration, uptake, and biomarkers); • indicates the method of assessment for that dimension by each study type.
A: Adams, Nieuwenhuijsen, and Colvile (2001), Adams, Nieuwenhuijsen, Colvile, McMullen, and Khandelwal (2001), Adams et al. (2002), Bean, Carslaw, Ashmore, Gillah, and Parkinson (2011), Berghmans et al. (2009), Bevan et al. (1991), Boogaard et al. (2009), Chan, Hung, and Qin (1994), Chertok et al. (2004), Dekoninck, Botteldooren, and Int Panis (2013), Dons et al. (2013), Farrar, Dingle, and Tan (2001), Gee and Raper (1999), Hatzopoulou, Weichenthal, Barreau, et al. (2013), Hatzopoulou, Weichenthal, Dugum, et al. (2013), Hertel et al. (2008), Hong and Bae (2012), Kaur and Nieuwenhuijsen (2009), Kaur et al. (2005), Kendrick et al. (2011), Kingham et al. (2013, 1998), Kleiner and Spengler (1976), McNabola et al. (2009a), Ragettli et al. (2013), Sitzmann, Kendall, Watt, and Williams (1999), Strauss et al. (2012), Thai, McKendry, and Brauer (2008); B: Dirks, Sharma, Salmond, and Costello (2012), Dons et al. (2012), Fajardo and Rojas (2012), Huang et al. (2012), Quiros et al. (2013), Rank, Folke, and Homann Jespersen (2001), Yu et al. (2012); C: Bernmark et al. (2006), de Nazelle et al. (2012), Zuurbier et al. (2010); D: O'Donoghue et al. (2007); E: van Wijnen et al. (1995); F: Cole-Hunter et al. (2012); G: Vinzents et al. (2005); H: McNabola et al. (2008); I: Int Panis et al. (2010); J: Bergmaschi et al. (1999), Nwokoro et al. (2012); K: Bos et al. (2011), Cole-Hunter et al. (2013), Jacobs et al. (2010), Jarjour et al. (2013), Strak et al. (2010), Waldman et al. (1977), Weichenthal et al. (2011, 2012); L: Zuurbier, Hoek, Oldenwening, Meliefste, van den Hazel, et al. (2011), Zuurbier, Hoek, Oldenwening, Meliefste, Krop, et al. (2011); M: Nyhan, McNabola, and Misstear (2014).
[a]'Constant' respiration means fixed respiration rates by mode or individual; 'variable' respiration means varying respiration by trip or greater level of detail.
[b]Includes all published papers of on-road bicyclists' pollution exposure with spatially explicit exposure concentration data.

Table 3 shows that there has been little assessment of the variability of bicyclists' respiration as they travel in an urban environment. If the variability in respiration is independent of exposure concentrations, then representative averages for each will suffice (assuming linearity). But there is likely to be spatial correlation between pollutant concentrations and bicyclist energy expenditure at locations such as intersections and hills, where both motor vehicles and bicyclists are required to generate more energy. There is also a potential correlation between exposure duration and exposure concentration at congested bottlenecks or busy intersections. At the route level, Cole-Hunter et al. (2013) found no significant differences in measured HR for routes with low and high proximity to traffic; they conclude that variability in UFP intake dose for bicyclists would be predominantly determined by exposure concentrations, not ventilation characteristics. But a wide range of bicyclists' respiration values have been reported (Section 4.1), and the lack of bicyclist intake dose studies considering variable respiration rates leaves the question open.

### 4.3.  *Modal Comparisons of Pollution Intake*

Int Panis et al. (2010) argues that comparisons of exposure concentrations by travel mode (as in Section 3.1) are 'not entirely relevant' because of the dominating effect of breathing differences among modes. Modal comparisons of pollution intake dose go beyond exposure concentrations by including respiration to compare intake dose rates per unit time. More detailed comparisons also consider the intake effects of travel duration differences, assessing intake doses per trip or unit travel distance. For faster trips, the time spent in an alternative environment is typically neglected; this aspect may be important when the air quality at the trip destination is poor. Inherent speed differences by mode are problematic for modal comparisons of intake rates by either normalization.

Table 4 summarizes the 12 published modal comparisons that include respiration, showing the median and range for ratios of bicycle to alternative mode intake or uptake doses. Dose ratios are presented separately for the eight studies that compare doses per unit distance and the five studies that compare doses per unit time (one assesses both). For most pollutants, studies that compare doses per unit distance find greater bicycle/car dose ratios than comparisons per unit time, as expected from bicyclists' lower travel speeds. This body of literature is still much smaller than modal comparisons of exposure, but for the most part two to five times higher ventilation rates and slower travel speeds for bicyclists compared to motor vehicle passengers outweigh any beneficial exposure concentration differences. Bicyclists' doses are less consistent when compared to pedestrians, which is not surprising because walking is another active travel mode with elevated respiration. Pedestrians typically have lower respiration rates (McNabola et al., 2007) but also lower speeds, with counteracting effects on intake rates per unit distance.

Few of the modal comparisons of dose directly measure on-road respiration or model respiration as a function of travel characteristics beyond mode. This is important because travel attributes such as road grade and speed affect respiration and inhalation rates for bicyclists but not motorized modes. Intake doses per trip will be further affected by duration changes with route and destination choices, which are normally not varied in modal comparisons (as discussed in Section 3.1). Furthermore, active travelers tend to have unique demographics

**Table 4.** Ratios of intake or uptake doses for bicyclists versus other modes

| | Alternative mode | Per unit distance[a] | | Per unit time | |
|---|---|---|---|---|---|
| | | $N$[b] | Median (Range) | $N$ | Median (range) |
| CO | Pedestrian | 1 | 0.80 | 0 | |
| | Car/taxi | 3 | 1.09 (0.36–4.67) | 1 | 0.87 |
| | Bus | 3 | 1.63 (1.07–4.67) | 0 | |
| | Rail | 1 | 7.00 | 0 | |
| VOC[c] | Pedestrian | 1 | 1.11 | 0 | |
| | Car | 1 | 0.81 | 4 (2 studies) | 0.71 (0.50–0.72) |
| | Bus | 2 | 1.60 (1.25–1.96) | 0 | |
| $NO_2$ | Car | 0 | | 1 | 3.08 |
| UFP | Pedestrian | 2 | 0.68 (0.51–0.84) | 0 | |
| | Car | 3 | 5.42 (1.00–10.42) | 1 | 2.09 |
| | Bus | 1 | 1.90 | 1 | 1.87 |
| $PM_{2.5}$ | Pedestrian | 4 | 1.13 (0.47–1.97) | 1 | 2.09 |
| | Car/taxi | 5 | 3.36 (1.38–10.88) | 1 | 1.70 |
| | Bus | 4 | 1.77 (1.06–4.78) | 2 | 3.14 (1.91–4.36) |
| | Rail | 1 | 2.56 | 1 | 2.29 |
| $PM_{10}$ | Pedestrian | 1 | 1.62 | 1 | 1.82 |
| | Car | 1 | 6.75 | 1 | 1.66 |
| | Bus | 1 | 3.21 | 2 | 2.13 (1.15–3.10) |
| | Rail | 1 | 3.06 | 1 | 2.21 |
| BC | Pedestrian | 1 | 0.81 | 0 | |
| | Car | 1 | 0.84 | 2 | 1.90 (1.36–2.44) |
| | Bus | 1 | 1.64 | 1 | 1.51 |

Notes: Sources, per unit distance: CO: de Nazelle et al. (2012), Dirks et al. (2012), Huang et al. (2012); VOC: McNabola et al. (2008), O'Donoghue et al. (2007); UFP: Int Panis et al. (2010), de Nazelle et al. (2012), Quiros et al. (2013); $PM_{2.5}$: Huang et al. (2012), Int Panis et al. (2010), McNabola et al. (2008), de Nazelle et al. (2012), Nyhan et al. (2014), Quiros et al. (2013); $PM_{10}$: Int Panis et al. (2010), Nyhan et al. (2014); BC: de Nazelle et al. (2012).
Sources, per unit time: CO: van Wijnen et al. (1995); VOC: Rank et al. (2001), van Wijnen et al. (1995); $NO_2$: van Wijnen et al. (1995); UFP: Zuurbier et al. (2010); $PM_{2.5}$: Nyhan et al. (2014), Zuurbier et al. (2010); $PM_{10}$: Nyhan et al. (2014), Zuurbier et al. (2010); BC: Dons et al. (2012), Zuurbier et al. (2010).
[a]Values are ratios of bicycle to alternative mode doses in mass, particles, or ppb per unit distance (i.e. per km or per trip) or per unit time (i.e. per hour of travel); the table includes all studies that directly compare pollutant intake or uptake between travelers by bicycle and other modes for similar trips.
[b]A single mean value (weighted by number of samples) was computed for studies reporting separate results by routes or times of day. VOC doses per unit time are from two studies, with one reporting three different compounds.
[c]Only reported values for BTEX compounds are included.

(Plaut, 2005), which could systematically impact respiration through physiological attributes such as sex and health condition (Adams, 1993).

## 5. Bicyclists' Air Pollution Uptake

A portion of inhaled pollutants are either absorbed (gases) or deposited (particles) onto the lining of the respiratory tract or into the bloodstream. Absorbed/deposited pollutants are then either expelled (through mucociliary clearance or desorption) or transported to body tissues. The air pollution uptake dose is the amount of pollutant that is not exhaled or expelled, but rather incorporated into the body (Figure 1).

**Table 5.** Factors that increase pollutant uptake

| Factor | Increased uptake with: |
| --- | --- |
| Exposure | |
|   Concentration | Higher concentrations |
|   Duration | Longer duration |
| Pollutant | |
|   Particle size | Smaller particles |
|   Particle hygroscopicity | More hydrophobic particles |
|   Gas solubility | More blood- and lipid-soluble compounds |
| Respiration/physiology | |
|   Breath volume flow rate ($V_E$) | Greater ventilation |
|   Depth of breathing ($V_T$) | Greater tidal volume |
|   Path of breathing | Oral breathing |
|   Cardiac output (lung perfusion) | Greater perfusion |
|   Metabolic rate | Higher metabolic rate |

Table 5 summarizes the factors that are expected to increase pollutant uptake for bicyclists. The first two factors reflect the exposure in terms of concentration and duration. The next set of factors in Table 5 is attributes of the pollutants that determine uptake dose (independent of travel characteristics). Particle size is important for PM uptake because deposition and clearance rates vary with particle size. UFP deposition is also influenced by the particles' growth characteristics in high humidity conditions such as in lung airways (hygroscopicity). Gas reactivity and solubility in blood and lipids are similarly important because they affect absorption and diffusion rates (Daigle et al., 2003; International Commission on Radiological Protection [ICRP] Task Group, 1994; Löndahl et al., 2007; McNabola et al., 2008; Ott et al., 2007; West, 2012).

Table 5 also summarizes the physiology and respiration factors that influence uptake. Intake dose is determined by $V_E$ and the exposure concentration; uptake dose is further influenced by the depth of respiration ($V_T$) and the amount of oral breathing. Greater uptake fractions of inhaled PM occur during deeper and more oral breathing (ICRP Task Group, 1994), which are associated with higher levels of exertion (Samet, Lambert, James, Mermier, & Chick, 1993; Weisman, 2003). Daigle et al. (2003) found that when subjects' $V_E$ increased from 11.5 to 38.1 L/min the deposition fraction (DF), the portion of particles that are not exhaled after inhalation, increased from 0.66 to 0.83 by number of particles and from 0.58 to 0.76 by mass of particles. Thus, a $V_E$ increase by a factor of 3.3 led to a total deposition increase by a factor of 4.5 due to a higher DF. Löndahl et al. (2007) found only small changes in DF for UFP (by less than 0.03) during exercise when compared to rest ($V_E$ of 33.9 versus 7.8 L/min), but both of these studies found that established models under-predicted deposition of UFP — especially during exercise.

Uptake rates for gaseous pollutants are also affected by the characteristics of the gas and the level of physical exertion. VOC and CO uptake rates are several times greater during exercise than at rest for a given exposure concentration. But the uptake *fraction* of inhaled gases tends to decrease with exertion level because gas uptake rates increase more slowly than intake rates with exercise (Astrand, 1985; Astrand, Engstrom, & Ovrum, 1978; Filley, MacIntosh, & Wright, 1954; Nadeau et al., 2006; Pezzagno, Imbriani, Ghittori, & Capodaglio, 1988). Diffu-

sion-limited gases such as CO are primarily impacted by the diffusing capacity of the lungs, which can increase by a factor of 3 during exercise (West, 2012). Uptake rates for perfusion-limited gases such as low-solubility VOC and $NO_2$ increase with ventilation and perfusion of the lungs, gas partial pressure differences between blood and air, and gas solubility in blood (Astrand, 1985; Csanády & Filser, 2001; Farhi, 1967; West, 2012). As blood concentrations approach equilibrium with inspired air, the uptake rate will fall to the steady-state rate of metabolic clearance (Csanády & Filser, 2001; Wallace, Pellizzari, & Gordon, 1993). Although exercise increases ventilation and perfusion, it also can decrease the rate at which pollutants are metabolized by reducing blood flow to the liver — reducing the steady-state uptake rate while simultaneously increasing blood concentrations (Astrand, 1985; Csanády & Filser, 2001; Kumagai & Matsunaga, 2000; Nadeau et al., 2006).

Detailed uptake models allow estimation of different locations/tissues of pollutant uptake, which is relevant because of varying susceptibility to negative health effects from air pollution uptake by different tissues. Common uptake models include body compartment and physiologically based pharmacokinetic models for gases and human respiratory tract models for both gases and PM (Heinrich-Ramm et al., 2000; Hofmann, 2011; ICRP Task Group, 1994; King et al., 2011; Ott et al., 2007; Wallace et al., 1993; Wallace, Nelson, Pellizzari, & Raymer, 1997). Uptake models are generally validated using much steadier air concentrations than have been observed in on-road environments, so it is not clear how applicable they are for on-road uptake analysis with highly transient exposure concentrations.

Uptake of air pollutants by bicyclists has been studied less than exposure concentrations or intake doses (6 of the 57 studies in Table 3 explicitly consider uptake). Vinzents et al. (2005) conservatively estimate deposition as linearly proportional to workload (on average 43% higher deposition of PM while bicycling than at rest). Int Panis et al. (2010) use DF that vary with $V_E$, $V_T$, and particle size, based on two previous studies of particle deposition (Chalupa, Morrow, Oberdorster, Utell, & Frampton, 2004; Daigle et al., 2003). Although other factors in Table 5 were not explicitly modeled, these reference studies used physically active subjects and traffic exhaust particles. Intake doses of UFP were 4.2–6.6 times higher for bicyclists than car passengers, while uptake doses were 5.1–8.3 times higher — despite lower or roughly equivalent exposure concentrations for bicyclists. $PM_{2.5}$ comparisons were similar, with intake doses 5.7–7.6 times higher for bicyclists than car passengers, but uptake doses 8.0–12.0 times higher.

McNabola et al. (2008) modeled uptake of VOC and $PM_{2.5}$ using the International Commission on Radiological Protection (ICRP) human respiratory tract model (ICRP Task Group, 1994) with on-road measured exposure concentrations and laboratory-measured respiration characteristics for bicycle, pedestrian, car, and bus modes. The ICRP model can include all relevant factors in Table 5 except lung perfusion, though the assumed fraction of oral breathing is not reported by McNabola et al. (2008). Bicyclists had the highest total lung deposition of $PM_{2.5}$ and the second highest absorption of VOC over similar trips to other modes. Breathing characteristics (frequency, tidal volume) and VOC solubility affected the uptake dose and the location of absorption, with more benzene absorbed deep in the lungs for bicyclists and pedestrians. Breathing differences also affected benzene absorption more than 1,3-butadiene absorption because of

benzene's lower solubility. McNabola et al. (2007) similarly model VOC uptake by bicyclists using different travel speeds, but with assumed (rather than measured) exposure concentrations. They found that higher bicycling speeds reduce VOC absorption over a fixed travel distance because the increase in respiration rate is smaller than the reduction in exposure duration.

The same ICRP model was also applied by Nyhan et al. (2014) to estimate $PM_{2.5}$ and $PM_{10}$ lung deposition for trips by bicycle, foot, bus, and train. Their estimates indicate that bicyclists' PM intake and uptake per trip is disproportionately higher than exposure concentrations compared to other modes. But the cross-mode ratios are equivalent for modeled intake and deposition, suggesting that only ventilation rate $V_E$ was varied by mode in the uptake model.

Bicyclists' uptake of traffic-related VOC was directly measured by sampling blood and urine concentrations of BTEX compounds (benzene, toluene, ethylbenzene, and xylenes) by Bergamaschi et al. (1999). They found significant increases of benzene and toluene in blood for bicyclists in urban areas, and significant increases of toluene and xylenes in urine. Although uptake was directly measured, respiration was not measured, and there was no discussion of pollutant intake or inhalation, which inhibits placement of their findings in the larger context of the emissions-health pathway (Figure 1). Nwokoro et al. (2012) directly measured uptake doses of BC by bicyclists and non-bicyclists (pedestrians and public transit riders) in London by sampling airway macrophages. They found significantly higher (63%) doses of BC for bicyclists, correlated with higher commute exposure concentrations. Bicyclists also had almost twice as long commute durations, and experienced 41% of daily BC exposure during the commute (when compared with 19% for non-bicyclists).

The few studies of bicyclists' pollution uptake suggest that PM uptake doses are disproportionally greater for bicyclists than intake doses or exposure concentrations when compared with other modes. Bicyclists' uptake doses of gaseous pollutants are also disproportionately higher than exposure concentrations when compared with other modes, but have yet to be directly compared with intake doses. Uptake dose is the closest measure of health risks for exposed travelers, but connections to health outcomes still require application of a dose–response function that reflects the toxicity of the pollutants, the susceptibility of the travelers, and other factors (Cho et al., 2009; ICRP Task Group, 1994).

## 6.   Health Effects of Bicyclists' Air Pollution Uptake

Linkages between long-term exposure to traffic-related air pollution and health impacts have been established, as described elsewhere (Bell, 2012; Brook et al., 2010; Brugge, Durant, & Rioux, 2007; Health Effects Institute, 2010; Nawrot et al., 2011; Pope & Dockery, 2006; Samet, 2007). Long-term health effects studies show elevated risk for development of asthma, reduced lung function, increased blood pressure, and cardiac and pulmonary mortality. An important gap for traveler health studies, though, is a lack of data on the health effects of chronic high-intensity but short-duration doses (Zuurbier, Hoek, Oldenwening, Meliefste, Krop, et al., 2011). Some evidence exists of effects on mortality and cardiovascular/pulmonary hospital admissions for short-term exposure to traffic-related air pollution in general, and particularly PM and UFP (Knibbs et al., 2011; McCreanor et al., 2007; Michaels & Kleinman, 2000; Peters et al.,

2004). A recent study indicates increased risk of acute myocardial infarction onset after travel specifically for bicyclists — though the risk is not higher than for other modes (Peters et al., 2013).

Health effects studies of bicyclists' exposure to air pollution have focused on respiratory and cardiovascular biomarkers following acute (0.5–2 h) exposures to traffic (11 studies of Types K–M in Table 3). Biomarkers are physiological indicators in the pathway of the morbidity and mortality outcomes studied in epidemiology; for example, blood cell counts can be indicators of systemic inflammation, and systemic inflammation is linked to cardiovascular disease (Brook et al., 2010). Unfortunately, even when acute health effects are recognized in the form of biomarkers, the broader health significance is often not known — especially in the context of chronic daily exposures.

Studies of bicyclists' biomarkers show inconsistent results, with 4 of 11 reporting insignificant acute effects and others reporting s*ome* cardiovascular or respiratory biomarker changes. No significant changes in bicyclists' respiratory or cardiovascular biomarkers were reported in four studies of acute on-road exposure (Jarjour et al., 2013; Waldman et al., 1977; Zuurbier, Hoek, Oldenwening, Meliefste, van den Hazel, et al., 2011; Zuurbier, Hoek, Oldenwening, Meliefste, Krop, et al., 2011). Jacobs et al. (2010) found a significant but small increase in a single indicator of blood inflammation for bicyclists, with 'unclear' health implications. Cole-Hunter et al. (2013) found significant differences in nasal and throat irritation between bicyclists in high-exposure and low-exposure routes, but no significant differences for airway inflammation biomarkers. Strak et al. (2010) found mostly insignificant changes in respiratory function biomarkers for bicyclists, though UFP and soot exposure were weakly associated with a biomarker of airway inflammation (exhaled NO) and degraded lung function. Weichenthal et al. (2011) found significant associations between UFP, ozone ($O_3$), and $NO_2$ exposures during travel and cardiovascular risk indicators (changes in heart rate variability), but no strong associations between in-traffic exposure and respiratory biomarkers. Further analysis of individual VOC in the data set found 'evidence of possible associations . . . for a small number of compounds' with biomarkers of lung inflammation, lung function, and heart rate variability (Weichenthal et al., 2012). Nyhan et al. (2014) found significant associations between decreased heart rate variability and $PM_{2.5}$ and $PM_{10}$ doses — stronger for bicyclists and pedestrians than other modes. Bos et al. (2011) took a different approach and found that PM exposure during bicycling can suppress a *positive* exercise-induced health biomarker associated with cognitive performance. Though again, the effects of chronic exposure are still unknown.

This review does not address the health impacts of bicycling-related crashes and physical activity, only air pollution uptake. However, a review of five recent health impact assessments for bicycling concludes that the physical activity benefits of bicycling far outweigh the crash safety and air pollution risks — by factors of 9–96 (Teschke, Reynolds, Ries, Gouge, & Winters, 2012). The air pollution risks in these assessments are based on extrapolations of epidemiological evidence for long-term health outcomes, and limited by the continued uncertainty of health effects of chronic daily uptake of air pollution by physically active travelers.

## 7.   Summary

This is the first review to specifically address bicyclists' health risks from traffic-related air pollution and to explicitly include intake and uptake doses in addition to exposure concentrations. Bicyclists' pollution *exposure concentrations* are highly variable, with median increases of up to 102% (for gaseous hydrocarbons) on high-traffic versus low-traffic routes. Bicyclists' relative exposure concentrations compared to other modes are inconsistent, varying by pollutant, facility, route, and city. Bicyclists' exposure concentrations are most affected by wind and proximity to motor vehicle traffic, though few studies have incorporated detailed, concurrent traffic data.

Bicyclists' pollution *intake doses* tend to be higher than motorized modes due to their two to five times higher respiration rates. Bicyclists' respiration and intake dose increase with bicycle travel speed and exertion, but only 12 of the 57 studies with spatially explicit bicyclist exposure concentration data include any measurement of respiration. Furthermore, only three of those studies consider variable bicyclist respiration rates, and there has been almost no assessment of the variability in respiration with trip characteristics (including correlation with exposure concentrations).

Bicyclists' pollution *uptake doses* are affected by the intake dose, pollutant characteristics, breathing depth and pathway, and other individual and physiological factors. Uptake rates tend to increase with exertion level, affecting bicyclists more than motorized travelers. There are clear links between traffic-related air pollution exposure and negative *health* outcomes in urban populations. However, the health effects of chronic daily air pollution uptake by bicyclists are still unknown. More research is needed on health impacts of pollution exposure because some studies of bicyclists' biomarkers show significant acute respiratory effects while other studies show insignificant effects.

To reduce exposure concentrations, spatial and temporal separation of bicyclists from motor vehicle traffic can be achieved with separated bicycle facilities, low-volume routes, and off-peak travel. These are potential 'win–win' strategies because bicyclists already prefer low-traffic routes and bicycle-specific facilities (Broach et al., 2012; Dill, 2009; Kang & Fricker, 2013; Wardman, Tight, & Page, 2007) and separated bicycle facilities could also improve safety (Lusk et al., 2011; Reynolds, Harris, Teschke, Cripton, & Winters, 2009; Teschke, Harris, et al., 2012). Regarding intake doses, other likely mitigation strategies would be to prioritize separation from traffic in locations where bicyclists' respiration is expected to be high (steep grades, for example) or to reduce energy expenditure requirements (by reducing required stops, for example) in locations where pollutant concentrations are known to be high.

## 8.   Research Gaps and Opportunities

This literature review reveals steady progress toward a better understanding of air pollution uptake by bicyclists. However, several significant research gaps deserve attention. Although the literature suggests that traffic-related air pollution uptake is higher for bicyclists than for travelers using motorized modes, persistent uncertainty in the intensity and effects of pollution uptake means that transportation planners and decision-makers are unable to consider bicyclists' air pollution risks in a precise way. More research is needed to provide better quantification

and understanding of the relative health benefits of alternative bicycle facility designs, bicycle network designs, and route options. Some research topics that can bring us closer to achieving these goals include

- study of the on-road variability of respiration and air quality for traveling bicyclists, including a broader array of pollutants (e.g. ground-level ozone);
- the impact of bicycle trip attributes such as road grade, road surface, travel speed, and number of stops on respiration rates for bicyclists;
- the impacts of bicycle facility design features on exposure concentrations (distance from motor vehicle travel lanes, physical barriers, intersection treatments such as 'bike boxes', etc.);
- the impacts of traffic flow characteristics on bicyclists' exposure concentrations, including traffic speeds, volumes, and queuing along arterials or at major intersections;
- inter-modal pollution exposure comparisons that apply more comprehensive and representative modal travel characteristics (trip location and distance, traveler demographics, route preferences) and that consider variable respiration (especially for active travelers);
- characterization of different bicyclist types (e.g. commuters and recreational riders) and demographic factors that can impact respiration or health effects; these factors include physiology (height, weight, and respiratory health), riding style (speed, acceleration, and response to grades), and equipment (weight, condition, and baggage);
- analysis of bicyclists' pollutant doses along different types of routes and facilities, to enable health impact assessments; and
- development of dose−response functions for health effects of chronic short-duration high-intensity air pollution exposure episodes.

## Acknowledgements

## Notes

1. Traffic data are reported in a companion paper, Kaur and Nieuwenhuijsen (2009).
2. Traffic data are only used in a companion paper, McNabola et al. (2009b).
3. It should be noted that metabolic energy expenditure during bicycling is the sum of energy expenditure for baseline functions and the rate of external work (Olds, 2001). Assuming that the baseline energy expenditure is roughly equal to the RMR, the MET can be expressed as a function of external power output $p$ as $MET = (1 + p/RMR)$. Thus, MET values increase linearly (but not proportionally) with the *external* power demands of bicycling.

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