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How do indoor environments affect air pollution exposure?

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1 Air pollution in urban areas is associated with adverse human health outcomes, including
2 cardiopulmonary disease and premature death. The relationships, determined largely through
3 epidemiological studies, drive regulatory policies for setting air pollution standards that are
4 designed to protect human health. Breathing predominantly occurs indoors and especially at
5 home. Therefore, indoor environments mediate the link between outdoor air pollution and human
6 exposure. In this perspective, we explore current understanding about the relationship between
7 air pollution, as characterized in monitoring networks, and inhalation exposures, which occur
8 largely indoors. Recognizing substantial and important gaps in knowledge, we suggest research
9 directions for advancing understanding at the nexus of outdoor air pollution, indoor
10 environments and exposure. We conclude with key points to consider when evaluating the
11 influence of indoor environments on air pollution exposure.

12
13 To illustrate the scale of concern, consider the most recent Global Burden of Disease study.
14 “Ambient particulate matter” (referring to PM_{2.5}) ranked as the environmental factor with the
15 highest adverse health consequences, with 2.9 million attributable premature deaths and 83
16 million disability-adjusted life years (DALYs) lost per year globally in 2017.¹ “Ambient ozone
17 pollution” contributed an additional 0.5 million premature deaths and 7.4 million DALYs per
18 year.

19
20 Buildings offer partial protection against pollutants of outdoor origin but enhance exposures to
21 particles and gases that are emitted or produced indoors. A survey of time apportionment for the
22 United States indicated that 87% of time on average is spent indoors (mostly in one’s own
23 residence), more than 10× higher than the proportion of time spent outdoors.² Consequently,

24 most “ambient particulate matter” and “ambient ozone pollution” is breathed indoors, albeit at
25 concentration levels below outdoors. Furthermore, epidemiological studies of the link between
26 outdoor air pollution and health generally do not effectively address the often-substantial
27 exposure to air pollutants of indoor origin (e.g., emissions from cooking, cleaning, furnishings or
28 the inhabitants themselves). Indoor organic compounds are of particular note, since their
29 concentrations tend to be much larger than those outdoors and include species with known
30 adverse health effects (e.g., carcinogens, teratogens, endocrine disruptors), as well as persistent
31 organic pollutants and ozone reactive compounds.

32

33 How do buildings modulate exposures to outdoor particulate matter, outdoor ozone, and
34 hazardous organic air pollutants, and what additional exposures are attributable to indoor
35 sources? In other words, how different is the exposure that happens because we mainly breathe
36 indoor air, not outdoor air? We contribute toward answering these broad questions by posing
37 and commenting on three more focused questions.

38

39 **Question 1. What chemical and physical transformations occur indoors that alter the form**
40 **and composition of outdoor air pollution?**

41 *Airborne particles* can span a diameter range covering about five orders of magnitude (0.001-100
42 μm). As outdoor particles penetrate building envelopes and deposit on indoor surfaces, the
43 smallest (ultrafine) and largest (coarse) particles are removed to a greater extent than particles in
44 the intermediate size range (fine).³⁻⁶ Hence, as illustrated in Figure 1, indoor particles of outdoor
45 origin have a narrower distribution, centered about the most penetrating and persistent airborne
46 particles. When inhaled indoors, a larger proportion of these fine particles can travel deep into

47 the respiratory tract. Being exposed to particles of outdoor origin while indoors shifts the pattern
48 of deposition in the respiratory tract owing to the effect of size-selective filtering of buildings.

49

50 The chemical composition of particles also changes with transport from outdoors to indoors
51 (Figure 1). Consider the partitioning of ammonia and nitric acid between the gas phase and
52 particle-phase ammonium nitrate.⁸ Transport to indoors is accompanied by several factors that
53 can each shift the gas-particle balance: change in temperature, loss of HNO₃ to indoor surfaces,
54 and increased NH₃ from indoor sources.⁹ Altering the abundance of NH₄NO₃ in particles can
55 shift particle pH. Also, given that outdoor and indoor relative humidity routinely differ,¹⁰ it is
56 anticipated that the water content of outdoor airborne particles would change when transported
57 indoors, which can also modify particle pH and size distribution. A change in pH can
58 substantially influence the partitioning of acidic and basic gas-phase species to particulate
59 water.^{11,12}

60

61 Another chemical transformation that occurs with outdoor-to-indoor transport is the shift in the
62 abundance and composition of semivolatile organic compounds (SVOCs) sorbed to airborne
63 particles.¹³⁻¹⁸ Certain SVOCs that are prominent in outdoor air, such as polycyclic aromatic
64 hydrocarbons (PAHs), can desorb from outdoor particles transported indoors.¹⁹ Other SVOCs,
65 which are prominent indoors, can sorb to particles that are transported into buildings. Common
66 examples of abundant indoor SVOCs are phthalate ester plasticizers, brominated flame
67 retardants, and perfluorinated surfactants.²⁰ Under conditions with minimal indoor particle
68 emissions, particles of outdoor origin have been shown to quickly sorb indoor SVOCs,¹⁶
69 resulting in larger mass fractions of particulate SVOCs indoors than outdoors.¹⁷

70

71 In summary, several features of indoor environments result in physical and chemical
72 transformations such that indoor particles of outdoor origin differ meaningfully in size
73 distribution and chemical composition from their outdoor precursors. Such changes might well
74 influence the health consequences associated with inhaling them.

75

76 *Ozone* reacts with surfaces it encounters as it is transported indoors and, subsequently, with
77 compounds on exposed indoor surfaces. Ozone also reacts, to a lesser extent, in indoor air. The
78 aggregate effect is that indoor ozone concentrations are commonly lower than those found
79 outdoors. The reactions that consume ozone generate both gaseous and condensed-phase
80 products. The indoor concentration of gas-phase reaction products can be larger than the indoor
81 concentration of ozone itself.²¹⁻²³ Indoors, humans inhale both ozone of outdoor origin and
82 indoor-generated oxidation products derived from outdoor ozone. Given the large fraction of
83 time spent indoors, the amount of ozone inhaled indoors is approximately equal to ozone inhaled
84 outdoors, and inhalation of products derived from outdoor ozone is comparable to or larger than
85 the total inhalation of ozone itself.²¹⁻²³ Notably, there is a strong link between indoor moisture
86 and the products of indoor ozone chemistry. As the indoor water vapor concentration increases,
87 ozone reactions with skin oil constituents have been shown to yield fewer secondary ozonides,
88 more carbonyls and an overall increase in gas-phase products.^{24,25} Many studies address ozone's
89 toxicity. However, the toxicity of many of its oxidation products is poorly characterized. This is
90 especially true for stabilized Criegee intermediates, secondary ozonides, epoxides, and
91 hydroperoxides. When considering ozone pollution and its health consequences, most studies

92 overlook the coincident indoor exposure to ozone-derived products and their potential
93 contribution to the mortality and morbidity attributed to outdoor ozone.

94

95 *Organic air pollutants* that are almost exclusively gaseous outdoors partition between the gas
96 phase and indoor surfaces,²⁶ reflecting surface-to-volume ratios that are markedly larger indoors
97 than outdoors.²⁷ Sorption serves to reduce indoor exposure to outdoor organic pollutants during
98 the peak of an outdoor episodic emission but extends the overall time of exposure to pollutants.
99 The indoor mixture of organic air pollutants results from a combination of indoor and outdoor
100 sources depending on ventilation and infiltration conditions. Importantly, there are many strong
101 indoor sources of organic pollutants (e.g., buildings and their contents, cooking, cleaning,
102 occupant metabolic emissions, personal care products, skin oil oxidation, consumer products,
103 indoor combustion), resulting in indoor concentrations that are commonly an order of magnitude
104 larger and chemically distinct from those outdoors (e.g., compounds emitted from vegetation,
105 motor vehicles, solid fuel combustion, industry or fires). Coupled with the large fraction of time
106 people spend indoors, inhalation of organic pollutants can be orders of magnitude larger indoors
107 than outdoors and is generally dominated by indoor sources.

108

109 **Question 2: How do building and human factors influence the nature and extent of**
110 **modulation of air pollution exposures?**

111 The *infiltration factor* quantifies the extent to which buildings are protective against airborne
112 particles of outdoor origin. This term represents the indoor-to-outdoor particle concentration
113 ratio when all indoor particles originate outdoors. The infiltration factor is controlled by loss
114 processes that occur passively (deposition to surfaces) or by design (filtration in mechanical

115 ventilation systems). A central-tendency estimate for the infiltration factor of PM_{2.5} is 0.5 and
116 studies report average values ranging from 0.3 to 0.8.²⁸ Buildings are much more effective at
117 limiting the outdoor-to-indoor transport of ultrafine and coarse-mode particles.²⁹ Overall, in
118 central tendency, buildings provide substantial but imperfect protection from outdoor particles.
119 Furthermore, there is considerable variability in infiltration factors across the building stock.

120

121 Buildings are also protective against outdoor ozone exposure, largely because ozone reacts
122 chemically with indoor surface materials. A compilation of empirical results studying the
123 indoor/outdoor ratio for ozone in buildings without known indoor sources suggests a central
124 tendency of about 0.25 with variation commonly spanning the range 0.1-0.6.^{23,30}

125

126 Important consequences ensue from the partial and variable protection provided by buildings
127 against outdoor air pollution. In epidemiological studies, actual inhalation intake of outdoor air
128 pollutants such as fine particles and ozone is lower than monitoring networks would indicate.
129 Furthermore, exposures are variable across populations owing to differences in the factors that
130 influence the degree of protection provided by buildings. For example, because of climate-
131 associated variation in building design and operation, systematic spatial differences occur in the
132 indoor-outdoor relationship of outdoor pollutants. Accounting for variability from location to
133 location in air change rates, augmented by the fraction of time spent indoors, has partially
134 explained observed regional differences in associated health effects for ozone,²² PM_{2.5},³¹ and
135 PM₁₀.³² Furthermore, there can be systematic variation in infiltration factors in the buildings
136 occupied by subpopulations, which may correlate with factors such as age, socioeconomic
137 conditions, and health status.

138

139 Prominent epidemiologic and risk assessment studies commonly overlook the roles of buildings
140 attenuating and modulating inhalation exposures of occupants. For example, Brauer et al.³³ did
141 not account for attenuation in exposures associated with buildings in their extensive effort to
142 estimate population exposures to outdoor PM_{2.5} and ozone for the Global Burden of Disease
143 2013 study. Burnett et al.³⁴ constructed an integrated exposure-response model for PM_{2.5},
144 synthesizing exposure and disease outcome information for “ambient air pollution, secondhand
145 tobacco smoke, household solid cooking fuel, and active smoking.” That effort neglected
146 building-associated attenuation of outdoor air pollution exposure, therefore biasing the outcome.
147 An assessment of how global mortality might shift with changes in outdoor PM_{2.5} concentrations
148 likewise neglects the role of buildings as attenuators and modulators of exposure.³⁵ Efforts to
149 incorporate the effects of buildings in health risk assessments for outdoor air pollution have been
150 reported,^{36,37} but these issues have not yet taken root to influence major epidemiological studies.

151

152 The uneven ability to characterize exposures well may influence the outcomes of air-pollution
153 epidemiology studies. For particles, the connection between PM_{2.5} and health effects understood
154 through epidemiological studies is superior to the knowledge for health effects of ultrafine and
155 coarse-mode particles. Part of the reason is that smoother spatial gradients occur outdoors for
156 PM_{2.5}, which is largely secondary with a longer atmospheric persistence time than coarse-mode
157 or ultrafine particles. Consequently, monitoring networks provide a more accurate representation
158 of the spatial and temporal distribution of outdoor PM_{2.5} than would be possible for other particle
159 size modes. Given that buildings generally provide a high degree of protection against ultrafine
160 and coarse particles, combined with significant indoor sources of these modes, we are currently

161 in a state of near blindness about actual inhalation exposure to ultrafine and coarse particles. The
162 same level of poor understanding applies to many of the pollutants for which indoor emissions
163 are the dominant contributors to exposure, such as most organic pollutants.

164

165 **Question 3: Which air pollutants have indoor sources leading to generally higher**
166 **concentrations and exposures indoors than outdoors, and which of these may be**
167 **particularly important for health?**

168 The mix of indoor, outdoor, primary and secondary chemicals is modulated through buildings
169 and the ways in which we construct and operate them. Since people spend ~70% of their time at
170 home, we focus here on residences. Every home may have a unique combination of smells and
171 activities, but in fact they have much in common that is generalizable. At their most basic,
172 houses are boxes, constructed out of a relatively small range of materials, that exchange mass
173 and energy with their surroundings. Buildings are operated to meet the requirements for thermal
174 comfort and respiratory metabolism of their occupants. With global supply chains, there is more
175 commonality in building materials and operations than might have been the case historically.
176 Standardized building materials and furniture are now available worldwide from rapidly growing
177 international vendors. Ventilation, filtration, and thermal comfort provisions are achieved
178 through a few core technologies. Thus, whereas the nexus between outdoor environments, indoor
179 air quality, and indoor exposure is richly complex, there are a core set of reasonably well-defined
180 scientific principles and engineering tools with which to make generalizable progress.

181

182 To elaborate, notwithstanding the wide variety of buildings, sources, human occupancy, and
183 activities, there is surprising commonality in indoor composition of organic chemicals in

184 residences.³⁸⁻⁴¹ Ample sources of VOCs and SVOCs cause their abundance in indoor air to be
185 greatly enhanced above what is present in outdoor air for a broad suite of chemical classes.
186 Typical sources include plastics and polymers (emitting plasticizers, flame retardants,
187 antioxidants), pressed wood products (formaldehyde, organic acids), synthetic carpets (flame
188 retardants, anti-stain agents, antioxidants), cleaning products, fragrances (terpenoids readily
189 oxidized to undesirable products), appliances (flame retardants, particles, thermal greases, oils),
190 other building materials, and common consumer products.⁴²⁻⁴⁸ Remarkably, it is now becoming
191 recognized that the ventilation of these chemicals from indoor environments can make
192 significant contributions to outdoor air pollutant loadings in cities.^{49,50}

193
194 Human beings are themselves potent mobile sources of volatile organic compounds (VOCs),
195 inorganic compounds and particles in indoor environments.⁵⁰⁻⁵³ We shed skin flakes and
196 microbes and we transfer skin oils to indoor surfaces. Several hundred bioeffluent VOCs are
197 known to be emitted via breath and skin.⁵⁴ Emissions of VOCs from personal care products are
198 ubiquitous. In the presence of oxidants such as ozone, a multitude of chemical byproducts can be
199 formed,^{24,55} some of which (e.g., 4-OPA) are known irritants.⁵⁶ As well as innately emitting into
200 their immediate environment, various human activities indoors, such as cooking, cleaning,
201 smoking, have been shown to introduce large quantities of gases and particles to indoor air.

202 Indoor sources are uncommon (although not rare) for ozone and include devices that produce a
203 corona discharge (e.g., electrostatic precipitators, photocopiers, and “ozone generators”).⁵⁷ For
204 particles, indoor sources are common and contribute substantially to exposures both acute and
205 chronic, with differing impacts on immune system responses.⁵⁸⁻⁶¹ Among the prominent
206 important indoor sources of particles are smoking⁶² and cooking.⁶³ Candle use has been

207 identified as an important source of indoor ultrafine particles.⁶⁴ Resuspension⁶⁵ and shedding⁶⁶
208 contribute to airborne coarse-mode particles indoors. While PM_{2.5} abundance inside residences is
209 often lower than outdoors, during specific types of events (e.g., smoking, cooking, candle use),
210 and via resuspension, much higher concentrations can occur indoors than outdoors.

211 Secondary sources of air pollutants can also be important indoors. Specifically, oxidation
212 products from outdoor ozone infiltrating and reacting with alkenes on exposed indoor surfaces,⁶⁷
213 in skin oil, and in the gas-phase are ubiquitous. Reactions with terpenes and terpene alcohols
214 used as scenting agents in a variety of indoor cleaners and “air fresheners” are common.⁴⁸
215 Additional reactions in the gas phase and on surfaces can occur sporadically because of occupant
216 activities.^{68,69} Noteworthy examples include addition of oxidants such as hypochlorite in bleach
217 and related products (chlorine chemistry), changing surface pH via cleaning with ammonia or
218 vinegar, and changing surface moisture through intentional humidification. Oxidants such as OH
219 and NO₃ are assumed to be at generally low concentrations indoors making them typically less
220 important as sources of secondary organics than is the case outdoors during daytime.^{70,71}

221 Kirk Smith aptly advised, that to advance scientific understanding and improve the human
222 condition, we need to “follow the risk”.⁷² Research is needed to determine which of the
223 numerous organic chemicals or chemical classes found indoors may be particularly important as
224 health risks. Among known candidates, PM_{2.5}, acrolein, and formaldehyde have been identified
225 as accounting for the vast majority of DALY losses caused by indoor air pollutants, with impacts
226 equal to or larger than those estimated for radon and secondhand tobacco smoke.⁷³ In current
227 morbidity and mortality studies, PM_{2.5} is assumed to be equitoxic, despite known dependencies
228 on chemical composition.⁷⁴ How indoor emissions and processes affect PM_{2.5} toxicity is a key

229 question for future research. Indoor sources of formaldehyde, as well as the health impacts of
230 this smallest aldehyde, have been extensively studied.⁷⁵ Carcinogenic chemicals (e.g., benzene,
231 benzo[a]pyrene (BaP), radon, nitrosamines) are another category of concern for indoor air
232 exposure.⁷⁶ Although PAHs may be elevated in outdoor particles, indoor combustion is also a
233 source, including of the carcinogenic benzo(a)pyrene. Nitrous acid has been shown to react with
234 nicotine and other third-hand smoke components to generate carcinogenic nitrosamines.⁷⁷

235 When chemicals enter the human body, some interact with our regulatory system disrupting the
236 autoregulatory processes that rely on chemical signaling.^{78,79} More than 100,000 new chemicals
237 have been introduced as constituents of commercial products in recent decades, including many
238 used deliberately or inadvertently in indoor environments.^{80,81} An emerging body of evidence
239 suggests some widely used commercial chemicals are endocrine disruptors.^{82,83} Some chemicals
240 of concern as endocrine disruptors have substantially higher concentrations indoors compared to
241 outdoors. Phthalates are a prime example.¹⁷ Human exposures to semivolatile organic
242 compounds are dominated by indoor environments both through inhalation and dermal
243 uptake.^{84,85} Diseases believed to be influenced by environmental exposures to endocrine
244 disrupting chemicals have increased over time, including asthma, allergy, Alzheimer's disease,
245 psychogenic processes, eating disorders, chronic obesity, and possibly autism.⁸⁶⁻⁸⁸ While indoor
246 exposure to many SVOCs that are suspected endocrine disruptors has increased, the clinical
247 relevance of these indoor exposures and the molecular basis for increased adverse health risks
248 remain subjects for future multidisciplinary investigations.

249 **Challenges and opportunities at the nexus of outdoor air pollution, indoor environments**
250 **and exposure.**

251 Human exposure to harmful air pollutants results from a combination of outdoor and indoor
252 sources. These are difficult to delineate since exposure occurs mostly indoors and especially
253 within the home. Although outdoor measurements of ozone and PM_{2.5} are used to link air
254 pollution to health effects, they are proxies for more complex chemical and physical processes
255 that act at the intersection of outdoor air pollution, indoor environments and exposure.

256 Understanding these processes should be a research focus to ensure future wellbeing.

257

258 Global trends show that the world's population is migrating from the countryside to cities. By
259 2050, some 70% of humanity will live in urban areas.^{89,90} This shift is occurring despite the per
260 capita mortality rate attributable to air pollution being approximately 50% higher in urban than in
261 rural areas.⁹¹ The resulting increased demand for urban housing and potential for rising levels of
262 outdoor pollutants including ozone⁹² will likely lead to smaller living spaces and tighter
263 structures in which indoor sources and chemical processing increase in importance. Lower
264 indoor-outdoor air-change rates are also anticipated as housing becomes more energy efficient
265 and heat exchange restricted. With continued warming of urban atmospheres,⁹³ air conditioning
266 will become more prevalent. In some of the world's hottest regions, such as the Eastern
267 Mediterranean and Middle East, home to over 400 million people, most already reside in urban
268 areas where life without active cooling is barely tenable.⁹⁴ Therefore, it seems likely that future
269 homes will tend to be increasingly isolated from the outside. Living and working conditions
270 described by temperature, humidity, and chemical exposure will be more directly influenced by
271 buildings and inhabitants. While effectively designed and operated mechanical ventilation

272 systems coupled with air-cleaning technologies can attenuate their impact, building occupants
273 will remain susceptible to exposures that originate from pollutants emitted indoors.

274

275 Improvements in outdoor and indoor air quality can improve human health. Landrigan et al.⁹⁵
276 estimated that in 2015, polluted air was responsible for 6.4 million deaths and economic losses of
277 US\$21 billion worldwide. Lowering urban and regional air pollution requires the concerted
278 action of a population that possesses considerable societal inertia. The continued use of the
279 outside atmosphere as a repository for pollutant emissions is therefore a lamentable but also
280 likely future condition as a modern-day tragedy of the commons.⁹⁶ A further complication for air
281 pollution control strategies is that the sources of outdoor PM_{2.5}, which is our clearest link to
282 premature mortality, vary considerably with location. In Asia, where most premature deaths
283 attributed to air pollution occur, residential energy use for heating and cooking is a substantial
284 source of emissions, while in Europe, parts of the USA, Russia and East Asia agriculture plays
285 an important role, and in large sections of the USA traffic and power generation are dominant
286 sources.⁹¹

287

288 People have more direct control over their exposure indoors than outdoors, and this potential for
289 control is especially germane in parts of the world that have serious outdoor air pollution issues,
290 such as India and China. Opportunities to intervene exist, modifying building systems to
291 deliberately reduce air pollution exposure and thereby improve occupant health. Activated
292 carbon filters have been demonstrated to be effective in controlling ozone.⁹⁷ High-quality
293 particle filters can substantially reduce indoor particle levels⁹⁸ and can be cost effective.⁹⁹⁻¹⁰¹
294 Intervention studies have documented improved outcomes for biomarkers of health through

295 building-level air-pollution mitigation,^{102,103} although overall health improvements have been
296 judged to be relatively modest compared to the substantial reduction in indoor PM using
297 filtration.¹⁰⁴ Nonetheless, building-level interventions can be applied with local control on a
298 much faster time scale than interventions for improving outdoor air pollution.

299
300 As isolation of the home environment from outside air is common and likely increasing,
301 architectural innovations have potential to play an important role in mediating chemical exposure
302 between our lungs and the environment. Cleansing facilities and practices (e.g., doormats, shoe
303 brushes, shoe removal) at the home entrance to limit importation of particles and chemicals into
304 the home, efficient range hoods, transitioning from gas to electric stoves, use of lower emitting
305 materials, frequent cleaning of clothing as protection from indoor exposure, and increased air
306 cleaning via filtration of recirculated air in whole-house HVAC systems or portable units in
307 selected rooms¹⁰⁴ are all possible adaptations. Such protective strategies can be adopted by
308 individuals, even while community-wide emission reductions of outdoor pollutants are pursued.

309

310 **Synthesis**

311 We highlight key points to consider when evaluating the influence of indoor environments on air
312 pollution exposure:

- 313 • Most of our inhalation of outdoor PM occurs indoors, and when indoors we are exposed
314 preferentially to outdoor fine PM, the size range that is most efficient at penetrating deeply
315 into our lungs.
- 316 • Buildings most effectively attenuate the ingress of outdoor ultrafine- and coarse-mode
317 particles. Indoor sources can make large contributions to inhalation exposures of the smallest

318 and largest particles. Epidemiological studies that rely on outdoor air concentrations have
319 elicited consistent associations (but not demonstrated causation) between PM_{2.5} and adverse
320 health outcomes. Studies that rely on outdoor concentrations as proxy measures of exposure
321 are likely to be less successful in assessing health consequences of exposure to ultrafine and
322 coarse mode particles.

- 323 • Inhalation of outdoor ozone occurs almost equally indoors and outdoors. Indoor inhalation of
324 the products of indoor reactive chemistry initiated by ozone originating outdoors can be
325 larger than the aggregate inhalation of ozone itself.
- 326 • The concentration of gas-phase organic compounds is commonly an order of magnitude
327 larger indoors than outdoors. In part, this reflects contributions from scented agents, cyclic
328 and linear siloxanes, plasticizers, flame retardants, perfluorinated surfactants, pesticides and
329 cooking emissions. Given the large fraction of time spent indoors, cumulative inhalation of
330 gas-phase organics occurs at approximately two orders of magnitude higher daily rate from
331 breathing indoor air rather than outdoor air.
- 332 • Outdoor PM_{2.5} transported indoors sorbs semivolatile organic compounds released from
333 indoor sources. When inhaled, these particles carry the sorbed organics deeper into the
334 respiratory tract than would be the case for the gas-phase counterparts.

335

336 Implications from our perspective include these three points:

- 337 • Science might be missing some of the important health consequences associated with air
338 pollution by failing to properly understand how buildings modulate air pollution exposures.
- 339 • The apparently equitoxic nature of PM_{2.5} might reflect inadequate understanding of inhaled
340 particle composition, in contrast to particle composition in outdoor air. Future studies that

341 aim to elucidate the most health-harmful components of inhaled PM_{2.5} should take account of
342 indoor modulation and modification of PM_{2.5} constituents.

343 • Indoor environments materially alter the link between outdoor air pollution and human
344 exposure. Core elements of understanding were identified several decades ago,¹⁰⁵ but the
345 lessons have not been widely learned. In failing to account for how indoor environments
346 affect air pollution exposure, epidemiologists are overlooking critical elements needed to
347 make the important transition from association to causation.

348

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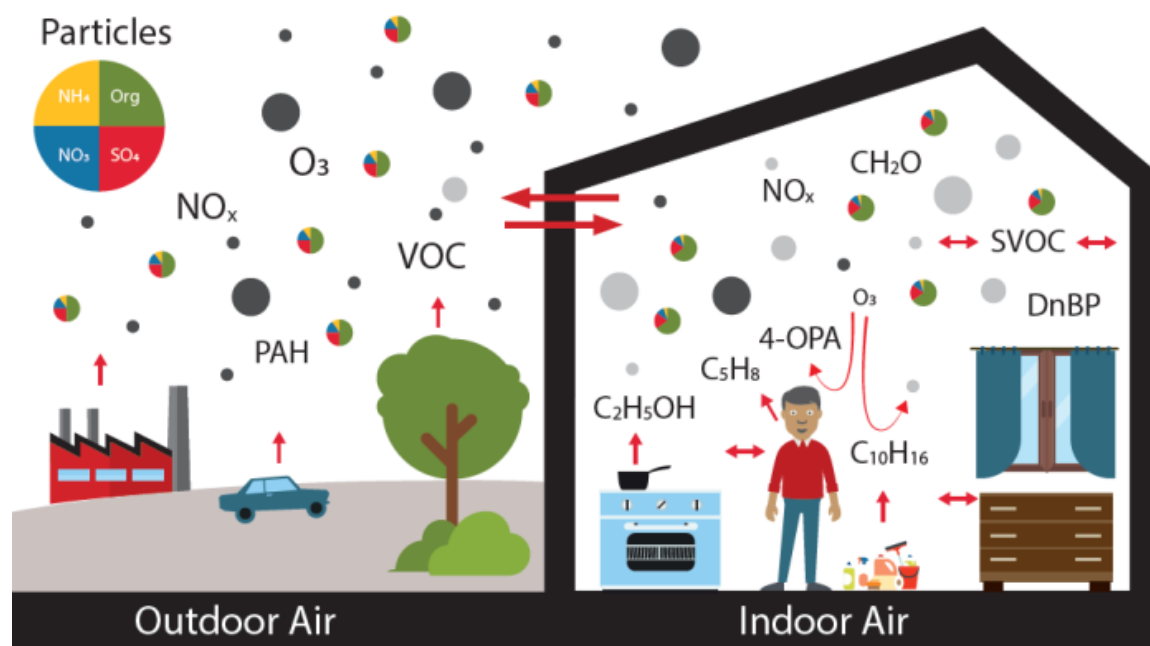
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679 **Figure 1.** Schematic illustration of selected air pollutant transformations that occur between
680 outdoor and indoor air. Pie charts indicate fine-mode particle composition (legend top left,
681 following color scheme of Zhang et al.⁷). Note outdoor versus indoor differences in particle size
682 distributions, particle composition, ozone, and sources. Solid black circles represent ultrafine-
683 and coarse-mode particles of outdoor origin; buildings attenuate their outdoor-to-indoor
684 transport. Solid light gray circles represent particles of indoor origin, some of which are
685 transported outdoors. Ozone tends to have a much larger concentration outdoors than indoors
686 (suggested by font size), reflecting reactions on indoor surfaces and in air that generate gaseous
687 and condensed-phase products. Multiple indoor sources of organic compounds result in indoor
688 concentrations much larger than those outdoors. Indoor organics partition among the air, airborne
689 particles, room and human surfaces.

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