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The Effect of Air Pollution on the Incidence of Asthma in School-Aged Children

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Abstract: Asthma is the most common chronic disease of childhood, and a growing body of epidemiological evidence indicates that outdoor air pollution contributes not only to the worsening of established disease but also to the development of new cases in children who were previously free of asthma. This article reviews the published literature and publicly available data on the effect of air pollution on the incidence of asthma in school-aged children, with particular attention to fine particulate matter, nitrogen dioxide, and ozone, and with a focus on the Indonesian urban context. Evidence was synthesised from peer reviewed cohort studies, meta analyses, burden of disease assessments, the World Health Organization global air quality guidelines of 2021, and Indonesian national health survey data. Meta analyses report modest but consistent increases in asthma incidence per unit increase in nitrogen dioxide and fine particulate matter, and global assessments attribute between roughly two and four million new paediatric asthma cases each year to nitrogen dioxide alone. The biological pathway runs through oxidative stress, airway inflammation, and interference with normal lung development during the years when the lungs are still growing. Indonesian cities record annual fine particulate concentrations many times higher than the World Health Organization guideline value, yet local incidence studies remain scarce, ecological in design, and constrained by limited monitoring. The review concludes that reducing traffic related pollution around homes and schools is a defensible prevention strategy, and that Indonesia needs prospective cohort research, denser exposure monitoring, and school level mitigation to convert global evidence into local protection.

Keywords: air pollution, asthma incidence, school-aged children, nitrogen dioxide, PM2.5, Indonesia

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INTRODUCTION

Asthma occupies a distinctive place in child health because it is at once very common, strongly shaped by the environment, and largely preventable in its avoidable fraction. The Global Asthma Network Phase I study, which standardised symptom measurement across countries, confirmed that asthma remains the most common chronic disease in children and reported that current asthma symptoms affected 9.1 percent of children aged 6 to 7 years and 11.0 percent of adolescents aged 13 to 14 years worldwide, with wide variation between countries and evidence that prevalence is still rising in several low and middle income regions [1]. The Global Burden of Disease study likewise places asthma among the leading causes of disability adjusted life years lost in childhood and adolescence, a burden carried disproportionately by settings where diagnosis and long term control are weakest [2]. Indonesia illustrates this pattern. The national basic health research survey,

Riskesdas 2018, recorded a doctor diagnosed asthma prevalence of 2.4 percent across all ages and 1.9 percent among children aged 5 to 14 years, figures that almost certainly understate the true burden because they depend on prior contact with diagnostic services [3]. For a school-aged child, asthma is not a minor inconvenience. It interrupts sleep, restricts play and sport, drives repeated visits to clinics and emergency departments, and is one of the most frequent medical causes of school absence, with consequences for learning that accumulate quietly over the years of schooling.

The environments in which children grow up have changed faster than the epidemiology that describes them. The World Health Organization has estimated that 93 percent of the world's children under the age of 15 years, some 1.8 billion children, breathe air in which pollutant concentrations exceed its guideline values, and that polluted air contributed to approximately 600,000 deaths of children under 15 years in 2016 through acute lower respiratory infections alone [4]. The guideline values themselves were tightened substantially in the 2021 update of the WHO global air quality guidelines, which set an annual mean of 5 µg/m³ for fine particulate matter (PM2.5) and 10 µg/m³ for nitrogen dioxide (NO2), reflecting evidence that harm occurs at concentrations once considered acceptable [5]. Measured against those values, Indonesian urban air is heavily polluted. The IQAir World Air Quality Report for 2024 placed Indonesia's population weighted annual PM2.5 concentration at 35.5 µg/m³, the highest in Southeast Asia, and recorded an annual mean of 41.7 µg/m³ for Jakarta, roughly eight times the WHO annual guideline value [6]. Children in such cities spend their school years breathing concentrations that the international evidence associates with measurable respiratory harm.

That air pollution worsens asthma in a child who already has the disease has been accepted for decades. The more consequential question, and the one this article addresses, is whether air pollution causes asthma to develop in children who did not previously have it. Brunekreef and Holgate argued at the turn of the century that the evidence linking traffic related pollution to respiratory ill health in children had become difficult to dismiss, while noting that demonstrating new onset disease is methodologically harder than demonstrating symptom aggravation [7]. The distinction matters for policy. Exacerbation research supports clinical management and short term alerts, whereas incidence research supports prevention, because every incident case averted removes a lifetime of disease rather than a single episode of it. Early European health impact assessment already treated traffic as a first order target: Künzli and colleagues estimated that air pollution accounted for 6 percent of total mortality in Austria, France, and Switzerland combined, that about half of the pollution related mortality was attributable to motorised traffic, and that traffic related pollution also drove more than half a million asthma attacks per year in the three countries [8]. Since that assessment, birth cohorts, school based studies, and successive meta analyses have progressively strengthened the incidence side of the evidence, and the question has shifted from whether pollution contributes to new asthma to how large that contribution is and where it can most effectively be reduced.

This article therefore reviews the effect of air pollution on the incidence of asthma in school-aged children, drawing exclusively on published literature and public data. It examines the pollutants of concern and their sources, the biological mechanisms that connect inhaled pollutants to a developing airway, and the methodological distinction between incidence and exacerbation. It then synthesises the quantitative evidence from cohorts and meta analyses, the windows of childhood during which vulnerability is

greatest, the exposure assessment problem posed by schools and traffic, and the estimates of how many new cases globally are attributable to pollution. A dedicated section considers the Indonesian urban evidence base and its gaps, and the article closes with the policy and school level measures that the evidence supports.

The focus on school-aged children is deliberate. Between the ages of roughly five and fourteen years, children are old enough for asthma to be diagnosed with reasonable reliability, since spirometry and standardised symptom questionnaires perform better than they do in toddlers, in whom wheeze is common and often transient. At the same time they are young enough that their lungs are still growing, their daily environment is structured by the school, and their exposure is substantially determined by decisions made by adults: where the family lives, where the school is built, and how the journey between them is made. Incidence measured in this age band therefore captures disease that begins during a modifiable window, in a setting, the school, that public policy can actually reach. Studies in younger and older groups are drawn on where they illuminate mechanism or timing, but the organising question throughout is what pollution does to the school-aged child and what schools and cities can do in response.

METHODS

This study is a structured literature review supplemented by public data, following the approach appropriate to a question for which the relevant primary evidence is dispersed across epidemiology, exposure science, toxicology, and official statistics. No new measurements were taken, no cohort was recruited, and no unpublished data were used. Every quantitative estimate reported in the article is attributed to the published study or official source from which it was taken, and modelled estimates are identified as modelled wherever they appear.

The literature base was assembled from peer reviewed sources indexed in international databases, prioritising material published by major international publishers and by established journals in respiratory medicine, environmental health, and exposure science. Priority was given to systematic reviews and meta analyses for quantitative effect estimates, to prospective cohort and birth cohort studies for questions of temporality, to the assessments of the Health Effects Institute and comparable expert panels for causal judgement, and to foundational reviews for mechanism. Burden figures were drawn from the Global Burden of Disease programme, whose comparative risk assessment framework quantifies the share of disease attributable to defined risk factors, including ambient particulate pollution [9], and from global attribution studies that apply concentration response functions to modelled exposure surfaces. Public data sources comprised the World Health Organization global air quality guidelines of 2021 and its report on air pollution and child health, the Indonesian Riskesdas 2018 national health survey published by the Ministry of Health, and the IQAir World Air Quality Report 2024, which aggregates government and validated low cost sensor monitoring. Inclusion required that a source report either an effect estimate for asthma incidence or exacerbation in children, a measured or modelled exposure concentration, a national prevalence figure from an official survey, or a guideline value from a recognised authority. Sources were excluded where the population was exclusively adult, where the outcome was a non respiratory endpoint without an asthma component, or where the report could not be traced to a verifiable publisher or official body. The synthesis is narrative and comparative rather than a new meta analysis, because exposure metrics, outcome definitions, and adjustment

sets differ across the underlying studies in ways that make repooling their estimates inappropriate.

RESULT AND DISCUSSION

1. Pollutants of Concern and Their Sources

The literature converges on a small group of pollutants as the principal agents of respiratory harm in children. Fine particulate matter, PM2.5, consists of particles with an aerodynamic diameter of 2.5 micrometres or smaller, a size fraction that penetrates deep into the lung and reaches the alveolar region. Nitrogen dioxide is a gaseous marker of combustion and, in cities, primarily of road traffic, although it also arises from gas cooking and other combustion sources. Ozone is a secondary pollutant formed in sunlight from nitrogen oxides and volatile organic compounds, so its peaks follow hot, bright afternoons rather than the morning traffic peak. Coarser particles (PM10), sulfur dioxide, carbon monoxide, and black carbon, the light absorbing component of soot, complete the set most often examined. Kampa and Castanas summarised the toxicological profile of these agents, noting that particulate matter and nitrogen dioxide act both as irritants and as carriers of reactive surface chemistry into the airways [10]. The Global Burden of Disease analysis by Cohen and colleagues estimated that ambient PM2.5 was responsible for 4.2 million deaths and 103.1 million disability adjusted life years worldwide in 2015, ranking it among the leading environmental risks to health [11]. The Lancet Commission on Pollution and Health placed such findings in a wider frame, concluding that pollution is the largest environmental cause of disease and premature death in the world and that its burden falls most heavily on low and middle income countries, where monitoring and control are weakest [12]. For children, the traffic related mixture deserves particular emphasis, because the school journey and the school day place children repeatedly in the near road environment where nitrogen dioxide, black carbon, and ultrafine particles reach their highest concentrations.

2. Biological Mechanisms: Oxidative Stress, Inflammation, and Airway Development

The plausibility of a causal effect on asthma incidence rests on mechanisms that are now well described. Guarnieri and Balmes, in their Lancet review of outdoor air pollution and asthma, set out how inhaled oxidant pollutants injure the airway epithelium, provoke inflammation, and heighten bronchial responsiveness, and how these processes can, with repeated exposure during lung growth, contribute to the remodelling that characterises established asthma [13]. Schraufnagel and colleagues, writing for the American Thoracic Society and the European Respiratory Society, traced the part one pathway from particle deposition through oxidative stress, in which reactive oxygen species overwhelm the antioxidant defences of the airway lining fluid, to epithelial barrier disruption, innate immune activation, and systemic inflammation [14]. The companion second part extended the account to clinical outcomes, emphasising that the child's lung is not a miniature adult lung: alveolarisation continues through childhood, airway calibre is smaller, breathing rates per unit of body mass are higher, and children spend more time active outdoors, so the delivered dose per unit of lung surface is greater than in adults exposed to the same air [15]. Pope and Dockery, in their critical review of the fine particle literature, showed how evidence from toxicology, controlled human exposure, and epidemiology forms connected lines that converge on cardiopulmonary harm, a coherence argument that supports reading the asthma incidence findings as more than

statistical coincidence [16]. Two mechanistic points carry particular weight for incidence. First, oxidative stress and allergic inflammation interact, so that pollution can act as an adjuvant that promotes sensitisation to inhaled allergens rather than merely irritating an already sensitised airway. Second, exposure during periods of rapid lung growth appears to shift the trajectory of lung development itself, which provides a biological route by which an exposure in childhood can produce a disease that persists. Mechanisms do not by themselves prove causation in populations, but they remove the objection that the epidemiological associations lack a credible pathway. A further mechanistic consideration concerns the composition of the mixture rather than its mass alone. Fresh traffic emissions are rich in ultrafine particles and black carbon, whose large surface area relative to mass carries adsorbed organic compounds and metals deep into the lung, and exposure science consistently finds that these traffic specific components track health outcomes more closely than total particle mass measured at a distance from roads. This is one reason nitrogen dioxide, which is itself an oxidant gas but also a faithful marker of the fresh traffic mixture, performs so consistently in the incidence literature reviewed below: the gas may be acting partly as an agent and partly as a tracer of the mixture that accompanies it. Genetic susceptibility modifies the response, since children whose antioxidant defence genes are less effective appear more vulnerable to oxidant exposures, which helps explain why population average effects conceal larger effects in susceptible subgroups. The practical consequence is that average risk estimates should not be read as the risk faced by every child, and that protection of the most exposed and most susceptible children yields benefits larger than the averages suggest.

3. Incidence Versus Exacerbation: Two Distinct Outcomes

Much public discussion of air pollution and asthma concerns exacerbation, the short term worsening of symptoms, emergency visits, and hospital admissions in children who already have asthma, typically demonstrated in time series and case crossover studies of daily pollution variation. That evidence is strong. Zheng and colleagues, systematically reviewing and meta analysing the time series literature on asthma emergency department visits and hospital admissions, found that short term increases in pollutants were consistently associated with exacerbation, with pooled relative risks per 10 μg/m³ increase of 1.008 for ozone, 1.014 for 24 hour nitrogen dioxide, and 1.010 for sulfur dioxide [17]. A third body of work concerns prevalence rather than onset or aggravation: Favarato and colleagues meta analysed within community studies and quantified the association between nitrogen dioxide, used as a marker of traffic related pollution, and the 12 month prevalence of asthma symptoms in children [18]. Prevalence designs cannot separate children whose disease was caused by exposure from children whose disease merely persists under exposure, so this evidence corroborates the burden without settling causation. Incidence is a different and more demanding outcome. It requires following children who are free of asthma at baseline and recording new diagnoses or new onset symptoms over years, which is why the incidence evidence rests on cohorts rather than on daily time series. The distinction is not pedantic. A pollutant could aggravate symptoms without causing disease, and a factor that causes disease in a small fraction of exposed children might be invisible in exacerbation studies. Conversely, an exposure that increases incidence has the larger lifetime consequence, because incident asthma in childhood frequently persists or relapses in adult life. This review therefore treats exacerbation evidence as supporting context, as evidence of biological activity in the

airway, but rests its conclusions about disease onset on the cohort and meta analytic literature addressed next.

4. Evidence on Asthma Incidence from Cohorts and Meta Analyses

The most comprehensive synthesis of the incidence literature remains the meta analysis by Khreis and colleagues, who combined 41 studies of traffic related air pollution and childhood asthma development and reported a risk ratio of 1.05 (95% CI 1.02 to 1.07) per 4 μg/m³ increase in nitrogen dioxide, 1.03 (95% CI 1.01 to 1.05) per 1 μg/m³ increase in PM2.5, and 1.08 per 0.5×10^{-5} m^{-1} increase in black carbon [19]. Individual exposure response estimates of this size appear small, but they apply to exposures that are essentially universal in cities, so their population impact is substantial. The systematic review and meta analysis of cohort studies by Anderson, Favarato, and Atkinson reached a compatible conclusion for nitrogen dioxide, reporting an incidence odds ratio of 1.07 (95% CI 1.02 to 1.13) per 10 μg/m³, while the corresponding estimate for PM10 was weaker and crossed the null at 1.06 (95% CI 0.97 to 1.17) [20]. Birth cohort evidence points in the same direction with finer exposure timing. Bowatte and colleagues meta analysed birth cohorts and found that PM2.5 exposure in early childhood was associated with later asthma, with an odds ratio of 1.14 (95% CI 1.00 to 1.30) per 2 μg/m³ increase, and an odds ratio of 1.20 per unit increase in black carbon [21]. The European ESCAPE project brought standardised exposure modelling to this question: Gehring and colleagues pooled four birth cohorts comprising 14,126 children and reported an odds ratio for incident asthma of 1.13 (95% CI 1.02 to 1.25) per 10 μg/m³ increase in nitrogen dioxide estimated at the birth address [22]. Not all syntheses are positive, and the exceptions are informative: Fuertes and colleagues meta analysed five European birth cohorts and found no increase in the prevalence of asthma at ages four and eight with increasing exposure, reporting an adjusted odds ratio of 0.91 (95% CI 0.74 to 1.11) per 10 μg/m³ increase in nitrogen dioxide at the birth address [23]. That analysis measured current prevalence rather than diagnosed incidence, in comparatively low pollution settings, which may explain its divergence from the incidence focused literature without overturning it. Individual regional cohorts add texture. In the Southern California Children's Health Study, Jerrett and colleagues found that within community variation in nitrogen dioxide carried a hazard ratio of 1.29 (95% CI 1.07 to 1.56) per 6.2 parts per billion for new onset asthma, an estimate larger than between community contrasts and a reminder that the finest exposure contrasts often carry the clearest signal [24]. In Toronto, the T-CHEQ study followed 1,286 children and reported a hazard ratio of 1.17 (95% CI 1.05 to 1.31) for asthma in relation to total oxidant exposure at the birth address [25]. Read together, these studies satisfy the considerations that matter most for causal reading: the exposure precedes the outcome, the association repeats across countries and designs, it strengthens with the traffic specificity of the exposure metric, and it aligns with the mechanistic account given above. The appropriate conclusion is not that pollution is the sole or dominant cause of childhood asthma, which is a multifactorial disease with strong genetic and allergenic components, but that it is a genuine contributory cause whose removal would prevent a meaningful number of new cases.

5. Windows of Vulnerability: Prenatal and Early Life Exposure

The timing of exposure appears to matter as much as its average level. The systematic review by Zhang and colleagues of prenatal air pollution exposure, covering 18 studies, found that exposure during pregnancy was associated with asthma in the offspring,

reporting an odds ratio of 1.07 for nitrogen dioxide and 1.08 (95% CI 1.05 to 1.12) for PM10, with estimates varying by trimester and by the child's age at assessment [26]. Such findings are biologically coherent: the fetal lung develops in stages that begin in utero, maternal inhalation exposes the placenta and the fetal circulation to inflammatory mediators and fine particles, and epigenetic and immune programming during this period can bias the child's later response to allergens and infections. The early school years constitute a second window, because it is during childhood and early adolescence that lung function grows towards its adult maximum. Gauderman and colleagues provided the most persuasive evidence that this growth is environmentally plastic: in the Southern California Children's Health Study, improvements in air quality over two decades were accompanied by measurably improved lung function growth in successive cohorts of children aged 11 to 15 years, and the proportion of children with clinically low lung function fell as nitrogen dioxide and particle concentrations declined [27]. Evidence that reducing pollution improves the growth trajectory of the lung strengthens the inference that pollution worsens it, and it converts the incidence question from an abstract risk estimate into an observable developmental effect. For school aged children specifically, the implication is that exposure reduction during the primary and early secondary school years is not too late to matter: the lungs those children carry into adult life are still being built while they sit in classrooms.

6. The Global Attributable Burden of New Childhood Asthma

Translating exposure response functions into case numbers clarifies the scale of prevention available. Achakulwisut, Brauer, and Anenberg estimated in The Lancet Planetary Health that nitrogen dioxide alone was responsible for approximately 4.0 million (95% uncertainty interval 1.8 to 5.2 million) new paediatric asthma cases per year globally, equivalent to 13 percent of global paediatric asthma incidence, with 64 percent of the attributable cases occurring in urban centres [28]. The attributable fraction varied sharply by setting, from a few percent in agricultural areas to more than 40 percent in the most polluted megacities, which is precisely the gradient that a traffic driven cause would predict. Anenberg and colleagues subsequently updated the assessment with improved exposure surfaces and estimated 1.85 million (95% uncertainty interval 0.93 to 2.80 million) new paediatric asthma cases attributable to nitrogen dioxide in 2019, again with about two thirds in urban areas, and showed that the urban attributable fraction fell from 19.8 percent in 2000 to 16.0 percent in 2019 as concentrations declined in many high income cities, while rising by 23 percent in South Asia and 11 percent in sub Saharan Africa [29]. These two estimates are not contradictory: they differ in exposure data, baseline years, and concentration response choices, and both place the preventable global burden in the millions of cases per year. Two cautions apply. The estimates are modelled, resting on concentration response functions derived largely from North American and European cohorts, and their transfer to Southeast Asian exposure mixtures is an assumption rather than a measurement. And because the underlying functions are driven mainly by nitrogen dioxide as a marker of the traffic mixture, the figures are best read as the burden of the traffic related mixture for which nitrogen dioxide stands proxy. Even with those cautions, the direction of travel is clear: where traffic pollution has fallen, the attributable share of new childhood asthma has fallen with it, and where it is rising, as in much of Asia, the share is rising too.

7. Exposure Assessment Near Schools and Traffic

The credibility of the incidence literature depends on how well exposure is measured, and the methodological literature is instructive. Hoek and colleagues reviewed land use regression modelling, in which monitoring at a limited number of sites is combined with geographic predictors such as road length, traffic intensity, and land use to estimate concentrations at children's homes and schools, and showed that such models reproduce spatial contrasts in nitrogen dioxide and particles well enough to support epidemiology at the within city scale [30]. This matters because pollution varies over tens of metres near busy roads, so city average concentrations from a handful of regulatory monitors can badly misclassify the exposure of a child whose school fronts a congested arterial road. The BREATHE project measured this directly in 39 Barcelona schools and found that traffic was the main source of school air pollution, that traffic related particles penetrated indoors readily, and that concentrations of PM2.5 at schools averaged about 1.7 times, and nitrogen dioxide about 1.2 times, the urban background [31]. The Health Effects Institute's expert panel on long term exposure to traffic related air pollution, reviewing the accumulated evidence, concluded that the epidemiological literature supports a causal relationship between long term traffic related pollution exposure and asthma onset in children, while noting continuing uncertainty about which components of the mixture drive the effect [32]. For Indonesian research and policy, the lesson is concrete. Exposure classification anchored to a single city monitor will understate contrasts between schools, whereas the feasible combination of targeted monitoring at schools, land use regression using road network data, and low cost sensors calibrated against reference instruments can resolve the near road gradients on which both risk and mitigation depend. Time activity patterns sharpen the point further. A school-aged child typically spends six to eight hours of each weekday at school, much of the remainder at home, and a concentrated period in transit, often at the morning and afternoon traffic peaks when concentrations are highest and when travel on foot or by motorcycle places the child's breathing zone close to exhaust sources. Exposure assigned only to the home address, as in many cohorts, therefore misses a systematic share of the child's dose, and exposure assigned to the school address captures a location where intervention is administratively feasible. Studies that combine home, school, and route exposure remain rare everywhere, and nonexistent in Indonesia, which is a further reason the local research agenda set out below gives exposure science a central place.

8. Evidence from Indonesia and Southeast Asian Cities

Indonesian evidence on pollution and childhood asthma is real but thin, and describing it accurately requires separating what has been measured from what has merely been assumed. The exposure side is well documented: the IQAir 2024 figures cited in the introduction, population weighted annual PM2.5 of 35.5 µg/m³ nationally and 41.7 µg/m³ in Jakarta, sit alongside official monitoring that consistently records exceedances of national standards in the capital region during the dry season [6]. The outcome side is documented mainly through survey prevalence, such as the Riskesdas 2018 figures, and through ecological analyses rather than through cohorts. Haryanto and colleagues examined children's respiratory health and air quality across Greater Jakarta for 2020 to 2022 and reported a mean PM2.5 concentration of 42.5 µg/m³ for the region, with 15,825 reported asthma cases and 73,694 pneumonia cases among children across 11 cities and districts over the period; their correlations between pollutant levels and asthma cases were weak to moderate and largely not statistically significant, a finding the authors and this review both read as reflecting the limits of aggregate ecological data rather than as

evidence of no effect [33]. At school level, Krismanuel and Tjhin compared 107 schoolchildren aged 6 to 12 years at elementary schools in Jakarta and Bandung and found that children in the high PM2.5 setting had markedly higher odds of respiratory tract infection, with an adjusted odds ratio of 7.88 (95% CI 3.23 to 19.25), although their outcome was respiratory tract infection rather than asthma incidence, the design was cross sectional, and the estimate cannot bear causal weight on onset [34]. Regional evidence from comparable Asian settings is stronger: in the China Children Homes Health study of 39,782 children aged 3 to 6 years across seven Chinese cities, Norbäck and colleagues found that doctor diagnosed asthma was associated with city level nitrogen dioxide, with an odds ratio of 1.16 (95% CI 1.02 to 1.33) per 10 μg/m³, and with living near major roads, extending the exposure response pattern observed in Western cohorts to East Asian urban mixtures, albeit in pre school children and with city level exposure assignment [35]. The gap is therefore specific and remediable. Indonesia has exposure levels at or above those of the settings in which incidence effects have been demonstrated, a school population of tens of millions, and national survey infrastructure, but it lacks a prospective cohort with individual exposure assignment, repeated lung function measurement, and standardised asthma ascertainment. Until such a study exists, Indonesian policy must rest on the transfer of international exposure response evidence to Indonesian exposure conditions, which is defensible for prevention but unsatisfying for local accountability, since it cannot show whether Indonesian interventions are working.

9. Policy and School Level Mitigation

The evidence supports a layered response in which emission control does the largest work and school level measures reduce the dose that remains. At the level of the city, the modelling study by Garcia and colleagues for Southern California showed that progressively stronger controls on nitrogen oxide emissions would prevent substantial numbers of nitrogen dioxide attributable new asthma cases in children, with the largest gains from the most aggressive scenarios, which demonstrates that the attributable burden responds to policy within planning horizons rather than only across generations [36]. Guideline anchored standard setting is the instrument that converts such findings into obligations: the WHO 2021 values give Indonesian regulators an evidence based target, and the distance between Jakarta's annual PM2.5 of 41.7 μg/m³ and the guideline value of 5 μg/m³ is a measure of the prevention opportunity, not merely of non compliance [5], [6]. At the level of the school and its surroundings, several measures follow directly from the exposure science. New schools should be sited away from high traffic corridors, and where relocation is impossible, classroom ventilation should draw air from the side of the building away from the road, with filtration considered for the most exposed schools, since the BREATHE findings show that outdoor traffic particles otherwise enter classrooms efficiently [31]. Traffic management around schools, including anti idling enforcement at drop off and pick up, staggered timing, and safe walking and cycling routes set back from carriageways, reduces both the concentration and the time children spend in the highest concentration microenvironment. Vegetation is a partial and context dependent measure: in a New York City analysis, Lovasi and colleagues found that street tree density was associated with lower asthma prevalence among young children, with a prevalence rate ratio of 0.71 per standard deviation increase in tree density, although that ecological design cannot establish that planting trees caused the difference, and poorly chosen planting in confined street canyons can impede dispersion [37]. Finally, schools need air quality communication systems that translate monitoring data into action,

adjusting outdoor activity on high pollution days, in the same way that heat and severe weather already modify the school day. None of these measures substitutes for emission reduction, but together they align the child's daily environment with what the incidence evidence says that environment should be.

CONCLUSION

The published evidence supports the conclusion that outdoor air pollution, and the traffic related mixture in particular, contributes causally to the development of new asthma in school aged children, through oxidative stress, airway inflammation, and disturbance of lung growth during the years when the lungs are still developing, with meta analytic estimates of increased incidence per unit of nitrogen dioxide and fine particulate matter that are modest for an individual child yet large in aggregate because exposure is universal, amounting to millions of attributable new cases globally each year. For Indonesia, where urban PM2.5 concentrations run at roughly seven to eight times the World Health Organization guideline value while local evidence remains ecological and sparse, the defensible course is to act on the international evidence now through emission control, traffic management around schools, considered school siting and ventilation, and air quality communication, while building the prospective cohort and school exposure monitoring capacity that would allow the country to measure its own incidence burden and verify that its interventions prevent disease rather than merely promise to.

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