World Health Organization Global Air Quality Guideline Recommendations: Executive Summary

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Introduction

As the most significant environmental threat to human health, air pollution exposure may account for as many as 1 in 5 deaths worldwide.^{1,2} The burden of disease disproportionately affects residents of low- and middle-income countries where air quality continues to worsen in contrast to high-income countries.¹ The World Health Organization (WHO) last issued health-based guidelines for major air pollutants in 2006: *Air quality guidelines – global update 2005. Particulate matter, ozone, nitrogen dioxide and sulfur dioxide*.³ Since then, methods of assessing air pollution exposure have improved.¹ Studies on the relationship between air pollution and health have been conducted in nearly all WHO regions, whereas most prior studies were of North American and European origin.¹ New evidence has linked air pollution with asthma, diabetes, reproductive outcomes, various neurocognitive endpoints, and other health conditions with which associations

were not previously suspected.^{1,4,5} In light of these developments, the WHO has released a global update to its air quality guidelines.

The purpose of the guideline is to provide key stakeholders and decision-makers with 1) evidence-based air quality guideline (AQG) levels, or the lowest levels of exposure that result in harms to health, for particulate matter 2.5 (PM_{2.5}), particulate matter 10 (PM₁₀), nitrogen dioxide, ozone, sulfur dioxide, and carbon monoxide in both indoor and outdoor environments; 2) interim target levels to guide the reduction of these pollutants in countries that significantly exceed AQG levels; and 3) good practice statements to guide mitigation efforts for specific types of PM that pose a health risk but lack sufficient evidence to derive AQGs.

Methods

Guideline development was initiated and overseen by a WHO steering group. AQG recommendations and other guidance were informed by systematic reviews and determined by the consensus decisions of a guideline development group (GDG) comprised of subject matter experts in the area of air quality and health. Evidence evaluation was guided by the Grading of Recommendations Assessment, Development and Evaluation approach.⁶ AQG levels were defined on the basis of moderate or high certainty evidence supporting a link between a pollutant and a particular health outcome over a long-term (annual mean or highest six-month average) or short-term (24-hour) period. Thus, long-term AQGs took precedence over short-term AQGs when both were considered. Additional methodological details can be found in the complete guideline text.

The AQG and interim target recommendations are summarized in **Table 1**. Previously established WHO AQGs that are not addressed in this update remain valid. Good practice statements for the management of certain types of health risk-associated PM – black carbon/elemental carbon (BC/EC), ultrafine particles (UFP), and sand and dust storms (SDS) – are listed in **Table 2**. Further discussion of the rationale and complete citations for the recommendations can be found in the guideline.

Guideline Recommendations

Classical Pollutants: PM_{2.5}, PM₁₀, O₃, NO₂, SO₂, CO

$1.1 \ PM_{2.5}$

 $PM_{2.5}$ are heterogeneous mixtures of fine inhalable particles defined by an aerodynamic diameter of less than or equal to $2.5\mu m.^{1,3}$ AQG and interim target levels were set based on the pollutant-attributed increase in all-cause non-accidental mortality and cause-specific mortality with high certainty of evidence.

- $AX\Gamma \lambda \epsilon \epsilon \lambda \ (\mu \gamma / \mu^3)$:
- Annual exposure: 5
- 24-hour exposure: 15
- $I\nu\tau\epsilon\rho\mu$ $Ta\rho\gamma\epsilon\tau\varsigma$ $(\mu\gamma/\mu^3)$:
- Annual exposure: 35, 25, 15, 10
- 24-hour exposure: 75, 50, 37.5, 25
- *Health outcomes* : all-cause mortality, cardiovascular mortality, respiratory mortality, lung cancer mortality
- Causality : all associations between $PM_{2.5}$ and health outcomes were determined to be causal or likely causal

$1.2~\mathrm{PM}_{10}$

 PM_{10} are defined by an aerodynamic diameter of less than or equal to $10\mu m.^{1,3}$ AQG and interim target levels were set based on the pollutant-attributed increase in all-cause non-accidental mortality or cause-specific mortality with high certainty of evidence. In situations where both $PM_{2.5}$ and PM_{10} measurements are available, priority should be given to $PM_{2.5}$ AQGs.

• $AX\Gamma \lambda \epsilon \epsilon \lambda \ (\mu \gamma / \mu^3)$:

- Annual exposure: 15
- 24-hour exposure: 45
- $I\nu\tau\epsilon\rho\mu$ $Ta\rho\gamma\epsilon\tau\varsigma$ $(\mu\gamma/\mu^3)$:
- Annual exposure: 70, 50, 30, 20
- 24-hour exposure: 150, 100, 75, 50
- *Health outcomes* : all-cause mortality, cardiovascular mortality, respiratory mortality, lung cancer mortality
- Causality: all associations between PM_{10} and health outcomes were determined to be causal or likely causal

$1.3 O_3$

Ozone is an oxidant species formed predominantly from nitrogen dioxide and non-methane volatile organic compounds in the atmosphere.^{1,3} Peak season AQG and interim target levels were set based on the pollutantattributed increase in all-cause non-accidental mortality, with moderate certainty evidence, and respiratory mortality, with low certainty evidence. 24-hour levels were set based on all-cause non-accidental mortality, with high certainty evidence.

- $AX\Gamma \lambda \epsilon \epsilon \lambda \ (\mu \gamma / \mu^3)$:
- Peak season exposure: 60
- 24-hour exposure: 100
- $I\nu\tau\epsilon\rho\mu$ $Tap\gamma\epsilon\tau\varsigma$ $(\mu\gamma/\mu^3)$:
- Peak season exposure: 100, 70
- 24-hour exposure: 160, 120
- *Health outcomes* : all-cause mortality, respiratory mortality, asthma-related hospital admissions and emergency room visits
- Causality:
- Peak season exposure : likely causal for respiratory effects; suggestive of causality for all-cause mortality
- 24-hour exposure : suggestive of causality for all-cause mortality; causal for respiratory effects

$1.4 \ NO_2$

 NO_2 is a trace atmospheric gas and strong oxidant that is both a threat to human health and implicated in climate change.^{1,3} Annual AQG and interim target levels were set based on the pollutant-attributed increase in all-cause non-accidental mortality, with moderate certainty evidence, and cause-specific respiratory mortality, with moderate to high certainty evidence. In addition, 24-hour levels were based on all cause nonaccidental mortality and asthma-related hospital admissions and emergency room visits, with high certainty evidence.

- $AX\Gamma \lambda \epsilon \epsilon \lambda \ (\mu \gamma / \mu^3)$:
- Annual exposure: 10
- 24-hour exposure: 25
- $I\nu\tau\epsilon\rho\mu$ $Ta\rho\gamma\epsilon\tau\varsigma$ $(\mu\gamma/\mu^3)$:
- Annual exposure: 40, 30, 20
- 24-hour exposure: 120, 50
- *Health outcomes* : all-cause mortality, respiratory mortality, asthma-related hospital admissions and emergency room visits
- Causality:
- Annual exposure : suggestive of causality for total mortality, likely causal for respiratory effects
- 24-hour exposure : suggestive of causality for total mortality, causal for respiratory effects

$1.5 \, \mathrm{SO}_2$

Sulfur dioxide is derived from fossil fuel combustion and is a major component of air pollution globally.^{1,3} 24-hour AQG and interim target levels were set based on pollutant-attributed increase in asthma-related

hospital admissions and emergency room visits with low certainty evidence, all non-accidental mortality with high certainty evidence, and respiratory mortality with moderate certainty evidence.

- 24-hour AQG level ($\mu g/m^3$): 40
- 24-ηουρ Ιντεριμ Ταργετς $(\mu\gamma/\mu^3)$: 125, 50
- *Health outcomes* : all-cause mortality, asthma-related hospital admissions and emergency room visits, respiratory mortality
- *Causality* : causal for respiratory effects, suggestive of causality for mortality

1.6 CO

Carbon monoxide is a product of incomplete combustion of carbon-rich fuels.^{1,3} 24-hour AQG and interim target levels were set based on pollutant-attributed increase in hospital admissions and mortality from myocardial infarction with moderate certainty evidence.

- 24-hour AQG level ($\mu g/m^3$): 4
- 24-ηουρ Ιντεριμ Ταργετς $(\mu\gamma/\mu^3)$: 7
- Health outcomes : hospital admissions and emergency room visits related to ischemic heart disease
- Causality : likely causal for cardiovascular effects

2. Other Pollutants: BC/EC, UFP, and SDS

BC/EC, a measure of airborne soot-like carbon, is formed from the incomplete combustion of fossil fuels, biofuel, and biomass. Both short-term and long-term exposures have been linked to adverse cardiovascular health effects and premature death.^{1,7}UFPs, defined as particulates with a diameter less than or equal to 0.1μ m, are largely generated from combustion. Short-term and long-term exposures have been associated with mortality and various morbidities.^{1,8} SDS exposure, which affects desert regions and countries affected by desert dust, may lead to an increased risk for inflammatory and allergic lung diseases such as asthma and cardiovascular mortality.^{1,9}

The GDG determined that evidence was insufficient to establish quantitative AQG and interim target levels. Instead, the following good practice statements were formulated to aid the management of the health and environmental concerns associated with these types of PM.

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Pollutant	Averaging Time	Interim Targets	Interim Targets	Interim Targets	Interim Targets	AQG	Eviden
$PM_{2.5}, \mu g/m^3$	Annual	35	25	15	10	5	High
	24-hour ^a	75	50	37.5	25	15	High
$PM_{10}, \mu g/m^3$	Annual	70	50	30	20	15	High
	24-hour ^a	150	100	75	50	45	High
O_3 , $\mu g/m^3$	Peak season ^b	100	70	-	-	60	Low-m
	8-hour ^a	160	120	-	-	100	High
NO_2 , $\mu g/m^3$	Annual	40	30	20	-	10	Modera
	24-hour ^a	120	50	-	-	25	High
${ m SO}_2,\mu g/m^3$	24-hour ^a	125	50	-	-	40	Low-hi
$CO, mg/m^3$	24-hour ^a	7	-	-	-	4	Modera

Table 1: AQG levels and interim targets for six key pollutants

 $^{\rm a}$ 99th percentile of the distribution of daily values (3-4 exceedance days per year)

^b Average of daily maximum 8-hour mean O_3 concentration in a period of six consecutive months with the highest six-month running-average O_3 concentration

Table 2: Good practice statements for BC/EC, UFP, and SDS

Pollutant	Good Practice Statements
BC/EC	Make systematic measurements of black carbon and/or elemental carbon. Such measurements should not replace or reduce the existing monitoring of pollutants for which guidelines currently exist. Undertake the production of emission inventories, exposure assessments and source apportionment for BC/EC. Take measures to reduce BC/EC emissions from within the relevant jurisdiction and, where considered appropriate, develop standards (or targets) for ambient BC/EC concentrations.

Pollutant	Good Practice Statements
UFP	Quantify ambient UFP in terms of particle number concentration (PNC) for a size range with a lower limit of [?] 10 nm and no restriction on the upper limit. Expand the common air quality monitoring strategy by integrating UFP monitoring into the existing air quality monitoring. Include size-segregated real-time PNC measurements at selected air monitoring stations in addition to and simultaneously with other airborne pollutants and characteristics of PM. Distinguish between low and high PNC to guide decisions on the priorities of UFP source emission control. Low PNC can be considered 10 000 particles/cm3 (24-hour mean) or 20 000 particles/cm3 (1-hour mean). Utilize emerging science and technology to advance approaches to the assessment of exposure to UFP for their application in epidemiological studies and
	UFP management.

Pollutant	Good Practice Statements
SDS	Maintain appropriate air quality management and dust forecasting programs. These should include early warning systems and short-term air pollutic action plans to alert the population to stay indoo and take personal measures to minimize exposure and subsequent short-term health effects during SDS incidents with high levels of PM. Maintain suitable air quality monitoring programs and reporting procedures, including source apportionment activities to quantify and characterize PM composition and the percentage contribution of SDS to the overall ambient concentration of PM. This will enable local authorities to target local PM emissions from anthropogenic and natural sources for reduction. Conduct epidemiological studies, including those addressing the long-term effects of SDS, and research activities aimed at better understanding the toxicity of the different types of PM. Such studies are especially recommended for areas whe there is a lack of sufficient knowledge and information about the health risk due to frequent exposure to SDS. Implement wind erosion control through the carefully planned expansion of green spaces that considers and is adjusted to the contextual ecosystem conditions. This calls for regional collaboration among countries in the regions affected by SDS to combat desertification and carefully manage green areas. Clean the streets in those urban areas characterized by a relatively high population density and low rainfal to prevent resuspension by road traffic as a short-term measure after intense SDS episodes with high dust deposition rates.