1st AFRICA/MIDDLE-EAST EXPERT MEETING AND WORKSHOP ON THE HEALTH IMPACT OF AIRBORNE DUST AMMAN, JORDAN, 2-5 NOVEMBER 2015

PARTICULATE MATTER AND HEALTH: Update on WHO's view on its impact on health with focus on mineral dust

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PM health impacts, Amman, Nov 2015

This presentation:

- PM levels and trends;
- Scientific evidence on health effects of particulate matter – results of recent research;
- Role of desert dust in causing health effects.

PM₁₀ and PM_{2.5} particle size



Sources of PM







Amman, 8 Dec 2014



Dust storm in Homs, Syria, 7.09.2015



Global decadal (2001-2010) PM_{2.5} **concentrations**



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Population-weighted PM_{2.5} long term means and trends

	2001-2010		1998–2012		
Region	PM _{2.5} (mean µg/m ³ ± SD)	Dust- and sea salt—free PM _{2.5} (mean µg/m ³ ± SD)	PM _{2.5} trend [µg/m ³ /year (95% CI)]	PM _{2.5} trend [%/year (95% CI)]	
Global North Africa/Middle East	26.4 ± 21.4 25.5 ± 10.7	21.2 ± 19.1 11.5 ± 3.6	0.55 (0.43, 0.67) 0.38 (0.17, 0.59)	2.1 (1.6, 2.6) 1.5 (0.7, 2.3)	



Van Donkelaar et al. 2015

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WHO AQG: GLOBAL UPDATE 2005: SUMMARY OF AQG VALUES



Pollutant	Averaging time	AQG value
Particulatematter		
PM _{2.5}	1 year	10 µg/m³
	24 hour (99 th percentile)	25 μg/m³
PM ₁₀	1 year	20 µg/m ³
	24 nour (99" percentile)	ου μg/m°
Ozone, O ₃	8 hour, daily maximum	100 µg/m³
Nitrogen dioxide, NO ₂	1 year	40 µg/m³
	1 hour	200 µg/m³
Sulfur dioxide, SO ₂	24 hour	20 µg/m ³
	10 minute	500 µg/m³

AQG levels recommended to be achieved everywhere in order to significantly reduce the adverse health effects of pollution

REVIHAAP: selected conclusions on PM

http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications

The scientific conclusions of the 2005 WHO Guidelines about the evidence for a causal link between $PM_{2.5}$ and adverse health outcomes in humans have been confirmed and strengthened and, thus, clearly remain valid.

- New studies on short- and long-term effects;
- Long-term exposures to PM_{2.5} are <u>a cause of cardiovascular</u> mortality and morbidity;
- More insight on physiological effects and plausible biological mechanisms linking short- and long-term PM_{2.5} exposure with mortality and morbidity;
- Studies linking long-term exposure to PM_{2.5} to several new health outcomes (e.g. atherosclerosis, adverse birth outcomes, childhood respiratory disease).

Review of evidence on health aspects of

air pollution – REVIHAAP Project

IARC: Air pollution causes cancer

The carcinogenicity of outdoor air pollution

In October, 2013, 24 experts from 11 countries met at the International Agency for Research on Cancer (IARC), Lyon, France, to assess the carcinogenicity of outdoor air pollution. This assessment was the last in a series that began with specific combustion products and sources of air pollution and concluded with the complex mixture that contains all of them. The results of this most recent assessment will be published as volume 109 of the IARC Monographs.¹ Outdoor air pollution is a mixture of The IARC Working Group unanimously classified outdoor air pollution and particulate matter from outdoor air pollution as carcinogenic to humans (IARC Group 1), based on sufficient evidence of carcinogenicity in humans and experimental animals and strong mechanistic evidence.

The findings regarding the carcinogenicity of outdoor air pollution as a mixture, and of particulate matter specifically, are remarkably consistent in epidemiological research, studies of cancer in experimental animals, and a to traffic or traffic emissions, in studies that were adjusted for tobacco smoking. However, most studies assessed exposure only by employment in occupations with potentially high exposure to outdoor air pollution, so the results did not weigh heavily in the evaluation.

The Working Group also reviewed evidence regarding the carcinogenicity of outdoor air pollution in experimental animals. As part of this process, the IARC's earlier evaluations of diesel engine



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For more on the IARC Monographs see http:// monographs.iarc.fr/

Meta-analysis of the association between long-term exposure to PM_{2.5} and all-cause (natural) mortality



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Mortality and long-term PM_{2.5} exposure Results of a Canadian cohort study (2.1 million adults, 1991-2001)



Long term PM_{2.5} exposure and cardiovascular mortality

Study	HR (per 1	95% CI) 0. ua/m ³	Weight
Harvard six cities	1.26	(1.14, 1.40)	8.43
ACS study	1.09	(1.03, 1.16)	13.31
ACS LA sub-cohort study	1.26	(1.00, 1.59)	2.63
Women's Health Initiative Study	→ 1.76	(1.25, 2.47)	1.30
Netherlands Cohort Study	1.04	(0.90, 1.21)	5.30
Health professionals follow-up study	1.03	(0.84, 1.27)	3.10
Vancouver cohort	1.07	(0.86, 1.33)	2.96
US trucking industry cohort	1.05	(0.93, 1.19)	6.79
Canadian national cohort	1.15	(1.07, 1.24)	11.50
California teachers study	1.07	(0.96, 1.20)	7.59
Rome longitudinal study	+ 1.06	(1.04, 1.08)	18.02
ACS California subcohort	1.12	(1.03, 1.23)	10.01
National English cohort	1.00	(0.85, 1.17)	4.82
Escape	0.98	(0.83, 1.16)	4.26
D-L Overall (I-squared=53.6%, p=0.0090)	1.10	(1.05, 1.16)	100.00

Forastiere et al, WHO 2014

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Long term exposure to PM and incidence of acute coronary events in ESCAPE

(100,166 people in 11 cohorts followed for average 11.5 years)





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Health indicators functionally related to PM_{2.5} or PM₁₀ exposure: HRAPIE project results

Effects of long-term exposure:

- Mortality, all (natural) cause, age 30+
- Mortality, CVDs, ischaemic heart disease, COPD, trachea, bronchus and lung cancer, age 30+;
- Post-neonatal infant mortality (all cause);
- Prevalence of bronchitis in children;
- Incidence of chronic bronchitis in adults.

Effects of short-term exposure:

- Mortality, all cause, all ages;
- Hospital admissions for CV and respiratory diseases, all ages;
- Restricted activity days, all ages;
- Work days lost, age 20-65;
- Incidence of asthma symptoms in asthmatic children, age 5–19 years.

http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications

Air pollution (PM) source types associated with health effects (conclusions of WHO REVIHAAP project)

- Carbonaceous material from traffic;
- Coal combustion (sulfate-contaminated particles);
- Oil or coal combustion in shipping, power generation, metal industry;
- Biomass combustion (including residential wood combustion);
- Traffic-generated dust, including road, brake and tyre wear;
- Desert dust episodes.

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Effects of Saharan dust on association between mortality and PM_{10-2.5}

Time series study in Rome, Italy, with 80 423 deaths in 2001-2004

Increase in risk of death (%) per IQR PM_{10-2.5} (10.8 µg/m³)



Risk of natural mortality associated with non-desert PM₁₀ and desert PM₁₀ in Southern Europe: MED-PARTICLES Study

Non-desert PM₁₀ (10 μ g/m³ increase)

Desert PM₁₀ (10 µg/m³ increase)



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Acute myocardial infarction (AMI) and Asian dust (AD) Odds ratios for AD days vs. non-AD days

A case-crossover study of hospitalization because of AMI among 3068 consecutive patients of 4 AMI centres in Fukuoka, Japan, and data for AD 2003-2010.



Matsukawa et al, Circ Cardiovasc Qual Outcomes. 2014

The nature of pollutant materials carried in dust storms

	Location	Nature of substance(s)		
	France Japan Japan	Radioactive cesium (¹³⁷ Cs) from Sahara Radioactive cesium (¹³⁷ Cs) from China and Mongolia Enriched uranium from Central Asia		
Location Biological material		Plutonium from Saharan atomic tests Heavy metals from China		
Kuwait & Iraq	Mycobacterium, Brucella, Coxiella Burnetii, Clostridium perfingens, Bacillus	Heavy metals from China Heavy metals Sulphates and nitrates		
West Africa Taiwan Japan Crete Korea Israel	frica Neissera meningitides Influenza virus Pollen spores Bacteria Bacteria Fungal communities	Polycyclic aromatic hydrocarbons Polycyclic aromatic hydrocarbons and fatty acids Phosalone from Aral Sea Heavy metals, organochlorine pesticides, Dioxins from Aral Sea Arsenic Dioxins and PCBs from China		
Iran	Fungi: Cladosporium, Alternaria, Aspergillus, Penicillium and Rhizophus	the section		
Turkey Israel Iran	Thermophilic bacteria (<i>Geobacillus</i>) Bacteria and fungi Bacteria and fungi			

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Goudi, Env Internat 2014

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Desert dust health effects: conclusions of WHO REVIHAAP project

- After the 2005 global update of the WHO air quality guidelines, several new studies reported positive associations between shortterm exposure to PM₁₀ or coarse particles and mortality during desert dust episodes;
- The results for cause-specific mortality or for hospitalizations have not been fully consistent for coarse particles and desert dust episodes;
- Evidence for an effect of desert dust on human health is increasing, but at the moment it is not clear whether crustal, anthropogenic, or biological components of dust are most strongly associated with the effects.

Conclusions

- Fine particulate matter (PM_{2.5}) causes cardiovascular diseases and rspiratory cancers, and is related to other health problems;
- Various sources (especially combustion) contribute to population exposure to PM and to its health effects;
- Health risk increases proportionally to the exposure level, also at relatively low PM concentrations (even below WHO AQG level);
- Desert dust contributes to the impacts of PM_{2.5} mass but scientific evidence on the type and magnitude of health effects <u>specific</u> to desert dust is scarce:
- More research conducted in desert dust affected countries is needed to elucidate aspects of effects specific to those exposures.



Three-year running average of satellite-derived PM_{2.5} concentrations over Middle East



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Long-term exposure to PM₁₀ and mortality. Dutch Environmental Longitudinal Study (DUELS) of 7.1 million adults (age 30+), 2004-2011



*RR(95%CI) per 10 µg/m³ PM₁₀, Adjusted for age, gender, marital status, region of origin, household income

Median $PM_{10} = 29 \ \mu g/m^3$, $IQR=2.5 \ \mu g/m^3$

Cardiovascular mortality excess risks (95% CIs) and IQR increases in sources of PM_{2.5} (lag 2) Case cross-over study in Barcelona, 2003-2007



Ostro et al, EHP 2011

Cause-specific mortality and Asian dust or suspended particulate matter (SPM) Relative risks (RR) in a two-pollutant model

		area	RR	(95% CI)		
	Asian dust			0.9	95 1	1.05
Time-series	circulatory d	isease		L		
analysis targeting	Lag 1	north	1.006	(1.001 to 1.011)		
analysis largeling		south	1.001	(0.999 to 1.004)	ľ	
ca. 1.4 million	I O C	.1	1.016	(1.002 + 1.020)		
neonle aged 65+	Lag 0-6	north	1.016	(1.002 to 1.030)	+0+	
people aged 03+	heart disease	south	1.000	(0.994 to 1.007)		
living in 47 cities in	Lag 0-2	north	1 0 1 7	(1.005 to 1.029)	⊢	
western Janan	Lug 0-2	south	1.003	(0.998 to 1.029)	HOH	
western Japan	cerebrovascular disease					
(2005-10).	Lag 1	north	1.004	(0.996 to 1.014)	⊢■⊣	
· · · ·		south	1.003	(0.999 to 1.008)		
	Lag 0-6	north	1.012	(0.990 to 1.035)	⊢_ ∎i	
		south	1.001	(0.990 to 1.012)	⊢¢	
	pulmonary d	isease				
	Lag 0-2	north	1.011	(0.999 to 1.023)		
		south	1.001	(0.996 to 1.007)	P	
	SPM			0	.9 1	1.1
	circulatory d	isease				_
	Lag 0-6	north	0.981	(0.944 to 1.020)		
SPM ≅ PM₀	arrhythmia	south	0.993	(0.977 to 1.010)		
0	Lag 2	north	0.983	(0.913 to 1.060)	· · · · · · · · ·	
		south	0.972	(0.926 to 1.022)		

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Immune responses to different particles in desert dust



Esmaeil et al. Am J Clin Exp Immunol 2014

Health effects of PM_{2.5} exposure

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Lungs

- Inflammation
- Oxidative stress
- Accelerated progression and exacerbation of COPD
- Increased respiratory symptoms
- Effected pulmonary reflexes
- Reduced lung function

Blood

- Altered rheology
- Increased coagulability
- Translocated particles
- Peripheral thrombosis
- Reduced oxygen saturation

Brain
Increased cerebrovascular ischemia
Heart
Altered cardiac autonomic function
Oxidative stress
Increased dysrhythmic susceptibility
Altered cardiac repolarisaion
Increased myocardial ischemia

Vasculature

- Atherosclerosis, accelerated progression and destabilisation of plaques
- Endothelial dysfunction
- · Vasoconstriction and hypertension

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