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ASPECTS OF AIR POLLUTION RELEVANT TO CHILD HEALTH

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OVERVIEW

1. General trends in air quality
2. Non-carcinogenic air pollutants and public health
3. Carcinogenic air pollutants and public health
4. The vulnerability of children

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TRENDS IN AIRBORNE CONCENTRATIONS

- **The London smog of 1952 caused about 4000 excess deaths as a result of exposure to smoke (particulate matter) and sulphur dioxide. It led to the Clean Air Acts of 1956 and 1968.**

- **Air quality in towns improved far faster than the reduction in national smoke and sulphur dioxide emissions due to reduction in domestic coal burning.**

- **The control of coal burning was concurrent with a large increase in road traffic which has brought its own problems and a different range of pollutants.**

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DEFINITION OF TERMINOLOGY

Airborne particles are measured as:

PM_{10} - approximates to particles less than 10 micrometres diameter, measured by weighing

$PM_{2.5}$ - less than 2.5 micrometres diameter (known as fine particles)

PM_{10} minus $PM_{2.5}$ - coarse particles

Black smoke - particles, usually less than 4 micrometres measured according to their blackness (a measure of combustion particles, but not secondary particles)

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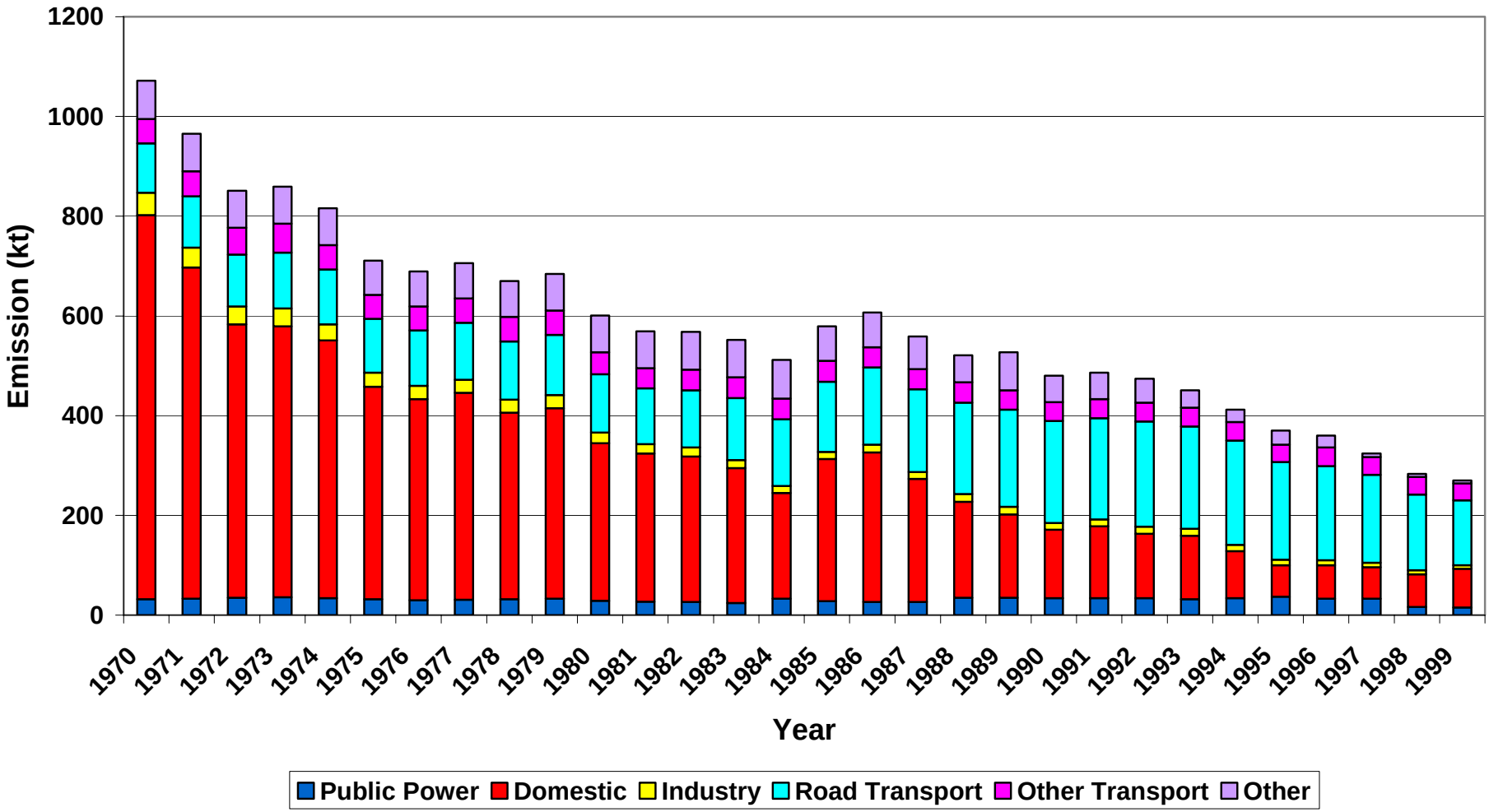
UK Emissions of Black Smoke, 1970-1999



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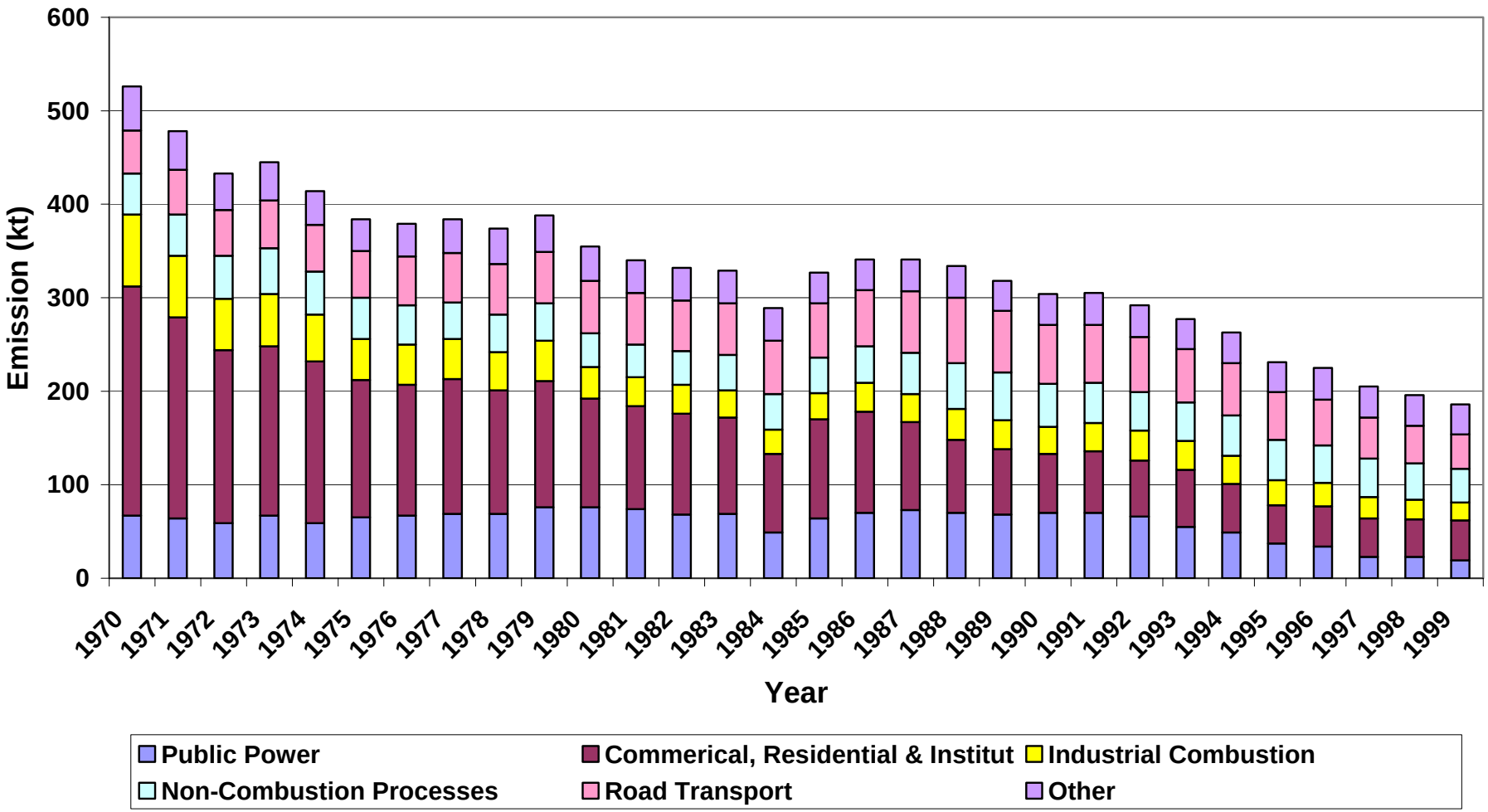
UK Emissions of PM10, 1970-1999



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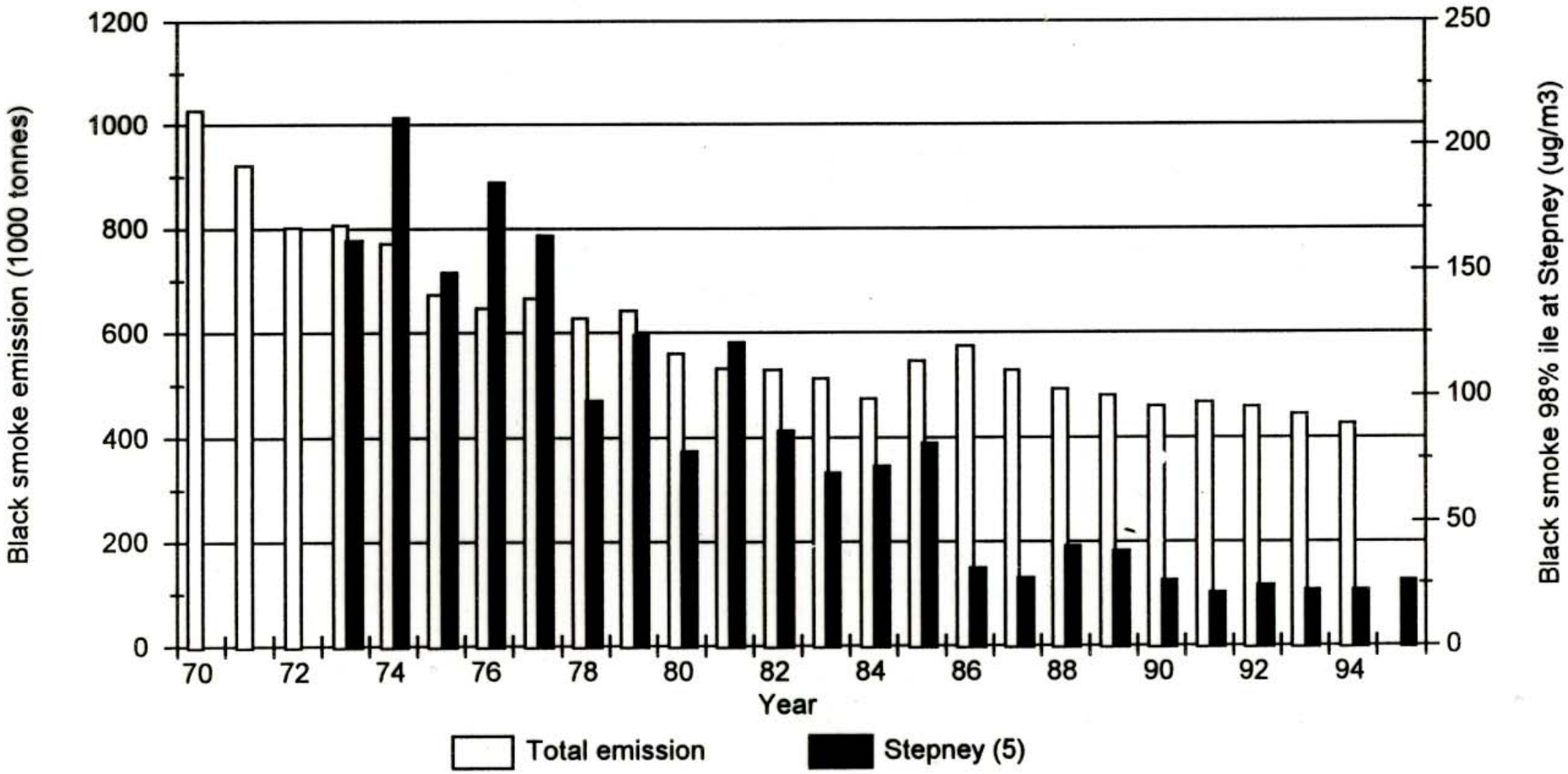
TRENDS IN BLACK SMOKE, 1970-1995



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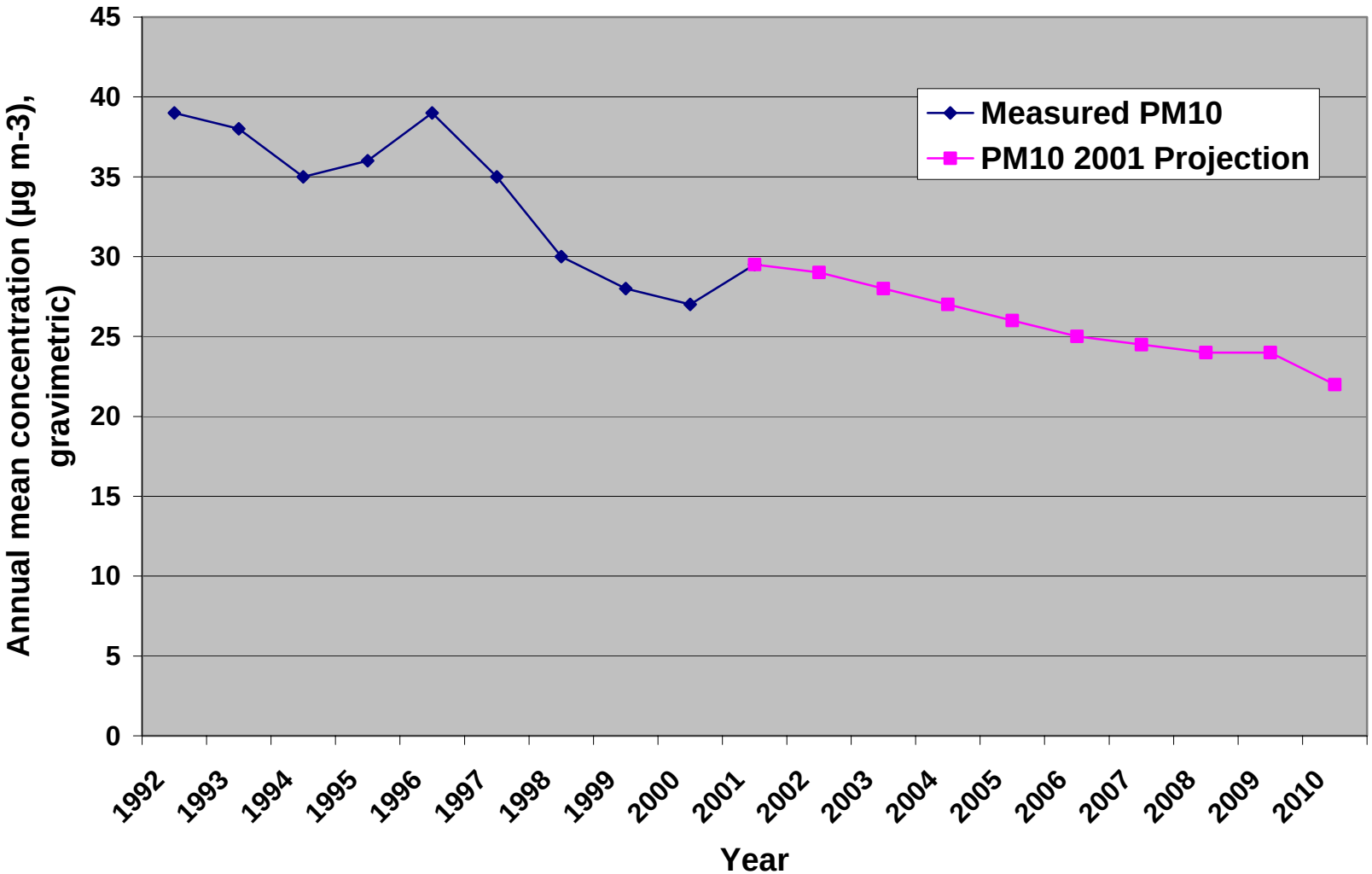
Past and Future Projected PM10 Concentrations at London Bloomsbury



Source: Mr J. Stedman, NETCEN, with support from DEFRA

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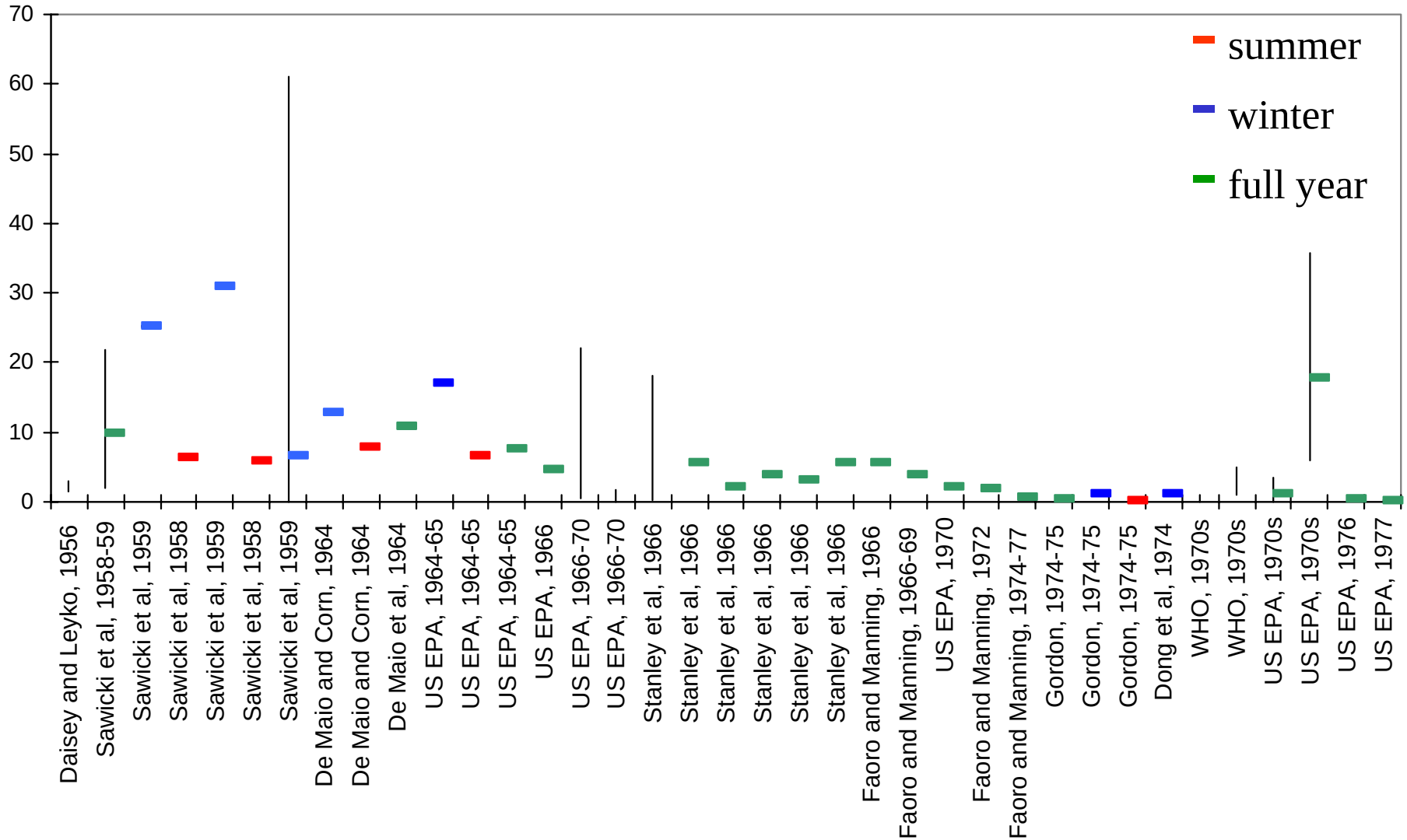
Range and mean benzo(a)pyrene concentrations (ng/m^3) found in ambient air in cities in the USA during the 1950s to 1970s



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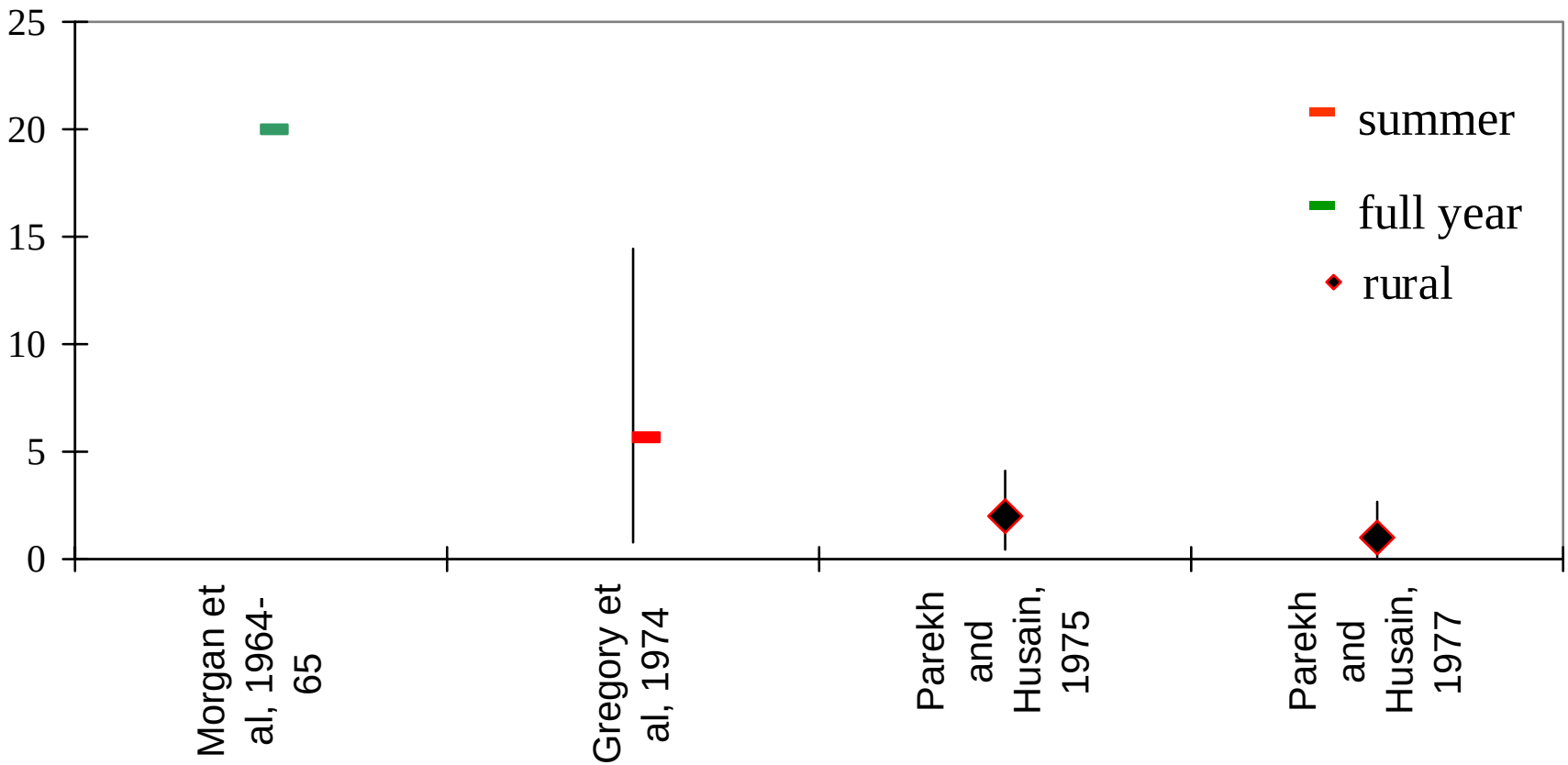
Range and mean arsenic concentrations (ng/m^3) found in ambient air in cities in the USA during the 1950s to 1970s



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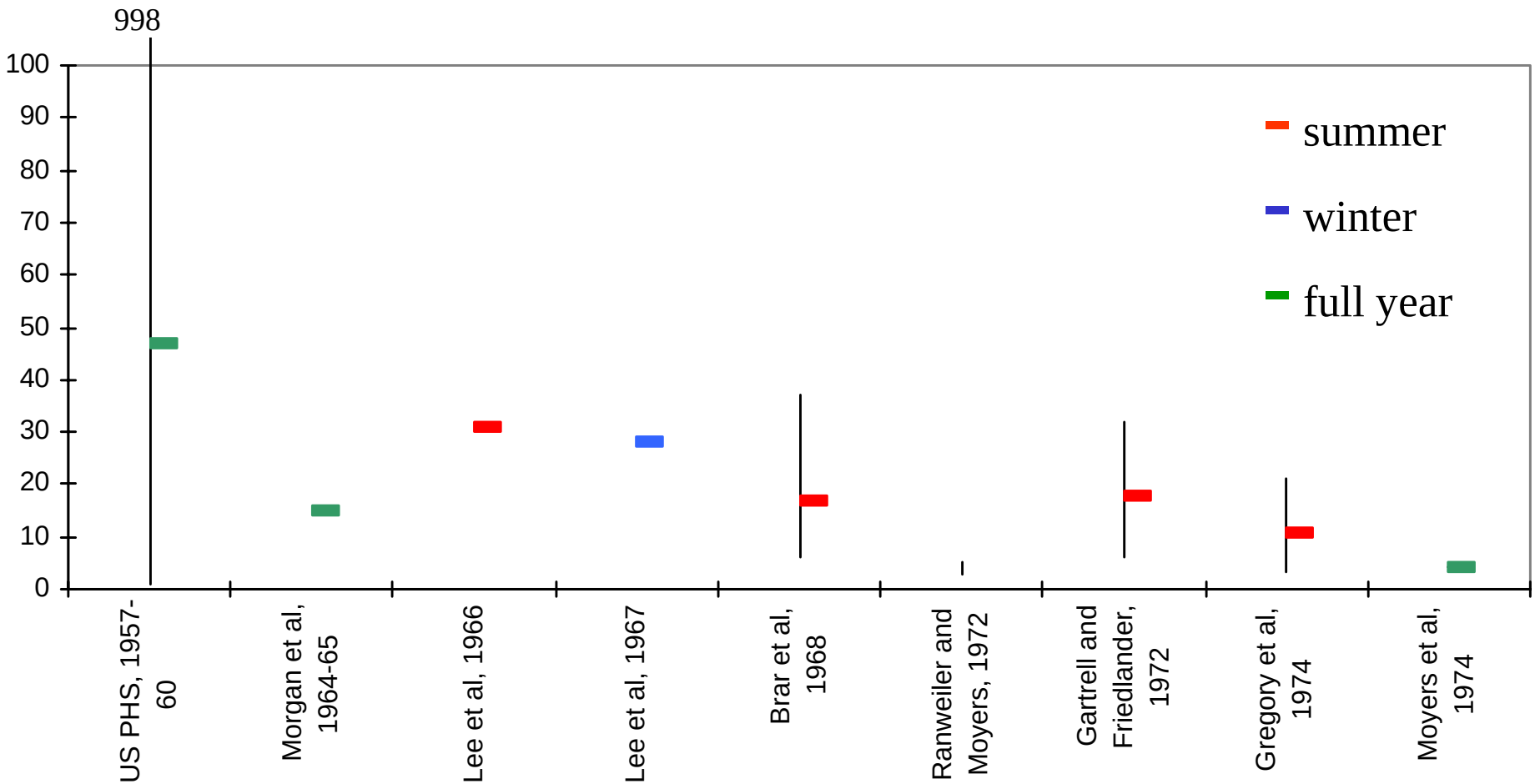
Range and mean chromium concentrations (ng/m^3) found in ambient air in cities in the USA during the 1950s to 1970s



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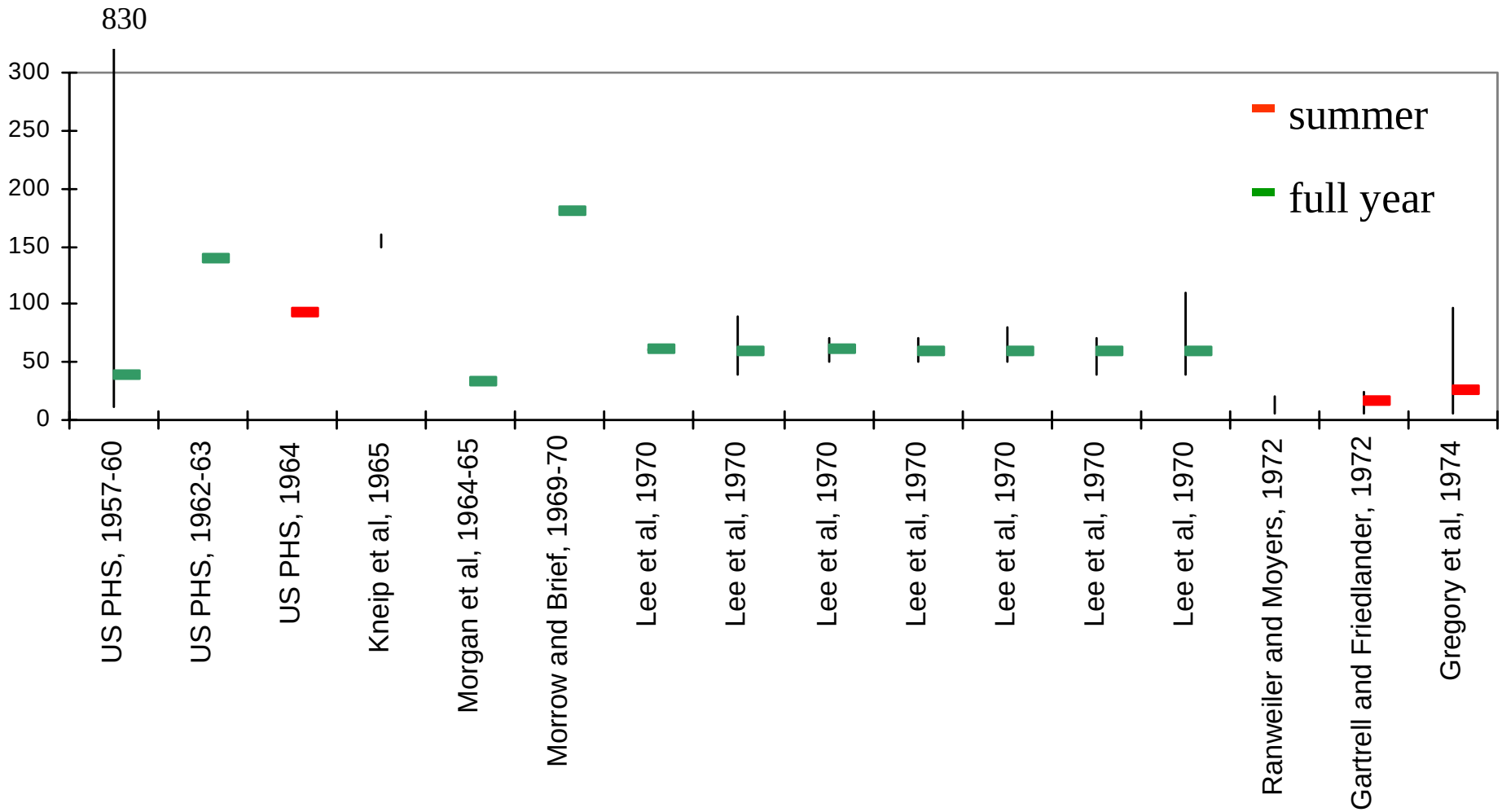
Range and mean nickel concentrations (ng/m^3) found in ambient air in cities in the USA during the 1950s to 1970s



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PUBLIC HEALTH IMPACTS OF AIR POLLUTION

What are the effects of current air pollution levels on public health?

- **Short-term (acute) effects – elucidated by time series studies**
- **Long-term (chronic) effects – some information from cohort studies**
- **Cancers induced by air pollutant exposure – extrapolations from occupational cancer data, and ACS study**

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SHORT TERM (ACUTE) EFFECTS

- **Elucidated through the "time series" epidemiological studies which relate day-to-day changes in air pollutant concentrations with changes in daily mortality and hospital admissions, frequently with a lag of up to three days between cause and effect.**
- **Department of Health Committee on the Medical Effects of Air Pollutants (COMEAP) has made quantification estimates of mortality and hospital admissions resultant from exposure to PM_{10}, sulphur dioxide, nitrogen dioxide and ozone.**
- **Biggest uncertainty is attached to who is dying prematurely and the extent of loss of life expectancy.**

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NUMBER OF DEATHS AND HOSPITAL ADMISSIONS FOR RESPIRATORY DISEASES AFFECTED PER YEAR BY PM_{10}*, SULPHUR DIOXIDE AND NITROGEN DIOXIDE IN URBAN AREAS OF GREAT BRITAIN

Pollutant	Health Outcomes	GB Urban
PM_{10}	Deaths brought forward (all cause)	8100
	Hospital admission (respiratory) brought forward and additional	10500
SO_2	Deaths brought forward (all cause)	3500
	Hospital admission (respiratory) brought forward and additional	3500
NO_2	Hospital admission (respiratory) brought forward and additional	8700

*** PM10: particulate matter generally less than 10 μm in diameter**

Estimated total deaths occurring in urban areas of GB per year = c430,000

Estimated total admission to hospital for respiratory disease occurring in urban areas of GB per year = c530,000

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NUMBERS OF DEATHS AND HOSPITAL ADMISSIONS FOR RESPIRATORY DISEASES AFFECTED PER YEAR BY OZONE IN BOTH URBAN AND RURAL AREAS OF GREAT BRITAIN DURING SUMMER ONLY

Pollutant	Health Outcomes	GB: threshold = 50 ppb	GB: threshold = 0 ppb
Ozone	Deaths brought forward: all causes	700	12500
	Hospital admission (respiratory) brought forward and additional	500	9900

FROM: COMEAP (1998)

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ESTIMATES OF NUMBER OF SUBJECTS EXPERIENCING HEALTH EFFECTS OVER A PERIOD OF THREE DAYS CHARACTERISED BY AN AVERAGE PM_{10} CONCENTRATION OF 50, 100 AND 200 $\mu g/m^3$

	Number of Subjects[a] Affected by Three-Days-Long Episode of PM_{10} at:		
Health Effect Indicator	**50 $\mu g/m^3$**	**100 $\mu g/m^3$**	**200 $\mu g/m^3$**
Mortality	**4**	**8**	**16**
Hospital admissions for respiratory conditions	**6**	**12**	**24**
Bronchodilator use among asthmatics	**1,400**	**2,800**	**5,600**
Symptom exacerbations among asthmatics	**1,000**	**2,000**	**4,000**

[a] Estimated number of subjects in an example population of 1 million people, having, on average in any period of 3 days: 80 deaths, 60 respiratory hospital admissions, 4,000 asthmatics using bronchodilators and/or experiencing asthma symptoms.

FROM: WHO (1995)

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LONG-TERM (CHRONIC) EFFECTS

- **Cohort studies carried out in North America demonstrate a loss of life expectancy associated with long-term exposure to airborne particles (PM_{10} or $PM_{2.5}$)**
- **Work at the Institute of Occupational Medicine (Edinburgh) has used the results of the American studies to predict loss of life expectancy across the U.K. population.**
- **Estimates relate to average loss of life expectancy and it is not known how this is distributed across the population, as it is unlikely to reduce all individuals' longevity to the same degree.**

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CHRONIC IMPACTS OF PARTICULATE MATTER EXPOSURE

- **DH have funded Institute of Occupational Medicine to calculate chronic impacts of particulate matter exposure on human health.**
- **Calculations use coefficients from the "American Cancer Society" study which related mortality rates in U.S. Cities to concentrations of airborne sulphate (151 cities) and fine particles – $PM_{2.5}$ (50 cities). Account was taken of an extended reanalysis of the ACS study data by the U.S. Health Effects Institute.**
- **For a birth cohort born in 2000 and followed for their lifetime, gain in life expectancy per 1 µg m^{-3} is from 0.5-4.5 weeks averaged across the population. Current background site $PM_{2.5}$ concentrations in London are around 18 µg m^{-3} equating to between 9 weeks and 18 months life expectancy gain from complete abolition of $PM_{2.5}$ exposure.**
- **Effects are greater than those due to short-term exposure.**

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Adjusted Mortality Relative Risk (RR) Associated with a 10 μg/m^3 Change in Fine Particles Measuring Less than 2.5 μm in Diameter

	Adjusted RR (95% CI)*		
Cause of Mortality	**1979-1983**	**1999-2000**	**Average**
All-cause	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)
All other causes	1.01 (0.97-1.05)	1.01 (0.97-1.06)	1.01 (0.95-1.06)

*Estimated and adjusted based on the baseline random-effects Cox proportional hazards model, controlling for age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure, and diet. CI indicates confidence interval.

Source: Pope et al. (2002), American Cancer Society Study

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CARCINOGENICITY OF AIRBORNE PARTICULATE MATTER

INTRODUCTION

- **In their extension to the ACS cohort study, Pope et al. (2002) report an increase of 8% in lung cancer mortality per 10 µg m^{-3} elevation in $PM_{2.5}$.**
- **Can this rate of lung cancer be explained by the concentrations of known carcinogens in US urban air?**
- **If not, should it be inferred that airborne particulate matter is carcinogenic irrespective of its chemical carcinogen content?**

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ACS II STUDY AND LUNG CANCER MORTALITY

Typical United States urban concentrations of chemical carcinogens (ng m^{-3})

Pollutant	Year			
	1960	1970	1980	1990
PAH (as B(a)P)	20	4	1	0.5
Cr (all valence states)	40	10	6	4
Ni	100	60	20	10
As	25	12	5	3

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ACS II STUDY AND LUNG CANCER MORTALITY

CONCLUSIONS (1)

If exposures in around 1960 are relevant to lung cancers causing deaths in 1982-1998, the cancer rates predicted from WHO unit risk factors may account for the lung cancer rates observed in in the ACS II Study in relation to $PM_{2.5}$ exposure.

Calculated rate for 1960 exposure **4.88 per 10^5**

Rate derived from Pope et al. study attributed to $PM_{2.5}$

- **1980 data** **7.2 (0.9-14.4) per 10^5**
- **1980-2000 data** **12.5 (3.6-20.5) per 10^5**

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Estimated Cancer Risk from Specific Air Pollutants Across the UK Population Annually*

Air Pollutant	Concentration	Unit Risk	Annual Cancer Rate
Benzene	1.9 µg m^{-3}	6 x 10^{-6} (µg m^{-3})$^{-1}$	10
1,3-Butadiene	0.15 µg m^{-3}	2.8 x 10^{-4} (µg m^{-3})$^{-1}$	36
Polycyclic Aromatic Hydrocarbons	0.3 ng m^{-3} (B(a)P)	8.7 x 10^{-5} (ng B(a)P m^{-3})$^{-1}$	22
Arsenic	1.5 ng m^{-3}	1.5 x 10^{-3} (µg m^{-3})$^{-1}$	2
Chromium	7 ng m^{-3}	4 x 10^{-2} (µg Cr(VI) m^{-3})$^{-1}$	240
Nickel	2 ng m^{-3}	3.8 x 10^{-4} (µg m^{-3})$^{-1}$	1
Particulate Matter ($PM_{2.5}$)	15 µg m^{-3}	0.8% (µg m^{-3})$^{-1}$	4030 (deaths)

*** Calculated upper bound assuming UK population of 60 million is exposed to typical concentrations**

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The Enhanced Vulnerability of Children to Toxicants in the Environment

- **Children have disproportionately heavy exposures to many environmental agents.**

- **Children's metabolic pathways, especially in foetal life and in the first months after birth are immature.**

- **Development processes are easily disrupted during rapid growth and development before and after birth.**

- **Children have more years of future life and thus more time to develop diseases initiated by early exposures.**

(from Landrigan et al., Environmental Health Perspectives, 112, 257-265 (2004)).

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The Enhanced Vulnerability of Children to Environmental Agents

- **Heavy metals**
  - **lead and IQ**
  - **prenatal exposure to methyl mercury**
- **Environmental tobacco smoke**
  - **lower birth weight**
  - **respiratory tract infections**
  - **lung growth and development**
- **Aeroallergens**
- **Endocrine disruptors**
- **Air Pollutants**

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Children's Health: Benefit of Air Pollution Abatement

Study of Wong et al., *Assessment of the Health Benefits of Air Pollution Reduction for Children*, Arch. Environ. Hlth, 112, 226-232 (2004) assesses the benefits to the U.S. of pollution reductions due to the Clean Air Act over the period 1990-2010.

Pollutant	Anticipated Concn. Decrease	Typical U.K. Urban Concentration
PM_{10}	2.85 µg m^{-3}	20 µg m^{-3}
Ozone	1.34 ppb	20 ppb
CO	1.68 ppm	2 ppm
NO_2	9.4 ppb	20 ppb
SO_2	1.15 ppb	2 ppb

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Children's Health: Benefits of Air Pollution Abatement

Calculated annual reductions by 2010 in health outcomes in children aged 1-16 in the U.S.

Asthma hospitalisation	**10,000**
Emergency department visits	**40,000**
School absences	**28×10^6**
Low birth weight	**10,000**

From Wong et al., Arch. Environ. Hlth, 112, 226-232 (2004)

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PARTICULATE MATTER

CHRONIC (LONG-TERM EFFECTS)

Avol et al. (2001): Studied children between 10 and 15 years of age who moved home between areas of differing PM_{10} concentration. Subjects who had moved to areas of lower PM_{10} showed increased growth in lung function and those who moved to areas of higher PM_{10} showed decreased growth in lung function.

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Childhood Exposure to Carcinogens: Higher Risk

In draft Supplemental Guidance, the USEPA is consulting on using higher cancer risk factors for children exposed to carcinogens. For agents causing cancer through a mutagenic mechanism, the following higher risks are proposed:

- **For exposures before 2 years of age, a 10-fold adjustment**
- **For exposures between 2 and 15 years of age, a 3-fold adjustment**
- **For exposures after 15 years of age, no adjustment**

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AIR QUALITY STANDARDS: HEALTH EFFECTS UPON WHICH STANDARDS ARE BASED

Pollutant	Health Outcome
Benzene	Leukaemia in exposed workers
1,3-Butadiene	Lymphoma and leukaemia in exposed workers
Polycyclic Aromatic Hydrocarbons	Lung cancer in exposed workers
Sulphur dioxide	Bronchoconstriction in asthmatics
Nitrogen dioxide	Reduced lung function in asthmatics; Increased prevalence of respiratory disease in children (annual limit value)
Ozone	Reduced lung function
PM_{10}	Increase in respiratory hospital admissions
Lead	Neurotoxic effects in children
Carbon monoxide	Exacerbation of angina in cardiovascular disease sufferers

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DO PARTICULATE AIR POLLUTANTS CAUSE HERITABLE MUTATIONS?

Somers *et al. demonstrate that exposure of laboratory mice to polluted air (near a major highway and two steel mills) leads to a 1.5 to 2-fold increase in germline mutation rates at expanded-simple-tandem-repeat (ESTR) DNA loci compared to a rural control site. The offspring of mice exposed to ambient air at the urban-industrial site inherited ESTR mutations of paternal origin 1.9 to 2.1 times as frequently as mice in control groups. Filtration of the air led to a substantial reduction in the effect showing that particulate rather than gaseous pollutants were responsible. PAH were suggested as a possible cause.**

Implications for humans: not yet known

*C.M. Somers et al., *Science*, 304, 1008-1010 (2004)

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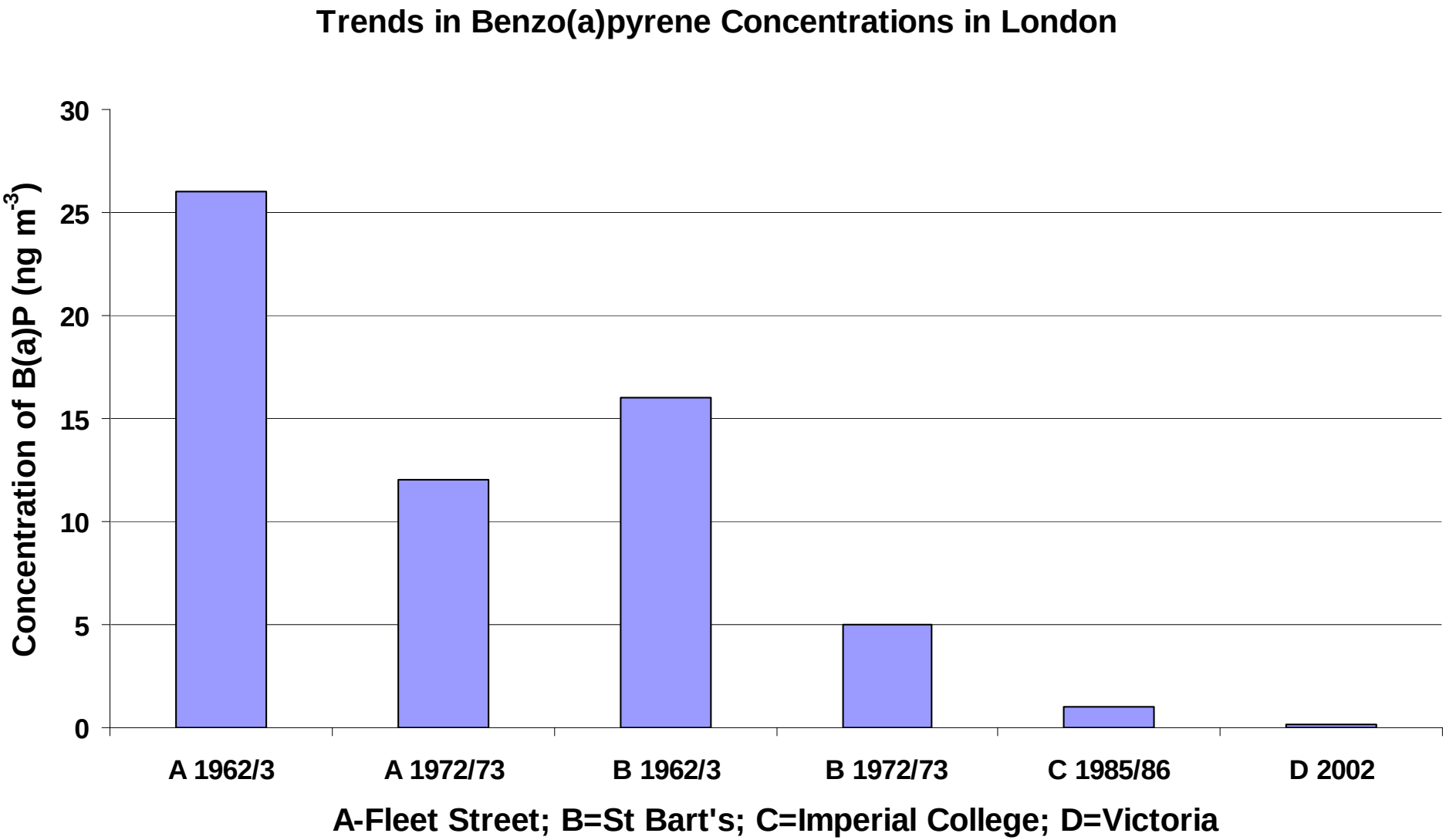

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Indoor Air Quality: Volatile Organic Compounds

Comparison of mean concentrations (μg m^{-3}) in smoking and non-smoking homes

Compound	Smoking Homes (N = 32)	Non-smoking homes (N = 32)
1,3-Butadiene	1.7	0.5
Benzene	16.3	11.5
Pyridine	0.9	not detected

From Y. Kim, S.J. Harrad and R.M. Harrison, *Environ. Sci. Technol.*, 35, 997 (2001)

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CONCLUSIONS

- **Air quality is improving, although much remains to be achieved.**

- **There is a substantial public health impact of exposure to current levels of air pollutants, affecting all age groups.**

- **Air pollutants contain a number of chemical carcinogens. Particulate matter may be the most important, although questions remain over the mechanisms, and its public health impact.**

- **The role of environmental tobacco smoke in children's exposure to air pollutants requires greater attention.**