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Forshaw, Rachel; Kharadi, Natalya; McLaughlin, Eoin

Working Paper

Cardiovascular Disease Mortality and Non-Particulate Air Pollution: Evidence from the 20th Century

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CARDIOVASCULAR DISEASE MORTALITY AND NON-PARTICULATE AIR POLLUTION:

EVIDENCE FROM THE 20TH CENTURY

Rachel Forshaw, Natalya Kharadi,

and Eoin McLaughlin

Date: December 2023

Keywords: Air pollution, Cardiovascular disease mortality, Economic growth, Environmental Kuznets Curve, Global public health, SO2 emissions

JEL: I15, N30, N50, Q53, Q54, Q56

Cardiovascular Disease Mortality and Non-Particulate Air Pollution: Evidence from the 20th Century

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Abstract

Air pollution is a global public health threat, responsible for more deaths annually than conventional lifestyle risk factors. While the link between particulate pollution and cardiovascular disease is well-established, evidence for gaseous pollutants remains limited. This study estimates the long-term population effects of a gaseous pollutant – SO_2 – from 1901 to 1975 in a panel comprising 29 countries distributed globally, contributing to the under-explored literature on its cardiovascular disease mortality impact. Across a comprehensive range of empirical specifications, we observe a robust economically and statistically significant rise in cardiovascular disease mortality for an increase in SO_2 emissions. We also contribute to the literature on economic growth and long-term health outcomes. Our historical perspective aligns with the call for more research on the effects of air pollution in developing nations. We highlight a complex trade-off: greater SO_2 emissions increases cardiovascular disease mortality but leads to short-term regional cooling and reduced global warming and as such its abatement may contribute to future climate-related deaths.

JEL: I15, N30, N50, Q53, Q54, Q56

Keywords: Air pollution, Cardiovascular disease mortality, Economic growth, Environmental Kuznets Curve, Global public health, SO2 emissions

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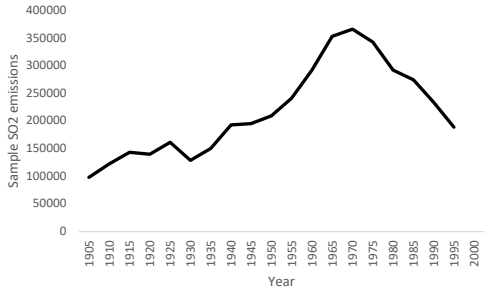
1. Introduction

Air pollution constitutes a significant global public health concern, resulting in over 3 million untimely deaths across the globe annually (Hadley et al., 2018). This number surpasses the fatalities caused by conventional risk factors for heart disease, such as obesity, diabetes, or smoking. Nearly the entire world population experiences measurable levels of outdoor air pollution stemming from traffic, industrial activities, and various other sources. Lelieveld et al. (2015) estimate that cardiovascular diseases (CVD) account for the majority – 60–80% – of air pollution-related deaths. The mechanism by which air pollution causes CVD are a series of harmful reactions in the blood vessels. It leads to increased oxidative stress throughout the circulatory system caused by highly reactive oxygen molecules which harm the cells that line the blood vessels, making them function less effectively and causing immune cells called monocytes to become more active. Additionally, air pollution triggers changes in certain types of fats in the blood that can contribute to the development of artery-clogging plaques. Furthermore, air pollution promotes the formation of blood clots by activating platelets, which are small cells involved in clotting. All of these effects of air pollution contribute to the risk of cardiovascular system problems.

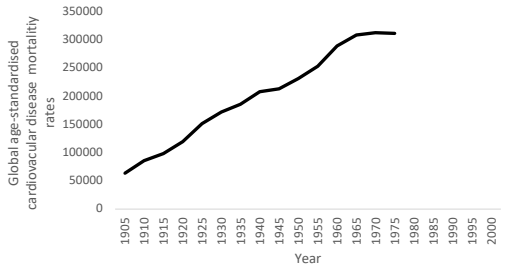
Europe and North American countries experienced an epidemic of cardiovascular disease in the latter twentieth century (Grimes, 2012). The rise in the CVD death rate coincided with increasing population levels and life expectancy; the death rate peaked in the late 1970s and fell thereafter (Jones and Greene (2013), Cutler et al. (2006)). The ultimate cause of decline in CVD is somewhat illusive, as the WHO MONICA project reported that "the major determinant of decline in CVD mortality is whatever drives changing coronary-event rates" (Tunstall-Pedoe et al., 1999). The current consensus is that the decline was due to improvements in prevention and detection of risk factors, pharmaceutical and medical technology, and "other factors yet to be identified" (Mensah et al., 2017). Concurrently, total global SO_2 emissions rose rapidly in the first half of the twentieth century, peaked in the 1970s and decreased substantially in the latter part of the twentieth century (Smith and Wigley, 2001), in line with changes in fuel use, declines in heavy industry, and commitments to international agreements such as the UNECE Convention on Long Range Transboundary Air Pollution, the European Union Sulphur Protocols, and Acid Rain protocols in the United States. Figure 1 compares historical trends in global SO_2 emissions (panel A) with age-standardised CVD mortality in a sample of 29 multi-region countries listed in table 2 (panel B).

Air pollution is composed of particulate matter (commonly measured as either PM_{10} or $PM_{2.5}$) and non-particulate or gaseous pollutants, such as nitrogen dioxide (NO_2), nitric oxide (NO), ozone (O_3), sulphur dioxide (SO_2) and carbon monoxide (CO). While there is good evidence of link between particulate pollution and CVD (see de Bont et al. (2022) for a recent review), evidence is lacking for gaseous or non-particulate pollutants. Bourdrel et al. (2017) examine the cardiovascular effects of outdoor air pollution in a systematic literature review of epidemiological and experimental studies. They find a significant link between long-term particulate exposure, and both short- and long-term exposure to nitrogen dioxide, and increases in CVD mortality. They note that, while there is a known connection between prolonged exposure to sulfur dioxide (SO_2) and respiratory-related deaths, its influence on CVD mortality remains uncertain. This lack of evidence is also echoed by Brook et al. (2010). Notable exceptions include Samoli et al. (2007), Wong et al. (2008) and Lef; all multi-city studies of the effects of air pollution on mortality in Europe, Asia, and the United States, respectively. All found gaseous pollutants were associated with excess CVD mortality. However - in contrast to the current study - they focus on short to medium-term effects.

The contributions of the current study are to add to the small literature on the links between CVD and gaseous or non-particulate SO_2 air pollution. To the best of our knowledge, it also is the first evidence concerning long-term population effects over multiple countries. Kobayashi, Y., and Santos, J.M. and Mill, J.G., et al. (2020) provides a comprehensive meta-analysis of the current evidence which suggests high heterogeneity in the association of SO_2 emissions and health outcomes across studies. They note that this is likely due to limited geographical variation of the available studies. Concretely, the current study provides evidence over the period 1901-1975. An important aspect of our study is the use of age-standardised mortality data compiled by Alderson



A: SO_2 emissions



B: Age-standardised CVD mortality

Figure 1. Historical trends in SO_2 emissions and CVD mortality

Panel A: Total sample SO_2 emissions (Gg SO_2) aggregated within 5-year intervals. Source: (Smith et al., 2011), authors' calculations. Panel B: Total sample age-standardised cardiovascular disease mortality calculated with a standard mortality ratio of 1000 aggregated within 5-year intervals. Source: Alderson (1981), authors' calculations. Both are comprised of the sample of 29 countries listed in table 2.

(1981). One limitation associated with this dataset is its temporal constraint, as it only allows analysis data up to the year 1975. However, age standardisation is essential for geographic and temporal comparisons of mortality given that CVD is predominantly a condition which stikes in later life. Research by GBD 2016 Causes of Death Collaborators (2016) underscores the importance of accounting for differences in age structures across populations: using non-standardised data for global rates of CVD deaths implies an increase of 14.5% over the period 2006-16; using age-standardised rates implies a *decrease* of 14.5% over the same period.

According to Atkinson et al. (2016), most research on air pollution primarily examines developed nations, highlighting the need for more studies in developing nations. This study's historical perspective contributes to the body of evidence on developing nations because, though our sample comprises presently developed countries, the time frame we investigate corresponds to their developmental phase. We find a robust, economically and statistically significant rise in CVD mortality for each percentage point increase in SO_2 emissions. Given the established links between health and economic growth (Bloom and Canning, 2000; Bloom et al., 2004; Weil, 2007; Bloom et al., 2018; Hansen and Strulik, 2017), we examine the impact of a country's level of development on CVD mortality. Our findings reveal an inverted U-shaped relationship indicating that, as income increases, mortality rates initially rise before the rate of change declines at more advanced stages of development. This finding has previously been established over the short-run in Europe (Spiteri and von Brockdorff, 2019), but to the best of our knowledge has not been established over the long-run, or globally. It is well-established that sulphur emissions from burning fossil fuels in the atmosphere lead to short-term cooling in specific regions and reduced global warming (Ripple et al., 2023; J. Hansen and Lacis, 1990). Climate change is itself a large and growing contributor to annual global deaths, estimated conservatively at 2 million since 1970, and impedes development by causing an additional $4.3 trillion in economic costs (United Nations News, 2023). Therefore we highlight an important trade-off which emerges as a consequence of our findings: countries that prioritise sulphur abatement as they develop will likely reduce CVD mortality, but could exacerbate deaths attributed to anthropogenic climate change.

2. Related Literature

Kampa and Castanas (2008) and Brunekreef and Holgate (2002) provide a review of literature focused on the health effects of air pollution. A consistent finding is that air pollution increases mortality and hospital admissions. Both epidemiological and animal model data indicate that systems most affected are the cardiovascular and the respiratory system. There is a well documented association between fine particulate matter $\leq 10\mu m(PM_{10})$ and $\leq 2.5\mu m$ $(PM_{2.5})$ air pollution and cardiovascular disease (Atkinson et al., 2014; Rajagopalan et al., 2018; Combes and Franchineau, 2019; Miller and Newby, 2020). An early landmark study documented a clear association between air pollution and cardiovascular mortality and morbidity in US cities (Dockery et al., 1993). Subsequent research has highlighted an association between air pollution and increased risk of myocardial infarction (Mustafić et al., 2012).

While there are a handful of studies that examine the longer-term links between SO_2 emissions, CVD, and other causes of mortality, each focus on a single geography. Abbey et al. (1999) examine a cohort of non-smoking Californian Seventh-day Adventists between 1977–1992, finding strong associations between SO_2 emissions and lung cancer mortality. Beelen et al. (2008) found no links between SO_2 and CVD, respiratory disease or lung cancer in a Dutch cohort between 1987 and 1996. Cao et al. (2011) found no links cardiopulmonary disease mortality or lung cancer in a cohort study in China between 1991 and 2000, Dong et al. (2011) also found no links in Shenyang between 1998 and for 2009, whereas Chen et al. (2016) found a positive assocation for lung cancer in Northern China between 1998–2009. Anthony Johnson Hedley et al. (2002) examine natural experiment stemming from a restriction of the sulphur content of fuel that occurred in 1990 in Hong Kong which led to an immediate fall in ambient SO_2. Using age-specific death rates they found a significant decline in deaths from all causes, respiratory and cardiovascular diseases. Katanoda et al. (2011) examined 3 Japanese prefectures between 1983 and 1985, finding a positive association with lung cancer and respiratory disease mortality. Nafstad et al. (2004) examined men between 40–49 years of age living in Oslo, Norway in 1972–1973, finding no significant association between SO_2 and respiratory disease, lung cancer, ischemic heart diseases or cerebrovascular diseases. Carey et al. (2013) examined a large cohort UK between 2003–2007, finding a positive association with all-cause mortality, respiratory deaths, lung cancer and cardiovascular deaths, a finding echoed for general mortality and respiratory illness by Elliott et al. (2007) between 1982 and 1998. Filleul et al. (2005) found no association between measures of mortality and SO_2 emissions in France between 1974 and 1976. The variety of relationships observed underscores the need for a comprehensive, long-term, cross-country study, such as the one presented in this paper.

The study closest to our own in methodology and topic is Spiteri and von Brockdorff (2019) who explore the relationship between cardiovascular disease mortality and economic development, as proxied by GDP per capita, in 27 European countries from 2004 to 2014. Their main finding is a statistically significant inverted U-shaped relationship, a "health Kuznets curve", where mortality rates first increase and then decline as income levels rise. The main specification is a fixed effect panel regression of cardiovascular disease per capita. Apart from GDP per capita and GDP per capita squared, Spiteri and von Brockdorff (2019) also find smoking and human capital to be two strong correlates, positive and negative respectively, with cardiovascular disease but with no statistically or economically significant effect for pollution, as measured by $PM_{2.5}$. Unlike the current study, they do not examine non-particulate pollution, or longer-term effects. This paper is related to the literature on the Environmental Kuznets Curve (e.g., Arrow et al., 1996; Stern, 1998) Specifying SO_2 emissions per capita as the dependent variable, Markandya et al. (2006) examine the relationship between emissions and GDP per capita going back to 1870 for twelve European countries, finding an inverted U-shaped relationship at both the aggregate and country levels. Ezzati et al. (2005) analyse risk factors for cardiovascular disease in a cross-national setting using parametric regressions, they also find evidence of inverted-U shaped relationships. The finding of an inverted-U, or Kuznets curve, are also found for obesity at the state level in the US (Grecu and Rotthoff, 2014).

Other related studies focus on the relationship between air pollution and different outcome variables. Arceo et al. (2016) find an increase in infant mortality due to air pollution in a developing country context using an instrumental variables strategy. They exploit the existence of thermal

inversions in Mexico, where a mass of hot air gets caught above a mass of cold air, trapping pollutants. Lohmann et al. (2023) conduct a randomised field experiment in Beijing, China where participants were randomly assigned to be surveyed on high pollution and low pollution days. They find no evidence of effects of air pollution on anti-social behaviour. O'Brien et al. (2018) estimate the causal relationship between air pollution and economic mobility in the US, finding that higher levels of air pollution in a person's birth year is associated with less upward economic mobility for children from low-income families. Clemens et al. (2017) find fetal growth in utero is reduced by exposure to air pollution.

There is also a well-developed literature that analyses the impact of cardiovascular disease on economic growth. At a cross-country level, Suhrcke and Urban (2010) find a negative relationship between increasing levels of cardiovascular disease in the working age population (15-64) and economic growth using panel data from 1960 to 2000. Similarly, Hyclak et al. (2016) find cardiovascular disease has a negative impact on economic growth over the period 2000 to 2012. At a subnational level, Hansen and Strulik (2017), using a difference-in-difference estimation strategy, find evidence that US states that experienced early declines in cardiovascular disease saw increases in life expectancy and educational attainment.

3. Methodology

To analyse the relationship between non-particulate air pollution and cardiovascular disease mortality over the long-term, we now detail the empirical models to be estimated. A first specification estimates a simple model of CVD mortality on sulphur dioxide emissions, equation 1:

$$\ln CVD_{it} = \beta_0 + \beta_1 lnSO2_{it} + \alpha_j + \gamma_d + \epsilon_{it} \tag{1}$$

where:
i = Country (where i = {1, 2, ...,29})
j = Region (where j={Europe, Asia, Americas, Oceania})
t = Five-year interval (where t={1900, 1905,...1970})
d = Decade where d={1900, 1905,...1970})
ln = Natural logarithm
CVD_{it}= Age-standardised male mortality attributed to cardiovascular disease in country i in five-year interval t.
$SO2$= SO_2 emissions in Gg in country i in five-year interval d.

In regression equation 2, we follow a specification in which we regress CVD on per capita income, allowing for the relationship to be non-linear via the inclusion of an additional term for squared per capita real GDP. This captures the inverted U-shaped curves first reported for income inequality by Kuznets (1955), environmental deterioration (Grossman and Krueger, 1995), and repeated for health outcomes by Spiteri and von Brockdorff (2019). Like Spiteri and von Brockdorff, in equation 2 we extend the basic setup of equation 1 to include a richer empirical model of health, based on the health production function of Grossman (1972) which outlines the importance of socioeconomic, lifestyle and environmental or contextual variables. To control for socioeconomic factors, we include GDP per capita. For lifestyle factors, we include a measures of cattle per capita and smoking rates per capita. Environmental controls include the numbers of doctors and infant mortality per 10,000 population. Section 4, below, explains each variable and their sources in more detail.

$$\ln CVD_{it} = \tilde{\beta}_0 + \tilde{\beta}_1 \ln SO2_{it} + \tilde{\beta}_2 \ln(gdp_{it}) + \tilde{\beta}_3 \ln(gdp_{it})^2 + \tilde{\beta}_4 \ln(imr_{it}) + \tilde{\beta}_5 \ln(cattlepc_{it}) + \tilde{\beta}_6 \ln(smokingpc_{it}) + \tilde{\beta}_7 \ln(doctors_{it}) + \alpha_j + \gamma_d + u_{it} \tag{2}$$

where:
i = Country (where i = {1, 2, ...,24})
gdp_{it} is Real GDP per capita (2011 prices) averaged over five-year interval t for country i
gdp^2_{it} is $\ln(gdp_{it})$ squared
imr_{it} is the under age five child mortality rate for country i averaged over t
$cattlepc_{it}$ is the under age five child mortality rate for country i averaged over t
$smokingpc_{it}$ is smoking per capita in country i averaged over t

Note that the number of countries is lower in this specification, due to no available smoking data for 4 countries (Chile, Iceland, Turkey and Yugoslavia).

Spiteri and von Brockdorff (2019) note a potential pitfall of the approach taken by model 2: possible reverse causality between CVD mortality and income per capita. As noted in section 2, there is a substantial literature which finds a effects of CVD on subsequent economic growth. We also note that GDP per capita is potentially a 'bad control', as described by Angrist and Pischke (2009). Figure 2 visualises the bad control problem as a directed acyclic graph. We are interested in the relationship between SO_2 emissions (SO_2) and cardiovascular disease mortality (CVD), which is also affected by exogenous independent variables contained in vector X, fixed at the time that the regressor of interest (SO_2) was determined. The edge between GDP and SO_2 is bidirectional, capturing the potential reverse causality caused by its inclusion. However, the edge from $SO_2 \rightarrow$ GDP is the root of the bad control problem. This reflects that nations with greater

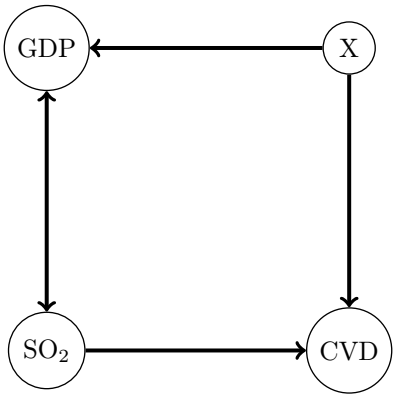


Figure 2. Bad control problem visualised as a Directed Acyclic Graph.
GDP=GDP per capita, SO_2= SO_2 emissions, X= Vector of exogenous controls , CVD= cardiovascular disease mortality.

pollution levels tend to exhibit higher GDP due to their adherence to a more resource-intensive or environmentally detrimental path of economic development. As such, GDP is a proxy control, i.e., it might partially control for omitted regressors but is itself affected by the variable of interest causing a 'collider bias' (Angrist and Pischke, 2009). Additionally, this creates another pathway for omitted variable bias in the presence of any pertinent variables in X that affect GDP but that are left out, due to data constraints, for instance.

To evaluate the robustness of our results to the challenges posed by issues of simultaneity and collider bias, we introduce a final specification in equation 3 which includes average male height by decade as a proxy for both population health and economic development. Average male height is commonly used as a surrogate for population health, which is predominantly determined by the consumption of protein- rich food and the prevailing disease environment (Steckel (2009), Floud et al., Baten and Blum (2014b), Grasgruber et al. (2014), Grasgruber et al. (2016)). Height is a common indicator in health studies, used primarily for its ability to convey long-run information on health status. It also is highly correlated with measures of standard of living and economic development, including GDP (Grasgruber et al. (2016)). As such, it parsimoniously captures the drivers of mortality in vector X in figure 2, i.e., the regressors of equation 2. In practice this has the advantage of allowing for a wider sample, as only three countries (Bulgaria, Iceland and Romania) are omitted due to data constraints, though the time dimension is shorter since the height variable is only available in decadal cohorts. Moreover, the use of this proxy circumvents both the simultaneity issue as well as the 'bad control' problem that arises when incorporating GDP measures. This is because height, a variable measured in adults and a result of the cumulative influence of one's environment across the childhood growth period, is fixed at the time that the regressor of interest (SO_2) is determined.

$$\ln CVD_{id} = \hat{\beta}_0 + \hat{\beta}_1 lnSO2_{id} + \hat{\beta}_2 lnheight_{id} + \alpha_j + \gamma_d + \mu_{id} \tag{3}$$

where:
$height_{id}$= Average male height in country i in decade d.

We use a random effects panel model for all regression specifications; fixed effects are not plausible for such a long time-frame since it would require the assumption that country effects remain unchanged for seventy five years, an assumption that we doubt is plausible. To account for unobserved heterogeneity along time and region dimensions we include decade- and region-fixed effects. We also account for serial correlation using robust standard errors clustered at the individual country level.

Category	Variable Name	Description	Source
Dependent Variable	Cardiovascular Disease Mortality	Age-standardised cardiovascular disease mortality, calculated with a standard mortality ratio of 1000.	Alderson (1981)
Variable of Interest	Non-particulate air pollution	SO_2 emissions in gigagrams, calculated using a bottom-up mass balance method	Klein Goldewijk (2015)
Socioeconomic control variable	GDP per capita	Real gross domestic product per capita in 1990 Geary-Khamis dollars	Maddison Project Database, version 2020
Lifestyle control variable	Cattle per capita	Numbers of cattle per capita	Mitchell (2013)
Lifestyle control variable	Smoking per capita	Total annual consumption of tobacco products divided by annual population estimates	Forey et al. (2002), Maddison Project Database, version 2020
Environmental control variable	Infant mortality rate	Deaths of infants under five years old per 1,000 live births	Gapminder
Environmental control variable	Doctors per 10,000 population	Proxy for healthcare expenditure and CVD mortality accuracy	Alderson (1981)
Socioeconomic, lifestyle, and environmental proxy	Height	Average adult height in decadal cohorts	Baten and Blum (2012)

Table 1. Description of variables and sources

4. Data

4.1. Cardiovascular Disease Mortality

As CVD is a leading cause of death for the elderly (Uemura, 1988), differences in age structures across populations skew direct comparisons of mortality data. The current study overcomes this issue by making use of age standardised mortality data. The Alderson (1981) dataset is unique due to its length and global geographical sample, sourcing mortality information from national statistics offices. The extent and data handling methods facilitate comparative analysis across time and space. This is as a result of the standardisation of mortality rates, where indirect standardisation has been the chosen method in order to account for "the force of mortality for different localities where the proportion of elderly varies" (Alderson, 1981, p. 81). Meaning the standardised mortality rates take into account the age distribution of the populations (Alderson, 1983). Cause-specific, age-standardised mortality statistics are available for a total of 29 countries spanning up to 7 decades in five-year intervals: 23 European countries, one Asian, 3 countries in the Americas and 2 in Oceania. Table 2 reports the countries, regions and observed time periods. For datasets that include the devolved regions of the United Kingdom, estimates for England and Wales are used to proxy the country as a whole since this area contains the majority of the United Kingdom's population. Descriptive statistics for all variables used in the analysis are presented in table 3.

4.2. Non-particulate Air Pollution

Historical sulphur dioxide emissions (in gigagrams) from anthropogenic sources have been estimated by NASA's Socioeconomic Data and Applications Center (SEDAC) (Smith et al., 2011). They provide annual country-level data for SO_2 emission levels from 1850 to 2005. The dataset has been constructed using a "bottom-up mass balance method" (Smith et al., 2011, p.1102). This approach aims to set total emissions equal to the difference between the sulphur content in sources, such as fuel, and the sulphur removed in the combustion process (Smith et al., 2011). The data reflects sulphur emissions arising from anthropogenic sources such as fossil fuel combustion, metal smelting, international shipping, pulp and paper processing, and other sources. It is adjusted so that calculations can be matched with data from individual country reports making estimates "consistent with the available country inventory . . . and. . . across years" (Smith et al., 2011, p.1102).

In figure 3, we illustrate a comparison of global SO_2 emissions with the subset of countries employed in our analysis, as detailed in table 2. Our sample effectively captures a significant portion of global emissions over the specified time period. The disparities primarily stem from the absence of China, Germany, and Russia in our sample. Nevertheless, we capture a large percentage of the level of emissions, and certainly capture the global trends over the time-period. We show that all findings are qualitatively unchanged whether specifying variables in levels or first differences.

4.3. GDP per capita

Data on income per capita is taken from Maddison Project Database, version 2020 dataset. This dataset provides estimates for population, GDP, and GDP per capita from 1 AD to 2018.

Country	Region	Time Periods
Australia	Oceania	11
Austria	Europe	6
Belgium	Europe	11
Bulgaria	Europe	3
Canada	Americas	11
Chile	Americas	14
Czechoslovakia	Europe	12
Denmark	Europe	7
Finland	Europe	10
France	Europe	7
Greece	Europe	7
Hungary	Europe	12
Iceland	Europe	9
Ireland	Europe	11
Italy	Europe	15
Japan	Asia	13
Netherlands	Europe	15
New Zealand	Oceania	14
Norway	Europe	15
Poland	Europe	5
Portugal	Europe	5
Romania	Europe	4
Spain	Europe	15
Sweden	Europe	13
Switzerland	Europe	15
Turkey	Europe	4
United Kingdom	Europe	15
USA	Americas	15
Yugoslavia	Europe	5
Total		299

Table 2. Data availability by country
Number of observations where both the dependent variable (CVD mortality) and the variable of interest (SO_2) is non-missing. Each observation represents a five-year interval between 1901-1975.

Table 3. Descriptive statistics

	Mean	Standard Deviation	Minimum	Maximum
CVD mortality	10189.23	3294.63	2980.00	20337.00
SO_2	1856.78	4503.14	0.06	29688.42
GDP per capita	8571.00	4828.00	1374.00	25330.80
Cattle per capita	0.52	0.58	0.02	3.11
Infant mortality	106.89	92.58	13.39	433.60
Doctors per 10,000 population	10.67	4.42	2.30	23.90
Smoking per capita	0.02	0.01	0.00	0.03
Height	173.67	4.76	159.60	182.30

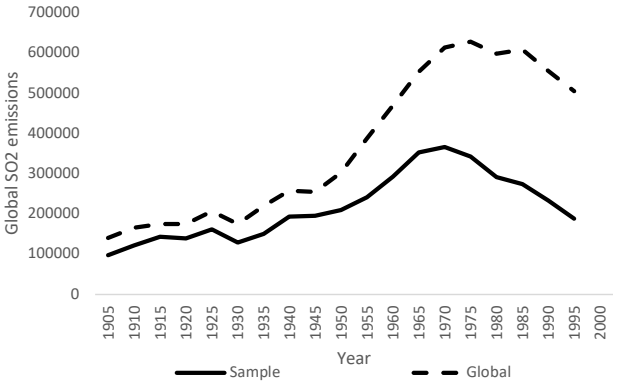


Figure 3. Sample vs Global SO_2 emissions
Total sample and global SO_2 emissions (Gg SO_2) aggregated within 5-year intervals. Source: (Smith et al., 2011), authors' calculations.

It differs from other sources with specific advantages due to its thorough temporal and spatial coverage (Mackenbach and Looman, 2013). It is renowned for its effort to compile GDP estimates from various sources to facilitate long-run comparisons across global regions by converting all estimates into a common currency: 1990 Geary-Khamis dollars (Bolt and van Zanden, 2014). For the countries investigated in this study GDP per capita data is complete after 1950 with few missing data points for a small proportion of the total sample for the preceding half-century. As the SO_2 dataset is reported every five years, we use five-yearly averages of GDP per capita. In this way, GDP per capita provides insight into the environment and budget constraints in which an individual was born. Due to the evidence illustrating that wealthier countries tend to be healthier, albeit with a non-linear Kuznets relationship, it is important to review the long-term trends in GDP per capita to form the basis of whether this relationship exists in the study sample and time period. As such, we also include a squared GDP term to capture the inverted U-shaped curves reported by previous literature (Spiteri and von Brockdorff, 2019).

4.4. Cattle

Cattle per capital is taken from Mitchell (2013). Baten and Blum (2014b) show that this measure is a key determinant of the biological standard of living from a global perspective. Throughout history, humans have consistently required substantial quantities of protein, and so we include cattle per capita to capture a measure relevant to general health and nutrition standards. However, the medical literature also indicates an adverse connection between increased red meat consumption and cardiovascular disease (Shi et al., 2023). It remains to be determined which of these effects dominates in the long-run.

4.5. Smoking rates per capita

Smoking rates are an important predictor of CVD mortality due to a substantial body of scientific research and several well-established mechanisms. While a comprehensive examination of this extensive body of research falls outside the purview of the present study, it is important to note that cigarette smoke is known to contain a multitude of detrimental compounds, such as nicotine and carbon monoxide. These substances have the potential to directly harm the cardiovascular system by fostering the development of atherosclerosis (the accumulation of plaque in arteries), elevating blood pressure, and inciting inflammation in blood vessels. Furthermore, the population attributable risk, which estimates the proportion of CVD cases that can be attributed to smoking, is substantial. Reducing smoking rates has been shown to have a significant impact on reducing CVD mortality rates at the population level. We use data from Forey et al. (2002) on total annual consumption of tobacco products, which we divide by annual population estimates from Maddison

Project Database, version 2020 and average within each five-year interval for each country in the sample.

4.6. Infant Mortality Rate

The infant mortality rates (IMR) used in this analysis has been sourced from Gapminder, and defined as deaths of infants under five years old per 1,000 live births, Other studies use mortality rates of children under one (e.g. Moradi and Baten (2005); Deaton (2007)) or post neonatal mortality (Bozzoli et al. (2009)) interchangeably, finding that results are not dependent on the measure used. The infant mortality rate is a good proxy for general health and healthcare access in the population, but its relationship with other mortality rates is less straightforward due to competing scarring and selection effects on the population of factors that affect the IMR rate (see Blum et al. (2022) for a discussion). We average the infant mortality rate within each five-year interval.

4.7. Doctors per 10,000 population

We include a measure of the number of doctors per 10,000 population in a given country, taken from Alderson (1981). The number of doctors in a country serves as a proxy for healthcare access and efficacy, and countries with more doctors are likely to have better access to medical care, including preventive and treatment measures for cardiovascular disease. Moreover, Stehbens (1987) and (Stehbens, 1990) notes that the mis-classification of the cause of death is a common cause of bias in epidemiological studies, particularly those involving CVD mortality rates. It is likely that the accuracy of cause of death recording increased over time in our sample. As such we control for this using number of doctors per 10,000 population, also aided by our empirical specification that includes across-country variation.

4.8. Height

We use average male height data from Baten and Blum's 2012 database which records height estimates across decadal birth cohorts, covering nearly two centuries and over 250 countries. The frequency of this data is lower, by decades rather than five-year intervals, resulting in a smaller sample size in the time dimension. The quality of the dataset has been examined by Baten and Blum (2012; 2014a) who examine differences by region, identifying potential sources of measurement error and sample selection due to gender, migration, and genetics. They find no significant sources of bias (Baten and Blum, 2012).

Height is frequently employed as a proxy for a broad range of variables in empirical studies. It provides insight into early life conditions and the cumulative influence of one's environment across the growth period (Fogel, 2012). Average height can reflect differences within and between households and countries that life expectancy or income per capita may overlook (Floud et al.). A study on agricultural production and average male height by Baten and Blum (2014b) found that the disease environment and nutrition are the main influencing factors on final adult stature, with the former having a more significant effect than the latter. The wider literature tends to agree that height developments over time can predominantly be explained by better living conditions through better control of disease (Deaton, 2013). The most influential socioeconomic factors on adult height are mortality and income (Dalgaard and Strulik, 2016; Deaton, 2013.) Hatton (2013) found that average height is negatively correlated the infant mortality rate in a sample of European countries spanning from the mid-nineteenth century to 1980. Adult height reflects the influence of childhood circumstances on adult health (Deaton, 2013). The explanatory power of height with respect to life expectancy has been investigated throughout the literature, with the most frequently cited result being produced by Baten and Komlos (1998) who estimated that "one additional centimeter in height equals...1.2 years additional life expectancy" (in Baten and Blum (2014b), p.162). Height is also a useful proxy for welfare, due to its correlations with other indicators such as life expectancy, income or mortality rates, it provides historical information on the biological standard of living (Baten and Blum, 2014a). Height, used as a proxy for better population health, has also been found to increase economic growth, (e.g. Arora, 2001; Bloom et al., 2004; Baten and Blum, 2012).

	(1)	(2)	(3)	(4)
$\ln(SO_2)$	0.349***	0.334***	0.110***	0.121***
	(0.043)	(0.050)	(0.028)	(0.019)
Decade fixed effects	No	No	Yes	Yes
Region fixed effects	No	Yes	No	Yes
N	299	299	299	299
R^2 (within)	0.54	0.54	0.78	0.78
R^2 (between)	0.04	0.33	0.16	0.53
R^2 (overall)	0.07	0.31	0.44	0.68

Table 4. Results for Equation 1.
Notes: Dependent variable is the natural logarithm of age-standardised cardiovascular disease mortality in country i averaged over five-year interval t. Estimated using a random effects model. Independent variable of interest is the natural logarithm of SO_2 emissions in country i at time t. Specifications are as follows by columns: (1) No fixed effects (2) Region fixed effects (3) Decade fixed effects (4) Region and decade fixed effects. *p $<$ 0.1; **p $<$ 0.05; ***p $<$ 0.01. Robust standard errors, clustered at the country level (29 clusters), are reported in parentheses.

5. Results

5.1. Baseline results

We first present the results from estimating equation 1, the baseline model in which we regress cardiovascular disease mortality on the natural logarithm of SO_2 emissions. The results are shown in table 4, with the findings from the random effects model including different specifications for decade and region fixed effects. Over all specifications, the estimated coefficient on $\ln(SO_2)$ is positive and both statistically and economically significant. The findings also indicate the importance of decade and region elements, as their inclusion substantially enhances the explained variance of the regressions. Additionally, their presence reduces the estimated magnitude of the link between CVD and SO_2 by more than half. This implies the presence of substantial common effects specific to each decade, reflecting global economic impacts experienced universally by countries. Likewise, region-specific fixed effects point to enduring common influences within global regions. Table 8 in the appendix shows our results are robust to using a specification in first-differences rather than levels.

5.2. Health production function & economic development

Table 5 reports the estimation results for equation 2, also reported in first-differences in 9 in the appendix. Over all but specification (2), the estimated coefficient on $\ln(SO_2)$ is again positive and significant, ranging between 7-9 percentage points increase in CVD mortality for a percentage point increase in SO_2 emissions. Like Spiteri and von Brockdorff (2019), we find evidence for a Kuznets inverted U-shaped relationship between CVD mortality and development as captured by estimated coefficients on GDP and GDP squared over specifications containing these terms and excluding decade fixed effects. Comparing the estimated coefficients between specification (5), where GDP measures are omitted, and specifications (1)-(4), which encompass GDP measures but exclude decade fixed effects, indicates a few noteworthy points. First, it suggests that a substantial portion of the temporal variation accounted for by decade fixed effects is also encompassed by fluctuations in GDP. Second, comparing columns (1)-(4) with column (5) suggests the presence of a downward bias in the estimated coefficient of interest on SO_2, an effect which is especially pronounced in the specification using first-differences in 9 in the appendix. This effect is likely due to simultaneous causality issues and bad control problem stemming from the inclusion of GDP measures, as discussed in section 3. The estimated coefficient on cattle per capita is positive and significant over most specifications, a finding which aligns with existing literature which finds that red meat consumption is associated with higher risk of CVD (see Shi et al. (2023) for a meta-analysis.) We find a significant negative relationship between infant mortality and cardiovascular disease, potentially reflecting a substitution effect in mortality causes, or a scarring effect of factors

	(1)	(2)	(3)	(4)	(5)
ln(SO_2)	0.077**	0.063	0.081***	0.076**	0.087**
	(0.035)	(0.045)	(0.027)	(0.037)	(0.039)
ln(GDPpc)	4.499*	4.542*	3.525	3.613	
	(2.601)	(2.607)	(2.219)	(2.319)	
ln(GDPpc)^2	-0.269*	-0.272*	-0.200	-0.206	
	(0.148)	(0.148)	(0.125)	(0.131)	
ln(imr)	-0.509***	-0.545***	-0.207**	-0.249**	-0.234***
	(0.092)	(0.099)	(0.095)	(0.107)	(0.078)
ln(cattlepc)	0.244***	0.133	0.264***	0.164**	0.193**
	(0.069)	(0.105)	(0.060)	(0.082)	(0.076)
ln(smokingpc)	-0.031	-0.053	-0.011	-0.024	-0.035
	(0.092)	(0.092)	(0.069)	(0.068)	(0.080)
ln(doctors)	0.341***	0.281***	0.069	0.049	0.017
	(0.088)	(0.086)	(0.069)	(0.074)	(0.075)
Decade fixed effects	No	No	Yes	Yes	Yes
Region fixed effects	No	Yes	No	Yes	Yes
N	165	165	165	165	165
R^2 (within)	0.58	0.60	0.67	0.68	0.66
R^2 (between)	0.72	0.78	0.81	0.84	0.86
R^2 (overall)	0.66	0.71	0.74	0.77	0.77

Table 5. Results for Equation 2.
Notes: Dependent variable is age-standardised cardiovascular disease mortality in country i averaged over five-year interval t. Estimated using a random effects model. Independent variable of interest is the natural logarithm of SO_2 emissions in country i at time t. Specifications are as follows by columns: (1) No fixed effects (2) Region fixed effects (3) Decade fixed effects (4) Region and decade fixed effects. (5) Region and decade fixed effects, omitting GDP measures. *p $<$ 0.1; **p $<$ 0.05; ***p $<$ 0.01. Robust standard errors, clustered at the country level (25 clusters), are reported in parentheses.

	(1) All causes	(2) All respiratory diseases	(3) Respiratory tract cancers
ln(SO_2)	0.013	0.039	0.070
	(0.016)	(0.049)	(0.071)
Controls (listed in table 5)	Yes	Yes	Yes
Decade fixed effects	Yes	Yes	Yes
Region fixed effects	Yes	Yes	Yes
N	178	173	131
R^2 (within)	0.61	0.85	0.94
R^2 (between)	0.74	0.77	0.49
R^2 (overall)	0.63	0.80	0.67

Table 6. Results for Equation 2 for non-CVD mortality causes.
Notes: Dependent variables are age-standardised mortality attributed to: (1) all causes, (2) all respiratory diseases, (3) respiratory tract cancers, in country i averaged over 5-year period t. Estimated using a random effects model. Independent variable of interest is the natural logarithm of SO_2 emissions in country i averaged over decade d. Controls include lnGDP, $\ln(GDP)^2$, ln(imr), ln(cattlepc), ln(smokingpc), ln(doctors). Specifications all include region and decade fixed effects. *p < 0.1; **p < 0.05; ***p < 0.01. Robust standard errors, clustered at the country level (25 clusters)are reported in parentheses.

that increase infant mortality on overall health. The lack of a significant relationship between per capita smoking rates and CVD mortality is somewhat surprising, though it is likely capturing offsetting substitution effects given that smoking causes many other types of disease that affect mortality rates including pulmonary and respiratory diseases, strokes and cancer. The estimated coefficients on doctors per 10,000 population is positive and significant in specifications excluding decade fixed effects. This potentially captures substitution effects away from mortality from other more readily treatable conditions, such as acute infections in populations with more doctors. It could also reflect a greater accuracy in recording deaths attributable to CVD when populations have higher levels of healthcare expenditure. However, the insignificant coefficients once decade fixed effects are included suggests that global innovations in general medical care are potentially more important than country-specific healthcare conditions in the long-run.

Table 6 shows results of estimating the same health production function (equation 2) specification for alternative mortality outcomes: all cause mortality, all respiratory diseases and respiratory tract cancers; the latter includes cancer of the larynx, trachea, bronchus and lung. We find no significant relationship between SO_2 emissions and these alternative mortality measures.

5.3. Alternative proxy: average adult height

Finally, table 7 (table 10 in first differences in the appendix) reports the estimation of equation 3 which uses average male height by decade as an alternative proxy for both population health and economic development while circumventing the issue of the 'bad control' problem and simultaneous causality bias that arises when incorporating GDP measures. In all but one of the specifications, point estimates are positive and significant, ranging in magnitude from 7 - 14 percentage points, a similar range as the two previous regression models. The estimated coefficient on height does not have a straightforward interpretation, given that it is a proxy for a multitude of factors. However, in the preferred specification of column 3, including decade- and region-fixed effects, height is not significantly different from zero, but adds significantly to the explained variance of the regression (when compared with column 4 of table 1.)

	(1)	(2)	(3)	(4)
ln(SO2)	0.123***	0.144***	0.045	0.068**
	(0.035)	(0.040)	(0.035)	(0.034)
ln(height)	18.944***	18.205***	5.707*	3.991
	(2.269)	(2.714)	(3.061)	(2.944)
Decade fixed effects	No	No	Yes	Yes
Region fixed effects	No	Yes	No	Yes
N	118	118	118	118
R^2 (within)	0.81	0.81	0.91	0.91
R^2 (between)	0.37	0.42	0.24	0.44
R^2 (overall)	0.62	0.63	0.69	0.77

Table 7. Results for Equation 3.
Notes: Dependent variable is age-standardised cardiovascular disease mortality in country i averaged over decade d. Estimated using a random effects model. Independent variable of interest is the natural logarithm of SO_2 emissions in country i averaged over decade d. Control is the natural logarithm of average male height i in decade d. Specifications are as follows by columns: (1) No fixed effects (2) Region fixed effects (3) Decade fixed effects. (4) Region and decade fixed effects. *p $<$ 0.1; **p $<$ 0.05; ***p $<$ 0.01. Robust standard errors, clustered at the country level (26 clusters), are reported in parentheses.

6. Conclusion

In conclusion, this study makes several significant contributions to the existing body of knowledge regarding the relationship between cardiovascular disease (CVD) and gaseous or non-particulate SO_2 air pollution. Notably, it expands the relatively small literature on this topic, shedding light on the previously understudied long-term population effects. Our investigation spans the period from 1900 to 1975, providing historical insights into this important health and environmental issue.

An important strength of our research is the utilisation of age-standardised mortality data from Alderson (1981), allowing us to make meaningful geographic and temporal comparisons of mortality, considering that CVD predominantly afflicts individuals in their later years. Furthermore, it is worth noting that the majority of air pollution research has traditionally focused on developed nations, leaving a significant gap in our understanding of its effects on developing nations. Our study, despite examining presently developed countries, provides a historical perspective that corresponds to their developmental phases. Our findings reveal a robust and statistically significant increase in CVD mortality for every percentage point rise in SO_2 emissions, indicating a substantial relationship between air pollution on public health. We find no significant relationship between SO_2 emissions and all mortality, respiratory disease mortality or respiratory tract cancer mortality. We also explore the relationship between a country's level of economic development and CVD mortality. Our results show an inverted U-shaped relationship, suggesting that lower levels of air pollution are associated with lower rates of mortality, as both increase rates initially rise before showing a declining rate of change in more advanced developmental stages. While this phenomenon has been observed in the short term in Europe Spiteri and von Brockdorff (2019), our study, to the best of our knowledge, is the first to establish this relationship over the long term and on a global scale.

Our research also highlights a complex trade-off that arises from our findings. Countries that prioritise reducing sulphur emissions as part of their development strategy are likely to witness a reduction in CVD mortality. However, this measure may inadvertently exacerbate deaths attributed to anthropogenic climate change, which is increasingly becoming a major contributor to global mortality and economic losses.

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A. Appendix

	(1)	(2)	(3)	(4)
ln(SO2) - ln($SO2_{t-1}$)	0.077**	0.078**	0.085**	0.084**
	(0.035)	(0.035)	(0.039)	(0.040)
Decade fixed effects	No	No	Yes	Yes
Region fixed effects	No	Yes	No	Yes
N	267	267	267	267
R^2 (within)	0.01	0.01	0.13	0.13
R^2 (between)	0.04	0.04	0.00	0.00
R^2 (overall)	0.01	0.01	0.11	0.11

Table 8. Results for Equation 1, in first differences.
Notes: Dependent variable is first differenced natural logarithm of age-standardised cardiovascular disease mortality in country i averaged over five-year interval t. Estimated using a random effects model. Independent variable of interest is the first differenced natural logarithm of SO_2 emissions in country i at time t. Specifications are as follows by columns: (1) No fixed effects (2) Region fixed effects (3) Decade fixed effects (4) Region and decade fixed effects. (5) Region and decade fixed effects, omitting GDP measures. *p < 0.1; **p < 0.05; ***p < 0.01. Robust standard errors, clustered at the country level (29 clusters), are reported in parentheses.

	(1)	(2)	(3)	(4)	(5)
ln(SO2) - ln($SO2_{t-1}$)	0.129	0.141	0.107	0.128	0.187**
	(0.111)	(0.109)	(0.137)	(0.133)	(0.079)
ln(GDPpc) - ln($GDPpc_{t-1}$)	4.573	4.314	2.230	1.799	
	(3.408)	(3.476)	(2.759)	(2.662)	
$\ln(GDPpc)^2$ - $\ln(GDPpc_{t-1})^2$	-0.268	-0.253	-0.118	-0.093	
	(0.189)	(0.191)	(0.147)	(0.140)	
ln(imr) - ln(imr_{t-1})	-0.038	-0.041	-0.040	-0.040	-0.075
	(0.060)	(0.060)	(0.088)	(0.090)	(0.064)
ln(cattlepc) - ln($cattlepc_{t-1}$)	0.216	0.220	0.219	0.227	0.240
	(0.209)	(0.206)	(0.218)	(0.213)	(0.209)
ln(smokingpc) - ln($smokingpc_{t-1}$)	-0.003	-0.003	0.007	0.011	0.020
	(0.021)	(0.018)	(0.031)	(0.030)	(0.031)
ln(doctors) - ln($doctors_{t-1}$)	-0.162	-0.231	-0.173	-0.291	-0.343
	(0.389)	(0.436)	(0.269)	(0.340)	(0.386)
Decade fixed effects	No	No	Yes	Yes	Yes
Region fixed effects	No	Yes	No	Yes	Yes
N	128	128	128	128	128
R^2 (within)	0.10	0.11	0.12	0.14	0.15
R^2 (between)	0.05	0.03	0.11	0.03	0.00
R^2 (overall)	0.09	0.09	0.13	0.12	0.11

Table 9. Results for Equation 2, in first-differences.
Notes: Dependent variable is the the first-differenced natural logarithm of age-standardised cardiovascular disease mortality in country i averaged over five-year interval t. Estimated using a random effects model. Independent variable of interest is the first-differenced natural logarithm of SO_2 emissions in country i at time t. Specifications are as follows by columns: (1) No fixed effects (2) Region fixed effects (3) Decade fixed effects (4) Region and decade fixed effects. (5) Region and decade fixed effects, omitting GDP measures. *p < 0.1; **p < 0.05; ***p < 0.01. Robust standard errors, clustered at the country level (25 clusters), are reported in parentheses.

	(1)	(2)	(3)	(4)
ln(SO2) - ln($SO2_{t-1}$)	0.112**	0.117**	0.122**	0.118**
	(0.056)	(0.055)	(0.058)	(0.058)
ln(height)	-0.481	-0.890**	0.610**	0.507
	(0.427)	(0.398)	(0.285)	(0.423)
Decade fixed effects	No	No	Yes	Yes
Region fixed effects	No	Yes	No	Yes
N	102	102	102	102
R^2 (within)	0.15	0.19	0.44	0.44
R^2 (between)	0.01	0.01	0.13	0.18
R^2 (overall)	0.07	0.10	0.38	0.39

Table 10. Results for Equation 3, in first-differences.
Notes: Dependent variable is the first-differenced natural logarithm of age-standardised cardiovascular disease mortality in country i averaged over decade d. Estimated using a random effects model. Independent variable of interest is the first-differenced natural logarithm of SO_2 emissions in country i averaged over decade d. Control is the natural logarithm of average male height i in decade d. Specifications are as follows by columns: (1) No fixed effects (2) Region fixed effects (3) Decade fixed effects. (4) Region and decade fixed effects. *p $<$ 0.1; **p $<$ 0.05; ***p $<$ 0.01. Robust standard errors, clustered at the country level (26 clusters), are reported in parentheses.