On Health Inequality in the Past and Present

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Abstract

This thesis explores socioeconomic gradients in health outcomes through evidence from the past and present. Studying health inequality is fundamental to revealing how socioeconomic factors create unfair variations in health outcomes across populations, which is essential for developing targeted interventions to promote equal opportunity for well-being. Historical analysis provides crucial insights into the long-run implications of socioeconomic inequalities on health, with particular significance during the mortality transition—a period characterised by unprecedented improvements in population health alongside profound transitions that established enduring patterns of health disparities that persisted for generations. Chapters 1 and 2 examine the past by analysing mortality dynamics in London and its constituent areas from the 1850s to the 1950s. Chapter 3 investigates present-day health inequality by focusing on a key health risk factor—physical (in)activity.

The first two chapters draw on novel datasets I constructed by digitising and compiling information and data from historical administrative records, including Medical Officer of Health reports, censuses, and Registrar General's reports. The final chapter leverages the Understanding Society survey, a nationally representative panel dataset that captures comprehensive and nuanced measures of socioeconomic status across UK households.

Chapter 1 utilises a new time series dataset of London mortality spanning 1841–1964, encompassing four mortality measures: crude mortality, corrected death rate (adjusted for age and sex composition), infant mortality, and non-infant mortality. The annual, long-term, and continuous mortality data illuminate an S-shaped decline curve, characterised by an initial period of slow reductions before faster reductions and ultimately a slowdown in improvements. This pattern is observed across all mortality measures, enabling analysis through a four-parameter logistic model. This formal statistical method yields key parameters that capture the underlying dynamics effectively. The inflection point, occurring around 1896 for crude mortality, indicates the point of the most rapid mor-

tality reduction. To explain the sigmoid pattern, an economic model is developed where higher socioeconomic groups adopt health technologies earlier, thereby experiencing mortality transitions earlier. The model underscores how socioeconomic inequality shapes the sigmoid dynamics of mortality decline.

Chapter 2 expands the analysis to constituent areas in London. The new panel dataset comprises three fundamental components: first, geographic areas whose boundaries remained stable for a long enough period; second, three mortality measures: crude mortality, infant mortality, and non-infant mortality for these areas; third, measures of socioeconomic inequality for these areas, including, for example, the proportion of professional occupations and middle-class residents. Application of the four-parameter logistic model reveals substantial spatial heterogeneity in mortality dynamics, with inflection points varying by decades across areas. The economic model developed in Chapter 1 predicts that this variation stems from socioeconomic differences—areas with more high-socioeconomic residents adopted health technologies earlier, facilitating earlier mortality transitions. Empirical testing through an augmented logistic model confirms this hypothesis, demonstrating a significant negative relationship between an area's socioeconomic status and the inflection point of the mortality dynamics. For example, areas that had a 1% higher proportion of individuals in professional classes experienced an inflection point in mortality declines around a year and a half earlier. These findings indicate that the mortality transition proceeded through a prolonged period of mortality divergence across socioeconomic groups before eventual convergence, rather than immediate convergence across social groups.

Taken together, the first two chapters provide the first formal examination of how socioeconomic inequality shaped the sigmoid pattern of mortality decline and generated the "divergence-convergence" pattern of mortality inequality during the period of mortality transition. This historical analysis yields twofold implications: it reveals how uneven access to health innovations historically perpetuated and amplified health disparities in today's developed economies, whilst providing an analytical framework for contemporary developing economies where ongoing epidemiological transitions, absent policies addressing socioeconomic inequalities, risk exacerbating existing health inequalities.

Shifting focus from historical London to contemporary UK, Chapter 3 investigates the distinct mechanisms through which education and household income shape physical activity (PA) patterns among working-age adults in the UK from 2015–2019, using Understanding Society survey data. Employing an ordered logit model to distinguish three PA levels—zero physical activity, engagement in at least some physical activity, and meeting WHO-recommended guidelines—the study yields three principal findings, contributing to the understudied aspects of the relationship between socioeconomic status and physical activity behaviour. First, while both education and income positively influence PA, their relative magnitudes differ substantially—the effect of upgrading from GCSE to degree-level education equals that of a four-and-a-half-fold increase in household disposable income, which suggests that educational interventions may be more effective at promoting physical activity than income support policies. Second, household disposable income has approximately twice the impact of household gross labour earnings on physical activity engagement, highlighting the necessity of using correct income measures and the effectiveness of redistributive policies. Third, women's physical activity is more strongly associated with educational attainment, while household income demonstrates a greater influence on men's activity patterns.

Drawing from this thesis, socioeconomic inequality functions as a crucial determinant of health outcomes, operating both through contemporaneous mechanisms and by shaping the long-run dynamics of health disparities. These findings underscore the importance of examining the persistent effects of socioeconomic conditions across temporal horizons to fully understand their implications for population health.

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Declaration

I declare that, except where explicit reference is made to the contribution of others, that this dissertation is the result of my own work and has not been submitted for any other degree at the University of Glasgow or any other institution.

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Introduction

Health inequalities represent systematic disparities in health outcomes between socioeconomic groups (Whitehead, 1992) that are "unfair, unjust, avoidable, and unnecessary" (Krieger, 2001; Marmot et al., 2010).[1] These disparities persist across multiple geographic scales—from neighbourhood-level differences within cities to global variations between nations—and throughout different historical periods into the present day (see e.g., Marmot, 2020; WHO, 2004; Kesztenbaum and Rosenthal, 2016; Costa and Kahn, 2015). Motivated by the pervasive and enduring health inequalities, my thesis focuses on mortality inequalities within London from the 1850s to the 1950s, and education- and income-based inequalities in physical activity among working-age adults in the UK from 2015 to 2019.

Historically, the high mortality rates before and during the 19th century were dominated by infectious diseases, with limited medical interventions available until the 1930s when effective vaccines and treatments emerged (Costa and Kahn, 2015; Sakai, 2018). This epidemiological landscape underwent a fundamental transformation, characterised by what Omran termed the "epidemiological transition" (Omran, 1971). From the late 19th century through the early 20th century, today's developed societies witnessed a gradual shift in mortality patterns—as infectious diseases came under control, degenerative and chronic conditions became the predominant causes of death. As a consequence of this transition, modern developed societies have entered a "post-medical era" (Health Education Unit, 1986), where individual behavioural choices have emerged as critical determinants of both health outcomes and health inequalities, thus becoming central to health policy formulation (Glorioso and Pisati, 2014).

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1. Variations in health outcomes attributable to genetic predispositions or biological determinants fall outside the scope of this thesis.

The United Kingdom has been particularly instrumental in identifying and addressing these evolving health inequalities (Marmot et al., 2010). As early as the 1840s, pioneering researchers like Edwin Chadwick systematically analysed administrative records to quantify mortality gaps across occupational groups, demonstrating stark differences in life expectancy, age at death, etc. (Chadwick and Flinn, 1842). This early foundation of systematic data collection laid the groundwork for successive generations of British researchers to examine the relationship between social class and health outcomes (Smith et al., 2016, p. 2). This tradition continues today through Understanding Society (UnSoc), which provides rich panel data on health and socioeconomic conditions. With its comprehensive and nationally representative sample of over 40,000 UK households, the survey captures extensive measures of health outcomes, health risks, health behaviours, and income measures, which enables robust analysis of health disparities across socioeconomic conditions. Chapters 1 and 2 of my thesis study mortality patterns and inequalities in London using newly created datasets drawn from historical administrative records, while Chapter 3 analyses contemporary physical activity inequalities using UnSoc data.

Chapter 1 systematically analyses the long-term dynamics of mortality reductions in London from the mid-19th to the mid-20th century. This period, known as the "mortality transition" in industrialised countries, saw an unprecedented substantial reduction in death rates—London's annual crude death rate fell from approximately 24 per thousand in 1840 to 13 per thousand by the 1930s. Despite the extensive studies documenting the decennial changes in mortality and the fundamental causes (see e.g., Dyson, 1996; Canning, 2011), the shape of the underlying dynamics remains understudied. This chapter addresses this research gap. To investigate these dynamics, I constructed a novel dataset tracking four mortality measures in London from 1841 to 1964: crude mortality, corrected death rate (adjusted for age and sex composition), infant mortality, and non-infant mortality. The data were sourced principally from Medical Officer of Health reports, with additional data taken from the Registrar General's reports and censuses. Although the source data are publicly available, some have not been accurately digitised and compiled comprehensively. The highly frequent (annual), long-term, and continuous mortality data reveal an S-shaped decline curve, characterised by an initial period of slow reductions before faster reductions and ultimately a slow-down in improvements. This sigmoid pat-

tern appears consistently across all mortality measures, suggesting the appropriateness of a four-parameter logistic model for analysis. The model provides informative parameters capturing key features of mortality dynamics, most notably an inflection point around 1896 for crude mortality, marking the point of most rapid mortality reduction. I then present an economic model of the diffusion dynamics of health progress with an underlying log-normal income distribution. This model successfully explains the observed sigmoid pattern—the uneven adoption of new health technologies across London's income distribution shaped the characteristic S-shaped decline in mortality rates during this period.

Chapter 2 expands the analysis of mortality dynamics to constituent areas in London, and examines the evolution of mortality inequality across these areas from the 1850s to the 1950s. Despite the consensus that there were significant differences in mortality between socioeconomic groups, there is debate surrounding the dynamics of mortality inequalities (see e.g., Antonovsky, 1967; Link and Phelan, 1995). This investigation requires a new panel dataset. The first challenge was identifying stable geographic units that persisted long enough for analysis to capture dynamics. By reconciling various administrative divisions, including Registration Districts, Parishes, Districts, Sanitary Districts, and Metropolitan Boroughs, I created consistent geographic boundaries that span nearly a century. This overcomes a key limitation in existing research, where changing administrative boundaries typically restrict analysis to shorter periods. Second, constructing three mortality measures: crude mortality, infant mortality, and non-infant mortality using data from municipal archives, parish records, and the National Census, I found that the sigmoid pattern demonstrated in Chapter 1 also broadly applies to the small areas, but that there is substantial variation regarding the period of fast reductions, marked by the estimated inflection points. To examine how these variations in mortality dynamics related to socioeconomic conditions, we revisit the model introduced in Chapter 1 and discuss its predictions with respect to the inflection point by socioeconomic conditions. In addition to predicting the sigmoid pattern, the model presented in Chapter 1 also predicts that areas with a higher proportion of higher-income residents would experience the period of rapid mortality reduction earlier, suggesting an earlier inflection point in trend mortality. Therefore, this framework suggests an increase in mortality inequality before this decreased. This prediction generates a clear testable empirical relationship:

the relationship between socioeconomic conditions and the inflection point should be negative, indicating initial divergence, rather than positive, which would suggest immediate convergence as soon as the mortality transition starts. I tested this using an extension of the logistic model that captures how initial conditions affect each parameter, specifically examining whether initial socioeconomic conditions significantly influenced the timing of the transition. The results confirm that populations with higher socioeconomic status experienced earlier mortality reductions, likely due to earlier adoption of public health improvements. For example, a 1% higher proportion of individuals in professional classes experienced an inflection point in mortality declines around a year and a half earlier. This timing differential suggests that the mortality transition saw a decades-long divergence in mortality rates across socioeconomic groups before eventual convergence, rather than immediate convergence.

Taking the first two chapters together, the mortality transition in today's developed economies demonstrates how differential access to health innovations perpetuated and amplified health disparities—an analytical lens through which we observe how contemporary developing economies, in their epidemiological transitions, risk exacerbating health inequalities absent interventions targeting socioeconomic disparities.

The epidemiological transition described by Omran (1971) reveals a shift in mortality patterns: while infectious diseases (including diarrhoea and tuberculosis) accounted for 50% of all deaths in England and Wales in 1860, with heart disease and cancer contributing only 3%, this ratio had reversed a century later, with infectious diseases declining to 15% and non-communicable diseases rising to 46%. Building on this historical context of evolving health challenges, the last chapter of this thesis turns to a contemporary factor of non-communicable disease risk: physical activity.

Chapter 3 examines how education and household income influence physical activity patterns among working-age adults in the UK between 2015 and 2019, using data from the Understanding Society survey. The analysis contributes to the literature on both socioeconomic determinants and physical activity outcomes. Education and income warrant separate investigations as they affect physical activity through different channels. Education shapes activity patterns through cognitive and psychological pathways while reflecting

broader social class dynamics. Income, particularly disposable household income—which UnSoc allows us to distinguish from gross income—directly captures purchasing power and consumption possibilities. Understanding their relative impacts is crucial for policy design, as interventions targeting education versus income require different approaches (Chevalier et al., 2013). I employ an ordered logit model to examine three distinct physical activity levels: zero physical activity, engagement in at least some physical activity, and meeting WHO-recommended guidelines. This classification is analytically important for two reasons. First, the effects of education and income levels vary distinctly across these physical activity thresholds. Second, each activity level independently generates differential health outcomes. The estimated odds ratios reveal three key insights—a) while more educated and affluent individuals are more likely to engage in physical activities and meet recommended PA guidelines, the income effects are modest—upgrading from GCSE-level to degree-level education multiplies the odds by 1.25. To achieve an equivalent effect through income alone would require a four-and-a-half-fold increase in household disposable income—an enormous increase that highlights the substantial impact of education relative to income. Therefore, educational interventions may therefore serve as a more effective policy lever than financial incentives for promoting physical activity. b) household disposable total income demonstrates double the impact of gross labour income on PA, underscoring the importance of precise income measurement for the effectiveness of redistributive policies. c) gender differences emerge clearly: education exhibits stronger associations with women's physical activity levels, while household income demonstrates more pronounced effects on men's physical activity.

In this thesis, each chapter is intended to be, as far as possible, self-contained. Each chapter thus contains its own literature review, description of the data and analysis methods, acknowledging links with earlier chapters as appropriate. All references are provided in a single reference list at the end of the thesis to comply with thesis formatting requirements at the University of Glasgow.

Drawing from this thesis, socioeconomic inequality serves as a fundamental determinant of health outcomes. Through both historical and contemporaneous channels, it shapes health variations across populations. Therefore, the examination of the persistent effects of socioeconomic conditions across temporal horizons proves critical for comprehending their multifaceted implications for population health.

Chapter 1

Mortality Dynamics in London During the Mortality Transition and a Model of the Diffusion of Health Progress

Abstract

London's mortality transition—marked by a substantial decline in death rates from the mid-19th to mid-20th century—provides a crucial case study of mortality dynamics in the first industrialised society. Despite the extensive research on the drivers and consequences of the mortality transition, there remains a lack of systematic understanding of mortality trend dynamics, and few studies have attempted to determine the precise timing of the transition. I constructed a novel dataset tracking four mortality measures in London from 1841 to 1964: crude mortality, corrected death rate (adjusted for age and sex composition), infant mortality, and non-infant mortality. The annual frequency and continuity of the mortality data enable the modelling of mortality dynamics, providing a comprehensive view of the transition's temporal evolution rather than discrete snapshots. Observing S-shaped transitions from high to low death rates, I employed a four-parameter logistic model, which demonstrates a good fit across all mortality measures and withstands rigorous statistical validation of standard errors. The notable similarity between crude and corrected mortality patterns shows that crude mortality is an appropriate measure of mortality during the period. One of the estimated parameters, the inflection point, which occurred around 1896 as measured by crude mortality, indicates both the mortality transition's midpoint and the timing of the fastest declines. It reveals that infant mortality decreased approximately two decades after crude mortality, suggesting that infant deaths were not the primary driver of London's mortality transition. I present a theoretical framework that demonstrates how socioeconomic inequality shaped the sigmoid pattern of mortality declines through the uneven diffusion of new health technologies across London's log-normal income distribution during this period. Further implications will be tested in the next chapter.

1.1 Introduction

During the second half of the 19th century and the early decades of the 20th century, technological progress in industrialised countries significantly alleviated the effects of frequent famines and enhanced overall health. For example, in London, the largest city in the UK, the annual crude death rate fell from around 24 per thousand persons in around 1840 to about 13 per thousand in the 1930s, without further appreciable reductions in the following decades. During this period, infectious diseases such as cholera and measles were brought under control, and chronic diseases such as heart disease and cancer became the predominant causes of death gradually. Omran (1977) coined the term "epidemiological transition" to describe this characteristic trend, while the general large decline in mortality is referred to as the "mortality transition." Marking a new era in human history, the mortality transition catalysed population welfare and well-being in these industrialised countries (e.g., the UK, the US, France) in the century that followed. As Deaton (2013, p. 24, 59) articulated, "health is the obvious starting point for an enquiry into well-being", and one crucial dimension of health is "the simple fact of being alive or dead". In England, life expectancy at birth increased from 32 years in the 1720s to 50 years in 1906, reached 60 years in 1930, and climbed to 77 years by 1996 (Galor, 2005). The increase in life expectancy had far-reaching effects, driving population growth while enhancing workforce productivity and economic development (e.g., Acemoglu and Johnson, 2007; Preston, 1975; Fogel, 1994; Bloom et al., 2003). Therefore, this historical episode has been the focus of extensive research over several decades, analysing mortality rate reductions in countries like the UK, France, and other today's developed economies while identifying the key contributing factors (see e.g., Antonovsky, 1967; McKeown et al., 1972; Szreter, 1988; Fogel, 1997; Deaton, 2013).

The initiation of the mortality transition requires advancements in public health, agricultural productivity driven by technological development, and education to promote individual awareness of health-promoting preventive measures such as nutrition and hygiene (Canning, 2011). Consequently, advancements in wealth stimulate the mortality transition, with the timing of this transition reflecting improvements in population welfare. In industrialised countries such as the UK and France, early access to life-saving meas-

ures, including vaccinations, drugs, and clean water, enabled the onset of the mortality transition as early as the 1860s (Lindert, 2000). In contrast, sub-Saharan countries did not witness major changes until a century later (Garenne and Gakusi, 2006). Therefore, historians regard the Industrial Revolution as the trigger for the "Great Divergence"—Britain and other northwestern European countries began to significantly surpass the rest of the world and the disparity continues to exist today (Deaton, 2013, p. 4).

Despite the extensive studies measuring the mortality transition and exploring its fundamental causes, few have attempted to understand the shape of the long-term underlying dynamics—a critical aspect given that this process spanned nearly a century and the timing of mortality reduction gains substantially influenced when their benefits materialised. I focus on the dynamics of mortality reductions during the mortality transition period in London, one of the most prosperous cities in the world in the 19th century.

To examine the dynamics of mortality, long-term time-series data spanning the period of mortality transition is necessary. First, I identified the Administrative County of London as the study area, given its stable boundary since 1889.[1] Then, through extensive archival research covering over 400 Medical Officer of Health (MOH) reports, I discovered that crude mortality data for London from 1841 to 1892 was visualised on a page in one of the MOH reports issued by the London County Council (LCC), with subsequent years' data reported in following LCC reports.[2] Building upon these crude mortality records, I expanded the dataset to incorporate additional mortality measures from the same period, with additional information meticulously collected from multiple sources and double-checked against the MOH reports of the LCC. While existing digitised collections provide valuable data—such as Populations Past,[3] which offers decennial data in census years, and other datasets that focus on specific mortality measures or shorter time periods—I have constructed a novel dataset incorporating four key mortality measures (crude death rate, infant mortality, non-infant mortality, and death rates corrected for age and sex compos-

1. The Administrative County of London corresponds to today's Inner London.
2. Some other MOH reports (issued by other local authorities) also include mortality for the County of London, but these sources provide only intermittent coverage rather than consistent annual records.
3. Source: Populations Past—Atlas of Victorian and Edwardian Population, The Cambridge Group for the History of Population and Social Structure (accessed December 2024), https://www.populationspast.org/imr/1871/#6/54.457/-4.098/bartholomew.

ition) for the Administrative County of London spanning 1841 to 1964, primarily sourced from annual MOH reports. This dataset uniquely covers the complete mortality transition period, extending beyond the beginning and end of the transition, which is essential to depict the dynamics.

I first conduct an empirical analysis of the dynamics in the reductions of four mortality measures for London, suggesting that these reductions follow sigmoid patterns. The sigmoid patterns are modelled using a four-parameter logistic model to estimate the S-shaped trend in mortality reductions, with a focus on the parameter that determines the inflection point, the year in which the trend changes convexity, and at which the decline is maximised. We selected and applied this modelling approach, using the four-parameter logistic model, to study mortality declines in our joint work (Angelopoulos et al., 2024) Therefore, the inflection point indicates the timing of fast declines during the mortality transition.[4] In London, the decline in crude mortality started around the 1850s, with a slow initial rate that picked up speed after the 1880s, culminating in an inflection point around 1896. After entering the 20th century, the pace of decline slowed, and crude mortality gradually transitioned to a new steady state at a much lower level before the Second World War. The dynamics of infant mortality also resemble the S-shaped curve, with the rapid decline, as measured by the inflection point, occurring around 1918, two decades later than for crude mortality.

Secondly, I analyse a mechanism that explains the emerging S-pattern. The key idea of this mechanism, which we developed and analysed in the joint work in (Angelopoulos et al., 2024), is that the S-shape pattern results from the technological diffusion of health progress, a process which depends on the distribution of income. Motivated by a large literature that studies diffusion dynamics arising from technological innovations (see e.g., Rogers, 1962; Stokey, 2021), our model predicts that the dynamics of the death rate during the mortality transition result from the diffusion of technological change across a population that differed in income, fueled by improvements in health and living conditions. In the model, agents decide whether to adopt new health technologies to lower

4. We developed and applied this modelling approach to different geographical areas in the joint work (Angelopoulos et al., 2024). In this chapter, I focus on London, while the next chapter examines its constituent areas, incorporating additional data I collected as a part of this thesis.

their probability of dying based on their annual personal income. Given a right-skewed log-normal income distribution in 19th-century London (Williamson, 1980), our model predicts sigmoid dynamics for the diffusion of health technology adoption rates, aligning with the empirical patterns of mortality decline.

The rest of the chapter is structured as follows: Section 1.2.1 introduces the theory of mortality transition and explores how progresses in health technologies drove this transformation, with particular emphasis on health improvements in London during the latter half of the nineteenth century in Section 1.2.2; Section 1.2.3 details the construction of a novel mortality dataset for London spanning the 1840s to 1960s, outlining data sources and defining four mortality measures; Section 1.2.4 presents the four-parameter logistic model to capture observed mortality trends; Section 1.2.5 analyses the estimated sigmoid mortality patterns at the city level; Section 1.3 develops an economic model of the diffusion dynamics of health progress, interacting with the underlying income distribution, and analyses its implications for the pattern of mortality declines; Section 1.4 summarises the key findings, situates them within the existing literature, and demonstrates how the proposed theory adequately explains the sigmoid mortality dynamics while acknowledging alternative theories.

1.2 Mortality Dynamics During the Mortality Transition

1.2.1 The Mortality Transition

This section introduces the mortality transition theory, examines its three major drivers—nutrition, education, and public health infrastructure, and demonstrates how earlier mortality transitions, beginning in the 18th and 19th centuries, led to significant health improvements.

The "epidemiologic transition theory" proposed by Omran (1971) describes the evolution of population change by emphasising the fundamental role of mortality patterns. For example, England and Wales, Japan, Ceylon, and Chile saw concomitant population growth and secular decline of mortality from the early 19th century to the late 20th century (Omran, 1971). One of the most important characteristics of the epidemiologic transition is the sharp reduction in communicable diseases and the gradual dominance of "degenerative and man-made diseases"[5] (Omran, 1971; Bhopal, 2016), which induces the overall decline in mortality. As Omran (1971) documented, in 1860, infectious diseases (including diarrhoea and tuberculosis) accounted for 50% of all deaths in England and Wales, with heart disease and cancer contributing to merely 3%. A century later, the scenario reversed, with the former dropping to approximately 15% and the latter escalating to 46%. This transformation resulted in a crude mortality rate decline of about 10 per 1,000 population during this century—McKeown et al. (1972) suggests that at least two-thirds of the mortality reduction in the first half of the 20th century can be attributed to the decline in deaths from infectious diseases.

The mortality transition marks an overall progress in public health throughout human history. Although the mortality transition in developing countries, facilitated by direct aid provided by international organisations, is largely exogenous, this was not the case in the 18th and 19th centuries in today's developed countries, where technological innovations catalysed the mortality transition (Dyson, 1996; Canning, 2011). Prior to the epoch of industrialisation and sustained economic growth, famines periodically hindered consistent mortality decline. Fogel (1997); Dyson (1996); Galor (2011) argued that throughout the ages, better living conditions and increasing population size inevitably led to pressures on agricultural wages per acre of land, causing starvation and subsequently, an increase in the death rate pushed the population back towards equilibrium. This phenomenon is called the "Malthusian trap". For example, from 1540 to 1820, the crude death rate in England fluctuated around 25–30 per 1,000 population, without any significant declines (Wrigley

5. Degenerative diseases are often due to the deterioration or damage of tissues or organs and are commonly seen in older populations. Man-made diseases are directly or indirectly caused by human activities like environmental pollution and lifestyle choices. Appropriate labels for the group of causes of death are further discussed by Mackenbach (1994).

and Schofield, 1989). According to Hinde (2003), the overall mortality experienced three phases—a definite but modest decline in mortality between 1780 and 1830; the decline stopped during the mid-nineteenth century; a decisive period of decline during the 1870s. I focus on this latter period of "decisive" decline.

Technological progress changed the centuries-lasting situation and drove an escape from the mortality trap through multiple channels. Thomas McKeown, in a series of papers and books published between 1955 and 1988, highlighted that, in a first step, the leap in agricultural technology boosted nutrition and caloric intakes (see e.g., McKeown et al., 1972; McKeown, 2005). Through the analysis of specific causes of death, McKeown and Record (1962, p. 110–119) argued that due to the minimal impact of effective environmental measures on exposure to airborne diseases before the 20th century, the great decline in deaths from airborne diseases[6] was largely attributed to an enhanced capacity, at the population level, to resist infection, driven by nutritional improvements. However, McKeown's hypothesis is contested. One of the major critiques concerns the differing impacts of pre-existing nutritional status on various infectious diseases (Conferees, 1983). While there were notable decreases in respiratory tuberculosis, typhoid, and diarrhoea, Szreter (1988) also noted significant declines in smallpox and scarlet fever, which have minimal links to nutrition, thereby casting doubt on the validity of the nutrition hypothesis. The critics disputed McKeown's categorisation of scarlet fever as airborne and argued that he exaggerated the role of pulmonary tuberculosis—these led to an overestimation of the airborne diseases and an underestimation of the water- and food-borne diseases (Harris, 2004). Recent research has utilised more comprehensive data to analyse nutritional impacts. For example, Schneider (2023), analysing rich microdata from the Foundling Hospital in London from 1892 to 1919, reported no influence of nutritional status on the morbidity rates for five infectious diseases (measles, mumps, rubella, chickenpox, and whooping cough), although for the duration of illness, a statistically significant effect of nutrition was found for measles and mumps. Secondly, the higher education levels demanded by the industrialised labour market contributed to improving overall health conditions (for the spread of schooling in history, see e.g., Goldin, 1998; Carpentier, 2003). The large

6. Airborne diseases include respiratory tuberculosis, bronchitis, pneumonia, scarlet fever, etc.

and positive correlation between education and health has gained consensus (see e.g., Ross and Wu, 1995; Grossman, 2017; Davies et al., 2018). In the historical context, for example, Lleras-Muney (2005) used compulsory education laws from 1915 to 1939 in the US as a quasi-natural experiment and concluded that an extra year of education lowers the probability of dying in the next 10 years by at least 3.6%. In addition, a panel of male twin pairs drawn from linked US census and death records in the first half of the 20th century supports the causal interpretation between education and mortality—an additional year of schooling extends the age at death by 4.8 months (Halpern-Manners et al., 2020). One of the possible mechanisms is the effect of education on health behaviours, including improved health practices and hygiene (Grossman, 2006; Sanchez et al., 2002). This first requires an understanding of the mechanisms of disease onset, such as the discovery of bacteria and infectious organisms, which is closely related to the third channel through which technological progress initiated the mortality transition. The third explanation of the mortality transition is the role of medicine, health technology, and the construction of modern public health infrastructure such as water sanitation (Angelopoulos et al., 2024). For example, exposure to midwifery licensing law at birth during the late 19th and early 20th century in the US[7] was associated with a 2.5% reduction in cumulative mortality rates over the following century (Noghanibehambari and Fletcher, 2023). The role of medical care innovations was contentious. Some believed that apart from smallpox vaccination, medical innovations had minimal impact on the recession of the plague and many other pandemics in Europe (Omran, 1971; McKeown et al., 1972). On the contrary, Canning (2011) emphasised the significance of developments in health knowledge, which have driven progress in the prevention and treatment of infectious diseases over the past three centuries. According to Mackenbach et al. (1988), from 1950 to 1984 in the Netherlands, medical care innovations contributed to mortality declines, without which the gains in life expectancy would shrink by one year in males and four years in females. Moreover, the timing of the mortality transition also aligns with the large-scale construction of sewage systems, driven by public legislation, which significantly aided in providing clean water and disposing of sewage (Hardy, 1984; Cicak and Tynan, 2015). The water-related public

7. Under these reforms, midwives were mandated to complete formal training, education, and gain experience, as well as adhere to a set of hygiene protocols, in order to acquire a midwifery license and legally practice midwifery (Noghanibehambari and Fletcher, 2023).

infrastructure effectively reduced mortality from intestinal infections which were spread through contaminated water and food from the 1880s (McKeown and Record, 1962). In particular, typhoid which is mainly transmitted through drinking water contaminated by the faecal wastes of infected people is closely associated with the quality and coverage of sewage systems (Beach et al., 2016). According to Hinde and Harris (2019), in England and Wales from the 1860s to the 1880s, mortality rates from typhoid and typhus were reduced by two-thirds, confirming the effectiveness of the sewage system.[8] However, this conclusion is not universally applicable. In the US between 1910 and 1930, water filtration led to a 15% reduction in mortality from diarrhoea among children under two for most of the year, except during the summer months (Anderson et al., 2020). Similarly, treating sewage and establishing bacteriological standards for milk did not alleviate the incidence of summer diarrhoea (Anderson et al., 2019). Aidt et al. (2023) provided an explanation—flush toilets did not become widespread in the UK until 1911, much later and more gradually than the supply of clean water. This implies limitations in the early sewage systems regarding waste disposal. See Section 1.2.2 for more information about health reforms in London.[9]

Large health progress during the period of mortality transition has been broadly documented. Western countries, today's developed countries, experienced the mortality transition starting from the 18th and 19th centuries (Lindert, 2000) while sub-Saharan countries saw main changes after a century (Garenne and Gakusi, 2006). As elaborated by Cervellati and Sunde (2015), the timing of the onset of the economic and demographic transition plays a crucial role in explaining the variations in development among different countries. In Lanciego, Spain, the 1890s marked the beginning of a clear and irreversible reduction in mortality rates, falling from 30 per 1,000 inhabitants to a third a century later (Sanchez et al., 2002). Wolleswinkel-Van den Bosch et al. (1997) differentiated causes of death into several clusters and documented that the vast majority of infectious diseases declined rapidly in the late 19th century in the Netherlands. After 1875, the total mortality rate decreased annually by around 1.1%. In Italy, the average life expectancy rose

8. For historical effects of clean water and sewage systems on health in France and US, see Kesztenbaum and Rosenthal (2017); Costa and Kahn (2015).
9. See Harris and Helgertz (2019) for a collection of research on the effect of urban sanitation measures on mortality.

from 37 years in 1887 to over 50 in the 1920s although WWI and the pandemic in 1918 interrupted the progress significantly (Caselli and Egidi, 1991).[10] However, although contemporary data has been broadly used to study health dynamics empirically (see e.g., Contoyannis et al., 2004; Haan and Myck, 2009), historical papers typically prioritise meticulous data description and documentation over modelling and elucidating the underlying dynamics of the mortality transition. The few studies have primarily examined the patterns of seasonal interdependence and secular trends during the period of the mortality transition using the Autoregressive Integrated Moving Average (ARIMA) models (see e.g., Land and Cantor, 1983; Munoz-Tuduri et al., 2006).

1.2.2 Health Progress in London

London is a good laboratory for studying the mortality transition. During the mortality transition phase, the UK, particularly London, became a pioneer of modern public health reforms, exerting a profound influence on other industrialised countries. The Sanitary Movement in Great Britain was notably marked by the 1842 publication of Sir Edwin Chadwick's *Report on the Sanitary Condition of the Labouring Population of Great Britain* (Wray, 2015). In the following half-century, the state became the guarantor of standards of health, leading to the enactment of numerous health reforms, as illustrated in Figure 1.2.1.

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10. Also see Condran and Cheney (1982); Hemström (1999); Carson et al. (2006).

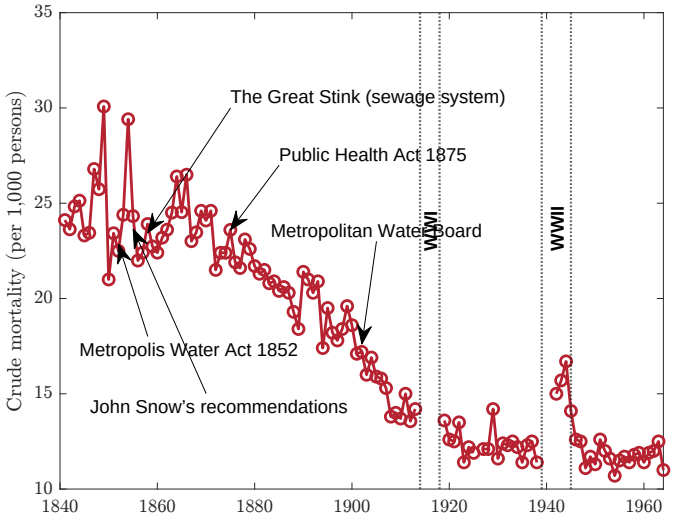

Figure 1.2.1: **Observed crude mortality and important public health reforms in London, 1841–1964.** The movement of crude death rate per 1,000 population (red line) and five significant public health-related issues and reforms implemented in London during the period of the mortality transition from 1841 to 1964. The construction and definition of the crude death rate are in Section 1.2.3.

Chadwick's report in 1842 catalysed the passing of the Public Health Act in 1848, which set up local boards of health tasked with appointing health officers, constructing sewer systems, and inspecting the quality of food available for sale. Firstly, the improvement of water quality underwent a prolonged exploration process. The Metropolis Water Act of 1852 was passed to regulate water supply companies in London—it mandated that water companies cease extracting water from the increasingly polluted Thames River and required them to filter the water before distribution (Cicak and Tynan, 2015). The Metropolis Local Management Act 1855 established the Metropolitan Board of Works (MBW), which replaced eight private water companies in 1904 (Hardy, 1984). Furthermore, in response to the Great Stink of the hot summer in 1858,[11] Sir Joseph Bazalgette (chief engineer of the MBW) initiated the construction of a comprehensive sewerage system for central London in 1859, which included an extensive network of pipelines under the streets of London and along the banks of the Thames to take sewage away from the river. The construction of the three river embankments was completed in 1864, and the sewer pipes were fully installed by 1875. The Public Health Act 1875 gave the local authorities the power to mandate the connection of residences to the principal sewerage network and

11. In summer 1858, London faced a severe sanitation crisis called the Great Stink. The combination of extreme heat and the Thames River, which served as an open sewer, created an overwhelming stench throughout central London.

prohibited the construction of new dwellings lacking this essential connection. In addition to policies, between 1875 and 1885, advancements in water filtration technology led to improvements in water quality, while innovations in the flushing mechanisms of water closets enhanced domestic sanitation (Wray, 2015).

The contribution of public health reforms in this period is generally validated by the substantial drops in the prevalence of several major infectious diseases in London. The most water-dependent diseases, such as cholera and dysentery, had experienced a marked decline by the mid-1880s. This decline underscores the effectiveness of measures taken to provide clean drinking water, including filtering river water and installing public standpipes (Aidt et al., 2023). The correlation between the stock of sewerage capital and the decrease in mortality rates became significant after 1880, while this pattern is not observed in the period from 1845 to 1884, which is hypothesised to be the timeframe before the impacts of the reforms were genuinely felt. According to Chapman (2019), between 1891 and 1900, the district which spent 6.6 pounds per capita on sanitation public goods (Hull Registration Districts) witnessed a rise in life expectancy; in contrast, Sunderland, with a lower investment of 3.2 pounds per capita, experienced stagnation in health improvements.[12]

Beyond the water-related reforms, several other interventions also led to notable advancements in health. The introduction of the Vaccination Act in 1840, which offered free vaccinations to the impoverished, marked a pivotal point. This was further reinforced by the 1853 Vaccination Act, requiring vaccinations for all infants under three months, a mandate expanded in 1867 to include children up to 14 years of age. Despite facing resistance from those who deemed vaccinations "un-Godly", resulting in potential fines or incarceration for non-compliance, these acts represented critical steps forward in disease prevention. Additionally, environmental health saw advances with the enactment of the Alkali Act in 1863, aimed at reducing harmful industrial emissions, such as gaseous hydrochloric acid, from the alkali industry. This period also witnessed the first efforts to combat food adulteration through the Food and Drugs Act of 1860, targeting the inclusion of hazardous substances in everyday food items.

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12. Hull was a registration district on the eastern coast of England, and Sunderland was a registration district in the northeast of England.

The nineteenth-century London saw numerous housing-related legislations. The 1844 London Building Act established fresh regulations aimed at enhancing housing quality, including revised standards for materials, the height of storeys, and the thickness of walls. Additionally, it mandated minimum dimensions for backyards, set alleys to have a minimum width of 20 feet, and required streets to be at least 40 feet wide or as wide as the tallest building on the street, whichever was greater. Over the next 14 years, the spirit behind these rules was extended nationwide. For example, the 1858 Public Health Act suggested further specifications, setting the minimum street width at 36 feet, mandating at least 150 square feet of space behind each house, and establishing a minimum gap of 10 feet between buildings at the rear, etc. In addition to enforcing connections to the mains sewerage system, the Sanitary Act of 1866 also set limits on cellar dwellings and established the definition of "overcrowding".

Beyond these legislative improvements, economic development brought substantial changes in nutrition. According to evidence from agricultural labourer families across England, while caloric intake showed no improvement between 1787–1796 and 1835–1846, this stagnation ended in the following decades. Calorie consumption peaked in the 1860s, coinciding with the "golden age of British agriculture" (Gazeley and Horrell, 2013). This nutritional improvement during the mid-Victorian period, resulting from the agricultural revolution, railway expansion, and economic development, led to higher life expectancy and lower incidence of degenerative diseases (Clayton and Rowbotham, 2009).

Reforms also addressed labour conditions and education. The Mines and Collieries Act of 1842 prohibited the employment of women and young girls in underground mines and set a minimum working age. The Elementary Education Act 1870 established school boards to build and manage schools in England and Wales, marking the first comprehensive attempt to achieve universal education. The Education Act 1876 went further by establishing School Attendance Committees to promote attendance and assist impoverished families with school fees. By 1880, education became compulsory for children aged 5–10, and the 1891 Elementary Education Act finally made primary education effectively free (Gillard, 2024). In England and Wales, the share of individuals who were unable to sign their names fell dramatically, from around 35% for men and 50% for women in the 1840s to less than

1% for both genders by the early 20th century (Schofield, 1973). These reforms collectively contributed to a gradual but meaningful enhancement of public health, education, and living standards by the turn of the 20th century, illustrating a comprehensive approach to societal progress.

1.2.3 Construction of the Mortality Dataset

In this section, I describe the construction of the London Administrative County mortality dataset, which contains mortality data across four variables: crude mortality (1841–1964), infant mortality (1858–1964), non-infant mortality (1860–1964), and corrected death rate (adjusted for age and sex composition, 1851–1964). This dataset contributes to historical mortality research by providing annual, continuous data covering nearly a century that facilitates analysis of mortality dynamics, while encompassing four distinct mortality measures.

1.2.3.1 Data Sources

The primary source of mortality variables we use is the reports of the Medical Officer of Health (MOH). The MOH for London was established in 1848. Around 5,800 annual reports and 275,000 tables covering the Greater London area, including the present-day City of London, 32 London boroughs and the predecessor local authorities for these boroughs have been photographed and digitised using Optical Character Recognition (OCR) by the Wellcome Library's collections. The Administrative County of London, corresponding to Inner London today, was established as part of the Local Government Act 1888 and its local authority is the London County Council (LCC). From 1892 until its dissolution in 1965, the LCC issued annual MOH reports that included data for the entire Administrative County of London and its constituent areas, which enabled comparisons of health and sanitation performance across the city. The online sources were released in February 2021.[13]

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13. Source: London's Pulse: The Medical Officer of Health reports 1848–1972 (accessed March 2023), https://wellcomelibrary.org/moh/about-the-reports/about-the-medical-officer-of-health-reports/.

In addition to the MOH reports, we also obtained information through existing databases accessible through the UK Data Service:

(a) **`cause_lon_ann_corr.xlsx`**, Great Britain Historical Database (GBHD): Vital Statistics for England and Wales 1840–1911 (Southall et al., 2022). This dataset was transcribed from the Annual Reports of the Registrar General. The data covers population, number of deaths, and number of deaths from over 50 specific causes in different administrative areas in the County of London from 1860 to 1920. The totals for population, number of deaths, and deaths by causes for all small areas[14] are aggregated annually to calculate the city-wide figures.

(b) **`mort_age_ew_reg_ann.xlsx`**, GBHD: Vital Statistics for England and Wales 1840–1911 (Southall et al., 2022). This dataset was transcribed from the Annual Reports of the Registrar General for England and Wales. It includes the number of births and deaths of persons aged under 1 in the County of London for time periods 1840–42, 1850–52, 1860–82, 1890–92, 1900–02, and 1908–10.[15]

(c) **`vital_ew_lgd_ann.xlsx`**, GBHD: Vital Statistics for England and Wales 1911–1973 (Southall and Mooney, 2022). This dataset was transcribed from the Annual Reports of the Registrar General.[16] It includes the number of births, number of infant deaths and infant mortality for small areas in London from 1911 to 1973. The totals for the number of births and infant deaths for all constituent areas are aggregated annually to calculate the city-wide figures from 1911 onwards.

(d) **`vital_ew_reg_ann.xlsx`**, GBHD: Vital Statistics for England and Wales 1840–1911 (Southall et al., 2022). This dataset was transcribed from the Annual Reports of the Registrar General. It includes the number of births among other demographic variables for the County of London.

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14. Here small areas refer to Registration Districts, sanitary districts, and Met.B.s, which vary across time. see detailed information in Section 2.3 in Chapter 2.
15. We do not use data for years before 1850. The number of births is not available before 1850.
16. Data from 1921 onwards are taken from the Registrar General's Statistical Review of England and Wales.

1.2.3.2 Mortality Measures

In this section, I describe the measures of mortality used in this chapter and their construction at the city level from the sources listed above. The main headline measure of mortality is the crude mortality rate (per 1,000 persons), which is available for the greatest number of years. I also construct infant mortality rates to examine whether they display the same dynamic patterns as crude mortality. To justify the robustness of crude mortality dynamics, I compute two additional measures: non-infant mortality rates to determine whether the crude mortality decline was solely driven by changes in infant mortality and corrected death rates to verify whether crude mortality patterns persist after controlling for demographic composition.

1.2.3.2.1 Crude mortality

The crude mortality rate is computed as the number of deaths divided by the living population. In this thesis, it is scaled such that crude mortality is defined as the number of deaths per 1,000 persons. For the period 1841–1892 inclusive, it is sourced from Diagram III, image page 26, Report of the Medical Officer of Health for London County Council (LCC) 1892.[17] For the years from 1892 to 1964, crude mortality and population figures were manually collected from the Medical Officer of Health (MOH) reports for LCC in each respective year.

1.2.3.2.2 Infant mortality

Infant mortality is defined as the number of infant deaths (deaths of children under 1 year) per 1,000 births. Where possibly, Database (c) was used to obtain the infant mortality rate, relying on annual MOH reports to complete the data series, and for the period from 1911 onwards. In Database (c), which covers the time periods 1840–42, 1850–52, 1860–82, 1890–92, 1900–02, and 1908–10, we keep `` `births' ``—total births; `` `i_death' ``—deaths under 1 year, and `` `i_rate' ``—death rate among infants under 1 per 1,000 births. City-

17. The diagram shows the mean death rate from 1841 to 1892 and the yearly percent deviations from this mean. These deviations are converted into absolute death rates.

level births and infant deaths are first determined by summing the annual statistics for all constituent areas. Then, infant mortality is calculated as `` `i_death'/`births' ``$\times 1,000$. Data gaps between 1892 and 1910, as well as data for subsequent years, were manually compiled from the MOH reports for the LCC for each respective year.

1.2.3.2.3 Non-infant mortality

Non-infant mortality is defined as the number of non-infant deaths above the age of 1. Here, this is expressed as 1,000 living persons above 1, which is calculated as:

$$\text{non-infant mortality} = \frac{\text{crude mortality} - \frac{\text{number of infant deaths}}{\text{population}}}{1 - \frac{\text{number of births} - \text{number of infant deaths}}{\text{population}}},$$

in which all elements have been obtained as discussed above. Specifically, population before 1892 is obtained from Database (a); number of births is obtained from Database (d) and Database (b), with missing values filled by using the MOH reports; number of infant deaths is obtained from Database (b) for the period before 1911 and Database (c) for period after 1911, with missing values completed using the MOH reports.

1.2.3.2.4 Corrected death rate

In addition to what the MOH report refers to as the "recorded death rate", now known as the crude death rate, they also report the "corrected death rate" from 1892 until WWI. The MOH explains that this is approximated based on the age and sex distribution of London at the last census and the death rate for each sex at each age period in England and Wales in the last decennium (Page 9, Report of the Medical Officer of Health for London County Council (LCC) 1892). To model the dynamics, I constructed a novel dataset by applying their calculation method to a long time period 1851–1964, as follows:

1. Collect data on the age and sex distribution of the population of London in each census year from 1851 to 1931;[18]

18. Data source: Census reports, Population, England and Wales, 1851 to 1931, http://www.histpop.org.

2. Collect data on the average age and sex distribution of the population, along with the average number of deaths within each age and sex group in England and Wales for each decade from 1851–1860 to 1931–1940;[19]
3. Using the data from the previous step, calculate the average death rate for each sex at each age period in England and Wales for each decade from 1851–1860 to 1931–1940, say y;
4. Applying 1 to 3 gives *standard death rate*, say x, i.e., a death rate which would be correct on the assumption that the rates of mortality at each age period and for each sex were identical with the mean rates in England and Wales in the last decennium;
5. Calculate the factor for correcting the recorded death rate as $\frac{y}{x}$;
6. Multiply crude mortality as discussed above by $\frac{y}{x}$ to get the corrected death rate.

Therefore, under the assumption that the rates of mortality at each age period and for each sex were identical with the mean rates in England and Wales in the last decennium, we can obtain the corrected death rates for London from 1851 to 1964.[20]

From the longer period of interest, 1851–1964, the LCC reports provide corrected death rates for the years 1892–1913. To validate our calculations, Figure 1.2.2 compares two sets of corrected death rates for the period 1892–1913: those we calculated by applying the MOH's methodology to our collected data as discussed above, and those originally reported in the MOH records. The values we calculated closely match those documented in the LCC reports (correlation= 0.994).

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19. Data source: supplement to Registrar-General's reports (England) for each decade from 1851–1860 to 1901–1910; the 20th Century Mortality Files from 1911–1920 to 1931–1940, https://webarchive.nationalarchives.gov.uk/ukgwa/20160111174808/http:/www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm:77-215593.
20. This application also assumes that the constitution of the population has remained unchanged during the past ten years, leading to some expected inaccuracies (Page 14, Report of the MOH for LCC 1910).

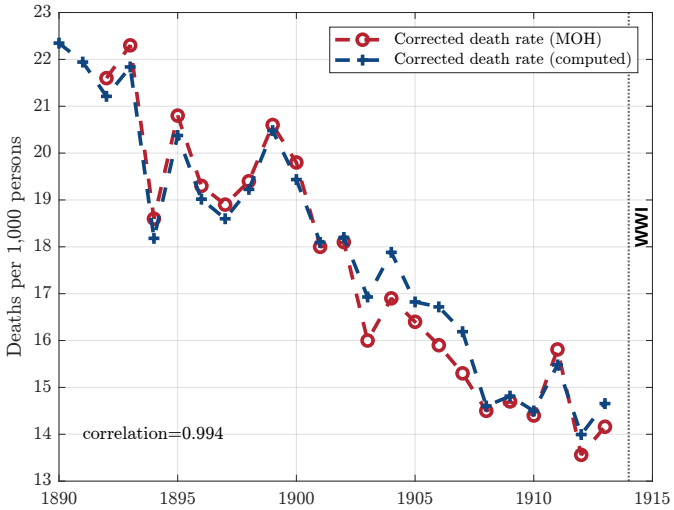

Figure 1.2.2: **Calculated vs. MOH-reported corrected death rates, London 1892–1913.** This figure plots the corrected death rates calculated using our collected data and the corrected death rates documented in the LCC reports from 1892 to 1913, the period for which the corrected death rate is available in MOH records. The values are very close, with only minor discrepancies observed annually.

Table 1.2.1 presents key mortality statistics for London from the mid-19th to mid-20th century, a pivotal period marked by significant public health reforms and the mortality transition. During this era, London experienced substantial declines across all mortality measures.

Table 1.2.1: Data availability and summary statistics of mortality measures, London

Variable	Available period	Min	Max	Mean	Std. dev.
Crude mortality (per 1,000 population)	1841–1964	10.70	30.08	18.01	5.24
Infant mortality (per 1,000 births)	1858–1964	21.00	172.48	109.28	55.47
Non-infant mortality (per 1,000 population above 1)	1860–1964	10.86	27.30	17.14	4.95
Corrected death rate (per 1,000 population)	1851–1964	11.20	31.43	19.43	5.27

1.2.4 Modelling Mortality Dynamics

In this section, I present the four-parameter logistic model used to analyse mortality dynamics in this chapter. Based on the mortality dataset constructed in the previous section, all four mortality measures exhibit an S-shaped transition from a high to low death rate equilibrium, making a logistic function a natural choice. This approach offers

valuable insights into key features of the mortality decline, including initial and final equilibrium levels, as well as the timing and speed of the fastest declines. In addition, I compare its fitted curve against a more flexible LOWESS trend estimation to validate that the logistic model does not impose unreasonable constraints on the shape of the fitted curve.

1.2.4.1 Sigmoid Mortality Dynamics

The mortality decline trend in Figure 1.2.1 exhibits an S-shaped transition from a high to a low death rate. This pattern motivates us to model the time series of the death rate using a four-parameter logistic function to depict the dynamic evolution and capture key features of the decline.[21] The logistic model we estimate is based on the *4-parameter logistic (4PL) model* or *Hill model* proposed by Hill (1910), which has been applied across various domains to model sigmoid dynamics. Examples include dose-response models (Gadagkar and Call, 2015), plant density-crop relationship (Mead, 1970) and population growth models (Pliška, 1987). Logistic models have also been used to describe, estimate and forecast the S-shape of the diffusion process of innovation (see classic studies of Griliches (1957); Mansfield (1961) and more recent applications in Meade and Islam (1998); Lotfi et al. (2014)). See detailed information regarding the theory of diffusion in Section 1.3.

In particular, defining $\overline{d}$ as the trend death rate during $t \in (-\infty, \infty)$,

$$\overline{d} = \delta_l + \frac{\delta_u - \delta_l}{1 + e^{\left(\beta(t-\tau_c)\right)}}, \tag{1.2.1}$$

where $\delta_u - \delta_l > 0$ and $\beta > 0$.

The four parameters are interpreted as follows:

1. The parameters δ_l and δ_u are the lower and upper asymptotes respectively.
2. The parameter τ_c is the inflection point at which the curvature of the trend-line changes sign, so the timing of the inflection point aligns with the maximum slope of the curve. It also corresponds to the year in which the death rate lies halfway between the upper and lower asymptotes.

21. The modelling of the mortality dynamics and results for London are also in the joint work (Angelopoulos et al., 2024).

3. Conditional on asymptotic mortality, β determines the maximum slope, i.e., at the inflection point.

See Appendix 1.5.1 for mathematical details of parameter interpretations.

We fit the four-parameter logistic model in Equation 1.2.1 to the mortality measure j in London during $t \in (1841, ..., 1964)$,[22] by applying Non-Linear Least Squares (NLS):[23]

$$d_t^j = \delta_l^j + \frac{\delta_u^j - \delta_l^j}{1 + e^{\left(\beta\left(t-\tau_c^j\right)\right)}} + u_t^j, \tag{1.2.2a}$$

where u_t is the error term measuring the discrepancy between the observed death rate and the fitted curve. The statistical inference of the four parameters $\boldsymbol{\Theta} = (\delta_l, \delta_u, \beta, \tau_c)$ requires careful consideration of the error structure in u_t.

Under standard assumptions, the error term is independently and identically distributed (*i.i.d.*) across time periods. We begin by relaxing the identical distribution assumption of the error term u_t to allow for heteroskedasticity (while maintaining independence across time periods), because the beginning of the period is characterised by larger fluctuations in the death rate, probably reflecting larger exposure of the population to random variations from the mean in the form of infectious disease outbreaks. In addition, improvements in public health over time also imply a lower probability of random shocks. Therefore, we calculate heteroskedasticity robust standard errors based on Davidson and MacKinnon (2004, p. 239); we use the HC3 variance estimator. See an explanation on the HC3 variance estimator in Appendix 1.5.2. This specification with heteroskedasticity-robust standard errors constitutes our baseline estimation.

Building upon this baseline heteroskedastic specification, we further relax the independence assumption by allowing for serial correlation in the error term. This extension accounts for the possibility that the shocks and other unobservable factors may persist over multiple periods. For instance, disease outbreaks can have mortality effects that span multiple years. To allow for this serial correlation, we model the error term in Equation 1.2.2a as

$$u_t = \rho u_{t-1} + \epsilon_t,$$

22. The range of t varies across different mortality measures.
23. To simplify notation, we omit superscript j in the presentation below.

where $\rho \in (-1, 1)$ and the idiosyncratic error ϵ_t is assumed to be independently distributed across time.[24] We then substitute u_t into 1.2.2a and estimate the parameters $\boldsymbol{\Theta} = (\rho, \delta_l, \delta_u, \beta, \tau_c)$ in the model

$$d_t = \rho d_{t-1} + \left[\delta_l + \frac{\delta_u - \delta_l}{1 + e^{\left(\beta(t-\tau_c)\right)}}\right] - \rho \left[\delta_l + \frac{\delta_u - \delta_l}{1 + e^{\left(\beta(t-1-\tau_c)\right)}}\right] + \epsilon_t. \tag{1.2.2b}$$

For both Equation 1.2.2a and Equation 1.2.2b, we estimate the parameters using the Gauss-Newton method to minimise the residual sum of squares, by employing the Stata package `nl` (Danuso et al., 1992; Royston, 1993). See an illustration of the Gauss-Newton method algorithm and the algorithm for uniqueness check of the solution in Appendix 1.5.2.

1.2.4.2 LOWESS Estimation

To demonstrate the appropriateness of the logistic model in capturing the shape of mortality decline, we compare the fitted curve with locally weighted scatterplot smoothing (LOWESS) on $\overline{d}$ in Equation 1.2.1, a non-parametric regression method (see also e.g., Costa and Kahn (2015) for an application using time series of death rates).

In LOWESS, the data consists of pairs $\left(t_k, \overline{d}_t(t_k)\right)$, where $t_k \in (1841, 1964)$. To fit a curve at a specific central point $\left(t_k, \overline{d}_t(t_k)\right)$, LOWESS considers a few data points surrounding it and assigns weights to the nearby data points based on their distances from this central point. The central point receives the highest weight, indicating its significance in the regression process, while other points receive decreasing weights as their distance from the central point increases.[25] Once the weights are assigned, LOWESS performs a weighted least squares regression model using the selected subset of data points. This procedure is

24. We again use the HC3 variance estimator to allow for heteroskedasticity in ϵ_t.
25. We apply the Stata command `lowess`, for which the weight function is the tricube weighting function $w(\kappa) = \left(1 - |\kappa|^3\right)^3$. $\kappa \in (0, 1)$ is the scaled distance of a given data point from the point on the curve being fitted.

repeatedly applied to each central point and ultimately a smooth curve that diminishes the impact of outliers is generated. The number of data points used is determined by the "bandwidth", the fraction of the total data points utilised in each local fitting. Following Costa and Kahn (2015), we use a bandwidth of 0.4.

The LOWESS-predicted trend mortality closely aligns with the solutions estimated using Equation 1.2.2a and Equation 1.2.2b, as illustrated in Appendix 1.5.3. Therefore, we conclude that the logistic specification successfully captures the underlying shape of mortality decline.

1.2.5 Analysis of City-Level Mortality Dynamics

In this section, I plot, describe and analyse the mortality dynamics at the city level for the four measures of mortality described in Section 1.2.3.2, which are fitted with the logistic model illustrated in Section 1.2.4.1.

Figure 1.2.3 plots four mortality measures for London between 1841 and 1964—crude mortality, corrected death rate (adjusted for age and sex composition), infant mortality, and non-infant mortality—with fitted logistic models overlaid. The respective inflection points are marked in blue and yellow respectively. As discussed in Section 1.2.4.1, the inflection point τ_c marks the middle point of the mortality transition. Prior to this year, the rate of decline in mortality was accelerating, and following this year, the curve shifted from concave to convex, indicating a slowdown in the rate of decline.

The first observation is that the data for all mortality indicators demonstrates significant declines during this period. For instance, crude mortality almost halved over a century from the 1840s to the 1960s, dropping from about 25 per thousand population to 12, while infant mortality reduced to one-eighth of its original rate over the century from the 1860s to the 1960s. Second, the trend of the corrected death rate resembles the trend of crude mortality, and the inflection points for both were around 1896, indicating that any changes in the age composition did not notably influence the mortality time series throughout that period. Third, the inflection point signalling the shift from concave to convex infant mortality trend occurred around 1919, approximately two decades after the transition was observed in crude mortality, suggesting that infant mortality decline was

not the primary driver of the early phase of the mortality transition. This finding from our systematic analysis of mortality dynamics contributes new insights to the historical literature, where comprehensive modelling of mortality transition patterns has been limited, as elaborated in Section 1.2. While direct studies of these dynamics are scarce, our results align with indirect historical evidence. For example, McKeown and Record (1962) documented the average decennial death rates in England and Wales in the last six decades of the nineteenth century, showing that while crude mortality began declining from 1861, infant mortality remained stagnant until the end of the 19th century—a pattern consistent with our finding of a delayed infant mortality inflection point. Davenport (2020) attributed the high mortality rate among young children in the mid-19th century to two factors: the high frequency and virulence of scarlet fever, and poor infant feeding practices (specifically wet-nursing and hand-feeding in urban areas). Similarly, Preston and Van de Walle (1978) found that in France's three largest urban areas (Seine, Rhŏne, and Bouches-du-Rhône), mortality improvements during the second half of the 19th century first occurred among ages below 15 or 20, notably excluding infancy, further supporting our observation of later mortality transition in infant mortality.[26]

More importantly, Figure 1.2.3 reveals a fundamental commonality across all mortality measures: they all follow a sigmoid (S-shaped) pattern of transition. As displayed in Table 1.2.2 and Figure 1.2.3, the four-parameter logistic functions Equation 1.2.2a and Equation 1.2.2b capture this S-shaped dynamic pattern with high precision (indicated by high R^2). Moreover, they provide very similar and statistically significant estimates, validating the two nonlinear functions in representing the time series of mortality transition in London. The four-parameter logistic model is further corroborated by Stewart (2024), who applied the model to Glasgow, England and Wales, and Scotland and found similar sigmoid patterns in the underlying mortality trends.

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26. In terms of mortality levels, several studies argue that the epidemiological transition led to the most profound changes in health and disease patterns among young females and children, as they are particularly sensitive to improvements in nutrition, hygiene, and water infrastructure (Dyson, 2010; Omran, 1971; Kesztenbaum and Rosenthal, 2017). While these scholars highlight the vulnerability of children during this transition, they did not specifically delineate the precise timing of infant mortality reductions.

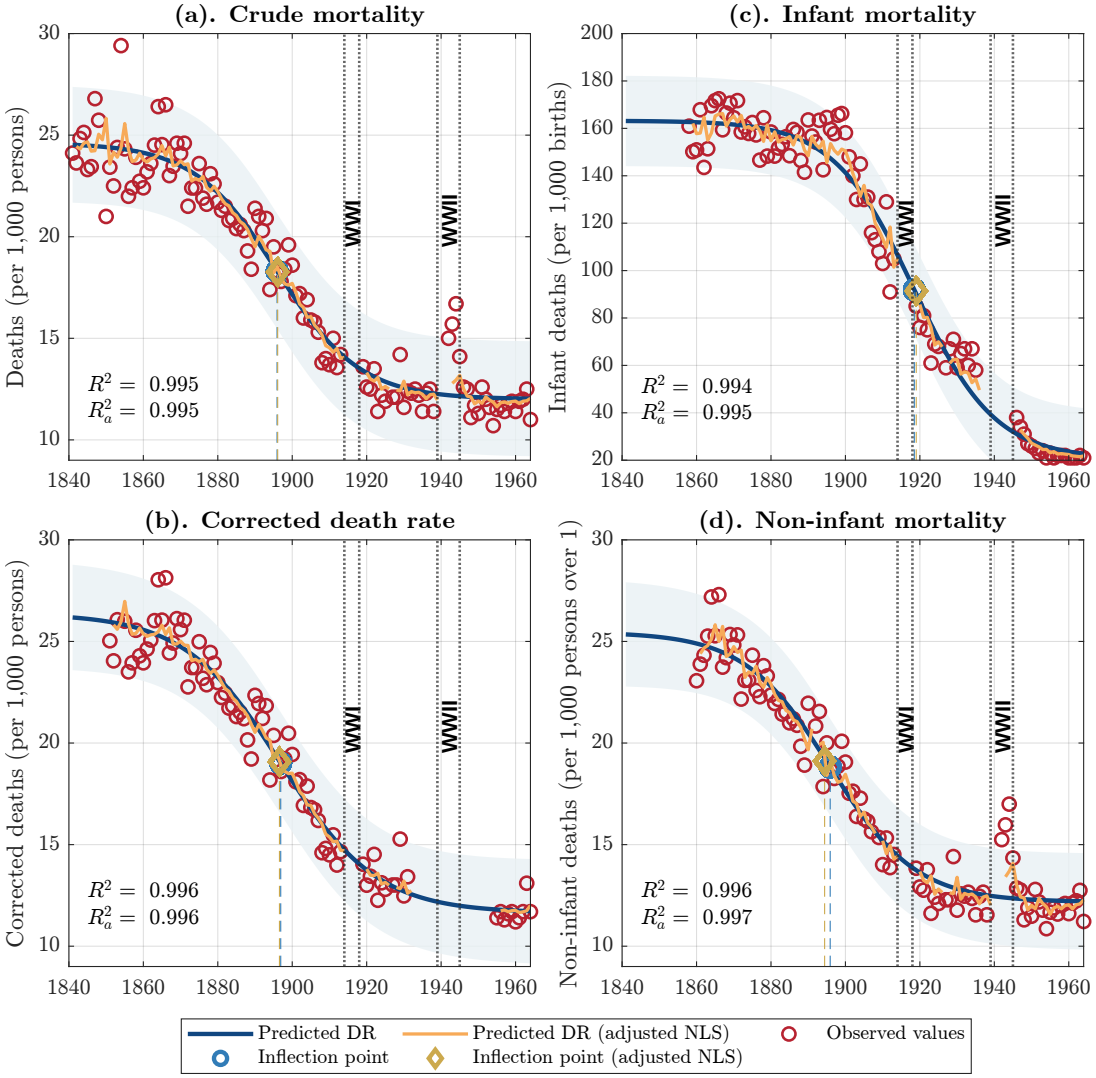

Figure 1.2.3: **Time series of four mortality rates for London.** Fitted logistic curves estimated by Equation 1.2.2a shown as blue lines and 95% prediction interval in blue shading; fitted logistic curves estimated by Equation 1.2.2b, which adjusts for serial autocorrelation, are shown as yellow lines. The corresponding inflection points are marked in blue and yellow respectively. Years corresponding to the First and Second World Wars are highlighted using vertical dashed lines, and R^2 values for the fit of the 4PL model (R^2) and the 4PL autoregressive model (R_a^2) are shown in the bottom left-hand corner of each plot.

Table 1.2.2: Estimated parameters of sigmoid trends for three mortality measures, London

	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
	Crude mortality		Corrected death rate		Infant mortality		Non-infant mortality	
ρ		0.235		0.195*		0.399***		0.395**
		(0.216)		(0.083)		(0.002)		(0.017)
δ_l	11.983***	11.852***	11.643***	11.666***	20.606***	19.194***	12.175***	11.914***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
δ_u	24.639***	24.690***	26.372***	26.491***	163.220***	163.598***	25.457***	26.316***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
β	0.088***	0.086***	0.077***	0.077***	0.092***	0.089***	0.087***	0.078***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
τ_c	1896.074***	1896.240***	1896.908***	1896.566***	1918.504***	1919.022***	1895.905***	1894.409***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
N	115	111	85	81	91	87	91	87
R^2	0.995	0.995	0.996	0.997	0.994	0.995	0.996	0.997

[a] The table shows the estimated parameters of Equation 1.2.2a in Columns (a) and Equation 1.2.2b in Columns (b) for four mortality measures. Standard errors are obtained using the heteroskedasticity-consistent covariance matrix (HC3) estimator (Davidson and MacKinnon, 2004, p. 200). p-values in parentheses, where * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

The four-parameter logistic function (Equation 1.2.2a) and the autoregressive variant (Equation 1.2.2b) contribute fundamentally to modelling mortality dynamics during the transition period. The key advantage of the models lies in their consistent, significant, and informative parameters, which reveal that the inflection points—representing the midpoint of the mortality transition in terms of both crude mortality and age- and sex-adjusted corrected mortality—occurred around 1896, approximately two decades before that in infant mortality. A crucial follow-up question emerges from this robust sigmoid pattern: what underlying processes drove these systematic mortality declines? This forms the focus of the next section.

1.3 Diffusion of Progress in Health and Mortality Dynamics

The sigmoid patterns discovered above motivate a framework grounded in technological diffusion research because diffusion processes typically predict characteristic sigmoid trajectories. This section begins with an explanation of technological diffusion theory and its supporting empirical evidence. This theory provides the requisite conceptual background for the economic model presented next, which is developed to elucidate the underlying sigmoid dynamics of mortality patterns.

1.3.1 Diffusion Processes

To explain the sigmoid dynamic patterns presented in Figure 1.2.3, we introduce the theory of the diffusion of innovations. In this context, Rogers (1962), defines diffusion as follows: "*diffusion* is the process by which an innovation is communicated through certain channels over time among the members of a social system". The adoption of a new idea, behaviour pattern, or technology within society does not occur simultaneously. Rather, it is gradually adopted by successive groups of consumers—innovators, early adopters, early majority, late majority, and laggards (Rogers, 1962, p. 150). These groups differ in their structural positions in the social system. According to Kerckhoff and Back (1965), different groups adopt the technology at different stages and for different reasons. For

example, innovators are characterised by their readiness to embrace risk, largely due to having little at stake. Early adopters are thought to require minimal information as they are driven primarily by evaluating the attributes of the innovation, while the larger subsequent majority adopts the innovation because of the imitation effect. Therefore, the timing of adoption varies across these groups (Dearing, 2009).[27]

The idea of diffusion has also been invoked in the context of urban sanitation progress. No progress can immediately and simultaneously reach everyone, as low-income families have limited ability to pay, for example, for basic water and sewer services. Worldwide, 1.1 billion people lack access to any type of improved drinking water source within 1 kilometre, and only about 42% of the rest with access have a household connection or yard tap (Devoto et al., 2012). Even in today's US, low-income households must spend an average of 9.7% of their disposable income, or work 9.5 hours at minimum wage, to afford basic monthly water and sewer services (Teodoro, 2019). In Paris in 1885, only 100 out of 65,000 buildings were connected to sewer lines (Kesztenbaum and Rosenthal, 2017). Kesztenbaum and Rosenthal (2017); Devoto et al. (2012) suggest that a major reason for this low connection rate in the 19th century was the cost, due to which there is a "threshold income" above which households are willing to pay at least the average cost of connecting to the sewer system. During this particular period when major improvements in population health occurred, the diffusion process has been observed. Sewer connections increased with two notable shifts—an early acceleration in the mid-1890s and a subsequent slowdown in the mid-1900s. By 1913, almost 70% of the buildings were connected. Although they did not comment on this, Kesztenbaum and Rosenthal (2017, Fig. 3) reveals that the shares of buildings connected to the sewers in various districts of Paris exhibit *S*-shaped curves. Moreover, apart from technological health innovations, individuals in higher social positions also tend to adopt new behaviours first. Examples include quitting smoking and adopting healthier diets in contemporary contexts, as well as practising home cleanliness

27. See substantial inequalities in the adoption of ChatGPT in Humlum and Vestergaard (2024).

and boiling milk in historical contexts (Lopez et al., 1994; Kesztenbaum and Rosenthal, 2014). The diffusion of these behaviours is more homogeneous because once their value is recognised, they can be adopted by everyone, given that their costs are relatively low (Kesztenbaum and Rosenthal, 2014).

As reviewed in Meade and Islam (1998), diffusion studies have revealed a mathematically consistent sigmoid pattern (S-shape) in the adoption process over time—the rate of adoption starts off slowly, accelerates due to the influence of the 5%–7% of social system members, and eventually slows down as it approaches the system's maximum capacity (Dearing, 2009). The classic study of Griliches (1957) demonstrated that the adoptions of hybrid corn in multiple states in the US from 1933 to 1956, as measured by the shares of corn acreage planted with hybrid seeds, fit the logistic curves characterised by origins, slopes and ceilings very well. Mansfield (1961) analysed the imitation rates of innovations requiring heavy equipment investments prior to cost reductions in four industries—bituminous coal, iron and steel, brewing, and railroads. Across various innovations, the diffusion was slow, with the time until half of the firms adopted the innovations varying from 0.9 to 15 years, and ultimately the adoption rate achieved 100%. More recent papers have applied S-shaped curves to investigate examples such as fibre consumption differentials in the US, the expansion of mobile telephony in Greece (Lotfi et al., 2014), and China's electric vehicle market (Yan and Sun, 2021).[28]

The era of the mortality transition, as discussed in Section 1.2.2, was triggered and catalysed by various technological progress in health. These included the construction of sewage systems, the medical revolution such as the development of vaccines, and reforms aimed at enhancing living conditions, etc. We present a theoretical model in which these technological health advancements interact with the need for individuals to make costly health-promoting decisions to reap the benefits of progress.

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28. See the application of logistic models for sigmoid dynamics across various domains, such as dose-response models (Gadagkar and Call, 2015), plant density-crop relationship (Mead, 1970), and population growth models (Morisita, 1965; Pliška, 1987).

1.3.2 Historical Inequality in Socioeconomic Conditions in London

Given that technological adoption patterns vary across groups, with wealthier individuals typically being early adopters, the pronounced socioeconomic inequality in Victorian London provides a setting that allows us to explore the theory of differential timing in health technology diffusion across social classes.

From around 1825, London became the most populous city in the world. In the period that followed, the development and urbanisation of London spurred its growth as a centre for international finance and trade. By 1891, the population rose to 5.56 million and the urban population increased to 72% (Weber, 1899). As Dickens (1949) portrayed it, "it was the best of times, it was the worst of times...", pointing to the fact that the nineteenth century was also a period of entrenched poverty and stark inequalities. In the UK, between 1900 and 1910, for wealth, the top 10% of private property owners held around 94% of all wealth (Szreter, 2021); and even in relation to income, the top 5% of households received nearly 40% of pre-tax income (Lindert, 2000). Many rich individuals resided in detached large estates flanked by parkland and private gardens. On the contrary, over 30% of the population subsisted below the poverty line (Pfautz, 1967), facing daily challenges in securing the basic necessities of life such as food, shelter, and clothing. A vast number of labourers were trapped in overcrowded conditions, living in back-to-back houses that were constructed with thin dividing walls (Clark, 2013). In the East End, Dorset Street, referred to as "the worst street in London", apparently "teemed with nasty characters—desperate, wicked, lecherous, razor-slashing hoodlums" (Rule, 2018). According to Williamson (1980), the Gini coefficients of total income exceeded 0.5 by the mid-19th century.

Understanding the form of socioeconomic distribution is essential because social stratification underpins the development of health inequalities (Mackenbach, 2012; Brown, 1977, p. 285). The present research has specified a large number of income distribution models, including the Pareto Type I model (Wold and Whittle, 1957), the log-normal distribution (Sargan, 1957), both of which are skewed to the right, along with several related

subsequent models.[29] In these two classic distributions, the long tails towards the higher values capture the small group of wealthy individuals holding a disproportionately large share of the total wealth. The left tail of the log-normal distribution suggests that low (nonzero) incomes are frequent, yet their likelihood decreases as they near zero, whereas the Pareto principle features a "cut-off" at the left end, implying that the majority have incomes close to a minimum value. Therefore, the Pareto law is broadly viewed as the model of high-income groups (Dagum, 1980, 2006, p. 237). However, empirically, the right tail of the distribution is commonly blurred in modern datasets due to the survey design not targeting potential high-income respondents, challenges in contacting them, or their refusal to participate (Jenkins, 2017). The "top incomes" literature therefore uses administrative record data on personal income tax returns instead of household surveys to understand the richest 1% (see e.g., Alvaredo, 2011; Atkinson and Piketty, 2007; Atkinson et al., 2011).

However, despite the rich measurements of inequality, historical data complicates the modelling of distribution before the onset of the mortality transition. The scarcity of distributional data in the 19th century concerning property and self-employed income forced researchers to infer overall income distribution trends from imperfect income and property tax data (Williamson, 1980). Some other studies relied on occupation data. For example, from the 1886 and 1906 Wage Censuses, Bowley (1937, p. 42) derived the respective earnings distributions of males in the UK which are log-normal because "*the lower quartile is about four-fifths and the upper quartile is about five-fourths of the median, and the lowest decile is about two-thirds and the highest decile is about three-halves of the median*". Based on Bowley's estimates, Williamson (1980) pointed out that the omission of agricultural labourers and service sector workers truncates the lower end of the overall earnings distribution, indicating that the left tail of the log-normal distribution was underrepresented. Apart from the pay differentials between occupations of various skills, Williamson (1985, Chap. 3) also pieced together the intra-occupational earnings distributions. According to Brown (1977, p. 287, 320), the earnings distributions for manual men across different

29. Early models of wealth distribution overlooked the high frequency of households and individuals possessing zero total wealth, as well as those with negative or nonexistent net wealth (see Dagum, 2006, for a review). I do not discuss these studies here because our paper concentrates on the distribution of socioeconomic conditions, not wealth.

occupations, industries, and regions have been log-normal since the first Wage Census in 1886. On the other hand, as mentioned above, the right tail of the distribution (top incomes) is under-covered in the data. In the historical context, wills with bequest and occupation information (Clark and Hamilton, 2006), probate grants (Bond and Morton, 2022), and rare surnames (Clark and Cummins, 2015) have been investigated to uncover the top incomes.

Based on the evidence of Bowley (1937); Brown (1977), we conclude that while the left tail was likely underrepresented due to the omission of agricultural and service workers, and the right tail was difficult to capture fully even with supplementary data from wills and probates, the core of the socioeconomic distribution in 19th century London likely followed a log-normal pattern.

The timing of engagement in the three crucial pathways—nutrition, education, and health technology—through which humans have been propelled into the era of low mortality varies along London's log-normal (asymmetric, right-skewed) income distribution, as wealthier individuals adopt these advancements earlier and in multiple ways (see Section 1.3.1).

1.3.3 A Model of Adoption of a New Health Technology

In this section, I describe a model in which the timing of health technology adoption depends on individuals' positions in the log-normal income distribution. At the macro-level, the log-normal income distribution underlies the sigmoid pattern in mortality dynamics—the extent of mortality declines depends on the share of the population adopting new health technology. The model presentation and analysis in this Section draw heavily from the joint paper (Angelopoulos et al., 2024).

Assume that adopting a new health technology involves an annual expenditure, denoted as ψ_k, to access the technological health improvements which increase survival probability, $s\left(h_k; \theta, I^i_{\psi,k}\right) \in (0,1)$, for all years after the decision to adopt the new technology in year $k \in [T_1, T_n]$ (e.g., $T_1 = 1840$ and $T_n = 1940$). In the survival function, the parameter $\theta \in (0,1)$ represents a baseline survival rate common to all individuals; h_k captures the new health technology for year k that can be accessed if expenditure ψ_k is incurred; $I^i_{\psi,k}$ is an indicator which equals to 1 if the individual i incurs expenditure ψ_k and zero otherwise.

We assume that adoption requires a financial outlay every year after the adoption, as opposed to a one-off expenditure, because this captures better the historical framework, in which the vast majority of households were renters so that health improvements related to accommodation would be reflected in the rent. Moreover, accessing progress in the form of improved treatment, care, medicine, nutrition and education, all relate to flows of expenses over time as opposed to one-off investments. The survival function $s(\cdot)$ is assumed to be continuously differentiable and strictly increasing in h_k if ψ_k is incurred and independent of h_k otherwise, and to satisfy $s\left(h_k;\theta,I^i_{\psi,k}=1\right) > s\left(h_k;\theta,I^i_{\psi,t}=0\right)$. As an example, consider the form $s\left(h_k;\theta,I^i_{\psi,k}\right)=\theta+h_k*I^i_{\psi,k}$, implying that $h_k\in(0,1-\theta)$.

There is a continuum of individuals i with exogenous income y^i, which is assumed to be the same for every year $t\in[T_1,T_n]$, and drawn from a density function f_y associated with a cumulative income distribution function $\overline{F}_y=1-F_y$, where $F_y(\overline{y})=Pr(y>\overline{y})$. Individual i derives utility from consumption c^i. Assuming that the individual anticipates the level of health technology to be given by h_k and the annual expenditure required to access it by ψ_k, for all years $t\geq k$, then in any year $k\in[T_1,T_n]$, the individual has expected lifetime utility

$$U_k=\sum_{t=k}^{\infty}\left(\beta*s\left(h_k;\theta,I^i_{\psi,k}\right)\right)^{t-k}u(c^i)=\left\{\frac{u(c^i)}{1-\beta*s\left(h_k;\theta,I^i_{\psi,t}\right)}\right\}, \tag{1.3.1}$$

where $\beta\in(0,1)$ discounts the future periods. The utility function $u(\cdot):(0,+\infty)\to\mathbb{R}$ is assumed to be twice continuously differentiable, strictly increasing and strictly concave, satisfying the conditions $\lim_{c\to0}u(c)=-\infty$, $\lim_{c\to0}u_c(c)=+\infty$ and $\lim_{c\to\infty}u_c(c)=0$. To focus on the importance of the distribution of income across the population, we abstract from complications associated with ageing, implying that the survival probability is not a function of time, so that Equation 1.3.1 is interpreted as reflecting a dynasty process in which an offspring replaces a living individual randomly over time, inheriting their income stream and survival probability.

In any year $k \in [T_1, T_n]$, an individual decides whether to incur expenditure ψ_k for all years $t \geq k$ to access the technological health improvements, given a resource constraint:

$$c^i + \psi_k * I^i_{\psi,k} = y^i. \tag{1.3.2}$$

The individual decides to undertake the expenditure ψ_k in year k for all years $t \geq k$ if there are net utility benefits from doing so:[30]

$$U^k\left(I^i_{\psi,k}=1\right) > U^k\left(I^i_{\psi,k}=0\right), \text{ i.e.,}$$
$$\left\{\frac{u(y^i-\psi_k)}{1-\beta * s\left(\theta, h_k; I^i_{\psi,k}=1\right)}\right\} > \left\{\frac{u(y^i)}{1-\beta * s\left(\theta, h_k; I^i_{\psi,k}=0\right)}\right\}.$$

Note that the decision of the individual is the same in any year $k' > k$ in this setup, as long as the model parameters do not change; hence, if the individual decides to adopt the technology in period k then she/he will also find it optimal to continue incurring the expense ψ_k and access the benefits of improved health technology h_k in all years following the year k.[31]

1.3.4 Adoption of New Health Technology Depends on Income

There is a threshold income level y^* such that individuals for which $y^i > y^*$ will choose to adopt health progress h_k and those with $y^i < y^*$ will not. To see this, define the net gain function from adopting the new technology

$$g\left(y^i, h_k, \psi_k\right) = \left\{\frac{u\left(y^i-\psi_k\right)}{1-\beta * s\left(h_k; \theta, I^i_{\psi,k}=1\right)}\right\} - \left\{\frac{u(y^i)}{1-\beta * s\left(h_k; \theta, I^i_{\psi,k}=0\right)}\right\},$$

30. The decision of the individual is the same in any year $k' > k$ in this setup, as long as the model parameters do not change, hence if the individual decides to adopt the technology in period k then it will continue to incur the expense ψ_k and access the benefits of improved public health technical progress h_k in all years following the year k.

31. In other words, a decision to commit to ψ_k for all years $t \geq k$ is time-consistent.

which has a positive derivative

$$g_{y^i}\left(y^i, h_k, \psi_k\right) = \left\{\frac{u_c\left(y^i - \psi_k\right)}{1 - \beta * s\left(h_k; \theta, I^i_{\psi,k} = 1\right)}\right\} - \left\{\frac{u_c(y^i)}{1 - \beta * s\left(h_k; \theta, I^i_{\psi,k} = 0\right)}\right\} > 0,$$

following from the assumptions on the functions $s(\cdot)$ and $u(\cdot)$. The properties of the utility function also imply that $\lim_{y^i \to +\infty} g\left(y^i, h_k\right) > 0$ and that $\lim_{y^i \to \psi} g\left(y^i, h_k\right) = -\infty$. By the Intermediate Value Theorem $g\left(y^i; \cdot\right) = 0$ has a solution, y^*_k, while strict monotonicity of $g\left(y^i; \cdot\right)$ implies that y^*_k is unique, meaning that $g\left(y^i, h_k\right) > 0$ for $y^i > y^*_k$ and that $g\left(y^i, h_k\right) < 0$ for $y^i < y^*_k$. A graphical representation of this solution is in Figure 1.3.1.

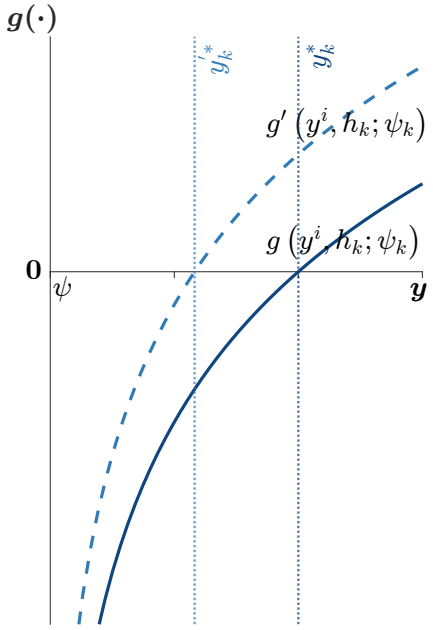

Figure 1.3.1: **Determination of the threshold for adopting health technology.** This figure illustrates the net gain function from adopting the new technology h_k in year k. Individuals whose income is higher than the threshold y^*_k will adopt new technology. Improvements in health technology which increase h_k and/or decrease ψ_k would shift the net gain function to the left i.e. from $g(\cdot)$ to $g'(\cdot)$. Consequently, this shift lowers the threshold from y^*_k to y'^*_k, leading to a greater proportion of individuals embracing the new technology.

The proportion of individuals who adopt health technology is determined by the properties of the income distribution function that describes the population. Income distributions across time and populations have been found to be asymmetric with a long right tail (for higher income individuals) and usually well approximately as log-normal. In Figure 1.3.2, we show how the income threshold y^*_k determines the proportion of individuals who adopt the new health technology when the income distribution is log-normal.

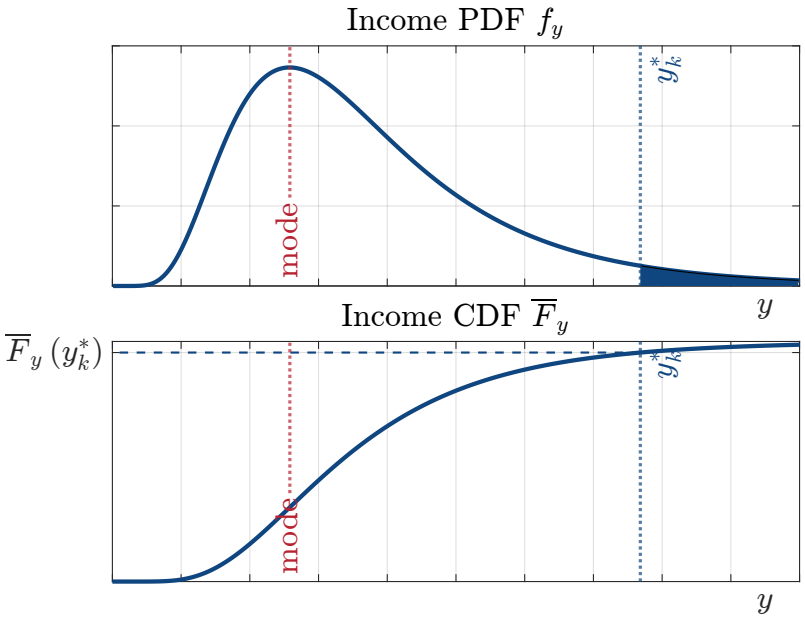

Figure 1.3.2: **Income distribution and adoption of health technology.** The figure shows a log-normal distribution of income y with its associated PDF f_y (*above*) and CDF $\overline{F}_y$ (*below*). The vertical line y_k^* represents the threshold beyond which an individual adopts the new health technology. The shaded area shows the fraction of individuals who adopt the new health technology and $\overline{F}_y\left(y_k^*\right)$ is the proportion of individuals who do not adopt the new health technology.

Empirical evidence across historical and modern contexts demonstrates that high-income individuals tend to be early adopters of new technologies. A compelling historical example comes from Paris in 1885. As depicted in Kesztenbaum and Rosenthal (2017, Fig. 3), the share of buildings connected to sewer lines in different districts in Paris increased at a relatively slow pace starting in 1885. This rate accelerated progressively until 1900, after which it began to decelerate gradually until 1913. This observation exemplifies the stylised fact that the adoption of new technologies over time typically follows an S-curve (Geroski, 2000). In modern times, this pattern persists in healthcare, where new public health interventions and programs, such as neonatal intensive care and surfactant therapy, initially benefit those of higher socioeconomic status (Victora et al., 2000). Two mechanisms can explain this earlier adoption by high-income individuals. First, individuals with higher socioeconomic status typically have higher education levels, leading to earlier awareness of preventative measures like hygiene and disease transmission (Kesztenbaum and Rosenthal, 2016; Floud et al., 2011). Second, the adoption timing varies due to differences in characteristics such as firm size and learning costs (Geroski, 2000; Davies, 1979; Hall and Khan, 2003). Both historical and contemporary evidence points to the

latter as the primary mechanism. Kesztenbaum and Rosenthal (2017) found that districts endogenously chose to equip streets with sewers, and landlords endogenously chose to make the connection based on their affordability.[32] Similarly, Victora et al. (2000) attributed the earlier adoption of public health interventions by higher socioeconomic groups to differences in access and availability.

> "In the history of diffusion of many innovations, one cannot help being struck by two characteristics of the diffusion process: its apparent overall slowness on the one hand, and the wide variations in the rates of acceptance of different inventions, on the other" (Rosenberg, 1972).

Having identified high-income individuals as early adopters, the next section examines how these innovations diffuse.

1.3.5 The Diffusion of Health Progress

In this setup, while the exogenous variables in the model do not change, the proportion of individuals who adopt the new technology, $F_y\left(y_k^*\right) = Pr\left(y > y_k^*\right)$ remains the same over time because the decision of the individual is the same in any year $k' > k$. In general, there are two types of changes that can lead to an increase in the proportion of individuals who access the new technology: technological progress in health and improvements in income. Both can lead to sigmoid dynamics of adoption of the new technology, i.e., of its diffusion across the population, consistent with the sigmoid dynamics of mortality declines seen in Section 1.2.5.

First, improvements in health technology, which increase h_k and/or reduce ψ_k, reduce the threshold level y_k^*. To see this, note that

$$g_{h_k}\left(y^i, h_k, \psi_k\right) = \frac{u\left(y^i - \psi_k\right)\beta * s_{h_k}\left(h_k; \theta, I^i_{\psi,k} = 1\right)}{\left(1 - \beta * s\left(h_k; \theta, I^i_{\psi,k} = 1\right)\right)^2} > 0,$$

32. Once the general population had access to relatively clean drinking water, exposure to the most lethal diseases was effectively controlled. Consequently, further efforts to extend filtration to all water sources provide diminishing benefits, making the remaining population less motivated to connect (Aidt et al., 2023).

following from the assumptions on the function $s(\cdot)$ and that

$$g_{\psi_k}\left(y^i, h_k, \psi_k\right) = \left\{ \frac{(-\psi_k)\, u_c\left(y^i - \psi_k\right)}{1 - \beta * s\left(h_k; \theta, I^i_{\psi,k} = 1\right)} \right\} < 0,$$

following from the assumptions on the function $u(\cdot)$. These improvements imply that the $g\left(y^i; h_k, \psi_k\right)$ curve shifts to the left, i.e., that a higher proportion of individuals decides to adopt the new technology (see Figure 1.3.1 for a graphical representation). Since improvements in health technology that decrease ψ_k have a similar effect with increases in h_k, and are likely strongly correlated (for example, improved sewage treatment that increases h_k is concurrent with extensions in sewage pipe network, making it cheaper for individuals' houses to be connected), we focus on the exposition and analysis of effects via h_k. These changes were substantial over the period we study, defining health technological progress, which a large research considers to be an important driver of the the epidemiological transition (see references in Section 1.2).

Technological progress in health over time, implying successive reductions in the threshold of income y^*_k that determines adoption of the health technology h_k, generates a technology diffusion pattern that is sigmoid given a right-skewed income distribution function such as the log-normal.[33] In particular, assuming a log-normal income distribution, then starting from a high y^*_k, as h_k rises, or, alternatively, as y^*_k falls, the rate of change of $F_y\left(y^*_k\right)$ with respect to y^*_k, i.e., the rate of change of the proportion of the population that adopts the new health technology as a function of the income threshold that this technology defines, increases first at an increasing rate and, after the mode of f_y, at a decreasing rate. This is a result of the convexity properties of the log-normal distribution f_y, which imply that $F_y\left(y^*_k\right)$ is a decreasing and concave function of y^*_k to the left of the mode of f_y and a decreasing and convex function to its right. Therefore, viewed as a function of time, $F_y\left(y^*_k\right)$, which we denote for clarity as $F_y\left(y^*_t\right)$, is initially convex and later concave,

33. Here, besides the implicit assumption that there is a positive relationship between income thresholds and time—as time goes by, the income threshold moves to the left, we assume that the marginal effect of the health progress on income thresholds is bigger before the inflection point than after the inflection point. In other words, for a typical individual i, on an annual basis, the marginal effects are diminished after the inflection point, or it necessitates additional years to move the income threshold. Under this assumption, the (threshold of) asymmetric log-normal income distribution exhibits a correspondence with the (inflection point of) observed symmetric mortality dynamics.

with an inflection point of changing convexity at the year where y_t^* equals the income associated with the mode of f_y. In other words, the dynamic pattern of the adoption rate is sigmoid over time, in effect the inverse of the plot of the CDF $\overline{F}_y$ in Figure 1.3.2, and thus similar to many empirical examples of diffusion dynamics of technological change in Rogers (1962); Stokey (2021).

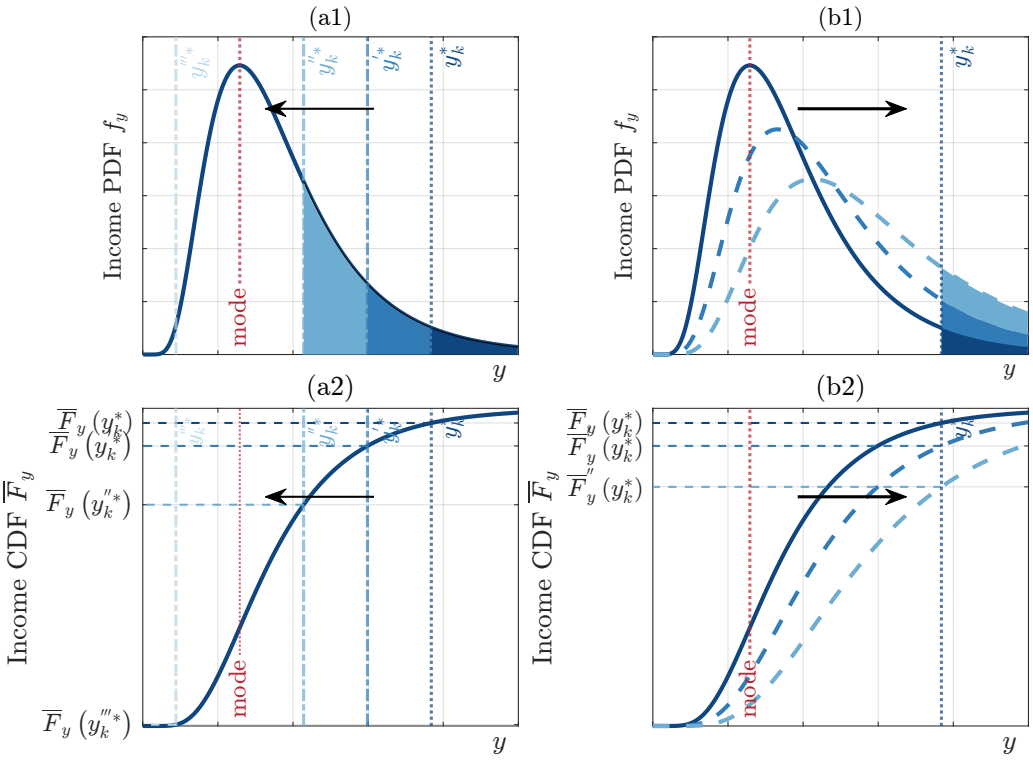

Figure 1.3.3: **Dynamics of adoption rate under technological progress in health and uniform increase in income.** Panels (a1) and (a2) illustrate the effect of technological progress in health on the threshold of income that determines the adoption rate (from y_k^* to $y_k'''^*$). Panels (b1) and (b2) illustrate the effect of successive uniform increases in income, shifting distributions to the right.

The dynamic process of technology adoption generated by successive stages of improvements in health technology is illustrated in Figure 1.3.3. In particular, in earlier stages of progress in health technology, the increases in the adoption rate are small, reflecting the relative flatness of the right tail of the income distribution, which implies that only a small proportion of individuals are near the threshold and in effect benefit by progress in health. The convexity of the income distribution implies that the adoption rate increases with further progress. Therefore, the initial part of the technological diffusion curve is convex (and can be relatively flat initially if the threshold is very high). With a log-normal income distribution, the convexity of which changes at the mode, the higher slope of the

density function, reflecting the higher rate of change in the proportion of individuals who adopt the new health technology, is thus near the mode of the distribution. Therefore, in the middle stages of health progress, when the income threshold has moved sufficiently closer to the mode of the distribution of income, improvements in health technology affect large proportions of individuals and thus the rate of adoption increases faster. Finally, in later stages, when the majority of the population has joined already, further improvements have a smaller impact as the proportions of individuals affected are again smaller. These stages are associated with the final part of the concave segment of the technological diffusion curve. In summary, the asymmetry of the underlying income distribution gives rise to sigmoid technological diffusion dynamics.

Changes in the income distribution function F_y that imply that more individuals are above the y_k^* threshold over time, can also generate sigmoid diffusion dynamics. This requires changes in the income distribution that imply a sufficiently large increase in y^i for at least a subset of the individuals for whom $y^i < y_k^*$. As an example, a uniform increase in income for all individuals i shifts the distribution F_y to the right, such that $y^i > y_k^*$ for more individuals (see (b1) and (b2) in Figure 1.3.3). Following the same logic as above, successive widespread, across the population, improvements in income would generate a sigmoid diffusion pattern. Note, however, that not all changes in the distribution of income generate sigmoid diffusion dynamics and that, in fact, increases in income for those with lower income do not, necessarily, increase the adoption rate, if the incomes of those benefiting do not rise sufficiently. Assuming a log-normal income distribution, changes in the distribution such that the mode of the distribution corresponds to a higher income would generally increase health technology adoption, by moving the inflection point of the income distribution closer to the income threshold. Such changes can arise from an increase in median income, reflecting, e.g., a more uniform increase in income as in the previous example, which shifts the income distribution to the right. They may also arise from changes that reduce the skewness of the distribution, for example by an increase in the proportion of high income individuals relative to low income individuals.[34]

34. The mode of the log-normal distribution is positively related to the median and negatively related to skewness.

The diffusion dynamics of progress in health determine the dynamics of declines in the death rate. The death rate in year t, defined across the population, is given by

$$d_t = 1 - \left[F_y\left(y_t^*\right) s_t^1 + \left(1 - F_y\left(y_t^*\right)\right) s_t^0 \right], \tag{1.3.3}$$

where $s_t^1 = s_{h_t}\left(h_t; \theta, I_{\psi,t}^i = 1\right)$ and $s_t^0 = s_{h_t}\left(h_t; \theta, I_{\psi,t}^i = 0\right) = \theta$ and the notation makes clear that both the adoption rate $F_y(y_t^*)$ and the survival probability for those who have adopted the new health technology, $s^1(t)$, are changing over time. It is convenient to express Equation 1.3.3 as

$$d_t = 1 - \theta - F_y\left(y_t^*\right) \overline{s}_t,$$

where $\overline{s}_t = s_t^1 - s_t^0$, with $\overline{s}_t > 0$ and $\overline{s}_t' > 0$, given the assumptions on $s(\cdot)$ and an increasing path of h_t over time. The rate of change (slope) of the death rate over time, d_t' is given by

$$d_t' = -f_y(y_t^*)\overline{s}(t) - F_y(y_t^*)\overline{s}'(t), \tag{1.3.4}$$

noting that $F_y' = f_y > 0$, so that $d_t' < 0$ given the properties of the remaining terms in Equation 1.3.4, while the slope of d_t' is given by

$$d_t'' = -F_y''(y_t^*)\overline{s}_t - F_y(y_t^*)\overline{s}_t'' - 2f_y(y_t^*)\overline{s}_t', \tag{1.3.5}$$

noting that $F_y'' = f_y'$.

The convexity properties of the death rate over time are therefore determined, via Equation 1.3.5, by the convexity properties of $F_y(y_t^*)$ and of $\overline{s}_t$. As explained above, $F_y''(y_t^*)$ is initially positive and later negative, with an inflection point of changing convexity, where $F_y'(y_t^*) = f_y(y_t^*)$ is maximised, at the year t where y_t^* equals the mode of f_y. We have made no assumptions regarding the convexity of $\overline{s}_t$. For example, it may seem plausible to assume that $\overline{s}_t$ was increasing over time also following a sigmoid function. In particular, the effect of more interventions on reducing mortality risk for those accessing the technology could increase with an increasing slope during the first stages of progress, perhaps because a number of interventions were needed for stronger effects to be observed; on the other hand, at the later stages, the marginal effect of new interventions could be smaller,

given that the survival probability must be bounded. Given the convexity properties of $\overline{s}_t$ over time, the diffusion dynamics associated with the technology adoption rate $F_y(y_t^*)$ generate sigmoid dynamics for the death rate d_t if there is a time period t^*, associated with $F_y(y_{t^*}^*)$ such that $d_t'' < 0$ for $t < t^*$ and $d_t'' > 0$ for $t > t^*$.

The inflection point in the death rate as function of time is observed at the year where the slope, d_t' is maximised, or else where $d_t'' = 0$.[35] The year where the inflection point is observed must thus satisfy

$$F_y'' \left(y_{t^*}^* \right) = - \frac{F_y \left(y_{t^*}^* \right) \overline{s}_{t^*}'' + 2 f_y \left(y_{t^*}^* \right) \overline{s}_{t^*}'}{\overline{s}_{t^*}}. \tag{1.3.6}$$

For example, assuming that health progress during the mortality transition generated an approximately linear survival probability as a function of time, i.e., that $\overline{s}_{t^*}'' = 0$, implies that the inflection point in the death rate must occur for a level y_t^* that is to the left of the level of income associated with the mode of the income distribution. Allowing for $\overline{s}_{t^*}'' = 0$ to be positive during at least the first decades of the mortality transition implies that the inflection point is even further to the left of the mode. Intuitively, the maximum reduction in mortality is achieved when the majority of the population have adopted the new health technologies and the impact of the latter on reducing mortality is maximised.

Moreover, these diffusion dynamics of health progress may vary substantially across age groups. In particular, the empirical evidence in Chapter 1.2.5 reveals that the mortality transition for infants occurred roughly two decades after the transitions in non-infant and crude mortality rates. This timing difference might be attributed to the nature of early public health interventions and medical advances (e.g., sanitation systems and basic

35. In a longer time series, there may be more than one inflection points, or indeed death rate need not even be a monotonic function of time. Although Equation 1.3.4 and Equation 1.3.5 are sufficiently generic to allow for these possibilities, here we focus on the specific episode of mortality declines with a monotonic death rate and a single inflection point.

medical care), which benefited older age groups earlier. According to Preston and Van de Walle (1978), infants did not see the same early benefits from improved sanitation as older children, since they typically don't encounter contaminated food and water until the second year of life.[36]

In summary, the technological diffusion model generates dynamic patterns of reductions in the death rate over the mortality transition that are consistent with both the empirical evidence we presented in Section 1.2.5 and with leading explanations of the drivers of the epidemiological transition.

1.4 Discussions and Conclusions

In this chapter, I constructed a novel historical dataset for London from municipal archives to analyse the dynamics of mortality reductions at the city level. Our findings reveal that mortality reductions in London from the 1840s to the 1960s followed sigmoid dynamics over time, characterised by an initial period of consistently high mortality rates, then a prolonged period of gradual declines followed by a phase of accelerated declines after and ultimately a gradual slow-down to a new equilibrium level. This pattern appears consistently across four mortality measures in London—crude mortality, corrected death rate (adjusted for age and sex composition), infant mortality, and non-infant mortality—as well as in other areas (see Stewart (2024) for evidence of sigmoid mortality patterns in Glasgow, England and Wales, and Scotland). This commonality illuminates the information conveyed by parameters estimated through the proposed four-parameter logistic model. In particular, the inflection point signals both the midpoint of the mortality transition and the timing of the fastest reduction. When comparing across mortality measures, the rapid reduction in infant mortality occurred around 1919, approximately two decades after the transition was observed in crude mortality (around 1896), suggesting that infant mortality decline was not the primary driver of the early phase of the mortality transition.

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36. To build on the earlier discussion in Section 1.2.5, the distinction between the extent of benefits and their timing is crucial. While numerous studies have shown that vulnerable age groups receive greater benefits from improvements in nutrition, hygiene, and water infrastructure (Dyson, 2010; Omran, 1971; Kesztenbaum and Rosenthal, 2017), there has been limited research examining the temporal sequence of these benefits across different age groups.

This empirical pattern is consistent with the predictions of an economic model of health progress diffusion, wherein endogenous adoption hinges on evaluating the costs and benefits of new health technologies. Initially, new health technologies were adopted by the most affluent segment of society, followed by a gradual diffusion to broader populations. This diffusion process started slowly but gained momentum over time until all the above-mode adopted the technology. This adoption pattern aligned with the early stage of the mortality transition, where death rates began declining from high levels—first at a slow pace, then at an accelerating rate, until reaching the inflection point. After that, both the adoption growth rate and mortality decline rate decelerated until reaching a new equilibrium.

Understanding the historical mortality transition in developed countries provides valuable insights for today's developing economies. These developing regions are experiencing similar epidemiologic, demographic, and technological transitions (Omran, 1971), and continue to face challenges in the distribution of health-improving infrastructure. Particularly, access to clean water and sanitation remains uneven across many regions in Asia, Africa, and Latin America (Banerjee and Duflo, 2007; Baisa et al., 2010; Galiani et al., 2009). While the historical lessons could inspire current public health challenges and development processes (Galor, 2005), systematic studies examining these historical transitions remain limited. I first contribute to constructing a novel historical mortality dataset for London from the 1840s to the 1960s, which is highly frequent (annual), continuous, and includes four mortality measures. Using this dataset, we further contribute to developing a four-parameter logistic model of mortality dynamics throughout this period. Our estimated coefficients provide a systematic quantification of the timing of the mortality transition, addressing a significant gap in the existing literature. Moreover, we propose a theoretical explanation for the observed sigmoid pattern in mortality decline by connecting it to health technology adoption, which diffuses dynamically across the population following the log-normal income distribution.

While our model identifies socioeconomic inequality, represented by log-normal income distribution, as the key driver of the sigmoid pattern of mortality decline (affluent individuals have earlier access to health innovations through multiple channels, see the discussions on mechanisms in Section 1.3.4), alternative explanations exist for this mortality trend. For instance, consider the adoption of sewer infrastructure in Paris, as documented by Kesztenbaum and Rosenthal (2017, Fig. 3). The proportion of buildings connected to sewer lines exhibited a characteristic S-shaped pattern: a slow initial uptake beginning in 1885, followed by accelerating adoption through 1900, and finally a gradual deceleration until 1913. This observation exemplifies the stylised fact that the adoption of new technologies over time typically follows an S-curve (Geroski, 2000). Therefore, if health progress diffusion inherently follows a sigmoid trend, the resulting mortality decline could display a sigmoid pattern irrespective of income distribution. In this light, our socioeconomic inequality-based theory, while sufficient to explain the observed mortality dynamics and consistent with empirical data, represents one possible explanation rather than the only explanation. The next section will subject our theoretical framework to further empirical testing.

1.5 Appendix

1.5.1 Sigmoid Mortality Dynamics Model

The four-parameter logistic function Equation 1.5.1 describes the dynamic evolution of the trend in London:

$$\overline{d} = \delta_l + \frac{\delta_u - \delta_l}{1 + e^{\left(\beta(t-\tau_c)\right)}}, \tag{1.5.1}$$

where $\delta_u - \delta_l > 0$ and $\beta > 0$.

The first-order derivative and the second-order derivative are:

$$\frac{d}{dt}\overline{d} = -\left(\delta_u - \delta_l\right) \frac{\beta e^{\left(\beta(t-\tau_c)\right)}}{\left(1 + e^{\left(\beta(t-\tau_c)\right)}\right)^2}; \tag{1.5.2}$$

$$\begin{aligned}
\frac{d^2}{dt^2}\overline{d} &= -\left(\delta_u - \delta_l\right)\beta \cdot \frac{d}{dt}\left(\frac{e^{\left(\beta(t-\tau_c)\right)}}{\left(1 + e^{\left(\beta(t-\tau_c)\right)}\right)^2}\right) \\
&= -\left(\delta_u - \delta_l\right)\beta \cdot \frac{\left(1 + e^{\left(\beta(t-\tau_c)\right)}\right)^2 \cdot \beta e^{\left(\beta(t-\tau_c)\right)} - 2\beta\left(e^{\left(\beta(t-\tau_c)\right)}\right)^2\left(1 + e^{\left(\beta(t-\tau_c)\right)}\right)}{\left(1 + e^{\left(\beta(t-\tau_c)\right)}\right)^4} \\
&= -\left(\delta_u - \delta_l\right)\beta^2 \cdot \frac{e^{\left(\beta(t-\tau_c)\right)}\left[1 - e^{\left(\beta(t-\tau_c)\right)}\right]}{\left[1 + e^{\left(\beta(t-\tau_c)\right)}\right]^3}.
\end{aligned} \tag{1.5.3}$$

The four parameters are interpreted as follows.

1. The parameters δ_l and δ_u are, respectively, the lower and upper asymptotes: $\lim_{t\to+\infty}\overline{d} = \delta_l$ and $\lim_{t\to-\infty}\overline{d} = \delta_u$.
2. τ_c has two properties:
   - It represents the year when the death rate lies halfway between the upper and lower asymptotes i.e. $\overline{d}\Big|_{t=\tau_c} = \left(\delta_l + \delta_u\right)/2$;[37]

37. In the biomedical literature, this parameter is commonly referred to as half maximum inhibitory concentration (IC50) or effective concentration (EC50) (An et al., 2019).

- It is the year of the inflection point at which the curvature changes sign. Based on the second-order derivative Equation 1.5.3, when $t < \tau_c$, $e^{(\beta(t-\tau_c))} < 1 \implies 1 - e^{(\beta(t-\tau_c))} > 0$ so that $\frac{d^2}{dt^2}\overline{d}\Big|_{t<\tau_c} < 0$. Similarly, $\frac{d^2}{dt^2}\overline{d}\Big|_{t=\tau_c} = 0$ and $\frac{d^2}{dt^2}\overline{d}\Big|_{t>\tau_c} > 0$. In other words, $\overline{d}(t)$ is concave when $t < \tau_c$ and convex when $t > \tau_c$. τ_c is the inflection point at which the curvature of the curve changes sign.
- $\frac{d^2}{dt^2}\overline{d}\Big|_{t=\tau_c} = 0$ implies that the first-order derivative Equation 1.5.2 is maximised at $t = \tau_c$, which means that the timing of the inflection point aligns with the maximum slope of the curve.

3. The parameter β shifts the slope of the trend in the death rate as Equation 1.5.2 illustrates. Since we assume $\delta_u - \delta_l > 0$ and $\beta > 0$, the slope at the inflection point is negative. For example, at the inflection point where $t = \tau_c$, the slope is:

$$\begin{aligned}\frac{d}{dt}\overline{d}\Big|_{t=\tau_c} &= -(\delta_u - \delta_l)\frac{\beta e^{(\beta(\tau_c-\tau_c))}}{\left(1+e^{(\beta(\tau_c-\tau_c))}\right)^2} \\ &= -(\delta_u - \delta_l)\frac{\beta}{4}.\end{aligned} \tag{1.5.4}$$

In addition, as described above, when the slope of the curve, $\frac{d}{dt}\overline{d}$ is maximal, $\frac{d^2}{dt^2}\overline{d} = 0$,

$$\begin{aligned}&\frac{d^2}{dt^2}\overline{d} = 0 \\ \implies &-(\delta_u - \delta_l)\beta^2 \cdot \frac{e^{(\beta(t-\tau_c))}\left[1-e^{(\beta(t-\tau_c))}\right]}{\left[1+e^{(\beta(t-\tau_c))}\right]^3} = 0 \\ \implies &1 - e^{(\beta(t-\tau_c))} = 0 \\ \implies &t = \tau_c, \text{ i.e., } \frac{d^2}{dt^2}\overline{d}\Big|_{t=\tau_c} = 0.\end{aligned} \tag{1.5.5}$$

Therefore, the maximum slope is reached at the inflection point when $t = \tau_c$, which is $-(\delta_u - \delta_l)\frac{\beta}{4}$. β plays an important role in determining the magnitude of steepness.

1.5.2 Estimation

For each mortality measure j in London, we fit the four-parameter logistic model Equation 1.5.1 by using the Gauss-Newton method.[38] The estimation process is facilitated by employing the Stata package **`nl'** (Danuso et al., 1992; Royston, 1993).

$$d_t = \delta_l + \frac{\delta_u - \delta_l}{1 + e^{\left(\beta(t-\tau_c)\right)}} + u_t, \tag{1.5.6a}$$

where u_t is the error term measuring the discrepancy between the actual value and the fitted curve. For each mortality measure j, u_t is assumed to be serially correlated and homoskedastic, as is commonly found in literature about nonlinear models (see Meade and Islam, 1998). However, as discussed in Section 1.2.5, we suspect that u_t is serially correlated and heteroskedastic.[39] To take into account the serial correlation, we model the error term in Equation 1.5.6a as

$$u_t = \rho u_{t-1} + \epsilon_t, \tag{1.5.6b}$$

where ϵ_t is assumed to be independently distributed across time. Then combining Equation 1.5.6a and Equation 1.5.6b, the statistical model of the sigmoid trend taking into account the serially correlated random shocks is Equation 1.2.2b:

$$d_t = \rho d_{t-1} + \left[\delta_l + \frac{\delta_u - \delta_l}{1 + e^{\left(\beta(t-\tau_c)\right)}}\right] - \rho\left[\delta_l + \frac{\delta_u - \delta_l}{1 + e^{\left(\beta(t-1-\tau_c)\right)}}\right] + \epsilon_t, \tag{1.5.6c}$$

where the error term ϵ_t is independently distributed and heteroskedastic.

To estimate Equation 1.5.6c using the Gauss-Newton method, we apply the algorithm below.[40]

Algorithm 1. *solving the four-parameter logistic model*

1. Start with initial guesses for the parameters $\mathbf{\Theta_0} = (\rho_0, \delta_{l,0}, \delta_{u,0}, \beta_0, \tau_{c,0})$.

---

38. We omit superscript j below to simplify the notation.
39. We also performed the Breusch-Godfrey test to confirm the presence of serial correlation.
40. Equation 1.5.6a can be estimated using the identical algorithm, with the modification of subtracting ρ.

2. Evaluate the predicted value at the guessed parameters and compute the in-sample prediction errors (residuals):

$$\boldsymbol{r_k} = \boldsymbol{d_t} - \hat{\boldsymbol{d}}_{\boldsymbol{t,k}},$$

where $\boldsymbol{d_t}$ is the observed value, $\hat{\boldsymbol{d}}_{\boldsymbol{t,k}}$ is the predicted value based on guesses of parameters in iteration k, $\boldsymbol{r_k}$ is the vector of residuals in iteration k.

3. Calculate the Jacobian matrix $\boldsymbol{J_k}$ containing the partial derivatives of the function with respect to $\boldsymbol{\Theta_k}$.

4. Update the parameter estimates using the residual and Jacobian matrix computed in Steps 2 and 3:

$$\boldsymbol{\Theta_{k+1}} = \boldsymbol{\Theta_k} + \left(\boldsymbol{J_k^T J_k}\right)^{-1} \boldsymbol{J_k^T r_k}.$$

The derivative of this Gauss-Newton formula is briefly explained below.
The Gauss-Newton algorithm minimises the sum of squares i.e., $RSS = \sum_{t=1830}^{T} \boldsymbol{r(\Theta)}^2$ by connecting to the concept of Taylor expansion. In the k-th iteration, the first-order Taylor expansion of $\boldsymbol{r(\Theta)}$ around current guess $\boldsymbol{\Theta_k}$ is:[41]

$$\boldsymbol{r(\Theta_{k+1})} \approx \boldsymbol{r(\Theta_k)} + \boldsymbol{J_k \Delta \Theta_k},$$

where $\boldsymbol{\Delta\Theta_k} = \boldsymbol{\Theta_{k+1}} - \boldsymbol{\Theta_k}$. The aim is to find $\boldsymbol{\Delta\Theta_k}$ to minimise the sum of squares of the right-hand side, i.e.,

$$\begin{aligned}
&\boldsymbol{\Delta\Theta_k} = \arg\min \frac{1}{2} \left\| \boldsymbol{r(\Theta_k)} + \boldsymbol{J_k \Delta\Theta_k} \right\|^2. \\
&\text{Let } f\left(\boldsymbol{\Delta\Theta_k}\right) = \min \frac{1}{2} \left\| \boldsymbol{r(\Theta_k)} + \boldsymbol{J_k \Delta\Theta_k} \right\|^2, \\
\text{then set } &\nabla f\left(\boldsymbol{\Delta\Theta_k}\right) = 0 \\
&\Longrightarrow \boldsymbol{J_k^T r(\Theta_k)} + \boldsymbol{J_k^T J_k \Delta\Theta_k} = 0 \\
&\Longrightarrow \boldsymbol{J_k^T J_k \Delta\Theta_k} = -\boldsymbol{J_k^T r(\Theta_k)} \\
&\Longrightarrow \boldsymbol{\Delta\Theta_k} = -\left(\boldsymbol{J_k^T J_k}\right)^{-1} \boldsymbol{J_k^T r_k},
\end{aligned}$$

so we adjust the parameter estimates by $\boldsymbol{\Delta\Theta_k}$.

---

41. The second-order Taylor expansion is applied in Newton's method. However, the Hessian matrices are computationally expensive, so the Gauss-Newton method neglects the term of Hessian matrix and applies the first-order Taylor expansion.

5. Check for convergence. If $\left|\boldsymbol{\Theta_{k+1}} - \boldsymbol{\Theta_k}\right| < \varepsilon$[42], terminate the iteration, otherwise go back to Step 2 with the updated parameters $\boldsymbol{\Theta_{k+1}}$.

This is a non-convex optimisation problem with (potentially) multiple local minima. Consequently, the choice of initial parameter estimates can lead to varied fitted models, or in some cases, may even cause the convergence process to fail (Gadagkar and Call, 2015; An et al., 2019). In software designed to fit the 4PL models, achieving accuracy is contingent upon the initial values of these parameters being close approximations to their ultimate values (Gadagkar and Call, 2015). Therefore, appropriate boundaries on the parameter spaces are normally established based on the sample values if not specified by the users (An et al., 2019).

Based on reasonable initial estimates, we contend that our local estimates provided by **Algorithm 1** are unique over a wide range. To confirm this uniqueness, we implement a grid search algorithm.

Algorithm 2. *ensuring the local uniqueness of estimates of the four-parameter logistic model*

1. Set initial guesses of parameters $\boldsymbol{\Theta_0}$ based on the empirical values[43]—set $\delta_{l,0}$ and $\delta_{u,0}$ as the minimal and maximal values of death rates in the sample i.e. $\delta_{l,0} = \bar{\delta}_l$, $\delta_{u,0} = \bar{\delta}_u$ where $\bar{\delta}_l$ and $\bar{\delta}_u$ are the minimal and maximal values of death rates of geography i across all sampled time periods; $\tau_{c,0}$ as the midpoint of the sample period i.e. $\tau_{c,0} = (t_{max} - t_{min})/2$, and β_0 as 0.1.[44] Set $\rho_0 = 0.5$. Apply **Algorithm 1** to identify the benchmark estimates $\boldsymbol{\Theta}^*$.

---

42. The default convergence criterion of `nl` is 1×10^{-5}.
43. Most computer software also uses the highest and lowest values in the dataset as initial upper and lower asymptotes, such as R packages `drc` and `nplr`. The other two parameters are computed based on the linear transformation of the traditional 4PL model (An et al., 2019).
44. Based on Equation 1.5.4, $\beta_0 = 1$ makes the slope at the inflection point around -1.

2. Define sensible parameter space for each parameter in
   $\boldsymbol{\Theta_{min}} = (\rho_{min}, \delta_{l,min}, \delta_{u,min}, \beta_{min}, \tau_{c,min})$ and
   $\boldsymbol{\Theta_{max}} = (\rho_{max}, \delta_{l,max}, \delta_{u,max}, \beta_{max}, \tau_{c,max})$:

$$\begin{aligned}
&\rho_{min} = 0.4, && \rho_{max} = 0.9\\
&\delta_{l,min} = \lfloor \bar{\delta}_l \rfloor, && \delta_{l,max} = \lceil \bar{\delta}_l \rceil,\\
&\delta_{u,min} = \lfloor \bar{\delta}_u \rfloor, && \delta_{u,max} = \lceil \bar{\delta}_u \rceil^{45},\\
&\beta_{min} = \beta^* - 2 \text{ s.e.}\left(\beta^*\right), && \beta_{max} = \beta^* + 2 \text{ s.e.}\left(\beta^*\right),\\
&\tau_{c,min} = 1870, && \tau_{c,max} = 1925.
\end{aligned}$$

3. Create evenly-spaced grid for each parameter $\boldsymbol{\mathcal{G}_\Theta} \in [\boldsymbol{\Theta_{min}}, \boldsymbol{\Theta_{max}}]$. The step between adjacent grid points is set as 5 for δ_l, δ_u and τ_c, as 0.1 for ρ and as 0.01 for β.
4. Take one point/parameter combination in the grid as the initial guess $\boldsymbol{\Theta_0}$ and apply **Algorithm 1** to get the resulted estimates $\tilde{\boldsymbol{\Theta}}$. If $\left|\tilde{\boldsymbol{\Theta}} - \boldsymbol{\Theta}^*\right| < \varepsilon$, take the next point in the grid and repeat this step, otherwise store the resulted estimates.

Heteroskedasticity of the standard error The error terms u_t in Equation 1.5.6a and ε_t in Equation 1.5.6c are assumed to be heteroskedastic. As displayed in Figure 1.2.3, the effect of random shocks is smaller in later years, probably because of improved public health measures, lower frequency of large infectious diseases outbreaks and the overall lower scale of mortality. Indeed, residual plots confirm that the residuals from both Equation 1.2.2a and Equation 1.2.2b have increased variance in earlier decades relative to later decades (see Figure 1.5.1 for an illustration in terms of crude mortality in London). Therefore, we compute standard errors that are robust to heteroskedasticity.

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45. R packages set the $\delta_l \in \left[\bar{\delta}_l - 2 \text{ s.e.}\left(\bar{\delta}_l\right), \bar{\delta}_l + 2 \text{ s.e.}\left(\bar{\delta}_l\right)\right]$ and $\delta_u \in \left[\bar{\delta}_u - 2 \text{ s.e.}\left(\bar{\delta}_u\right), \bar{\delta}_u + 2 \text{ s.e.}\left(\bar{\delta}_u\right)\right]$. In our dataset, $\left[\lfloor \bar{\delta}_l \rfloor, \lceil \bar{\delta}_l \rceil\right]$ and $\left[\lfloor \bar{\delta}_u \rfloor, \lceil \bar{\delta}_u \rceil\right]$ provide wider ranges.

The variance-covariance matrix of the estimates under homoskedasticity is $\sigma_u^2 \left(\boldsymbol{J}^T \boldsymbol{J}\right)^{-1}$, where $\boldsymbol{J}$ is the Jacobian matrix. In the presence of heteroskedasticity, the heteroskedasticity-consistent estimator (HCE) is

$$\hat{\mathbb{V}}_{\text{HCE}} = \left(\boldsymbol{J}^T \boldsymbol{J}\right)^{-1} \left(\boldsymbol{J}^T \boldsymbol{D} \boldsymbol{J}\right) \left(\boldsymbol{J}^T \boldsymbol{J}\right)^{-1},$$

where $\boldsymbol{D}$ is a diagonal matrix and the t-th element is $D_{tt} = \sigma_{\epsilon,t}^2$, the variance of the error term for the t-th observation. The elements of $\boldsymbol{D}$ are unknown and need to be estimated. Davidson and MacKinnon (2004) proposed the HC3 estimator where $\hat{\sigma}_{\epsilon,t}^2 = \frac{\epsilon_t}{(1-h_t)^2}$, where h_t is the leverage of time t measuring the influence of each data point on the estimation. Therefore, the denominator inflates the variance of outliers so that the HC3 estimator counteracts their undue influence. Davidson and MacKinnon (2004, p. 239) highlight that the HC3 estimator yields the most conservative confidence intervals, particularly advocating for its use in the context of nonlinear least squares (StataCorp LLC, 2023). We employ the HC3 estimator to adjust the standard errors by using an option `` `vce(hc3)' `` built in the `` `nl' `` package (StataCorp LLC, 2023).

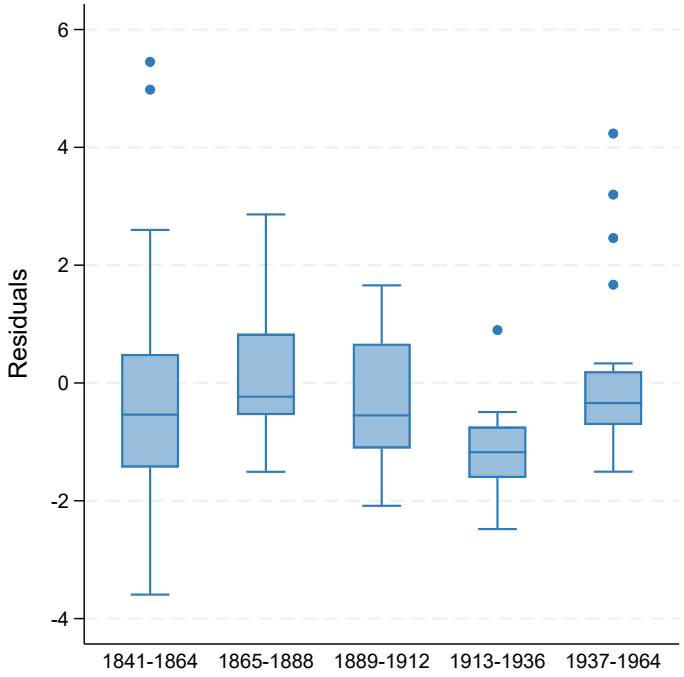

Figure 1.5.1: **Residuals plots of crude mortality for London.** This box plot shows the dynamic of the spread of residuals (u_t in Equation 1.2.2a) across time in London. The heights of the boxes, indicating residual variance, exhibit a decreasing trend over time, reflecting the presence of heteroskedasticity.

1.5.3 LOWESS Regression—Estimated Curves

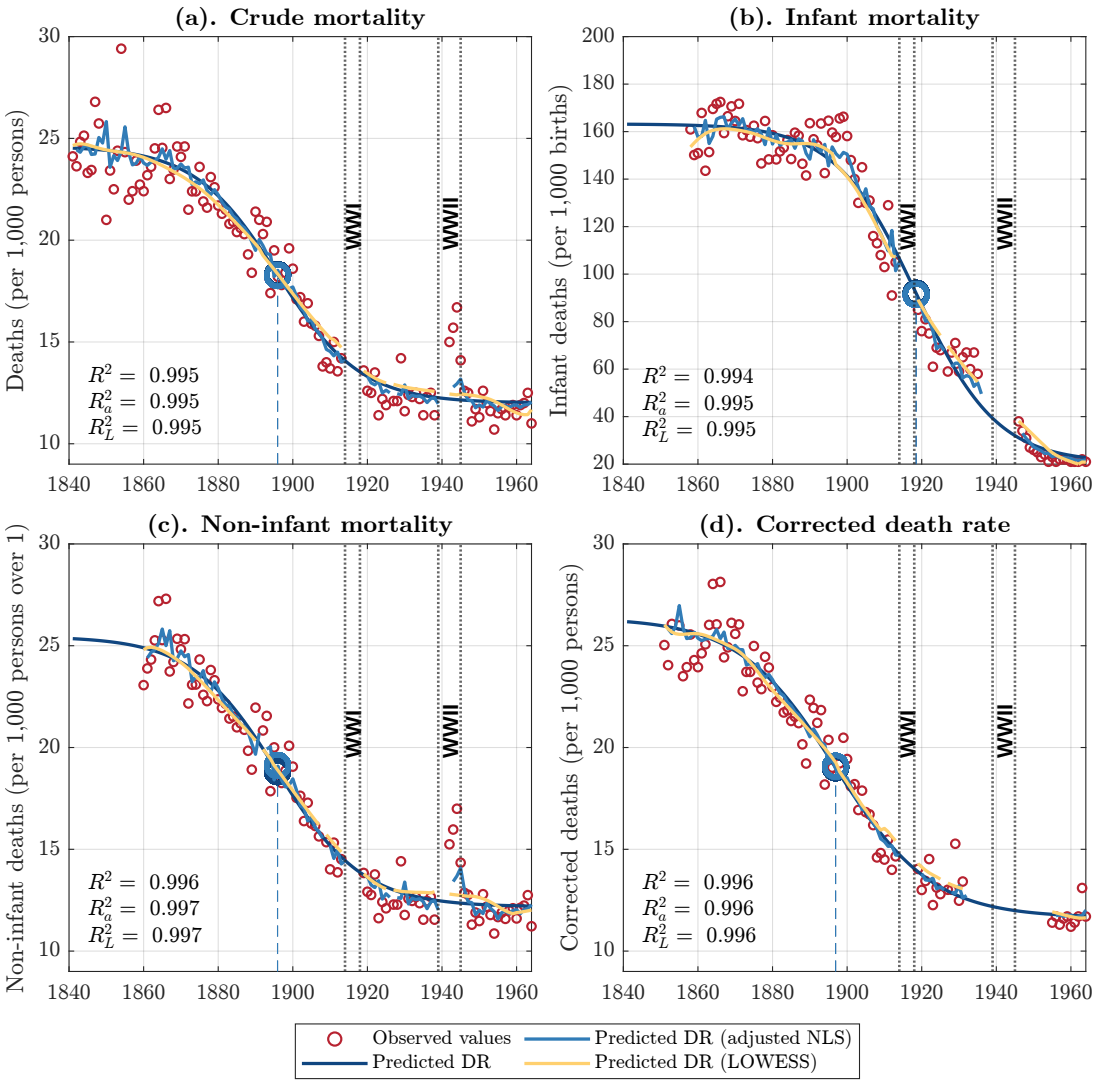

Figure 1.5.2: **Comparative fitted curves of four mortality measures for London.** This figure compares the fitted logistic curve, fitted logistic curve adjusted for serial correlation, and the LOWESS-fitted curve (bandwidth = 0.4). The results are all very similar.

Chapter 2

Heterogeneity in the Dynamics of the Mortality Transition in London

Abstract

By the mid-20th century, mortality inequality had declined substantially in the industrialised areas that had undergone the mortality transition (e.g., London), signalling the final stages of the mortality transition. In this chapter, I examine the evolution of mortality inequality across London's constituent areas from the 1850s to the 1950s, investigating the pathway toward ultimate convergence. To this end, I construct a novel dataset of annual death rates for the small constituent areas of London using data from municipal archives, parish records, and the National Census. The analysis in this chapter reveals that the sigmoid pattern, which applies to London as a whole (as demonstrated in the previous chapter), also broadly applies to its constituent areas. However, there is substantial variation in the timing of rapid mortality reductions, as marked by the estimated inflection points. The model of health progress diffusion with an underlying log-normal income distribution, which was presented in the previous chapter, predicts that areas with generally better socioeconomic conditions—which correlate strongly with the adoption rate of health technologies—experienced earlier mortality declines. This prediction implies an initial divergence in mortality levels between areas during early health technology adoption, followed by subsequent convergence. To provide a further test of this prediction, analysis in this chapter uses the statistical model that expresses the four mortality dynamic parameters from the previous chapter as functions of socioeconomic inequality measures. To quantify area-level socioeconomic disparities, I construct measures capturing the tails of the income distribution in each area, extending the panel dataset to link mortality dynamics to socioeconomic inequalities in each area. The consistently negative and statistically significant marginal effects of socioeconomic conditions on inflection points, robust across multiple measures and specifications, indicate that there was inequality—by decades—in the time advantage that higher social classes had to access the benefits of lower mortality.

2.1 Introduction

It is well documented that by the mid-20th century, there had been a substantial convergence in health inequalities relative to the preceding period in the industrialised areas that had undergone the mortality transition. Understanding the path to this reduction in inequality over the century-long transition is crucial because the long-time dimension implies that those who had earlier access may have enjoyed its benefits decades earlier than others. To illuminate the dynamics of mortality inequalities, in this chapter, I construct a novel dataset for London that tracks mortality trends across areas with varying socioeconomic conditions. This approach allows the estimation between socioeconomic conditions and the timing of mortality declines, thus testing directly the hypothesis that convergence in mortality inequalities started with the mortality transition, against the alternative that mortality inequalities increased during the early phase of the mortality transition.

During the mortality transition period, large disparities in life expectancy, infectious disease mortality, infant mortality, and all-cause mortality have been detected between regions in the US, the UK, and France (e.g., Kesztenbaum and Rosenthal, 2016; Costa and Kahn, 2015; Lee, 1991). Some literature also highlighted the heterogeneity in the timing of the mortality transition process (e.g., Lee, 1991). Socioeconomic conditions are acknowledged as the key driver of the mortality gradient during the transition period. For example, research suggests that individuals with better socioeconomic conditions benefited more from the overall progress since the threat of virulent diseases was eliminated in around 1750 (Bengtsson and Van Poppel, 2011). This advantage manifested through multiple channels: better housing and nutrition associated with higher socioeconomic classes contributed to lower mortality rates and extended life expectancy (see e.g., Cage and Foster, 2002; Kesztenbaum and Rosenthal, 2016; Jørkov, 2015), while greater wealth provided earlier access to public health infrastructure and educational resources that enabled better health practices (Kesztenbaum and Rosenthal, 2017; Floud et al., 2011).

Despite the consensus that there were significant differences between groups in how populations benefited from health progress, there is debate surrounding the dynamics of mortality inequalities. Some studies found convergence trends, with diminishing disparities in France's *départements* (1880s–1910s) and US life expectancy in the early 20th century (Kesztenbaum and Rosenthal, 2016; Bonnet and d'Albis, 2020; Peltzman, 2009). Others observed persistent gaps, such as constant infant mortality differences in the UK (Woods et al., 1988), or even divergence (Lee, 1991). Two competing theoretical frameworks emerge: the "divergence-convergence" hypothesis by Antonovsky (1967) suggests narrowing mortality inequality from 1850 onwards, while the "fundamental cause theory" by Link and Phelan (1995) argues that socioeconomic mortality differentials remained stable as wealthy groups maintained superior access to health resources.

To estimate the relationship between socioeconomic conditions and the timing of mortality declines, I first construct a novel dataset comprising long-term mortality series from the 1850s to the 1930s for 26 constituent areas in London, alongside measures of their socioeconomic distributions from the initial decades of this period. I focus on London as the largest city in the modern world at the time and a pioneering example of urban mortality transition, with detailed historical records available. First, the boundaries of these areas remained consistent over a long time span—a notable achievement given that many existing studies are constrained to shorter time periods due to administrative boundary changes. Constructing these consistent geographic units required extensive archival research to track areas that underwent multiple jurisdictional changes and name modifications across different local authorities. I consulted historical GIS datasets alongside detailed urban planning maps to assess boundary modifications, distinguishing between substantive changes and minor adjustments (such as the inclusion or exclusion of parks and mountains) that would not materially affect our socioeconomic analysis. This meticulous approach made possible the creation of comparable geographic units spanning nearly a century, overcoming a key data limitation. Second, based on administrative data held in municipal archives and the Great Britain Historical Database (GBHD), this dataset contributes to enabling a comprehensive analysis of mortality inequality throughout London's entire transitional period—most studies of inequalities during the mortality transition analyse the dynamics of inequality by comparing disparities over a short period

before and after the transition, leaving the complete evolutionary arc of mortality inequality from the start of the transitional process understudied. Third, by constructing measures of socioeconomic inequalities from National Census data and the Booth reports, and meticulously matching these to areas with mortality records, this dataset also allows analysis of how socioeconomic inequalities shaped the varying patterns of mortality decline across London's constituent areas.

Based on the novel dataset, in this chapter I first estimate the trend mortality for each area of London and find that the reductions in mortality follow a sigmoid pattern, extending the application of the four-parameter logistic model used to describe the city-level dynamics in Chapter 1 to small areas. More importantly, the dynamics of the reductions vary markedly between areas, with over one decade of variation in the timing of the period of rapid reductions. This implies an initial period of increased divergence in mortality differentials between areas, as some areas entered the period of rapid reduction earlier, conditional on initial mortality, before later convergence. In particular, we observe that areas that started with low mortality in the 1850s typically experienced reductions in mortality earlier.

To examine how these variations in mortality dynamics related to socioeconomic conditions, we developed a test of the hypothesis that convergence started as soon as the mortality transition happened in the joint work Angelopoulos et al. (2024). For this purpose, the model introduced in Chapter 1 is revisited and I present its predictions with respect to the inflection point by socioeconomic conditions. The model predicts that areas with a higher proportion of higher-income residents should see the period of rapid mortality reductions begin sooner, suggesting an earlier inflection point in trend mortality. Therefore, this framework suggests an increase in mortality inequality before the period of convergence.

This hypothesis creates a testable prediction about the relationship between socioeconomic conditions and the inflection point—a positive relationship indicates immediate convergence, while a negative relationship suggests initial divergence. As in Angelopoulos et al. (2024), I here use an extension of the logistic model that captures the dependence of each parameter on initial conditions to examine the statistical significance of initial

socioeconomic conditions for the timing of the transition. Estimating the model suggests that populations with higher socioeconomic status, who likely adopted improvements in public health and living conditions earlier, experienced earlier reductions in mortality. The disparity in timing produced a divergence in mortality rates by socioeconomic conditions for several decades before eventual convergence.

The rest of the chapter is structured as follows: Section 2.2 reviews the related literature about mortality differentials and their dynamics, and mortality inequalities that connects these differentials to socioeconomic conditions and their dynamics; Section 2.3 details the construction of a comprehensive panel dataset encompassing London's constituent areas. This novel panel dataset spans a crucial century of demographic transition—from the 1850s through the 1950s—and incorporates four mortality measures to capture the dynamics of mortality for each area. It also includes multiple indicators of socioeconomic inequality, with particular emphasis on measurements at the beginning of the mortality transition period; Section 2.4 presents empirical evidence on the heterogeneity of mortality trends, captured by variations in the estimated inflection points, across the constituent areas, Section 2.5 links the variations in mortality dynamics to heterogeneous socioeconomic inequalities across areas and predicts that mortality inequality increased at the beginning of the mortality transition period before beginning to decrease. This prediction is examined through descriptive analysis in Section 2.6.1 and rigorous statistical testing in Section 2.6.2. Conclusions are provided in Section 2.7.

2.2 Related Literature

In this section, I review two streams of literature: first, mortality differentials and their evolution over time; second, mortality inequalities that connects these differentials to socioeconomic conditions and how they have evolved over time.

2.2.1 Heterogeneity in Mortality

As described in Chapter 1, the mortality transition in industrialised economies played a pivotal role in boosting population well-being, welfare, and growth during the second half of the 19th century and the first half of the 20th century, setting the stage for continued progress in the following century. However, the aggregate progress did not benefit everyone fairly. Extensive research has identified heterogeneities in multiple geographic dimensions during the period of the mortality transition we investigate i.e., from the 1850s to the 1930s.

Apart from obvious heterogeneity in mortality across countries, variations were also noted within countries, including disparities between age groups, races, sexes, and geographic regions.[1] In the 20th century in the US, higher mortality was observed among blacks, foreign-born whites and in urban areas (Haines, 2011). One major reason lies in the gaps in infectious diseases. African Americans suffered from higher infectious disease mortality, and the southern region experienced a later epidemiological transition (Feigenbaum et al., 2019). Differences can also be seen across occupational classes, but the pattern had shifted with changes in the social environment (Haines, 2011). Spatial variations in mortality in the Netherlands were discussed by Wolleswinkel-van den Bosch (1998)—during the last three decades of the 19th century, mortality declined rapidly in the rural areas of the southwestern and northern provinces, while the northern areas slowed down the pace of progress in the 20th century. Variation between geographic regions also existed in France—from the 1860s to 1880s: life expectancy at age five in Paris was four to five years lower than in other French areas and this gap did not close until the 1930s (Kesztenbaum and Rosenthal, 2016).

Heterogeneities have also been uncovered within smaller areas during this period. In the Sundsvall region in Sweden, the urban areas and males, especially unmarried and widowed men were mortality-disadvantaged in the 19th century (Edvinsson and Lindkvist, 2011). In Paris, the gap in life expectancy between the worst and best neighbourhoods (*quartiers*) in the early 1880s reached 15 years. Costa and Kahn (2015) confirmed the large variations in infectious mortality and overall mortality across wards within New York City

1. See discussions about disparities between age groups in e.g., Section 1.2.5 in Chapter 1.

and Philadelphia from 1900 to 1930. Conducting research on smaller regions effectively describes the aggregate area like New York City via a distribution rather than aggregate indicators. Studying small-area differences in the dynamics or timing of the mortality transition allows us to abstract away from large-scale macro-level changes—such as access to technologies or economic shocks—that affect comparisons between cities or countries, making this analysis valuable for understanding underlying causes of differences in the timing of the mortality transition.

To summarise, the existing literature has exposed substantial evidence of mortality heterogeneity within and across different spatial units at various time points (or within a relatively short timeframe). Such heterogeneity can be traced back to the mid-19th century and did not disappear in the following century regardless of the mortality transition. This persistent heterogeneity underscores the need for a systematic examination of how mortality heterogeneity evolved throughout the mortality transition, which I review in the following section.

2.2.2 Dynamics of Mortality Heterogeneity

The time-varying dynamics of mortality across countries have been discussed. Santosa et al. (2014) pointed out the heterogeneous epidemiological transition across continents and countries in terms of timing, pace and underlying mechanisms. For example, most countries like Spain, Japan and Mexico experienced mortality reduction during the 20th century, whereas the health transition in sub-Saharan Africa did not begin until the 1930s (Garenne and Gakusi, 2006).

There are debates about the dynamics of mortality within countries. The differences between *départements* within France decreased from the 1880s to 1910s (Kesztenbaum and Rosenthal, 2016). Similarly, the 1881–1980 period is regarded as the "century of convergence" by Bonnet and d'Albis (2020) when comparing across the same spatial units because from the late 19th century to the 1920s, there was a decrease of about six years in the gap in life expectancy between the top and bottom *départements*. However, before 1880 and after 1980, alternating divergence and convergence in life expectancy among regions are observed (Bonnet and d'Albis, 2020). In the UK, Woods et al. (1988) claimed uniform

infant mortality trends across the country, whereas a different pattern was identified by Lee (1991)—from the 1860s, there was an improvement in the regions with the lowest infant mortality rates during the second half of the 19th century, while rates increased elsewhere such as Scotland and South Wales. This resulted in a divergence of infant mortality until 1921/31, followed by convergence towards equality in the later period. Moreover, comparing the general trend of infant mortality of constituent counties at ten-year intervals from 1861 to 1971, which first increases and then consistently decreases, demonstrates that regions with lower infant mortality rates tend to experience an earlier peak in infant mortality. Germany has also revealed differences in the timing of significant stages in the mortality transition process, where the geographic variations in terms of health indicators mirror the differences in the timing of declines in crude death rate and infant mortality between states (Haines and Kintner, 2000). In the US, inequality in life expectancy between states decreased in the first few decades of the 20th century (Peltzman, 2009).

Furthermore, whether mortality heterogeneities within smaller areas increased or decreased during the mortality transition period lacks a clear consensus. Within Paris, the large gap in life expectancy between neighbourhoods did not close until 1925, which contrasts with the conclusion generated by comparisons between *départements* (Kesztenbaum and Rosenthal, 2016). While larger states showed a divergence in infant mortality rates before 1921/31, as noted by Lee (1991), inequality was found to decrease since the 1890s when analysing smaller areas, over 600 registration districts, in England and Wales (Congdon et al., 2001). In New York City and Philadelphia from 1900 to 1930, Costa and Kahn (2015) identified convergence between wards in terms of infectious diseases like typhoid fever, diphtheria and measles, but all-cause mortality did not exhibit the same pattern. Geographical heterogeneities of the median age at death among elders also remained constant in the Sundsvall region in Sweden from the mid-19th century to the end of the 19th century (Edvinsson and Broström, 2012).

The preceding analysis focuses on mortality heterogeneity (unconditional on SES) and its dynamics across demographic or geographic groups. The next two sections turn to mortality inequality—analysing how mortality rates systematically vary across socioeconomic groups and how these socioeconomic gradients in mortality dynamics have evolved over time.

2.2.3 Health Inequality in Historical Context

Higher socioeconomic conditions are thought to promote better health conditions via a range of mechanisms as a result of enhanced housing, clothing and nutrition. According to Sundin (1995), in Linköping, Sweden, infant mortality rates among illegitimate children were consistently higher than those of the middle class, craftsmen, and lower class from 1752 to 1842, but the relative relationships among these three groups were not stable. In America, the infant mortality rate for families in which the father's annual income was less than 450 dollars was 108 (per 1,000 live births) higher than those where fathers earned over 1,250 dollars (Preston and Haines, 1991). A similar gradient was also discovered in Ipswich, England, at the end of the 19th century, between the elite families employing a domestic servant and labourer families (Razzell and Spence, 2006). Between 1911 and 1961, Glasgow experienced a notably higher infant mortality rate than Edinburgh, with Cage and Foster (2002) suggesting that a substantial portion of this discrepancy can be traced back to the involuntary overcrowding that occurred in the first half of the 20th century. In particular, improvements in housing conditions are thought to have contributed to reductions in mortality. For example, Kesztenbaum and Rosenthal (2016) proxied housing conditions by rents and discovered its close relationship with life expectancy in Paris, revealing that even a slight rise in the proportion of impoverished individuals within a neighbourhood could lead to a decrease in life expectancy by around three years. Furthermore, when comparing different neighbourhoods, it becomes evident that health improvement occurred at varying times along the socioeconomic gradient. The most affluent neighbourhoods experienced significant progress between 1881 and 1891, and again between 1891 and 1901. Meanwhile, the poorest areas saw little to no improvement prior to 1901. It is thought that nutrition also probably played a role in health during the mortality transition. It brought taller statures to states with higher average wealth

in the 19th century US (Carson, 2010). A study of skeletal remains from 19th-century Copenhagen indicates that males were more susceptible to nutritional fluctuations, as evidenced by statistically significant differences in their heights among cemeteries representing the middle/high class, paupers and navy employees (Jørkov, 2015). Higher income also provides the capacity to hedge against certain external health shocks. For example, Lindeboom et al. (2010) showed that individuals from lower social classes were more severely impacted by exposure to the mid-19th century Potato Famine in the Netherlands at birth compared to those from higher social classes, as reflected in their shorter life expectancy.[2]

On the other hand, some empirical evidence for particular time spans and places challenges the importance of socioeconomic conditions in mortality heterogeneity. Southern Sweden and Saguenay in Canada did not exhibit any social differences in working-age mortality during the industrialisation period (Bengtsson and Van Poppel, 2011; Gagnon et al., 2011). Additionally, the causal link between SES and mortality was questioned by (Bengtsson and Van Poppel, 2011), and the effects of the social class of origin on mortality are not statistically significant for either males and females born between 1850 and 1922 in the Netherlands (Schenk and Van Poppel, 2011).

2.2.4 Dynamics of Health Inequality

Unlike the uniform agreement that wealth creates health advantages, how socioeconomic mortality differentials changed over time remains controversial. In the agricultural society until around 1650, the social class differentials in mortality and life expectancy were generally thought to be trivial because of the high prevalence of virulent diseases and the absence of effective treatments (Antonovsky, 1967; Woods and Williams, 1995; Bengtsson and Van Poppel, 2011). According to Antonovsky (1967)'s theory, there were three phases with respect to class mortality inequalities in the Western world: the absence phase prior to 1650, the widening phase between 1650 and 1850, and the narrowing phase from 1860 onwards.[3] This "divergence-convergence" hypothesis has received some theoretical and

2. Also see Scholte et al. (2015); Van den Berg et al. (2016), etc.
3. According to Hinde (2003), the overall mortality experienced three phases: a definite but modest decline in mortality between 1780 and 1830; the decline stopped during the mid-nineteenth century; a decisive period of decline during the 1870s.

empirical support (see Haines et al., 2011). For example, Bengtsson and Van Poppel (2011) identified a convergence in both absolute and relative mortality differences in the US during the 20th century, as well as in the Netherlands and Alghero, Italy after 1900. The variations in life expectancy within Paris also fell, but the rate of convergence was sluggish (Kesztenbaum and Rosenthal, 2016).

An alternative framework for understanding the relationship between socioeconomic conditions and health over time is the "fundamental cause theory" of Link and Phelan (1995). According to this theory, socioeconomic mortality differentials are relatively static over time because, despite changes in the nature of available resources, the upper classes' access to superior resources remained constant (see supportive evidence Breschi et al., 2011, for Alghero in the period 1866–1925). Differences in resource availability between socioeconomic groups thus explain why wealth is the "fundamental cause" of diseases and mortality, even if the mechanisms evolve over time (Link and Phelan, 1995)—during the 19th century, rich individuals had earlier access to public health advancements and improved nutrition; in the 20th century, they invested more time and financial resources in maintaining a healthy lifestyle (Mackenbach, 2012).

The socioeconomic gradient in health becomes particularly evident during transformative periods such as the mortality transition, which saw the development of significant new technologies. As stated in Deaton (2013, p. 1), "*inequality is often a consequence of progress*"—someone always benefits first from new inventions or knowledge. In extreme cases, rich people even hindered the roll-out of new technologies like water infrastructure for the poor to prevent their migration to cities (Feler and Henderson, 2011). Such behaviours cost temporary inequalities (Deaton, 2013, p. 7). Kesztenbaum and Rosenthal (2017) noted that from 1885 to 1913, the most affluent districts in Paris consistently had the highest sewer connection rates, which resulted from decisions by renters, landlords, and the city's sanitation department based their choices on perceived affordability. In fact, the sewage system benefited wealthier Parisians first and did not extend to poorer areas until the 20th century. Consequently, the adoption of sewers exacerbated spatial inequality within Paris during the period of mortality transition. In the contemporary context, transformative technologies also affect inequality. For example, new public health

interventions and programmes (e.g., neonatal intensive care and surfactant therapy) initially benefit those of higher socioeconomic status, leading to early increases in inequity ratios for coverage, morbidity, and mortality. These inequities only improve later when the wealthy have achieved new minimal levels for morbidity and mortality, and the poor gain better access to these interventions (Victora et al., 2000). In addition, accelerated automation and the introduction of new tasks exacerbate inequality by diminishing the employability of unskilled workers in the short run (Acemoglu and Restrepo, 2018). On the other hand, the progress in Artificial Intelligence (AI) is expected to contribute less to widening inequality, since its influence is more uniformly distributed across diverse demographic groups (Acemoglu, 2024).

Despite extensive research on the evolution of health inequalities reviewed above, no studies have explicitly examined whether inequalities steadily converged from the start of the mortality transition or followed a divergence-convergence pattern, a key question this chapter addresses.

London provides a well-suited context for this analysis because a) the late 18th and early 19th centuries saw infectious disease outbreaks mainly in urban areas due to overcrowding because of urbanisation and poor infrastructure which was not yet prepared to support the increased population (Cain and Hong, 2009). Therefore, the subsequent mortality declines also occurred in these regions; b) major health improvements, such as the establishment of sewage systems, began in cities.[4] Cross-city comparisons are less insightful because cities started from different baselines and experienced varying construction progress; c) the health indicators of a country or state capture only key moments and are more susceptible to external influences that can affect the entire region, potentially obscuring underlying heterogeneity. Only a few research conducted within cities because of a lack of historical data. Moreover, among these studies, Costa and Kahn (2015) and Kesztenbaum and Rosenthal (2016), for example, only focused on several decades.[5] We

4. See detailed background of London in Section 1.2.2 in Chapter 1.
5. Costa and Kahn (2015) looked at New York City for 1900–1927 and Philadelphia for 1900–1930. Kesztenbaum and Rosenthal (2016) investigated Paris for 1880–1913.

aim to explore the entire period of the mortality transition, with particular emphasis on the half-century period from the 1870s to the 1920s with significant variations because the pattern of dynamics is time-varying and key timings might be neglected if the time period is restricted.

2.3 Construction of Datasets

This section details the methodology for constructing the panel dataset used to examine the relationship between mortality dynamics and socioeconomic inequalities in constituent areas in London.

Section 2.3.1 first explains the procedure of constructing geographic areas whose boundaries remained consistent for nearly a century, addressing a significant limitation in existing research where administrative boundary changes often restrict studies to shorter time periods. Through extensive archival research, I identified and clarified the geographic boundaries of different administrative divisions, including Registration Districts, Parishes, Districts, Sanitary Districts, and Metropolitan Boroughs, each representing constituent areas under different jurisdictional frameworks and time periods. I then cross-referenced GIS datasets with detailed urban planning maps to determine boundary stability over time and documented any modifications, including their specific rationale and scope. This rigorous approach resulted in comparable geographic units that maintain consistent boundaries across the study period, thereby overcoming a critical data limitation.

Section 2.3.2 documents the construction of three mortality variables: crude mortality, infant mortality, and non-infant mortality. The process requires meticulous data management to address several challenges. Historical sources, primarily the Great Britain Historical Database (GBHD) and the Medical Officer of Health (MOH) reports from 1850 to 1935, need extensive harmonisation to match the consistent geographical units constructed in Section 2.3.1. The data construction process addresses measurement challenges including institutional mortality bias, digitisation errors in MOH reports, and missing infant mortality statistics through careful cross-validation and compilation of multiple sources.

Section 2.3.3 describes the development of measures capturing socioeconomic inequalities from two sources. The data draws on the Great Britain Historical Database (GBHD) and the social surveys of Booth (1889) and Llewellyn-Smith and Booth (1930). While Booth's granular mapping of poverty in Victorian London is instrumental for understanding the socioeconomic landscape, its empirical application remains limited. Areas covered in these two sources also require spatial matching to the consistent geographical units constructed in Section 2.3.1. From these historical records, I construct measures capturing the tails of socioeconomic distribution to quantify inequality.

2.3.1 Construction of Consistent Geographic Areas

To analyse the mortality dynamics between the middle of the 19th century and the first decades of the 20th century, I study geographies that remained consistent over time and for which mortality and socioeconomic data were available. A well-acknowledged obstacle to the use of historical time series for London areas is that the boundaries of administrative areas in London experienced multiple and significant changes from the late 19th century to the early 20th century (Rafferty, 2021). I thus identify and trace over time areas for which the available data refer consistently to the same geographic unit. I provide an overview of how I worked here.

2.3.1.1 Geographic Units Based on Registration Districts

During the 19th century, statistical data on vital events (births, deaths and marriages) were collected and reported at the level of Registration Districts in the form of censuses and reports from the Registrar-General.[6] These Registration Districts serve as the foundation for the creation of the areas I study. The shapefiles for these districts are sourced from the Great Britain Historical Database (Southall et al., 2023),[7] and background information on each area over time can be found in the Great Britain Historical GIS Project

6. A registration district, established under the Births and Deaths Registration Act 1836 and later legislation, was a geographic area set up to systematically record (i.e., register) births, marriages, and deaths for civil purposes.
7. Source: Great Britain Historical Database: Digital Boundaries for Registration Districts of England and Wales, 1851–1911 (accessed February 2024), http://doi.org/10.5255/UKDA-SN-9032-1.

(GB Historical GIS / University of Portsmouth, 2024)[8] and UKBMD. I keep all Registration Districts from 1850 to 1900, but aggregate data from smaller constituent areas to the larger ones encompassing them, if these can be traced consistently over time. For instance, the area in our sample named "Southeast" comprises the combined Registration Districts of Greenwich and Lewisham between 1837 and 1868, and Greenwich, Lewisham, and Woolwich between 1868 and 1900. If I can have the required data for both a larger area and its constituent smaller areas, I keep the smaller areas if I have data for at least two consistently over time, otherwise, I keep the larger area. In total, I have identified 38 areas for which the boundaries did not change between 1850 and 1900. I ensure that the areas I include in the panel analysis do not overlap.

2.3.1.2 Geographic Units Based on Parishes and Districts

The Metropolis Management Act 1855 created the Metropolitan Board of Works (MBW), with the primary task of upgrading infrastructure. The MBW worked via organising London into smaller areas which were based on pre-existing *parishes*, in many cases combining parishes into larger *districts*. I have a map for parishes and districts under the jurisdiction of the MBW for the year 1884,[9] and background information on all parishes over time can be found in the Great Britain Historical GIS Project (GB Historical GIS / University of Portsmouth, 2024).[8] In the second half of the 19th century, the boundaries of almost all parishes or districts remained unchanged unless specifically noted otherwise. Alongside the MBW, the Act required the relevant local authorities to appoint Medical Officers of Health (MOH) with the responsibility to conduct sanitary works and collect relevant population and health statistics. They prepared annual MOH reports for parishes or districts from 1855 onwards.

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8. Source: GB Historical GIS / University of Portsmouth, *A Vision of Britain through Time* (accessed February 2024), https://www.visionofbritain.org.uk.
9. Data Source: London, England: parishes, districts, and extra-parochial places, 1884 (Raster Image), Harvard University, Harvard Map Collection, https://hgl.harvard.edu/catalog/harvard-g5754-l7f7-1884-s3-sh-1/citation.

By comparing the MBW map of parishes and districts with the shapefiles for Registration Districts, I can map all 38 Registration Districts to areas under the jurisdiction of the MBW—parishes, districts, or a combination thereof—between 1850 and 1891. For these 38 Registration Districts I can find MOH data from reports, either directly for the area for which the MOH was appointed to or for a sub-division of that area, because MOH reports in some cases also included information for constituent parishes. There exist more parishes and districts, other than these included in the 38 areas. By reviewing the MOH reports for larger areas, I can in some cases gather data on their sub-divisions.[10] I have identified 4 extra areas, corresponding to parishes or their combinations, for which I have MOH reports separately from the other 38.

Previous scholars dedicated significant efforts to utilising parish-level data. Based on the CGKO (Cambridge Group Kain Oliver) map (Kain and Oliver, 2020) and the GIS developed by Burton and Southall (2004), *The Occupational Structure of Britain c.1379–1911* research programme created the 1851EngWalesParishandPlace (EWCP) GIS dataset for boundaries of parishes and smaller places in 1851 (Satchell et al., 2023). To make use of the occupation data in the earlier decades, Shaw-Taylor et al. (2010) linked the occupation dataset to the GIS data, which is called "spatial matching". By utilising base units smaller than individual parishes, often to identify very small plots of land, Kain and Oliver's boundary data facilitates the comparison of diverse sources at the most finely-grained geographical scale. Next, they employ a *Transitive Closure Algorithm* to determine the lowest common unit between parish polygons in different years—*mappable units*. The position and area of mappable units as displayed on a map fluctuate based on the sources consulted, indicating that they do not correspond to any singular system of land boundary or to boundary locations at a determined time frame (Kitson et al., 2012). Also using this method, Heblich et al. (2020) obtained 183 mappable units for the LCC from 1801–1891. Then, for the years before 1921, they allocate the data of the mappable units to the larger 1921 areas, by weighting the values for each mappable unit by its share of the geographical area of the 1921 areas.

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10. For example, data on the parish of Battersea is sourced from the Reports of the Medical Officer of Health for Wandsworth District, which encompassed Battersea, Clapham, Wandsworth, Putney, Streatham, Tooting, and Balham.

I downloaded the shapefile of parishes and smaller places in 1851 from UK Data Service (Satchell et al., 2023).[11] By comparing it with the map for parishes and districts for the year 1884, I am aware of changes to boundaries that took place during the first half of the 19th century, e.g., the Parish of Mile End Old Town. This comparison acts as additional evidence supporting (GB Historical GIS / University of Portsmouth, 2024).

2.3.1.3 Geographic Units Based on Sanitary Districts

Following the enactment of the Public Health Acts of 1872 and 1875, 41 sanitary districts were formed, in most cases corresponding to parishes, each overseen by a sanitary authority tasked with overseeing public health issues within its area.[12] In London, the sanitary districts corresponded to the areas defined and managed by the MBW.[13] As a result, despite the formal establishment of sanitary districts post-1872, the preparation of MOH reports continued for the same geographic units as outlined in the previous section. The boundaries of Sanitary Districts did not change until 1900 (GB Historical GIS / University of Portsmouth, 2024). Using a map of sanitary districts, downloaded from the MOH report of London County Council 1892 P13 (image),[14] I can relate the sanitary districts to the 42 areas constructed above. In some cases, a sanitary district corresponds to one area, in others a combination of sanitary districts corresponds to one of the 42 areas. The administrative County of London, corresponding to Inner London today, was established as part of the Local Government Act 1888 and its local authority is the London County Council (LCC). After 1892, the LCC began to compile data from all of London's sanitary authorities, enabling a comparison of performance throughout the city.

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11. Since the shapefile includes polygons of various administrative units including Registration Districts, Registration sub-districts, and parishes, I aggregate the polygons of smaller places with the same parish name indicated by `PAR`.
12. In 1894, two boundary changes happened and there were 43 sanitary districts. Firstly, the parish of Plumstead was removed from the district to be governed by a parish vestry and the district was renamed Lee. Secondly, the Hackney District formed under the Metropolis Management Act 1855 was abolished. Under the Metropolis Management (Plumstead and Hackney) Act 1893, the Civil Parishes of Hackney and Stoke Newington separated again, and both remained as Met.B.s after 1901. Neither change affects our areas.
13. Data Source: Table 4. 'Sanitary Areas, Distinguishing Those Under Vestries, Board of Works, and Other Authorities; Area; House and Population in 1891, and Population in 1881.' In *1891 Census, Population. Registration Areas and Sanitary Districts, England and Wales, Vol. II.*
14. Data Source: Report of the Medical Officer of Health for London County Council 1892, image P13 (accessed February 2024), https://wellcomelibrary.org/moh/report/b18252412#?c=0&m=0&s=0&cv=0.

2.3.1.4 Geographic Units and Data Sources, 1850–1900

In summary, since 1850, I can track over time and until 1900, 42 consistently defined areas of London. I gathered mortality data from several sources, corresponding to these areas: from the UK Data Service, where the areas are recognised as Registration Districts; from MOH reports dated between 1855 and 1892, in which these areas are considered parishes/Districts; and from MOH reports for LCC from 1892 to 1900, where these areas are acknowledged as Sanitary Districts. After 1900, data are recorded at the level of Metropolitan Boroughs, which I can map to the earlier areas, as I explain below.

2.3.1.5 Geographic Units Based on Metropolitan Boroughs

The London Government Act 1899 divided the County of London into 28 metropolitan boroughs (Met.B.s), with effects taking place from 1901 onwards. After 1901, demographic and health data were reported for Met.B.s. The shapefile of Met.B.s is downloaded from ONS Geography (2023).[15] I imported the shapefiles for Registration Districts and Met.B.s into QGIS and confirmed that 22 Met.B.s in effect correspond to 22 of the 38 Registration District areas; in addition, by using the MBW map I confirm that 4 additional Met.B.s correspond to the 4 extra areas I constructed based on parishes. There were very small changes between Registration Districts and Met.B.s in 1901. In most cases, these refer to a small number of streets changing between Registration Districts and Metropolitan Boroughs and in some cases that appeared to have bigger changes, we confirmed, following examination of the urban planning characteristics of each area, that these refer to geographic features that should not have a sizable impact on population numbers.[16] The 16 Registration Districts that were not mapped to a Met.B. are generally smaller than the Met.B.s. I can find data for them post-1901 by consulting the individual MOH reports for the Met.B.s to gather data on respective "sub-divisions" that can be mapped

15. Data Source: LAD (Dec 1961) in England and Wales BFC Boundaries (accessed February 2024), https://geoportal.statistics.gov.uk/datasets/ons::lad-dec-1961-in-england-and-wales-bfc-boundaries/about.

16. This is done using *Digimap*, providing details of urban planning from 1860s to 1920s. For example, by importing the shapefiles of Registration Districts and Metropolitan Boroughs into Digimap, I observe that the changes in the boundary of the Registration District of Hampstead relative to the Metropolitan Borough of Hampstead, refer to modifications to the northern and eastern borders that traverse hills and ponds. Consequently, I consider the Registration District of Hampstead and the Metropolitan Borough of Hampstead as a single polygon.

to Registration Districts.[17] Finally, by comparing the shapefile of the Met.B.s, the map of parishes, and the shapefiles of Registration Districts, I discovered that the combined area of the Met.B. of Greenwich and the Met.B. of Deptford, excluding Chalton & Kidbrooke (Kidbrooke detached included), matches the area of the Registration Districts of Greenwich and the Greenwich District. Therefore, this area can be traced back to 1850, making the total number of areas 43. Inspection of the time series plots for mortality rates for these 43 geographic areas between 1850 and 1935 does not suggest a structural change after 1901. In addition to these 43 areas, there are six additional Met.B.s that do not match the previously identified areas, making 1901 the earliest year for which data is available for these boroughs. Ultimately, 49 areas are included in our sample.

2.3.2 Construction of Mortality Dataset

The primary mortality data source is the reports of the Medical Officer of Health (MOH) as introduced in Section 1.2.3 in Chapter 1. Besides the London County Council, other local authorities also published the MOH reports before and after the establishment of LCC. I scrutinised the MOH reports between 1850 and 1935 to identify the local authority units that they refer to. Between 1855 and 1891, the reports were issued by the Medical Officer of Health (MOH) for parishes and districts as discussed in Section 2.3.1.2. Matching the report with its corresponding area on the map requires accurate identification of the local authority. I normally rely on the cover page of the original report. This is because while the Wellcome Library assigns titles to each report, there can be cases of incorrectness in these titles. For example, "Lewisham 1874" is actually for the Limehouse District. In addition, the names of the local authorities sometimes changed over time. For example, what was known as the Registration District of St George the Martyr in the reports of 1896 and 1897 is also referred to as St George (Southwark). This information is sourced from GB Historical GIS / University of Portsmouth (2024), and the geographical area reported in the MOH reports is also reviewed for additional verification when available. As described in Section 2.3.1.3, the London County Council (LCC) started producing MOH reports at the city level and the level of sanitary districts in 1892. From 1901

17. See Data Appendix for details on minor changes in boundaries between "sub-divisions" and corresponding Registration Districts.

onwards, the Metropolitan Boroughs (Met.B.s) along with the City of London took over the responsibility for sanitary matters from the sanitary districts, continuing until their abolition in 1965. The MOH reports of LCC facilitate data collection significantly by consolidating statistics from various constituent areas in London.

The majority of MOH reports for London have been digitised through OCR technology, and the resulting Excel spreadsheets can be downloaded. However, due to the challenges in printing and preserving historical documents, these downloaded spreadsheets often feature numerous inaccuracies and have missing information. Therefore, I cross-checked the information included in the OCR-constructed tables against the original reports. I found many errors, especially for the digitised reports of the earlier decades, although this differs across the areas. In our datasets, I have corrected these errors by manual checking of all entries and these contain the information as recorded in the MOH reports. As we explain in more detail below, I have also worked with the MOH reports directly because these provided the information to adjust crude mortality rates for deaths in institutions. The MOH reports for larger areas ("parent areas") often contain information on smaller constituent areas ("child areas") and I collect this as well when available. For example, data for Mile End Old Town, Whitechapel, St George in the East, and Limehouse prior to 1892 were gathered from their respective Medical Officer of Health (MOH) reports, while data post-1892 for these areas were obtained from the MOH reports of the Met.B. of Stepney, of which they were constituent parts (see Figure 2.3.1).

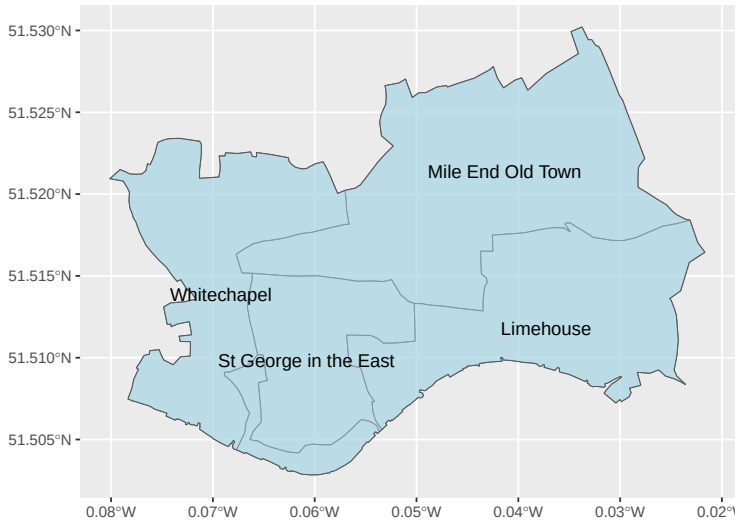

Figure 2.3.1: Map of the Met.B. of Stepney and its constituent areas

In addition to the MOH reports, I also obtained information through existing databases accessible through the UK Data Service:

(a) **`cause_lon_ann_corr.xlsx`**, Great Britain Historical Database (GBHD): Vital Statistics for England and Wales 1840–1911 (Southall et al., 2022). This dataset was transcribed from the Annual Reports of the Registrar General and then corrected for deaths in institutions by Graham Mooney (Mooney et al., 1999). The data covers population, number of deaths, and number of deaths from over 50 specific causes in all Registration Districts in the County of London from 1860 to 1884, all sanitary districts from 1885 to 1900, and all Met.B.s from 1901 to 1920.

(b) **`mort_age_ew_reg_ann.xlsx`**, GBHD: Vital Statistics for England and Wales 1840-1911 (Southall et al., 2022). This dataset was transcribed from the Annual Reports of the Registrar General for England and Wales. It includes the number of births and deaths of persons aged under 1 in all Registration Districts, Sanitary Districts and Met.B.s for time periods 1840–42, 1850–52, 1860–82, 1890–92, 1900–02, and 1908–10.[18]

(c) **`vital_ew_lgd_ann.xlsx`**, GBHD: Vital Statistics for England and Wales 1911–1973 (Southall and Mooney, 2022). This dataset was transcribed from the Annual Reports of the Registrar General.[19] It includes the number of births, number of infant deaths and infant mortality for Met.B.s from 1911 to 1973.[20]

(d) **`vital_ew_reg_ann.xlsx`**, GBHD: Vital Statistics for England and Wales 1840–1911 (Southall et al., 2022). This dataset was transcribed from the Annual Reports of the Registrar General. It includes the number of births among other demographic variables.

To compile these datasets, I systematically assigned unique identifiers to the consistent geographic units constructed according to Section 2.3.1. Subsequently, I mapped these identifiers to correspond with the Registration Districts, sanitary districts, and Met.B.s featured across the three datasets.

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18. I do not use data for years before 1850. The number of births is not available before 1850.
19. Data from 1921 onwards are taken from the Registrar General's Statistical Review of England and Wales.
20. I do not use data for years after 1935.

2.3.2.1 Crude Mortality

Crude mortality is defined as the number of deaths per 1,000 persons. It is collected from the MOH reports and complemented by the UK Data Service, source (a).

There has been a concern about the geographical precision of mortality data before 1911 in the Registrar General's Returns, due to the failure to reallocate institutional deaths to place of residence. Therefore, death rates in areas hosting public institutions such as hospitals, particularly those specialising in infectious diseases, were likely exaggerated (Mooney et al., 1999; Rafferty, 2021). There are two alternative methods to "correct" the consequential distortions. The first method takes advantage of the comprehensive details in the MOH reports. As Rafferty (2021) demonstrated, although the quality of information depends on the authors' preference, there were attempts to redistribute institutional deaths of non-residents. Therefore, when collecting data from the MOH reports, I was conscious of this problem and aimed to select the most impartial figures available. For example, the Report of the MOH for Shoreditch, Parish of St. Leonard 1891, P160 stated, "... 3,163 deaths were registered as having occurred in this Parish; from these are deducted the deaths of 421 non-parishioners who died in various institutions and elsewhere in the District, but the deaths of 800 parishioners who died in other parts of the Metropolis have to be added, making a total of 3,042 inhabitants of Shoreditch who died during the year. This is equivalent to an annual death rate of 24.8 per 1,000 inhabitants". In this case, I record 3,042 as the number of deaths and 24.8 as the crude mortality rate. Although certain reports did not fully execute the reallocation process, I continue to rely on the figures provided by the MOH. This trust stems from their direct access to contemporary information, which seems to be more dependable than any retrospective analyses (Rafferty, 2021). For example, the Report of the MOH for Kensington 1879, P46 stated, "of these 110 deaths, 106 were of non-parishioners, which, following custom, I retain in the vital statistics by way of compensation for the deaths of parishioners that may have taken place in public institutions or elsewhere outside the parish". After 1885, our confidence in

the data from the MOH reports is well-founded. This is because as stated in Report of the MOH for LCC 1892 P9, "*in the year 1885 the Registrar-General began to distribute to the several London sanitary districts to which they belong the deaths from all causes and from certain zymotic diseases occurring in London*".

For each observation, I also collected the population from the MOH reports. To ensure the consistency of definition in population, I use "estimated population at the middle of the year". In cases where the crude mortality rate is not provided, it is calculated by dividing the number of deaths by the population (multiplied by 1,000). When calculating the crude mortality data for "parent" areas by summing up smaller consistent areas, both population and the number of deaths of smaller areas are necessary.

As discussed in Section 2.3.1.5, we can trace 43 geographic areas between 1850 and 1935. From these, I successfully collected or calculated crude mortality data for 24 areas from the MOH reports. Before 1901, 3 of these 24 areas were identified as "sub-divisions" within larger parishes/districts, with their data sourced from their "parent" areas. One area was classified as a "parent" region, compiling its data by summing up information from its "sub-division" areas. The remaining 20 areas had data directly gathered from their respective MOH reports. After 1901, 11 out of the 24 areas were "sub-divisions" of larger Met.B.s, with their data acquired from "parent" areas. 2 areas were recognised as "parent" regions, assembling their data by aggregating results from the "sub-division" areas. The data for the other 11 areas as well as the 6 extra Met.B.s were directly obtained from the MOH reports for the LCC.

The second method to "correct" the distortions induced by public institutions uses indirect estimation techniques. Mooney et al. (1999) redistributed cause-specific deaths using institutional records and their "corrected" dataset is publicly accessible through the UK Data Service, source (a). I matched the Registration Districts, Sanitary Districts and Met.B.s in this dataset[21] to the 38 corresponding Registration Districts constructed as explained in Section 2.3.1. Using the two variables `` `total' `` and `` `pop' `` in this dataset, the crude mortality is calculated as `` `total'/`pop' `` $\times 1,000$.

21. In this dataset, the column `row_type` records the "type of unit", including Registration Districts, Sanitary Districts and Metropolitan Boroughs.

Data for all of the 38 areas can be obtained before 1901, in which 8 are "parent" areas. After 1901, 27 out of 38 areas continue to have traceable data up until 1920.

Despite the relatively large sample size, this dataset is limited in various perspectives that render its standalone use unsuitable for this paper. First, the dataset covers the years 1860 to 1920. The absence of data for the two decades prior to 1860 and following 1920 hinders the ability to estimate non-linear trends in several areas, particularly where the inflection points occur relatively early or late. Second, if the "parent" area is represented in the dataset for certain years, data for the "sub-divisions" is not accessible. For example, while data for the Met.B. of Stepney before 1901 can be compiled by aggregating the "child areas", the data for the four constituent areas post-1900 is not available.[22] Therefore, unlike the data I collect from MOH reports, this dataset alone does not reflect the heterogeneity within these "parent areas". Third, despite meticulous redistribution of the patient's residence and the hospital's catchment area based on hospital registers as detailed in Mooney et al. (1999), the potential for inaccuracies remains. This is due to the often ambiguous addresses listed on death certificates and the necessity of relying on mathematical approximations. Consequently, while this dataset is more systematically organised, it falls short in accuracy compared to the data sourced from the Medical Officers of Health, who possessed the most comprehensive understanding of the areas under their jurisdiction.

Therefore, to obtain crude mortality, I use the data collected from the MOH reports as the foundational dataset, supplementing any missing values with the crude mortality rates from Database (a). Ultimately, I obtained 2,694 crude mortality rates for 52 areas.[23] See Table 2.3.1 and Table 2.8.1 in Appendix 2.8.1.1.2 for detailed information on data availability. See Figure 2.8.1 in Appendix 2.8.1.1.1 for the consistent areas constructed and spatial distribution of crude mortality across these areas in London.

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22. This dataset encompasses a wider range of areas compared to the MOH reports because it successfully incorporates several peripheral London areas. For instance, MOH reports for areas like Poplar in the northeastern part of London are not available before 1893, yet they are included in this dataset.
23. These areas include both "parent areas" and "child areas" which may overlap, but I make sure that overlapping geographies do not appear simultaneously in any regressions. The baseline results are based on the combination of two datasets, and I confirm the robustness of results through the exclusive use of MOH data (Angelopoulos et al., 2024).

Beyond crude mortality, when comparing mortality rates across different areas, it is useful to consider potential differences in the age and sex structure of populations. This is due not only to the varying mortality rates but also to different health behaviours among various age and sex groups (Hollingshaus et al., 2019; Salomon and Murray, 2002). The absence of age-sex specific mortality counts and population denominators for London's constituent areas in the nineteenth century precludes the construction of annual corrected death rates (see Section 2.8.1.1.3 in Appendix 2.8). Instead, I examine infant and non-infant mortality rates as alternatives. This is because the primary interest in demographic composition stems from the possibility that crude mortality transition might be primarily driven by infant mortality decline, whereby areas with higher proportions of infants would experience earlier mortality transitions regardless of their socioeconomic conditions.

2.3.2.2 Infant Mortality

Infant mortality is defined as the number of infant deaths (deaths of children under 1 year) per 1,000 births. It is obtained by combining the MOH reports, Database (b), Database (c), and Database (d).

In Database (b), I keep `` `tot_birth' ``—total number of births; `` `d_0' ``—the deaths of persons aged under 1. So infant mortality for Met.B.s from 1911 to 1935 is calculated as `` `d_0'/`tot_birth' ``$\times 1,000$. In Database (c), which covers the time periods 1840–42, 1850–52, 1860–82, 1890–92, 1900–02, and 1908–10, I identify 27 areas that share identical boundaries out of the 49 consistent areas established. I keep `` `births' ``—total births; `` `i_death' ``—deaths under 1 year, and `` `i_rate' ``—death rate among infants under 1 per 1,000 births.

Based on the compiled dataset, I aggregate the number of births and the number of infant deaths across small areas to obtain the number of births and the number of infant deaths in the respective "parent" areas. Additionally, I filled as many gaps as possible between 1850 and 1910 by collecting data from the MOH reports for 38 areas that functioned both as Registration Districts and parishes, along with an additional 4 parishes and districts. During this process, our initial step was to collect data on infant mortality rates that were directly provided. If such data were unavailable, I then collected the number of

infant deaths and calculated the infant mortality rates using the variable `birth_tot' in Database (d), which is the number of births for all Registration Districts from 1850 to 1910. I do not have information on how to adjust infant mortality figures from the Registrar General reports for mortality taking place in institutions.

Ultimately I obtained 1,360 infant mortality rates for 49 areas, of which 438 were collected from the MOH reports. See Table 2.3.1 and Table 2.8.1 in Appendix 2.8.1.1.2 for detailed information on data availability.

2.3.2.3 Non-Infant Mortality

Non-infant mortality is defined as the number of non-infant deaths above 1 per 1,000 persons above 1, which is calculated as:

$$\text{non-infant mortality} = \frac{\text{crude mortality} - \frac{\text{number of infant deaths}}{\text{population}}}{1 - \frac{\text{number of births} - \text{number of infant deaths}}{\text{population}}},$$

in which all elements have been obtained as discussed above—crude mortality and population is obtained as discussed in the above section about crude mortality; number of births is obtained from Database (d) and Database (b), with missing values filled by using the MOH reports; number of infant deaths is obtained from Database (b) and Database (c), with missing values filled by using the MOH reports.

Ultimately I obtained 1,016 non-infant mortality rates for 31 areas. See Table 2.3.1 and Table 2.8.1 in Appendix 2.8.1.1.2 for detailed information on data availability.

Table 2.3.1: Data availability and summary statistics of mortality measures, constituent areas in London

Variable	# of available areas	Min	Max	Mean	Std. dev.
Crude mortality	52	8.15	42.42	18.65	5.16
Infant mortality	49	38.79	264.55	141.05	41.06
Non-infant mortality	31	7.71	43.84	21.63	5.33

2.3.3 Construction of Socioeconomic Dataset

I use a range of variables to capture the socioeconomic conditions of London and its constituent areas in each decade from the 1860s to the 1880s. Firstly, I use measures of the proportion of male residents who belong to different professional classes. This information is extracted from National Census data for 1861 and 1871 which are publicly accessible through the UK Data Service:

(a) `occ_1861_ew.xlsx`, GBHD: Census Data, Occupational Statistics, 1841-1991 (Gatley et al., 2022). This dataset was transcribed from the Census of England & Wales Census, 1861. *Population Tables, Vol II, Part 1*, p. 21-34, Tables 11 & 12, "Occupations of males aged 20 years and upwards in districts" & "Occupations of females aged 20 years and upwards in districts".

(b) `occ_1871_ew.xlsx`, GBHD: Census Data, Occupational Statistics, 1841-1991 (Gatley et al., 2022). This dataset was transcribed from the Census of England & Wales Census, 1871. *Population Abstracts, Vol III*, p. 22-23, Table 13, "Occupations of males and females aged 20 years and upwards in superintendent registrars' districts, arranged in classes and orders".

From these two datasets, I calculated the proportions of six occupational classes for males—"professional", "domestic", "commercial", "agricultural", "industrial", and "indefinite and non-productive"—by dividing the respective number of males in each class by the total number of working-age males. Figure 2.3.2 displays the occupation structure in London for the years 1861 and 1871, highlighting the persistent social class inequality. Throughout this decade, the occupational distribution remained largely unchanged. Table 2.3.2 lists the occupations classified as the "professional" class in 1861 and 1871 Censuses. This variable proxies the upper tail of the socioeconomic distribution.

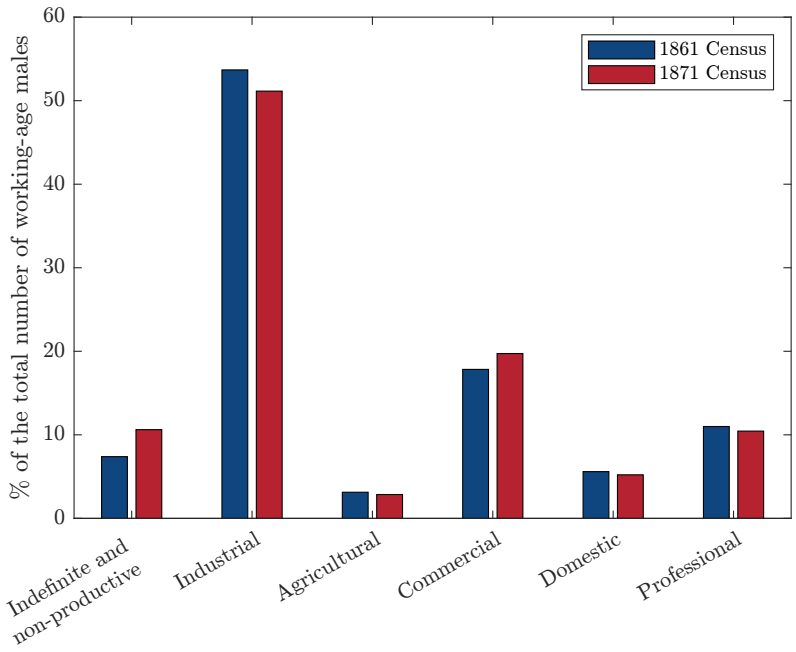

Figure 2.3.2: **Occupation structure in London, 1861 and 1871.** This figure shows the proportions of six occupation groups of the total number of working-age males in London reported in the 1861 and 1871 censuses. The occupation structure did not change significantly in this decade.

Table 2.3.2: Occupational classification of the professional class, 1861 and 1871 Censuses

	Orders	Sub-orders
1	Persons engaged in the general or local government of the country	Officers of National Government Officers of Local Government Officers of East India and Colonial Service
2	Persons engaged in the defence of the country	Army Navy
3	Persons engaged in the learned professions or in literature, art and science (with their immediate subordinates)	Clergymen, ministers, and church officers Lawyers, law court officers, and law stationers Physicians, surgeons, and druggists Authors and literacy persons Artists Musicians, teachers and music Actors, actresses Teachers Scientific persons

[a] Source: Census of England & Wales Census, 1861. *Population Tables, Vol II, Part 1*, p. 12, Table 8, "Occupations of males and females under 20, and 20 years of age and upwards—in classes, orders, and sub-orders"; Census of England & Wales Census, 1871. *Population Abstracts, Vol III*, p. 12, Table 10, "Occupations of males and females under 20, and 20 years of age and upwards—in classes, orders, and sub-orders".

I gathered data on the proportion of the professional class in 1861 across 36 areas, including 9 "parent" areas where the data was derived by summing up the figures from the "child" areas; similarly, for 1871, I collected this measure for 27 areas, with 9 of these being "parent" areas. I also use the proportion of the "industrial" class (see Table 2.8.3 in Appendix 2.8.1.2 for the list of the occupations) to gauge the lower tail of the socioeconomic distribution. Furthermore, I use the ratio of the professional class to the industrial class to indicate inequality.

Another set of variables measuring socioeconomic conditions is based on reports of Booth (1889) and Llewellyn-Smith and Booth (1930). Charles James Booth is generally regarded as an important contributor to the progress of social science and the measurement of social deprivation (Davies, 1978; O'Day and Englander, 1992). He conducted a thorough inquiry into the conditions of workers and the "degree of poverty" in London from 1886 to the early 20th century. The first edition of the survey named "*Life and Labour of the People in London*" was published in 1889 (Booth, 1889).[24] It pictured Victorian London and examined the level of deprivation with multiple social indicators which are inspiring even for modern research (Davies, 1978). One of the most striking products of the inquiry which contributes to the progress of social science (O'Day and Englander, 1992) is the "Poverty Map of 1889".[25] To create the map, each street was classified into an ascending series of seven social and economic grades described by letters from **A** to **H**. Then, the distribution of poverty was visualised using a colour code. For example, black is used on streets classified as Class **A**. Table 2.3.3 lists the short descriptions of each class.

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24. Charles Booth's "Life and Labour of the People in London" is a 17-volume work published between 1889 and 1903.
25. Source: LSE Library's Charles Booth archive (accessed March 2024), https://booth.lse.ac.uk/map/14/-0.1174/51.5064/100/0..

Table 2.3.3: Classifications of social classes in two surveys

Booth's Survey			New Survey	
in poverty	**A**	the lowest class of occasional labourers, loafers, and semi-criminals	**P**	poverty class
	B	casual earnings		
	C	intermittent earnings		
	D	small regular earnings		
in comfort	**E**	regular standard earnings	**U**	unskilled
	F	higher class labour	**S**	skilled
	G	lower middle class	**M**	middle class
	H	upper middle class		

This classification system was established not solely based on income but was primarily influenced by the "living standard". Class **A** consists of some occasional labourers, street-sellers, loafers, criminals, and semi-criminals. Booth sympathetically described their living, stating, "*their food is of the coarsest description, and their only luxury is drink. It is not easy to say how they live; the living is picked up, and what is got is frequently shared; when they cannot find 3 pences for their night's lodging, unless favourably known to the deputy, they are turned out at night into the street, to return to the common kitchen in the morning*". Class **B** is classified as "very poor," indicating "*living in a state of chronic want*". Classes **C** and **D** are characterised as "poor," signifying individuals and families "*living under a struggle to obtain the necessaries of life and make both ends meet*". The designated income range for these classes lies between 18 to 21 shillings per week, highlighting the economic challenges faced by these groups. Class **C** encompasses lower-income artisans, street vendors, and owners of small retail establishments. This segment is particularly susceptible to fluctuations in economic conditions, notably the periodic downturns in trade that exacerbate their vulnerability. In contrast, Class **D** families possess a somewhat stable income, regarded as sufficient to categorise their financial inflow as regular, offering them a marginally more secure economic footing. Further up the economic hierarchy, Class **F** is noted for earning an income that ranges from 30 to 45 or 50 shillings. This classification indicates a higher economic status, characterised by a greater degree of financial stability and security. Class **G** includes individuals such as shopkeepers, small business owners, and subordinate professional men. This class is regarded as "a hard-working, sober, energetic class". All above Class **G** is lumped together in Class

H, defined as the "servant-keeping class". They live in houses "*which, owing to their high rental, are not scheduled by the School Board visitor*", and "*there are many streets as to which the visitors have not even the names in their books*".[26] Figure 2.3.3 exhibits the distribution of these social classes in London.[27]

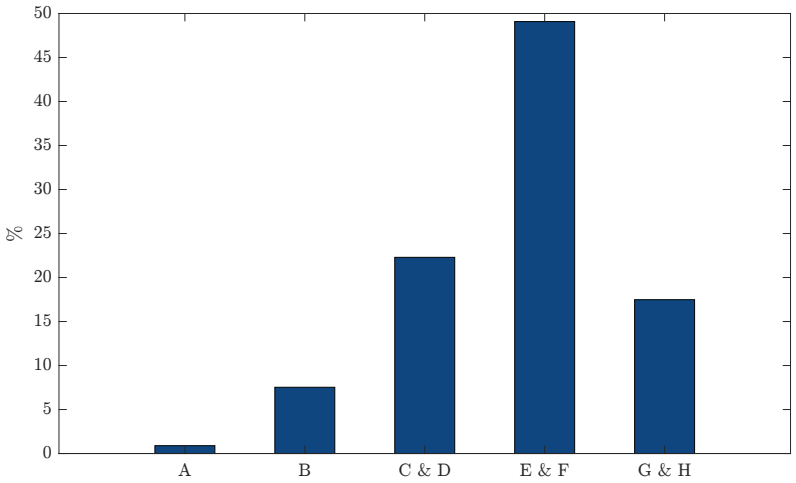

Figure 2.3.3: **Proportions of different social classes in London, 1880s.** This figure shows the proportions of different social classes in London.

Based on the classifications for each street, the proportions of people in each social class in each Registration District were reported in Booth (1889), which provides valuable insights into the socioeconomic structure of London during the 1880s. I digitised the data from Labour and life of the people, *Vol I, Appendix A*, p. 130-133, Tables II & III, "Classification of the population of London, by Registration Districts and School Board Divisions" & "Classification of Registration Districts by percentages, in order of poverty".[28] These two tables classify Classes **A**-**D** as "in poverty" and Classes **E**-**H** as "in comfort". I collected and computed the proportion of the "comfort" class as a measure of the upper tail of the distribution in 17 Registration District, in which 4 are "parent" areas.

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26. Source: Life and labour of the people in London. *First Series: Poverty, Vol I*, p. 33-62, (accessed March 2024), https://archive.org/details/b21357547/page/60/mode/2up?view=theater.
27. Figure 2.3.2 and Figure 2.3.3 visualise distributions from data, but they do not tell the full story of inequality in London at that time. See Section 1.3.2 in Chapter 1 for detailed discussions.
28. Source: Labour and life of the people. *Vol I, Appendix A*, p. 130-133, Tables II & III, "Classification of the population of London, by Registration Districts and School Board Divisions" & "Classification of Registration Districts by percentages, in order of poverty", https://wellcomecollection.org/works/ba3zsu3e/items?canvas=5.

To analyse the four decades of transformation from the late 19th century to the 1930s, which is the post-war period, Sir Hubert Llewellyn Smith, one of Booth's assistants published *New Survey of Life and Labour* in 1929-30 (Llewellyn-Smith and Booth, 1930). For the sake of a longitudinal comparison, the geographic boundaries in Booth's reports — Registration Districts were mapped to Met.B.s and the figures were recalculated at the level of Met.B.s (Llewellyn-Smith and Booth, 1930, p. 101-106, Vol III). In this report, as displayed in Table 2.3.3, Class **P** corresponds to Classes **B**-**D**; Class **U** corresponds to Class **E**; Class **S** corresponds to Class **F**; and Class **M** corresponds to Class **G**-**H**. Therefore, I supplement the proportion of the "comfort" class from Booth's report for those Registration Districts for which it is available with the sum of the proportions of Classes **U**, **S**, and **M** from (Llewellyn-Smith and Booth, 1930, p. 101-106, Vol III) for the Met.B.s. With the help of the New Survey, I acquired data for an additional 14 areas. In total, I gathered data on the proportion of the "comfort" class, the proportion of the "poverty" class, and the ratio of the "comfort" class to the "poverty" class in the 1880s for 31 areas, including 8 "parent" areas.

Table 2.3.4 summarises the data availability and key descriptive statistics of all socioeconomic measures explained above.

Table 2.3.4: Data availability and summary statistics of socioeconomic measures

Variable	# of areas available	Min	Max	Mean	Std. dev.	P90/P10
1861 Census						
% Professional	36	3.29	37.82	10.89	8.34	7.36
% Industrial	36	37.24	71.21	52.70	9.82	1.68
% Professional/% Industrial	36	4.62	99.15	23.40	22.51	11.00
1871 Census						
% Professional	27	3.14	30.09	10.54	6.01	5.19
% Industrial	27	35.57	69.80	50.68	8.21	1.50
% Professional/% Industrial	27	4.64	83.65	22.03	16.81	5.55
Booth Data 1880s						
% Middle	31	3.32	41.30	17.52	10.43	7.54
% Poverty	31	13.52	44.59	30.49	7.67	1.96
% Middle/% Poverty	31	8.28	251.42	70.52	59.18	14.22

[a] Data availability as well as the key statistics of measures of socioeconomic conditions. % Professional/% Industrial and % Middle/% Poverty are scaled by a factor of 100. For example, % Professional/% Industrial = 99.15 means the percentage of the professional class is 0.99 times the percentage of the industrial class.

2.4 Empirical Evidence: Variations in Mortality Dynamics Across Areas

This section inspects the variations in the dynamics of mortality reductions among small areas of London by using the dataset constructed in Sections 2.3.1 and 2.3.2. The dataset encompasses three mortality measures: crude mortality, infant mortality, and non-infant mortality for over 30 areas and covering, for most of these, the period 1850–1935.

Equation 1.2.2a in Chapter 1 was used to estimate the dynamics of reductions in mortality in London, as displayed in Figure 1.5.2. To examine the heterogeneity in dynamics of mortality, we next apply Equation 1.2.2a to the mortality trends of constituent areas within London, using three different mortality measures between the 1850s and the 1930s. Figure 2.4.1 illustrates the heterogeneity within London using six selected constituent areas, where Figure 2.4.1a, Figure 2.4.1b, and Figure 2.4.1c plot crude mortality, infant mortality, and non-infant mortality respectively.[29] This application first reassures that mortality declines during the transition period display a common sigmoid pattern (S-shaped) across different mortality measures and geographical units. Second, echoing the patterns shown in Figure 1.5.2, infant mortality lagged behind crude mortality by approximately 12–25 years, which confirms that the crude mortality decline was not solely driven by changes in infant mortality in London and these constituent areas.

The heterogeneity in the dynamics paths of mortality is striking in London, which is mainly reflected by the substantial differences in the timing of inflection points. As demonstrated in Chapter 1 (Section 1.2.5), the inflection point, the point where the logistic curve's convexity shifts and the most rapid decline occurs, signifies, in some sense, the midpoint of the mortality transition.

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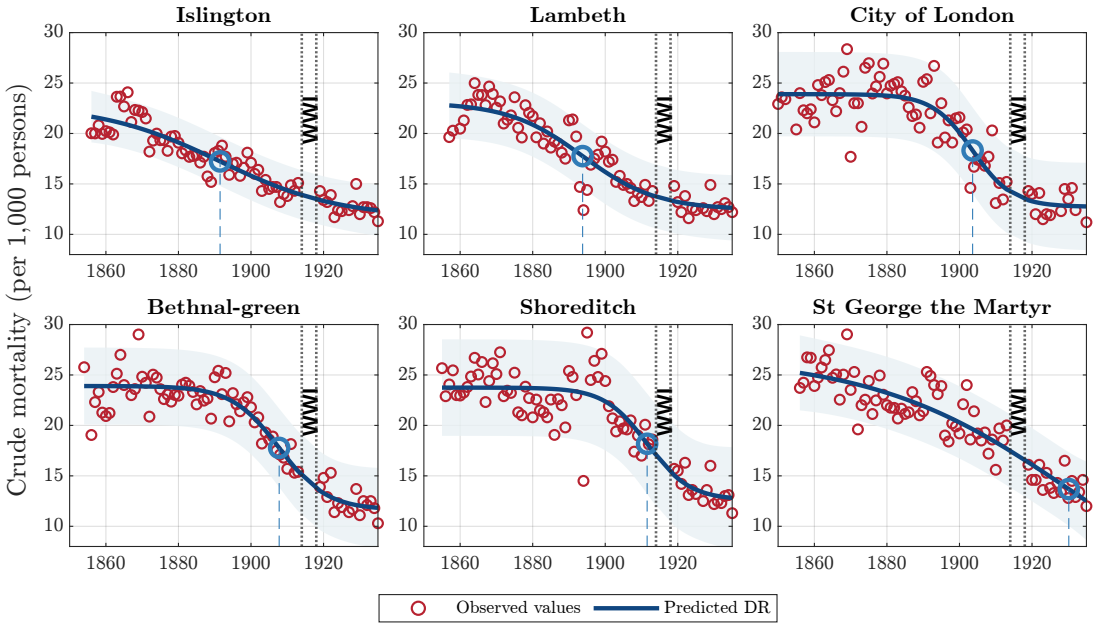
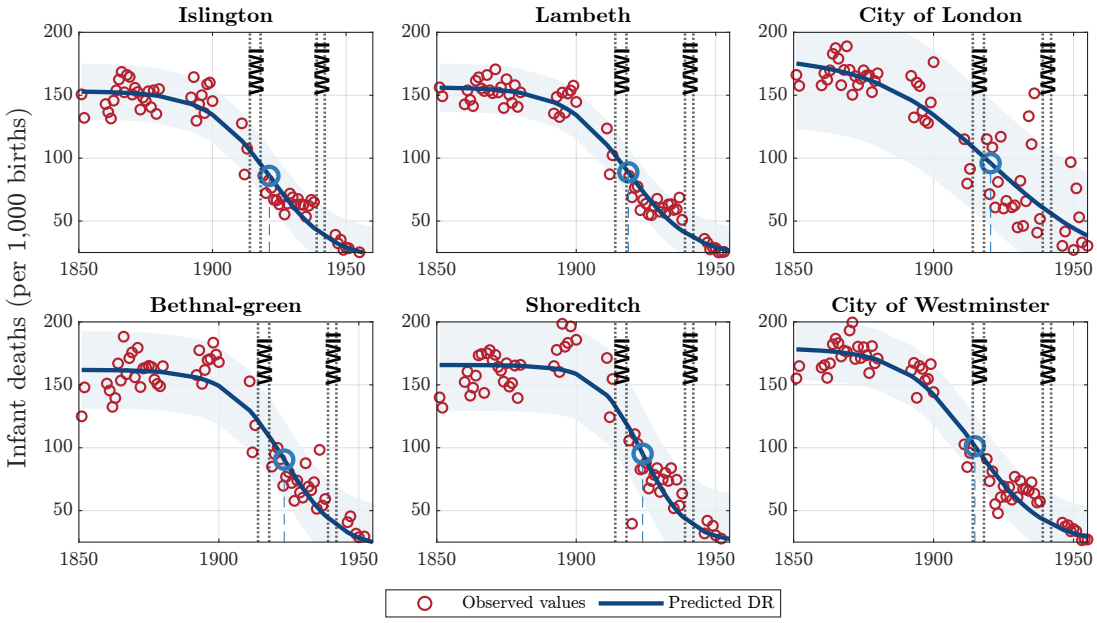
29. see Figure 2.8.2 and Figure 2.8.3 for trends of crude mortality in all areas in Appendix 2.8.3.

(a) Crude mortality, 1850–1935



(b) Infant mortality, 1860–1950



(c) Non-infant mortality, 1850–1935

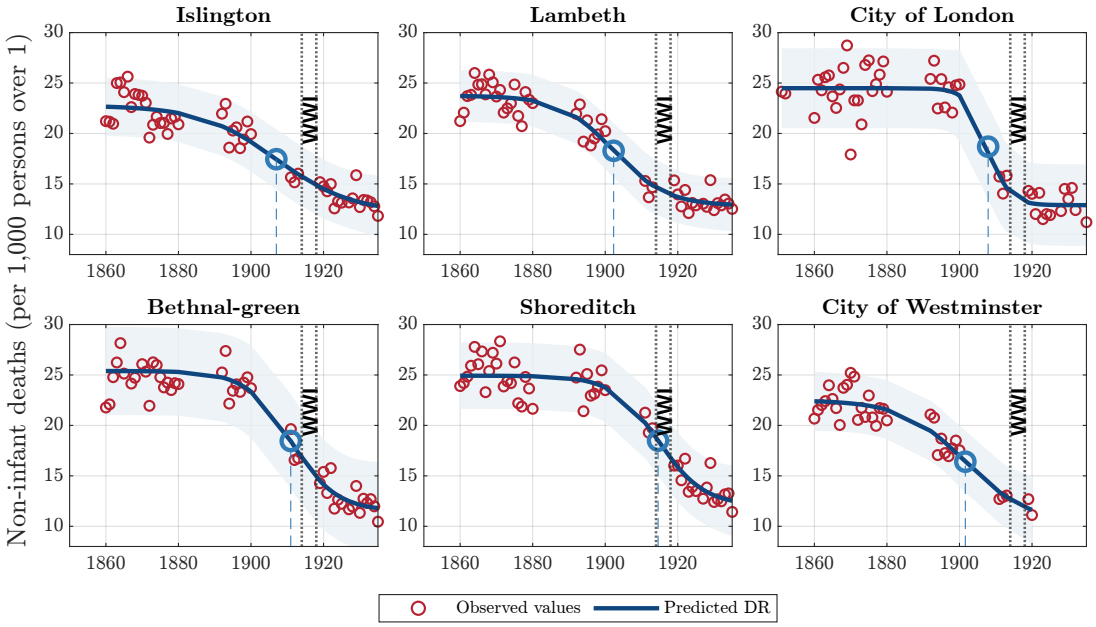


Figure 2.4.1: **Time series of three mortality rates for selected areas in London.** Fitted logistic curves shown as blue lines and 95% prediction intervals in blue shading; the inflection points are marked with blue circles. The six selected areas are not identical across mortality measures since infant mortality is not available for a long enough period of St George the Martyr.

Figure 2.4.1a reveals that there can be a discrepancy of nearly forty years in the inflection points, with Islington transitioning in 1891.48 compared to St George the Martyr in 1930.14, suggesting that certain areas experienced a significantly delayed decline in crude mortality and were stuck in a relatively long period of slow reductions. In terms of infant and non-infant mortality in Figure 2.4.1b and Figure 2.4.1c, although the gaps in inflection points are narrower, disparities of up to two decades persist.

Moreover, Figure 2.4.2 plots the coefficient of variation of crude mortality across all areas with available data, suggesting that mortality heterogeneity across all areas in London increased until around 1900, before dropping afterwards. In other words, the majority of the mortality transition period did not feature convergence in mortality; instead, several decades after the health improvements that triggered the mortality transition saw a rise in mortality disparities between different areas of London.

Similar patterns of mortality variations are found for Glasgow (Stewart, 2024), and see detailed empirical analysis and additional findings in the joint paper (Angelopoulos et al., 2024).

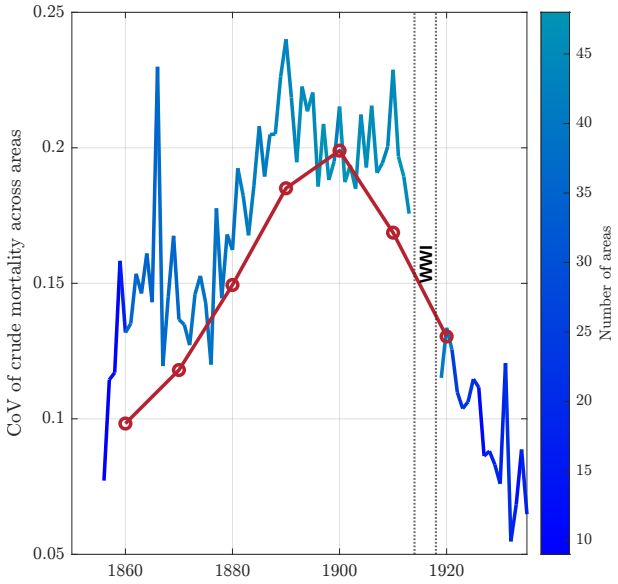


Figure 2.4.2: **Coefficient of variation of crude mortality across areas for London.** The blue time series shows the coefficient of variation across all areas in London. Due to boundary changes and data issues discussed in Section 2.3, the areas (and the number of areas) used to calculate this coefficient vary over time. The red line shows the coefficient of variation across 26 areas that consistently existed every ten years from 1860 to 1920. Both series show an initial increase followed by a decrease.

The coefficient of variation patterns for infant and non-infant mortality in Figure 2.4.3 echo the initial rise followed by decline observed in crude death rates across regions. However, the series shift from an upward to downward trajectory around 1920, approximately two decades after the corresponding reversal in crude mortality rates. This reiterates that the infant mortality decline, despite its considerable magnitude, did not initiate the crude mortality transition.

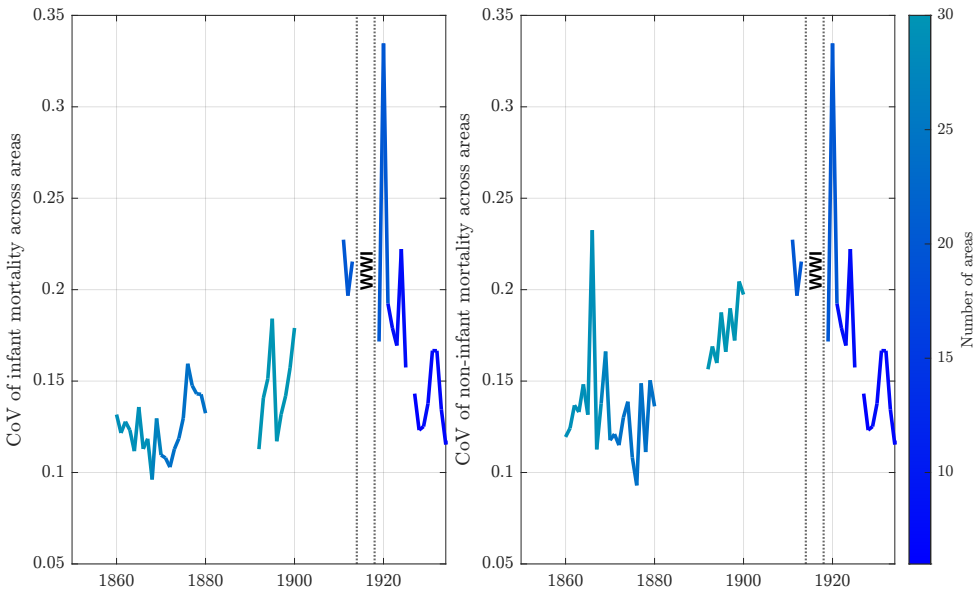


Figure 2.4.3: **Coefficient of variation of infant and non-infant mortality across areas for London.** The blue time series show the coefficient of variation of infant mortality (left) and non-infant mortality (right) across all areas with available data in London. The series display discontinuities when the number of areas with available data in a given year falls below five.

2.5 Model Predictions: Socioeconomic Conditions and Mortality Dynamics

The observed pattern of widening and subsequent narrowing mortality differentials illustrated above raises the question of whether initial socioeconomic disparities at the beginning of the mortality transition period could have shaped these mortality differentials. This section explains how the theory of mortality dynamics and income-driven health diffusion in Chapter 1 yields clear predictions about the relationship between socioeconomic status and variations in mortality trajectories, formally asking the question: is income inequality also the fundamental driver of the heterogeneity of mortality dynamics in London?

Section 1.3 of Chapter 1 presents a technological diffusion model where an individual's adoption of health technology depends on a cost-benefit evaluation. The log-normal income distribution across the population (see discussions in Section 1.3.2) then produces sigmoid mortality dynamics. Using this framework, we next examine the model's predictions concerning the impact of socioeconomic variations on observed mortality dynamics across populations.

Consider N geographic areas, whose populations are characterised by different socioeconomic conditions that are captured by differences in the log-normal distributions of income. All areas and populations have, in principle, exposure to the same technological progress in health. In poorer areas with a smaller share of higher income individuals and thus a long right-tail for the distribution of income, during the initial stages of the mortality transition the income threshold for adoption of health progress y_t^* is distanced from the mode of the distribution, situated on the flat section of the right tail of f_y^L. The opposite occurs in more affluent areas, where a relatively larger proportion of the population has higher incomes, meaning that during the initial stages of the mortality transition, the income threshold for adoption of health progress is relatively closer to the mode of the distribution. Therefore, both $F_y^L(y_t^*)$ and $f_y^L(y_t^*)$ will be smaller for the poorer areas during the initial phase of the mortality transition. This implies from Equation 1.3.4 that, ceteris paribus, the decline in mortality starts earlier in the higher-income areas which also have lower levels of mortality. Moreover, from Equation 1.3.6 we know that the inflection point occurs when y_t^* is to the left or near the level of income associated with the mode of the distribution of income. Hence, in areas with a bigger distance between the mode and the income threshold the year in which the inflection point is reached will be delayed. These points are illustrated for two populations in Figure 2.5.1.

The different rates of adoption of health progress and its changing pattern across time in areas differentiated by socioeconomic conditions have been documented in empirical evidence. As depicted in Kesztenbaum and Rosenthal (2017, Fig. 3), while the percentage of buildings connected to sewers increased across all Paris districts from 1885 to 1913,

the wealthiest neighbourhoods consistently maintained the highest connection rates. Furthermore, the affluent districts initially adopted direct connections more quickly, but by 1906, the rate of sewer adoption had become slightly faster in poorer districts and slightly slower in richer ones, which reflects our points here.[30]

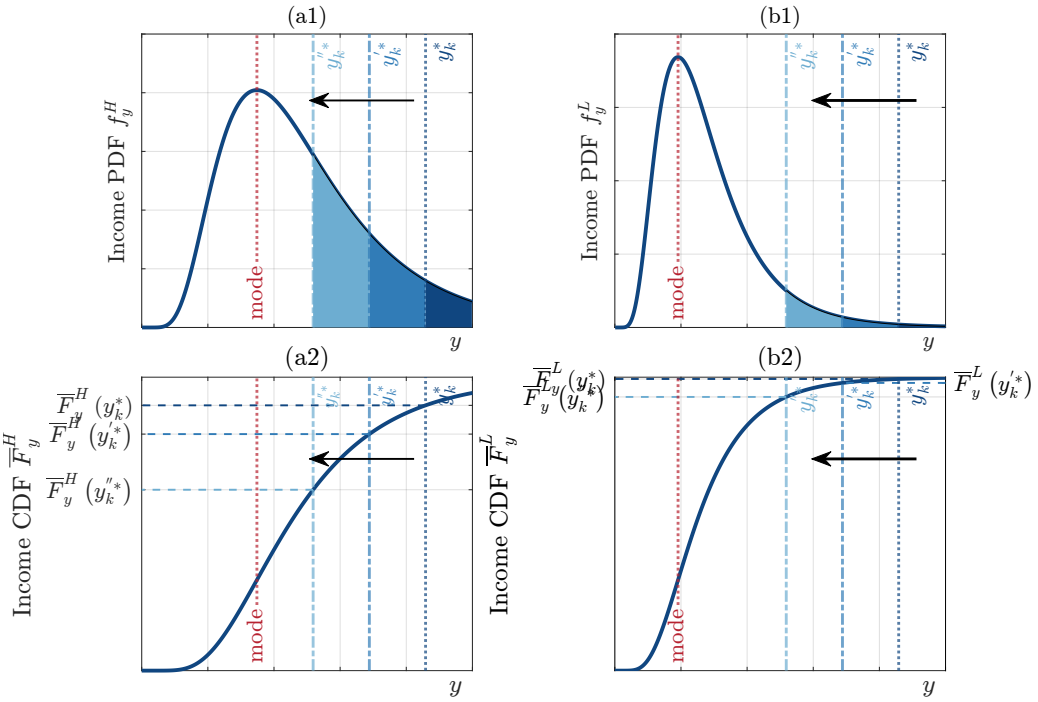


Figure 2.5.1: **Dependence of diffusion dynamics of health progress on the distribution of income.** The figure shows the effect of progress in health in a high-income area ((a1) and (a2)) and a low-income area ((b1) and (b2)). The transitions from y_k^* to $y_k''^*$ result in more significant expansions of the shaded areas in (a1) compared to (b1) and more substantial decreases in F^H in (b1) compared to F^L in (b2). In earlier stages, the same technological progress in health (reflected by the shifts from y_k^* to $y_k''^*$) implies higher adoption in the high-income area.

The technology diffusion model we have developed predicts that areas that have a higher proportion of high-income (relative to low-income) individuals—such that the skewness of the distribution is lower and the mean and median are higher—should have a faster reduction in mortality during the first phase of the mortality transition and an earlier inflection point of the dynamics of the death rate.[31] More generally, the model predicts

30. Kesztenbaum and Rosenthal (2017) collected data on the share of buildings connected to the sewer from 1881 to 1914 from the *Annuaires statistiques de la ville de Paris*. No similar data for London exists to our knowledge. The closest we found is Mooney (1994, Table 2.2), but we cannot conduct systematic research using this data. See Section 2.6.2.2.3 for details.

31. The mean of the log-normal distribution is a positive function of both the median and the skewness.

that in areas with generally better socioeconomic conditions, mortality rates fall earlier, in particular, these areas would benefit earlier from health progress. The implication of these effects is that mortality levels between the areas would initially diverge at the beginning of the process of health technology progress, before starting to converge.

2.6 Empirical Evidence: Socioeconomic Conditions and Mortality Dynamics in a Panel of Areas

Following our analysis of heterogeneity in mortality dynamics in Section 2.4, Section 2.5 explains theoretical predictions regarding how this heterogeneity is shaped by the socioeconomic status of each area, drawing on the framework developed in Chapter 1. Here, we test the prediction visually and descriptively in Section 2.6.1, and conduct a formal analysis using a statistical model in Section 2.6.2.

2.6.1 Stylised Facts

Before examining dynamics, Figure 2.6.1 visualises mortality inequality at the beginning of the mortality transition (1860–1865). The analysis proceeds in three steps: First, crude mortality rates are averaged for each area across the six-year period. Second, these averaged area-level mortality rates are ranked and divided into four quartiles, with the first quartile (the "bottom 25 quartile") comprising areas exhibiting the lowest mortality rates. Finally, the figure displays the average proportions of professional class (blue bars) and industrial class (red bars) within each mortality quartile, revealing the socioeconomic gradient of mortality.

Figure 2.6.1 indicates a clear inverse relationship between socioeconomic status and mortality rates—the professional class (blue bars) was predominant in the group with the lowest crude mortality (represented by the leftmost bars), whereas many individuals in industrial occupations resided in areas with the highest death rates.

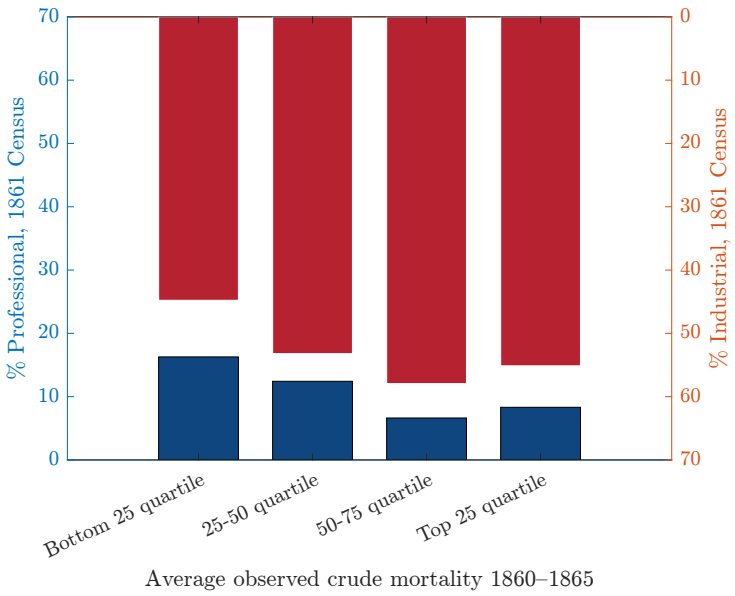


Figure 2.6.1: **Average proportions of professional and industrial classes across mortality quartiles derived from average crude mortality rates between 1860 and 1865.** First, crude mortality rates are averaged for each area over 1860–1865. Second, these averaged area-level mortality rates are ranked and divided into four quartiles, where the "bottom 25 quartile" includes areas with the lowest mortality rates. Finally, for each mortality quartile, the figure displays the average proportions of professional class (blue bars) and industrial class (red bars), revealing the socioeconomic gradient of mortality.

Given the cross-sectional evidence of mortality inequality in Figure 2.6.1, Figure 2.6.2 illustrates the evolution of mortality across different social classes from 1860 to 1920. Areas are first divided into four socioeconomic quartiles based on the proportion of the professional class in 1861, and their respective (estimated) crude mortality rates are tracked until 1920.[32] First, all four quartiles experienced striking mortality declines over this period. Second, the black dashed line shows the gap between the most affluent quartile (dark blue) and the poorest quartile (red curves), demonstrating that the dynamics of mortality inequality were not monotonic—it initially increased and then decreased until the 20th century. The same pattern is observed when comparing the lowest 25% quartile with the 25–50% quartile, and the 50–75% quartile with the highest 25% quartile.

32. 34 areas are used to plot Figure 2.6.2, so each quartile has eight or nine areas.

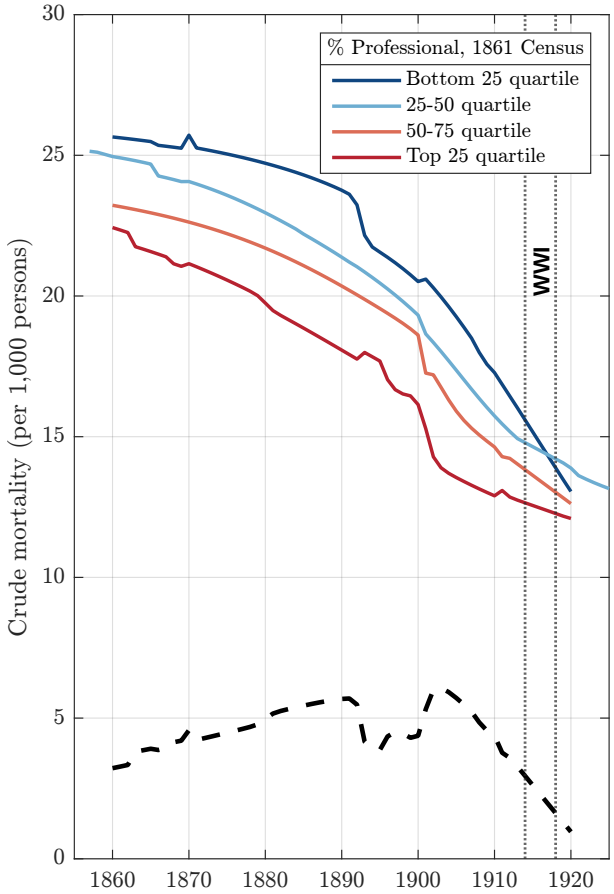


Figure 2.6.2: **Annual averages of trend crude mortality in four socioeconomic quartiles across areas in London, 1860–1920.** The constituent areas are ranked based on the proportion of professionals in 1861 and divided into four quartiles. The average trend crude death rates, estimated using the logistic function Equation 1.2.2a, are then plotted for each quartile. The dark blue curve at the top represents the quartile with the lowest proportion of professional class, while the red curve at the bottom signifies the quartile with the highest proportion of professional class. The black dashed line illustrates the gap between the dark blue and red curves, which visualises the dynamics of mortality inequality.

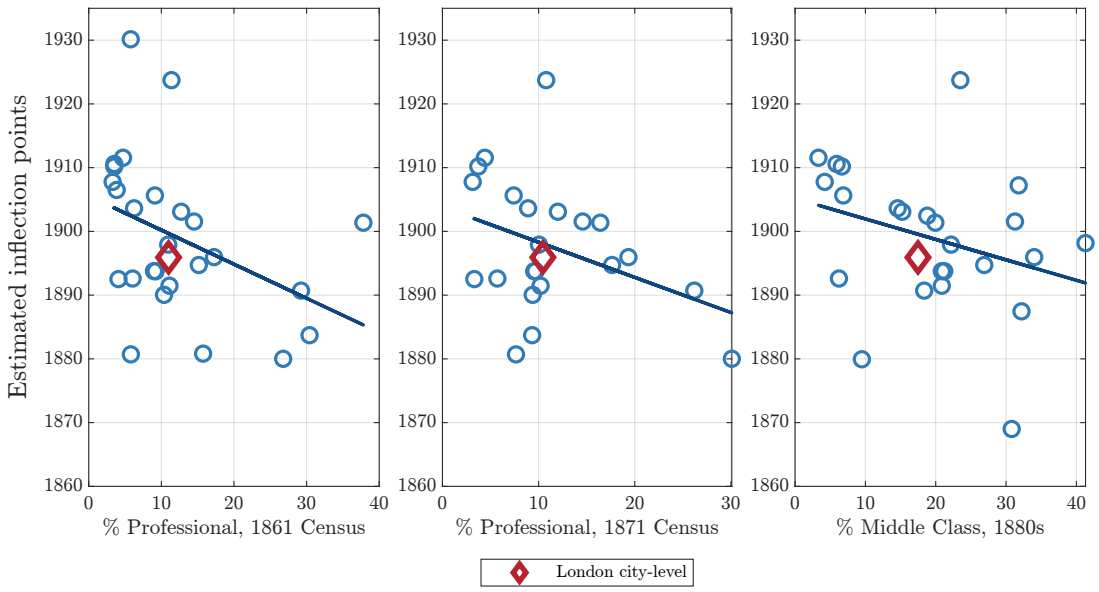


Figure 2.6.3: **Socioeconomic conditions vs. estimated inflection points.** This figure plots the negative relationship between the socioeconomic conditions and the estimated inflection points for each small area. The dark blue solid lines represent the linear fitted lines. Areas for which the corresponding estimated inflection points fall out of the sample (either before 1850 or after 1935) are considered invalid and are excluded. The red diamonds mark the London city-level using dataset introduced in Section 1.2.3 in Chapter 1.[33]

In order to understand how socioeconomic conditions elucidate the "divergence-convergence" dynamics of mortality inequality between areas, Figure 2.6.3 links the heterogeneity of mortality dynamics seen in Figure 2.4.1a and the measures of socioeconomic distributions. In particular, Figure 2.6.3 illustrates the negative relationship between the socioeconomic conditions and the inflection points estimated by the four-parameter logistic function Equation 1.2.2a for each small area (see Figure 2.8.10 in Appendix 2.8.4 for equivalent figures of infant mortality and non-infant mortality). In other words, richer areas experienced a shorter period of initially slow mortality declines and saw fast reductions earlier. Among the six selected areas in Figure 2.4.1a, three of them (*Panel A* in Table 2.6.1) had more than 10% of their population in professional occupations. In contrast, the other three (*Panel B*) had a lower proportion of high-social-class residents, with inflection points occurring decades later. Therefore, mortality rates across areas initially diverged before poor areas saw fast reductions as well, which eventually led to convergence.

33. City-level data is not involved in the linear fit estimation (shown as the blue line).

Table 2.6.1: Statistics of six selected constituent areas in London

Area	Average crude mortality 1860-1865	% Professional, 1861 Census	Estimated inflection point of crude mortality
Panel A			
Islington	21.70	20.72	1891.48
Lambeth	22.72	16.16	1893.77
City of London	23.90	12.03	1903.62
Panel B			
Bethnal-green	23.68	4.62	1907.78
Shoreditch	24.44	6.80	1911.55
St George the Martyr	25.51	9.14	1930.14

2.6.2 Statistical Model and Results

Building upon the stylised facts presented in Section 2.6.1, we now develop a statistical model to formally test the theory in Section 2.5. In particular, a positive relationship between socioeconomic conditions and the inflection point indicates immediate convergence of mortality inequality, while a negative relationship suggests initial divergence.

As illustrated in Figure 2.5.1, the shape of the income distribution to the right of the mode determines the adoption rate of new health technology during the earlier period. Therefore, we employ measures of socioeconomic distributions which reflect better the concentration of wealth within higher income brackets. We use socioeconomic measures for the first decades of the mortality transition, and which pre-date the inflection point of the trend mortality, which took place around 1900 at the city level (see Figure 1.2.3). This approach allows for an examination of whether area-specific socioeconomic conditions prior to the 1890s can account for variations in the timing of the inflection points across different small areas within London.

2.6.2.1 Baseline Model

To implement the empirical test, I employ an extension of the four-parameter logistic model to the panel of areas in London. We developed and implemented this model in our joint work in Angelopoulos et al. (2024) in both London and Glasgow. Here, I contextualise the statistical framework and examine results from applying these specifications to the

panel of areas of London, using the data I collected. In particular, we make the four parameters in Equation 1.2.1 a function of the area-specific measures of socioeconomic inequalities:

$$\overline{d} = (\delta_l + \gamma_{\delta_l} S) + \frac{(\delta_u + \gamma_{\delta_u} S) - (\delta_l + \gamma_{\delta_l} S)}{1 + e^{\Big(\big(\beta+\gamma_\beta S\big)\big(t-\big(\tau_c+\gamma_{\tau_c} S\big)\big)\Big)}}, \tag{2.6.1}$$

where $\overline{d}$ is the trend death rate; S is the variable measuring socioeconomic conditions that capture relevant properties of the distribution of income; $\delta_u, \delta_l, \beta, \tau_c$ are parameters that control the general shape of mortality dynamics, as explained in Section 1.2.4.1 (see Equation 1.2.1); γ_j for $j = \delta_u, \delta_l, \beta, \tau_c$ are parameters controlling the impact of socioeconomic conditions—relating to the distribution of income—on mortality dynamics. See Appendix 2.8.2 for detailed mathematical interpretations of the model.

Then, we fit the modified logistic model in Equation 2.6.1 to the mortality measure j in London during $t \in (1850, ..., 1935)$,[22] by applying the Non-Linear Least Squares (see Section 1.2.4.1 in Chapter 1 for detailed discussions of NLS):[23]

$$d^j_{i,t} = \left(\delta^j_l + \gamma^j_{\delta_l} S_i\right) + \frac{\left(\delta^j_u + \gamma^j_{\delta_u} S_i\right) - \left(\delta^j_l + \gamma^j_{\delta_l} S_i\right)}{1 + e^{\Big(\big(\beta^j+\gamma^j_\beta S_i\big)\big(t-\big(\tau^j_c+\gamma^j_{\tau_c} S_i\big)\big)\Big)}} + \nu^j_{i,t}, \tag{2.6.2a}$$

where $\nu_{i,t}$ is the error term measuring the discrepancy between the actual value and the fitted curve. $\nu_{i,t}$ includes area-specific random shocks $u_{i,t}$ which for each geography i, is serially correlated because random shocks to mortality such as disease outbreaks have persistent effects. Therefore, we model $u_{i,t}$ as

$$u_{i,t} = \rho u_{i,t-1} + \epsilon_{i,t}, \tag{2.6.2b}$$

where $\rho \in (-1, 1)$ and the idiosyncratic error $\epsilon_{i,t}$ is assumed to be independently distributed across time (not serially correlated for each geography i). Then the statistical model of the sigmoid trend taking into account the serially correlated random shocks is[34]

$$d_{i,t} = \rho d_{i,t-1} + \left[\left(\delta_l + \gamma_{\delta_l} S_i\right) + \frac{\left(\delta_u + \gamma_{\delta_u} S_i\right) - \left(\delta_l + \gamma_{\delta_l} S_i\right)}{1 + e^{\left(\left(\beta+\gamma_\beta S_i\right)\left(t-\left(\tau_c+\gamma_{\tau_c} S_i\right)\right)\right)}} \right] - \rho \left[\left(\delta_l + \gamma_{\delta_l} S_i\right) + \frac{\left(\delta_u + \gamma_{\delta_u} S_i\right) - \left(\delta_l + \gamma_{\delta_l} S_i\right)}{1 + e^{\left(\left(\beta+\gamma_\beta S_i\right)\left(t-1-\left(\tau_c+\gamma_{\tau_c} S_i\right)\right)\right)}} \right] + \epsilon_{i,t}. \tag{2.6.2c}$$

In addition, as illustrated in Section 1.2.4.1 in Chapter 1, $u_{i,t}$ and $\epsilon_{i,t}$ are allowed to be heteroskedastic because of two reasons: first, the larger fluctuations in death rates at the beginning of the period; and second, the probability of random shocks varying across areas i.e., the variance of the error term might be a function of the area. Therefore, we estimate both Equation 2.6.2a and Equation 2.6.2c using Non-Linear Least Squares (NLS) and compute standard errors that are robust to heteroskedasticity (see Appendix 1.5.2 in Chapter 1 for details). See an illustration of the Gauss-Newton method algorithm and the algorithm for uniqueness check of the solution in Appendix 2.8.2.

2.6.2.2 Model Extensions

2.6.2.2.1 City-level trend-adjusted mortality

To account for possible random shocks to the death rates in Equation 2.6.2a–Equation 2.6.2b that have a common component, we also estimate the model using an adjusted mortality measure instead of crude mortality for area i in year t, calculated as the death rate minus the deviation of the city-level death rate in that year from the city-level trend—$\tilde{d}_{i,t} = d_{i,t} - \left(d_t - \hat{d}_t\right)$, where the deviation of the city-level death rate from the city-level

34. When estimating Equation 2.6.2c, we also use additional specifications to check robustness. We allow ρ to be affected by socioeconomic conditions, i.e., Equation 2.6.2b is modified as $u_{i,t} = \left(\rho + \gamma_\rho\right) u_{i,t-1} + \epsilon_{i,t}$. See additional results in Angelopoulos et al. (2024).

trend, $d_t - \hat{d}_t$,[35] captures common fluctuations in the death rate across areas (e.g., an epidemic). They are thus purged from the error term of the model. The results from NLS estimation of the model using excess mortality are shown in Columns (1)–(3) in Table 2.6.6.

2.6.2.2.2 Geographic controls

Beyond socioeconomic conditions, geographic factors may exert additional influence on mortality. Figure 2.8.1 in Appendix 2.8.1.1.1 visualises the spatial heterogeneity of average crude mortality from 1860 to 1865 (beginning of the mortality transition) and from 1920 to 1930 (end of the mortality transition). First, the central part of London (e.g., Stepney, Holborn, St Olave) is the darkest, indicating the highest relative crude mortality. In contrast, the western side, which is upstream of the Thames (e.g., Wandsworth, Hammersmith), falls within the bottom 20% quartile in terms of crude mortality. Second, the south bank of the Thames is generally healthier, especially after the mortality transition.

Despite the spatial variations, the darkest regions remained the darkest after eight decades, implying that their relative ranking in terms of crude mortality was unchanged. In other words, geographic factors, to a large extent, constitute a fixed effect that affects the level of mortality rather than influencing the temporal evolution of mortality patterns. Therefore, in order to mitigate the confounding effects of geographic factors and isolate the impact of income distribution, we introduce regional fixed effect by adding five dummy variables, which are assigned a value of 1 for areas classified as "Central", "North", "South", "East", and "West" regions, respectively, and a value of 0 for all other areas. The classification of five regions is according to the MOH reports.[36]

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35. d_t is the observed crude mortality in London in year t in Equation 1.2.2a in Chapter 1; $\hat{d}_t$ is the estimated crude mortality based on Equation 1.2.2a.
36. Source: Report of the Medical Officer of Health for London County Council 1892, P3 (accessed February 2024), https://wellcomelibrary.org/moh/report/b18252412#?c=0&m=0&s=0&cv=0.

We also consider other geographic measures such as the shortest distance from each area's centroid to the Thames. As illustrated in Figure 2.6.4, we first identify the centroid of each geometry i.e., polygon/constituent area (marked by black dots), and then compute the distance between the points and the nearest point on the Thames.

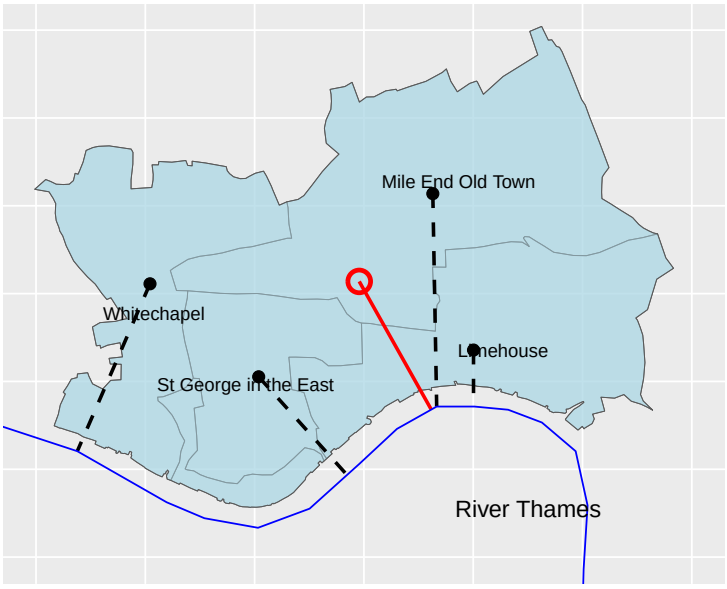

Figure 2.6.4: **Nearest distances from the River Thames to the Met.B. of Stepney and its constituent areas.** In this figure, the red circle marks the centroid of the "parent" area (the Met.B. of Stepney) and black dots mark the centroids of the constituent areas. The corresponding (dashed and solid) lines are the closest distance to the Thames, which are used as a geographic control in the regression.

To explicitly account for geographic effects, we allow $\nu_{i,t}$ in Equation 2.6.2a to include effects G_h, i.e., $\nu_{i,t} = G_h + u_{i,t}$, where h can be equal to i or can be a broader area that comprises several areas i, working at a local scale within the city but that may be common for a subset of areas, generating correlation in $\nu_{i,t}$ across areas. Then, Equation 2.6.2c becomes

$$d_{i,t} = \rho d_{i,t-1} + \left[(\delta_l + \gamma_{\delta_l} S_i) + \frac{(\delta_u + \gamma_{\delta_u} S_i) - (\delta_l + \gamma_{\delta_l} S_i)}{1 + e^{\Big((\beta+\gamma_\beta S_i)\big(t-(\tau_c+\gamma_{\tau_c} S_i)\big)\Big)}} \right] - \rho \left[(\delta_l + \gamma_{\delta_l} S_i) + \frac{(\delta_u + \gamma_{\delta_u} S_i) - (\delta_l + \gamma_{\delta_l} S_i)}{1 + e^{\Big((\beta+\gamma_\beta S_i)\big(t-1-(\tau_c+\gamma_{\tau_c} S_i)\big)\Big)}} \right] + (1-\rho)\, \boldsymbol{\zeta}^\intercal \boldsymbol{G}_h + \epsilon_{i,t}, \tag{2.6.3}$$

where $\boldsymbol{G}_h$ is a vector containing characteristics that may be common for several geographic areas and $\boldsymbol{\zeta}$ is a vector with the associated coefficients. Therefore, when including the five dummies for "East", "North", "South", "West", and "Central", we have four corresponding estimates ζ_E, ζ_N, ζ_S, and ζ_W (see e.g., Table 2.6.2, ζ_C is omitted). When including the shortest distance from each area's centroid to the Thames (see Section 2.6.2.2.2 for the illustration), we have one estimate ζ in Table 2.6.6.

2.6.2.2.3 Adoption of new health technologies

Using an economic model alongside a statistical model, we argue that those on the right side of the income distribution skew are the earliest adopters of new health technologies, leading them to reach the inflection point earlier. However, we do not have direct data on the adoption of new health technologies, such as the percentage of households connected to sewage systems, with access to clean water, proper ventilation, modern medical treatments, etc. Some research relies on data from the Local Taxation Returns to assess how infrastructure investment contributed to the decline in mortality (e.g., Chapman, 2019; Harris and Hinde, 2019; Aidt et al., 2023). Other research has explored specific public health efforts across London boroughs (or cities in the UK). For example, Antman (2023) studied water quality differences across English parishes; Wray (2015) examined variations in water supply sources and the adoption of chlorination by the Metropolitan Water Board in 1916; and Troesken et al. (2021) investigated the transition from intermittent to constant water supply in London.[37] Nonetheless, we do not employ these data, as it remains unclear how many households benefited from the government investments or the specific health improvements in each parish.[38] In addition, as demonstrated by Aidt et al. (2023), in Britain, the gradual improvements in water supplies and sewerage were

37. See Fletcher and Noghanibehambari (2023); Anderson et al. (2022); Alsan and Goldin (2019) for evidence of the US.
38. For example, Troesken et al. (2021, Table 1) provides a summary of the differing times at which the eight London water companies first introduced filtration. These companies supplied water to multiple parishes that we cannot disentangle from each other, and within each parish, we lack information on the number of households that had access to piped water.

accompanied by the incremental extension of these services to households. As a result, there is limited documentation of the population's access to different qualities of water and waste disposal, making it difficult to develop consistent time series data on water and sewerage provision and quality across most towns.

The most relevant data we have found is Mooney (1994, Table 2.2), which includes sanitary works data, such as the number of houses connected to sewers, houses supplied with water, and cesspools, conducted after the appointment of the Medical Officer of Health (MOH) from 1855 to 1980. We do not conduct systematic research using this data because: a) the author could not obtain all MOH reports, so the table includes only seventeen areas, with missing information marked as "numerous"; b) the original data source is the *National Association for the Promotion of Social Science*, which relies on self-reported information from the Medical Officers of Health, who may have incentives to over-report their own sanitary works; c) our aim is not to disentangle different health innovations but to conceptualise the diffusion pattern of all health innovations, including public/local water infrastructure and private new products like flush toilets. In addition, the MOH reports contain statistics and narratives related to the sanitary efforts in each region. For example, in St. Pancras, water was provided to 23 houses in 1856, 24 more were connected in 1860, and 9 houses were supplied in each of the years 1870 and 1880.[39] However, conducting systematic research with this data is challenging because: a) such data is limited to a few areas, each emphasising different aspects of sanitary works. For instance, Paddington focused on the length of new brick pipe sewers, the number of gulleys constructed and cleaned, etc. No single variable is consistently available across all, or even most, areas in any given year; b) As noted earlier, the data was collected by individual inspectors who were unable to visit every house in the parish and may have been inclined to overstate the extent of the works.

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39. Data Source: Report of the Medical Officer of Health for St. Pancras 1856, P19; Report of the MOH for St. Pancras 1860, P9; Report of the MOH for St. Pancras 1870, P6; Report of the MOH for St. Pancras 1880, P31 (accessed February 2024). These reports also include the number of houses that have been repaired, cleansed, whitewashed, had overcrowding abated, and had ventilation improved, etc.

Therefore, after careful examination and cross-referencing of literature and datasets, these sources proved unsuitable for inclusion in this chapter. This is the reason why we employ the measures of socioeconomic distributions in the baseline model based on the rich literature suggesting that socioeconomic inequality drives disparities in access to information and technological adoption (see discussions in Section 1.4). As an alternative, we use two overcrowding indicators—defined as the percentage of individuals residing more than two per room in tenements with fewer than five rooms—to proxy for housing conditions, namely the potential for access to clean water and ventilation (see results in Columns (4)–(5) in Table 2.6.6.[40]

2.6.2.3 Results

Among the 49 areas I construct in Section 2.3.1, 20–27 areas are considered in the panel regressions because of data issues. First, as explained in Section 2.4, we apply the four-parameter logistic model Equation 1.2.2a to capture the mortality trends of constituent areas within London. While the model works for the majority of areas, insufficient temporal coverage in some areas precludes meaningful estimation of mortality dynamics. However, in the panel regressions, we partially retain these areas. In particular, we restrict the sample to areas exhibiting downward mortality trends, leveraging the commonality of sigmoid mortality patterns across regions.[41] This exploits the panel structure's capacity to pool information across areas, potentially compensating for temporal limitations in individual series.[42] Second, only a part of the areas have available socioeconomic inequality measures. Figure 2.8.2 in Appendix 2.8.3.1 plots the dynamics of crude mortality for all areas that have at least one socioeconomic measure and are included in any of the regressions. In contrast, Figure 2.8.3 depicts areas with mortality data but no socioeconomic data.

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40. Source: Report of the Medical Officer of Health for London County Council 1902, P5–P9 (accessed February 2024), https://wellcomelibrary.org/moh/report/b18252412#?c=0&m=0&s=0&cv=0.
41. The trends of crude mortality in all areas have a downward pattern, so this restriction does not exclude any areas in panel regressions in terms of crude mortality.
42. I also conducted robustness checks using a more restricted sample—areas for which the corresponding estimated inflection points using Equation 1.2.2a fall out of the sample (either before 1850 or after 1935) are considered invalid and are excluded i.e., areas used to plot Figure 2.6.3. See the results in Column (9) in Table 2.6.6.

2.6.2.3.1 Baseline results

Table 2.6.2 summarises the estimation results of the model Equation 2.6.2a (Columns (1)–(3))–Equation 2.6.2c (Columns (4)–(9)) for crude mortality using three socioeconomic variables S_i capturing the extent of right-skewness of the underlying socioeconomic distribution. Columns (7)–(9), which take into account the regional fixed effect discussed in Section 2.6.2.2.2 additionally i.e., Equation 2.6.3 show the benchmark results.

The γ_{τ_c} coefficients across all columns in Table 2.6.2 are negative and statistically significant, indicating that areas with more professional or middle-class male populations are expected to reach the inflection point earlier. This substantiates the hypothesis, which has been predicted by the model in Section 2.5 and suggested by stylised facts in Section 2.6.1, that mortality differentials between regions would initially diverge during the early phases of health technology advancement, prior to initiating convergence. As shown in Appendix 2.8.2, $\left(\tau_c + \gamma_{\tau_c}\overline{S_i}\right)$ is the inflection point at which the curvature of the curve changes sign at the aggregate-level and γ_{τ_c} measures the marginal effect of socioeconomic condition of area i on the inflection point. In practical terms, Column (7) indicates that a one-percent increase in the proportion of professionals within an area in 1861 would result in the inflection point occurring 1.473 years earlier.

While our primary specifications utilise socioeconomic measures in 1861 preceding the mortality transition to mitigate potential reverse causality concerns, these findings prove robust to alternative temporal measurements. Specifically, analyses employing socioeconomic indicators from 1871 and the 1880s yield qualitatively similar results, albeit with a modestly reduced sample size in later periods. This temporal consistency in effect sizes across different measurement periods reinforces the structural nature of the relationship between socioeconomic inequality and mortality dynamics, rather than reflecting measurement artifacts specific to the 1861 baseline.

We also note that the estimates for γ_{δ_l}, which capture the association between S_i and the asymptotic mortality levels that emerge in the decades following the end of the sample, are positive and not statistically significantly different from zero for the professional class in 1861. This finding indicates that variation in socioeconomic conditions during the

Table 2.6.2: Estimated effects of socioeconomic inequality on crude mortality dynamics, 1850–1935

$d_{i,t}$: crude mortality									
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
S_i	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s
ρ				0.619***	0.605***	0.544***	0.593***	0.525***	0.510***
				(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
δ_l	4.777	8.872***	10.903***	6.713	8.103***	11.939***	10.019***	10.205***	13.208***
	(0.194)	(0.000)	(0.000)	(0.200)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{δ_l}	0.207	0.225***	0.034**	0.125	0.244**	-0.038	0.147	0.293***	-0.106***
	(0.488)	(0.000)	(0.024)	(0.760)	(0.036)	(0.301)	(0.369)	(0.000)	(0.005)
δ_u	25.159***	27.591***	25.932***	24.865***	27.690***	24.406***	26.438***	30.341***	25.076***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{δ_u}	0.282***	-0.322***	-0.168***	0.304	-0.334**	-0.075**	0.034	-0.376***	-0.102***
	(0.004)	(0.000)	(0.000)	(0.127)	(0.019)	(0.034)	(0.808)	(0.006)	(0.003)
β	0.057***	0.080***	0.093***	0.065***	0.077***	0.156***	0.080***	0.071***	0.158***
	(0.000)	(0.000)	(0.000)	(0.003)	(0.008)	(0.000)	(0.001)	(0.001)	(0.000)
γ_β	-0.001*	0.001	0.001	-0.002	0.001	-0.003***	-0.001	0.002	-0.003***
	(0.078)	(0.565)	(0.323)	(0.203)	(0.668)	(0.009)	(0.488)	(0.553)	(0.001)
τ_c	1,931.131***	1,913.355***	1,908.367***	1,926.005***	1,913.564***	1,909.667***	1,916.618***	1,916.256***	1,909.333***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{τ_c}	-3.053***	-1.296***	-0.464***	-2.788**	-1.213***	-0.557***	-1.473***	-1.580***	-0.560***
	(0.000)	(0.000)	(0.000)	(0.023)	(0.005)	(0.000)	(0.001)	(0.000)	(0.000)
ζ_E							-0.804	-2.416***	-0.368
							(0.172)	(0.000)	(0.496)
ζ_N							-2.852***	-2.975***	-0.043
							(0.000)	(0.000)	(0.921)
ζ_S							-1.893***	-3.345***	-1.053**
							(0.000)	(0.000)	(0.013)
ζ_W							-2.728***	0.969	1.524***
							(0.000)	(0.273)	(0.001)
$\tau_c + \gamma_{\tau_c}\overline{S_i}$	1905.213	1901.339	1901.265	1902.387	1902.307	1901.139	1904.139	1901.598	1900.760
N	1,577	1,175	1,288	1,518	1,137	1,237	1,518	1,137	1,237
# of areas	27	20	25	27	20	25	27	20	25

[a] The table shows the estimated effects of measures of socioeconomic inequality on crude mortality in administrative areas of London for Equation 2.6.2a–Equation 2.6.2c using the Non-Linear Least Squares. The dependent variable is the crude death rate $d_{i,t}$ in area i and year t. Standard errors are obtained using the heteroskedasticity-consistent covariance matrix estimator (Davidson and MacKinnon, 2004, p. 200). p-values in parentheses, where * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The reported p-values are based on two-sided tests. Given the directional hypothesis—both model and stylised empirical evidence suggest initial mortality divergence followed by convergence i.e., negative γ_{τ_c}—the one-sided tests would yield even stronger statistical significance.
[b] The results are generated using the whole sample, which is also the restricted sample for which the observed values display downward trend. The death rates are collected from the MOH reports and UK Data Service (see Sections 2.3).
[c] $\overline{S_i}$ is the weighted average of respective socioeconomic conditions across all areas for which such data are accessible, where weight is the population in area i in 1861, 1871, and 1881 respectively for three socioeconomic variables. $\tau_c + \gamma_{\tau_c}\overline{S_i}$ is the estimated inflection point (see Appendix 2.8.2).

1860s across regions did not generate persistent effects on mortality patterns towards the middle of the 20th century. Such evidence is consistent with the model prediction of eventual convergence as health innovations diffused across the population. This eventual convergence is also observed in Kesztenbaum and Rosenthal (2016); Costa and Kahn (2015).

Furthermore, Figure 2.6.5 compares the coefficient of variation of a) the observed crude mortality, b) crude mortality predicted by the four-parameter logistic function Equation 1.2.2b, and c) crude mortality predicted by the panel regression Equation 2.6.2c i.e., Column (7) in Table 2.6.2 across all areas in London. The latter two resemble the empirical trend which elucidates that mortality heterogeneity across all areas in London increased until around 1910, before dropping afterwards. In addition, c) implies that socioeconomic inequality accounts for 60%–80% of the variations in the observed crude mortality rates.

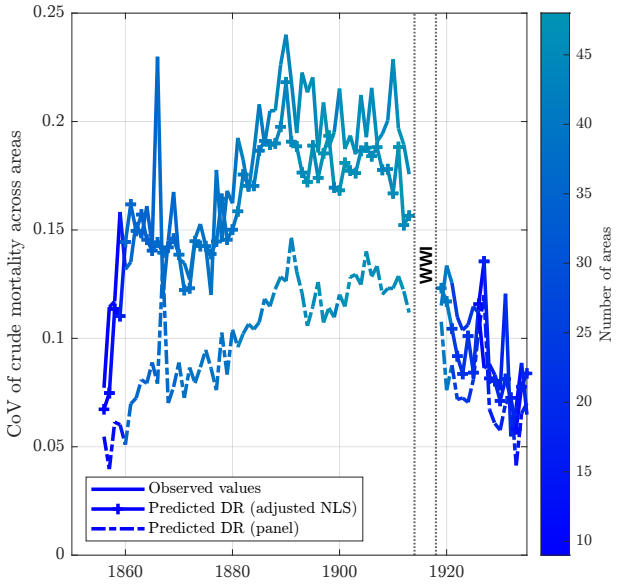

Figure 2.6.5: **Coefficient of variation of observed vs. predicted crude mortality across areas for London.** This figure shows the coefficient of variation of a) observed crude mortality;[43] b) crude mortality predicted by the four-parameter logistic function Equation 1.2.2b, and c) crude mortality predicted by the panel regression Equation 2.6.2c across all areas in London. Due to boundary changes and data issues discussed in Section 2.3, the areas (and the number of areas) used to calculate this coefficient vary over time. Both b) and c) predict the increasing-decreasing trend well.

43. For the sake of comparison, Figure 2.6.5 plots the coefficient of variation of observed crude mortality across a sub-sample of areas which are involved in the panel regression using % Professional, 1861 Census as the socioeconomic measure (Column (7) in Table 2.6.2), so the solid line is not identical with Figure 2.4.2, which uses the whole sample.

The formal panel analysis reaffirms the validity of the economic model of health progress diffusion dynamics developed in Section 1.3 of Chapter 1, in which socioeconomic inequality is a key driver of the sigmoid mortality dynamics and the disparities in mortality trends across different regions. While other theories may explain the sigmoid pattern of mortality dynamics at the city level, they are less equipped to capture the "divergence-convergence" pattern observed in mortality trends. As discussed in Section 1.4 of Chapter 1, if the adoption of new technologies follows an S-curve, mortality rates at the city-level will mirror this pattern. However, without accounting for socioeconomic inequality, we must assume that health improvements affect all populations uniformly. In this case, mortality reductions would be faster in areas that had higher initial mortality i.e., areas with more people at risk, and mortality inequality should decrease from the beginning, contradicting the empirical evidence. Thus, our explanation is essential and necessary for understanding the "divergence-convergence" pattern in the spatial heterogeneity of mortality dynamics.

Moreover, if we allow for relocation between areas i.e., residential sorting, the importance of diffusion dynamics in predicting the "divergence-convergence" pattern will be enhanced. High-income individuals systematically move to affluent areas, and vice versa, which increases the proportion of high-income individuals in rich areas and reduces it in poorer regions (see e.g., Heblich et al., 2021; Lee and Lin, 2018; Banzhaf and Walsh, 2008; Depro et al., 2015). Consequently, this spatial sorting delays the inflection point in poor areas and strengthens the "divergence-convergence" pattern.

2.6.2.3.2 Addressing demographic composition concerns

In addition to crude mortality, as elaborated in Section 2.3.2, there exists a possibility that crude mortality transitions were primarily driven by declining infant mortality rates, given the severity of infant mortality at the period. Evidence suggests that fertility rates—and thus likely the proportion of infants in the population—were inversely related with income (see e.g., Haines, 1979; Woods and Smith, 1983). Under this hypothesis, areas

with a higher proportion of poorer families, also likely had a higher proportion of infants, might experience earlier mortality transitions regardless of their socioeconomic conditions. Such a demographic mechanism would confound the explanatory power of socioeconomic inequalities in determining mortality dynamics.

To dilute this concern, we repeat the baseline estimation model Equation 2.6.2a–Equation 2.6.2c for infant and non-infant mortality respectively, and further examine age- and sex-corrected mortality measures.

Table 2.6.3 and Table 2.6.4 present the results of using infant and non-infant mortality as dependent variables respectively.[44] The results are very similar to Table 2.6.2—γ_{τ_c}s are negative and significant for both infant and non-infant mortality. In addition, γ_{τ_c}s exhibit smaller magnitudes when infant mortality is the dependent variable, relative to specifications using either crude or non-infant mortality rates. Therefore, age composition appears to have minimal influence on mortality dynamics. The smaller effects of socioeconomic inequalities on the inflection points in infant mortality may also imply that infants experience relatively modest gains from health innovations. This potentially explains the observed lag in infant mortality transition at both the city level and the area level (see discussions in Section 1.3.5 and empirical evidence in Sections 1.2.5 and 2.4).

Although the time series of age- and sex-corrected mortality is too short to be used to estimate the panel model (see Appendix 2.8.1.1.3), I plot these corrected death rates for all areas in Appendix 2.8.3.2. These corrected death rates show highly similar trends and exhibit strong correlations with crude mortality rates across all areas, endorsing the latter as the key mortality measure.

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44. Table 2.6.3 and Table 2.6.4 include only those areas where the observed mortality rates show a declining trend. In other words, if an area has infant mortality data for just two or three years, making it difficult to detect a declining trend, it will be excluded from the sample.

Table 2.6.3: Estimated effects of socioeconomic inequality on infant mortality dynamics, 1852–1935

$d_{i,t}$: infant mortality						
	(1)	(2)	(3)	(4)	(5)	(6)
S_i	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s
ρ				0.450***	0.440***	0.434***
				(0.000)	(0.000)	(0.000)
δ_l	71.021***	69.216***	66.779***	77.675***	85.942***	75.107***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{δ_l}	-0.862	-0.563	0.067	-1.111	-1.503	-0.525**
	(0.137)	(0.346)	(0.551)	(0.309)	(0.149)	(0.013)
δ_u	177.283***	173.556***	168.203***	185.092***	188.449***	171.611***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{δ_u}	-1.407***	-1.072***	-0.464***	-2.254***	-2.189***	-0.694***
	(0.000)	(0.000)	(0.000)	(0.002)	(0.000)	(0.003)
β	0.234***	0.231***	0.223***	0.271***	0.281***	0.271***
	(0.000)	(0.000)	(0.000)	(0.006)	(0.003)	(0.003)
γ_β	-0.006*	-0.005	0.001	-0.006	-0.006	0.001
	(0.074)	(0.130)	(0.629)	(0.504)	(0.518)	(0.838)
τ_c	1,915.515***	1,916.048***	1,913.047***	1,913.842***	1,914.112***	1,911.407***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{τ_c}	-0.569***	-0.616***	-0.110**	-0.590*	-0.622**	-0.143*
	(0.001)	(0.000)	(0.032)	(0.083)	(0.038)	(0.089)
ζ_E				-2.742	-8.106	-0.154
				(0.561)	(0.100)	(0.975)
ζ_N				0.433	-3.764	-1.494
				(0.937)	(0.413)	(0.739)
ζ_S				-6.325	-12.407***	-3.535
				(0.230)	(0.005)	(0.423)
ζ_W				16.371**	24.597***	17.812***
				(0.023)	(0.003)	(0.001)
$\tau_c + \gamma_{\tau_c}\overline{S_i}$	1910.525	1910.736	1911.381	1908.684	1908.759	1909.255
N	731	707	803	682	662	746
# of areas	19	17	25	19	17	25

[a] The table shows the estimated effects of measures of socioeconomic inequality on infant mortality in administrative areas of London for Equation 2.6.2a–Equation 2.6.2c using the Non-Linear Least Squares. The dependent variable is the infant mortality rate $d_{i,t}$ in area i and year t. Standard errors are obtained using the heteroskedasticity-consistent covariance matrix estimator (Davidson and MacKinnon, 2004, p. 200). p-values in parentheses, where * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The reported p-values are based on two-sided tests. Given the directional hypothesis—both model and stylised empirical evidence suggest initial mortality divergence followed by convergence i.e., negative γ_{τ_c}—the one-sided tests would yield even stronger statistical significance.

[b] The results are generated using the restricted sample for which the observed values display downward trend. The death rates are collected from the MOH reports and UK Data Service (see Sections 2.3).

[c] $\overline{S_i}$ is the weighted average of respective socioeconomic conditions across all areas for which such data are accessible, where weight is the population in area i in 1861, 1871, and 1881 respectively for three socioeconomic variables. $\tau_c + \gamma_{\tau_c}\overline{S_i}$ is the estimated inflection point (see Appendix 2.8.2).

Table 2.6.4: Estimated effects of socioeconomic inequality on non-infant mortality dynamics, 1852–1935

$d_{i,t}$: non-infant mortality						
	(1)	(2)	(3)	(4)	(5)	(6)
S_i	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s
ρ				0.544***	0.470***	0.371***
				(0.000)	(0.000)	(0.000)
δ_l	11.325***	11.593***	14.008***	11.243***	12.423***	8.861**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.015)
γ_{δ_l}	0.119**	0.082	0.303***	0.172	0.118	-0.457***
	(0.013)	(0.154)	(0.000)	(0.298)	(0.303)	(0.001)
δ_u	28.643***	28.233***	29.119***	29.081***	29.075***	27.187***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{δ_u}	-0.383***	-0.327***	-0.558***	-0.338***	-0.302***	-0.019
	(0.000)	(0.000)	(0.000)	(0.001)	(0.000)	(0.818)
β	0.121***	0.124***	0.858*	0.137*	0.131**	0.077***
	(0.000)	(0.000)	(0.077)	(0.081)	(0.026)	(0.000)
γ_β	0.002	0.002	-0.048*	-0.001	0.001	-0.002***
	(0.547)	(0.655)	(0.058)	(0.923)	(0.881)	(0.001)
τ_c	1913.430***	1912.401***	1901.906***	1,911.310***	1,909.860***	1,908.949***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{τ_c}	-1.072***	-0.830***	-0.243***	-0.906*	-0.646*	-0.903**
	(0.000)	(0.001)	(0.000)	(0.075)	(0.067)	(0.026)
ζ_E				0.118	-0.195	1.065
				(0.880)	(0.787)	(0.269)
ζ_N				-1.764***	-2.494***	-0.452
				(0.004)	(0.000)	(0.499)
ζ_S				-1.071*	-1.934***	-0.490
				(0.089)	(0.001)	(0.475)
ζ_W				-0.035	2.100**	3.344***
				(0.966)	(0.020)	(0.000)
$\tau_c + \gamma_{\tau_c}\overline{S_i}$	1903.799	1904.343	1897.974	1903.189	1903.597	1923.596
N	674	605	525	613	552	478
# of areas	20	16	13	20	16	13

[a] The table shows the estimated effects of measures of socioeconomic inequality on non-infant mortality in administrative areas of London for Equation 2.6.2a–Equation 2.6.2c using the Non-Linear Least Squares. The dependent variable is the non-infant mortality rate $d_{i,t}$ in area i and year t. Standard errors are obtained using the heteroskedasticity-consistent covariance matrix estimator (Davidson and MacKinnon, 2004, p. 200). p-values in parentheses, where * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The reported p-values are based on two-sided tests. Given the directional hypothesis—both model and stylised empirical evidence suggest initial mortality divergence followed by convergence i.e., negative γ_{τ_c}—the one-sided tests would yield even stronger statistical significance.

[b] The results are generated using the restricted sample for which the observed values display downward trend. The death rates are collected from the MOH reports and UK Data Service (see Sections 2.3).

[c] $\overline{S_i}$ is the weighted average of respective socioeconomic conditions across all areas for which such data are accessible, where weight is the population in area i in 1861, 1871, and 1881 respectively for three socioeconomic variables. $\tau_c + \gamma_{\tau_c}\overline{S_i}$ is the estimated inflection point (see Appendix 2.8.2).

2.6.2.3.3 Extended model results

To summarise, socioeconomic inequalities at the beginning of the mortality transition period exhibit robust associations with variations in mortality dynamics across areas in London over the subsequent century—initially manifesting as mortality divergence during the early diffusion of health technologies, before transitioning to convergence as the mortality transition approached its terminal phase.

Table 2.6.5 employs alternative socioeconomic variables that measure the lower tail of the socioeconomic distribution—the proportion of the industrial class and the proportion of the poverty class (see Table 2.6.5), as well as the ratios of the proportion of the professional class to the industrial class and the proportion of the middle class to the poverty class (Angelopoulos et al., 2024). Table 2.6.6 presents results from additional robustness checks—using the observed death rate minus the deviation of the city-level death rate in that year from the city-level trend as the dependent variable to account for possible random shocks to the death rates at the city level (Columns (1)–(3)), using overcrowding data as proxies for potential access to clean water and ventilation (Columns (4)–(5)), using the shortest distance from each area's centroid to the Thames rather than five regional dummies (Columns (6)–(8)) to control for geographic effects, and constraining the sample to areas where the corresponding estimated inflection points fall within the interval 1850 to 1935 (Column (9)).

Across these specifications, the γ_{τ_c} coefficients demonstrate robust and consistent patterns. When examining measures that capture the lower tail of the socioeconomic distribution, we observe consistently positive and statistically significant γ_{τ_c}, as evidenced throughout Table 2.6.5 and in Columns (4)–(5) of Table 2.6.6. This pattern inverts—yielding negative and statistically significant estimates—across Columns (1)–(3) and (6)–(9) in Table 2.6.6, when analysing the upper segments of the socioeconomic distribution.

Table 2.6.5: Estimated effects of socioeconomic inequality (lower tail) on crude mortality dynamics, 1850–1935

$d_{i,t}$: crude mortality						
	(1)	(2)	(3)	(4)	(5)	(6)
S_i	% Industrial, 1861 Census	% Industrial, 1871 Census	% Poverty, Booth Data 1880s	% Industrial, 1861 Census	% Industrial, 1871 Census	% Poverty, Booth Data 1880s
ρ				0.548***	0.526***	0.510***
				(0.000)	(0.000)	(0.000)
δ_l	13.689***	71.564***	11.626***	12.253*	20.176***	13.508***
	(0.000)	(0.000)	(0.000)	(0.051)	(0.001)	(0.000)
γ_{δ_l}	-0.069	-1.278***	0.003	-0.037	-0.175	-0.061
	(0.312)	(0.000)	(0.934)	(0.727)	(0.121)	(0.520)
δ_u	24.192***	34.554***	14.498***	28.640***	37.195***	18.962***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{δ_u}	-0.001	-0.138	0.252***	-0.077	-0.225**	0.122
	(0.973)	(0.257)	(0.000)	(0.277)	(0.019)	(0.179)
β	-0.004	0.015	0.130***	-0.016	-0.082	0.087
	(0.895)	(0.739)	(0.005)	(0.744)	(0.407)	(0.302)
γ_β	0.001**	0.000	-0.001	0.002	0.003	0.000
	(0.027)	(0.520)	(0.666)	(0.112)	(0.152)	(0.889)
τ_c	1,869.156***	1,779.477***	1,889.845***	1,871.350***	1,847.482***	1,883.925***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{τ_c}	0.673***	2.458***	0.412***	0.605**	0.949***	0.581***
	(0.000)	(0.000)	(0.000)	(0.042)	(0.003)	(0.003)
ζ_E				1.205*	2.009**	1.055*
				(0.067)	(0.017)	(0.087)
ζ_N				-0.829	-0.452	-0.081
				(0.169)	(0.565)	(0.885)
ζ_S				-0.131	-0.977	-1.099**
				(0.823)	(0.203)	(0.044)
ζ_W				-2.695***	-0.844	-0.766
				(0.000)	(0.404)	(0.161)
$\tau_c + \gamma_{\tau_c}\overline{S_i}$	1907.408	1914.630	1903.113	1905.790	1899.620	1902.626
N	1,577	1,175	1,288	1,518	1,137	1,237
# of areas	27	20	25	27	20	25

[a] The table shows the estimated effects of measures of socioeconomic inequality on crude mortality in administrative areas of London for Equation 2.6.2a–Equation 2.6.2c using the Non-Linear Least Squares. The dependent variable is the crude death rate $d_{i,t}$ in area i and year t. Standard errors are obtained using the heteroskedasticity-consistent covariance matrix estimator (Davidson and MacKinnon, 2004, p. 200). p-values in parentheses, where * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The reported p-values are based on two-sided tests. Given the directional hypothesis—both model and stylised empirical evidence suggest initial mortality divergence followed by convergence i.e., negative γ_{τ_c}—the one-sided tests would yield even stronger statistical significance.

[b] The results are generated using the whole sample, which is also the restricted sample for which the observed values display downward trend. The death rates are collected from the MOH reports and U.K. Data Service (see Sections 2.3).

[c] $\overline{S_i}$ is the weighted average of respective socioeconomic conditions across all areas for which such data are accessible, where weight is the population in area i in 1861, 1871, and 1881 respectively for three socioeconomic variables. $\tau_c + \gamma_{\tau_c}\overline{S_i}$ is the estimated inflection point (see Appendix 2.8.2).

Table 2.6.6: Estimated effects of socioeconomic inequality on crude mortality dynamics, robustness checks, 1850–1935

	$\tilde{d}_{i,t}$: city-adjusted crude mortality			$d_{i,t}$: crude mortality					
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
S_i	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s	% Over-crowding, 1891 Census	% Over-crowding, 1901 Census	% Professional, 1861 Census	% Professional, 1871 Census	% Middle, Booth Data 1880s	% Professional, 1861 Census
ρ	0.710***	0.656***	0.668***	0.492***	0.491***	0.586***	0.549***	0.533***	0.470***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
δ_l	12.353***	13.248***	15.081***	14.339***	15.713***	11.581***	10.580***	12.358***	6.632***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.007)
γ_{δ_l}	0.000	0.125**	-0.136***	-0.095	-0.168**	0.032	0.246*	-0.019	0.438***
	(1.000)	(0.024)	(0.000)	(0.145)	(0.031)	(0.789)	(0.072)	(0.588)	(0.005)
δ_u	24.965***	30.733***	25.730***	18.541***	20.698***	26.418***	28.186***	24.740***	28.115***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{δ_u}	0.384	-0.458***	-0.125***	0.242***	0.250***	-0.039	-0.217**	-0.061*	-0.425***
	(0.157)	(0.003)	(0.002)	(0.000)	(0.000)	(0.750)	(0.050)	(0.087)	(0.000)
β	0.083***	0.087***	0.170***	0.162***	0.136***	0.085***	0.083***	0.155***	0.062***
	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)	(0.006)	(0.000)	(0.008)
γ_β	-0.002**	0.001	-0.003***	-0.003***	-0.002***	-0.001	0.001	-0.002*	0.003
	(0.012)	(0.643)	(0.000)	(0.001)	(0.000)	(0.549)	(0.714)	(0.053)	(0.325)
τ_c	1,928.256***	1,917.105***	1,908.738***	1,894.618***	1,892.584***	1,911.555***	1,909.572***	1,909.190***	1,913.452***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
γ_{τ_c}	-3.522***	-1.812***	-0.517***	0.487**	0.768***	-0.921**	-0.867**	-0.535***	-1.078*
	(0.001)	(0.000)	(0.000)	(0.014)	(0.001)	(0.014)	(0.041)	(0.000)	(0.081)
ζ_E	-1.153*	-2.886***	-1.024	-0.838	-2.514***				1.027
	(0.094)	(0.000)	(0.198)	(0.119)	(0.000)				(0.240)
ζ_N	-2.011***	-2.891***	-0.840	-2.626***	-3.750***				-0.406
	(0.001)	(0.000)	(0.153)	(0.000)	(0.000)				(0.653)
ζ_S	-1.943***	-3.252***	-1.346**	-1.062**	-2.412***				-0.682
	(0.002)	(0.000)	(0.035)	(0.026)	(0.000)				(0.408)
ζ_W	-1.121	2.440***	1.325**	-2.461***	-3.504***				1.817
	(0.128)	(0.004)	(0.025)	(0.000)	(0.000)				(0.141)
ζ						-0.652***	-0.703***	-0.238***	
						(0.000)	(0.000)	(0.005)	
$\tau_c + \gamma_{\tau_c}\overline{S_i}$	1898.347	1900.197	1900.765	1906.581	1908.402	1903.754	1901.525	1900.995	1903.669
N	1,474	1,111	1,186	1,474	1,474	1,518	1,137	1,237	557
# of areas	27	20	24	26	26	27	20	25	11

[a] The table shows the estimated effects of measures of socioeconomic inequality on crude mortality in administrative areas of London for Equation 2.6.2a–Equation 2.6.2c using the Non-Linear Least Squares. For Columns (1)–(3), the dependent variable is the observed death rate minus the deviation of the city-level death rate in that year from the city-level trend, i.e., $\tilde{d}_{i,t} = d_{i,t} - \left(d_t - \hat{d}_t\right)$, where d_t is the observed crude mortality in London in year t and $\hat{d}_t$ is the trend crude mortality in London in year t. Standard errors are obtained using the heteroskedasticity-consistent covariance matrix estimator (Davidson and MacKinnon, 2004, p. 200). p-values in parentheses, where * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The reported p-values are based on two-sided tests. Given the directional hypothesis—both model and stylised empirical evidence suggest initial mortality divergence followed by convergence i.e., negative γ_{τ_c}—the one-sided tests would yield even stronger statistical significance.

[b] The results are generated using the whole sample, which is also the restricted sample for which the observed values display downward trend. The death rates are collected from the MOH reports and UK Data Service (see Section 2.3).

[c] $\overline{S_i}$ is the weighted average of respective socioeconomic conditions across all areas for which such data are accessible, where weight is the population in area i in 1861, 1871, and 1881 respectively for three socioeconomic variables. $\tau_c + \gamma_{\tau_c}\overline{S_i}$ is the estimated inflection point (see Appendix 2.8.2).

2.7 Discussions and Conclusions

In this chapter, I first expand the time-series mortality dataset for London in Chapter 1 to a panel of almost 30 constituent areas in London, for which a) the geographic boundaries remained unchanged from the 1850s to the 1930s, b) mortality data exists in sufficient consecutive years to identify downward declines, and c) measures of socioeconomic inequalities at the beginning of this period exist.

Based on this novel dataset, we observed that mortality reductions in these constituent areas also manifest as sigmoid curves. The inflection points on these curves, therefore, serve to summarise the variation in the timing of mortality transitions. Our data revealed two primary findings regarding mortality dynamics. First, we documented substantial heterogeneity in the inflection points across different areas. Second, the mortality coefficient of variation exhibited a non-monotonic trajectory—increasing first, then exhibiting a decline.

To explain this pattern, we applied the economic model of health progress diffusion developed in Chapter 1. By introducing areas with different proportions of high-income residents, the model predicts that areas with better socioeconomic conditions would adopt new health technologies earlier, thereby experiencing mortality transition earlier. This implies that socioeconomic inequality is the main driver behind the "divergence-convergence" pattern observed in mortality heterogeneity.

To formally test this, we incorporated skewness measures of socioeconomic distribution into the logistic function, making the four parameters defining mortality declines vary with socioeconomic inequalities. Our findings confirmed that the marginal effects of higher socioeconomic status concentration on inflection points of the mortality decline trends are negative and statistically significant. In other words, areas with a higher proportion of residents in higher-class occupations or higher social classes at the beginning of the mortality

transition experienced earlier transitions.[45] Since reductions in mortality are prerequisites for economic growth, human capital development, and improvements in social welfare (see Section 1.2.1 in Chapter 1), the unequal timing at which these conditions were achieved led to enduring inequalities that persisted for generations.

Our theory and the formal statistical tests explain and capture the inequalities in mortality dynamics using socioeconomic inequalities. While other theories, such as the S-curve model of technology diffusion, may explain the sigmoid patterns in mortality trends observed in the previous chapter, they provide insufficient explanatory power for the empirical relationship between inflection points and socioeconomic inequalities demonstrated in this chapter.

This chapter has several limitations that warrant acknowledgement. The first constraint stems from data scarcity, particularly the absence of age-sex-corrected mortality rates across the 30 constituent areas over a long enough time period, owing to the unavailability of annual demographic composition data, which were not systematically collected in the 19th century for London. Second, the fertility pattern is not a focus in our analysis, presenting a promising avenue for future research. Furthermore, the theoretical framework employed here, while generating predictions that align with empirical observations, is somewhat stylised, as historical data limitations preclude direct calibration of parameters governing health technology diffusion and income distribution dynamics.

45. This conclusion holds across various mortality measures, socioeconomic variables, and robustness tests.

2.8 Appendix

2.8.1 Data Appendix

2.8.1.1 Construction of Mortality Dataset

2.8.1.1.1 Visualisation of spatial heterogeneity of crude mortality in London

46. This figure illustrates the general spatial heterogeneity in London. The displayed areas are not necessarily involved in the panel regressions. For instance, the largest area in the southeast represents a combination of multiple areas, such as Greenwich and Plumstead. Due to significant changes in authorities and boundaries in this region, we display Southeast as a whole for comparison purposes. In addition, the displayed areas are not necessarily the smallest I can get. In the panel analysis, for example, the City of Westminster in the top left corner is further divided into small parishes so that I can utilise the richest possible data depending on availability.

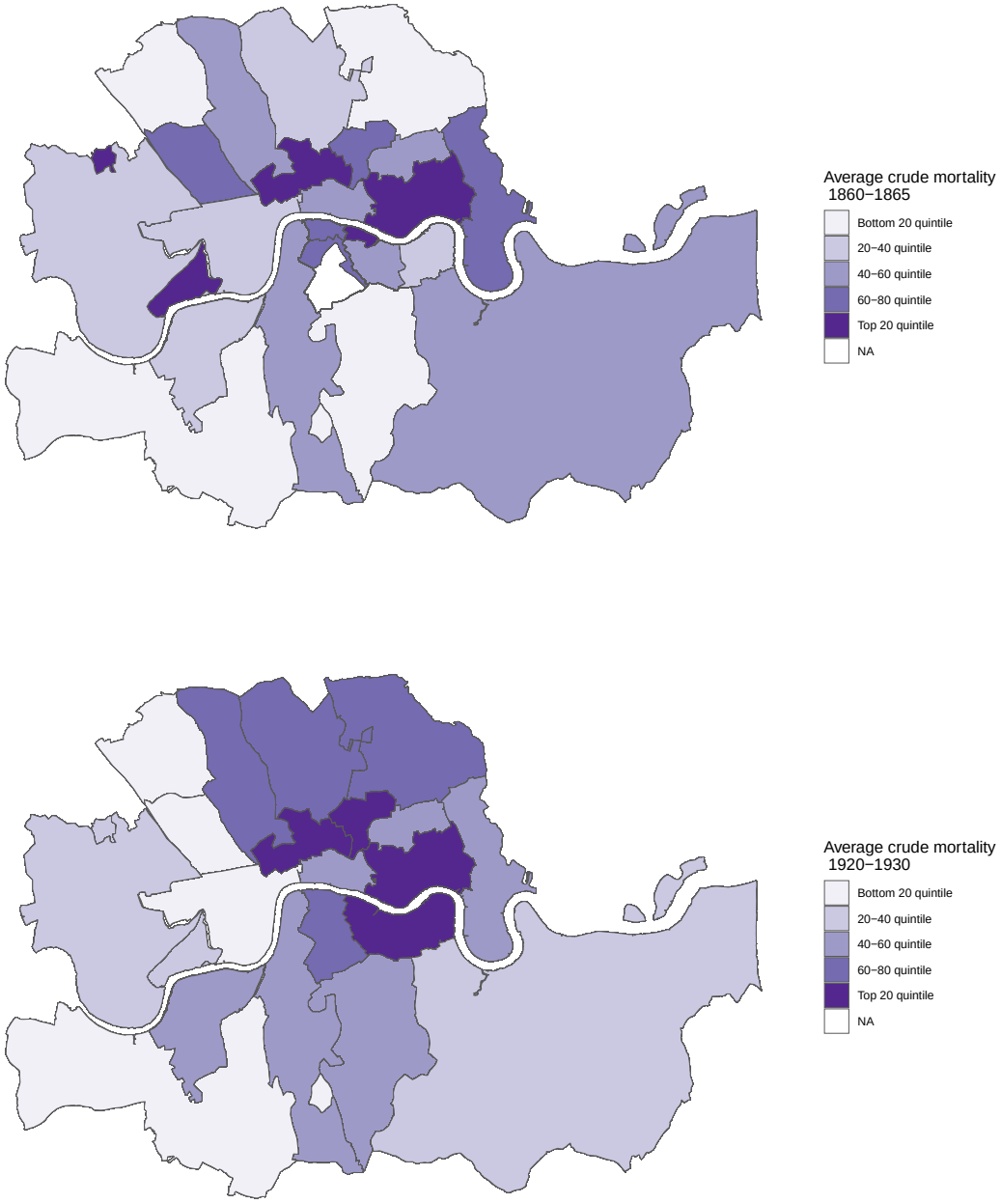


Figure 2.8.1: **Spatial heterogeneity of average crude mortality at the beginning and end of the mortality transition in London.** Average crude mortality 1860–1865 (above) and average crude mortality 1920–1930 (below), showing persistent spatial gradients in mortality rates across areas.[46]

2.8.1.1.2 Data availability of main mortality measures

Table 2.8.1: Data availability of mortality measures

	Crude mortality		Infant mortality		Non-infant mortality	
Time period ranges	# of areas	Average # of years available	# of areas	Average # of years available	# of areas	Average # of years available
Available pre- and post-1901						
1850s–1930s	17	67.24	20	85.50	1	48.00
1850s–1920s	3	58.00	2	58.00		
1860s–1930s	2	67.50			7	47.86
1860s–1920s	13	59.15	2	52.00	9	33.89
1870s–1920s	2	47.50			2	20.50
1880s–1930s	3	50.67				
1880s–1920s	1	39.00				
1890s–1930s			8	62.75		
1890s–1920s	2	26.50				
1890s–1910s	1	22.00				
Available pre- or post-1901						
1850s–1900s			11	23.82		
1860s–1900s	3	41.00			10	22.10
1870s–1900s	1	31.00	1	19.00	1	20.00
1860s–1890s					1	11.00
1900s–1920s	4	20.00				
1910s–1930s onwards			5	54.00		
Sum	52		49		31	

[a] This table shows the data availability of three mortality measures for all areas constructed. For most areas, we can track the mortality data for a relatively long period crossing the approximated inflection point at the city level.

2.8.1.1.3 Corrected death rate

As illustrated in Section 1.2.3.2.4 in Chapter 1, the corrected death rates correct variations in population composition, allowing for the comparison of death rates across different small areas in London. We apply the method of calculation described on Page 9 of Report of the Medical Officer of Health for London County Council (LCC) 1892 as follows:

1. Collect data on the age and sex distribution of the population of area A in each census year from 1851 to 1931;[47]

47. Data Source: Census reports, Population, England and Wales, 1851 to 1931, http://www.histpop.org.

2. Collect data on the average age and sex distribution of the population, along with the average number of deaths within each age and sex group in England and Wales for each decade from 1851–1860 to 1931–1940;[48]
3. Using the data from the previous step, calculate the average death rate for each sex at each age period in England and Wales for each decade from 1851–1860 to 1931–1940, say y;
4. Applying 1 to 3 gives *standard death rate* for A, say x, i.e., a death rate which would be correct on the assumption that the rates of mortality at each age period and for each sex were identical with the mean rates in England and Wales in the last decennium;
5. Calculate the factor for correcting the recorded death rate for A as $\frac{y}{x}$;
6. Multiply crude mortality as discussed above by $\frac{y}{x}$ to get the corrected death rate for A.

Therefore, under the assumption that the rates of mortality at each age period and for each sex were identical with the mean rates in England and Wales in the last decennium, we can obtain the corrected death rates for each area from 1851 to 1964.[49] See Table 2.8.2 for the summary statistics.

Figure 2.8.9 in Appendix 2.8.3.2 plots the crude mortality and corrected death rates for all available areas in London. The data exhibit highly similar trends and strong correlations across all areas, endorsing crude mortality as the key mortality measure.

Table 2.8.2: Data availability and summary statistics of corrected death rates, constituent areas in London

# of available areas	Min	Max	Mean	Std. dev.
48	9.60	45.08	21.40	5.63

48. Data Source: supplement to Registrar-General's reports (England) for each decade from 1851–1860 to 1901–1910; the 20th Century Mortality Files from 1911–1920 to 1931–1940, https://webarchive.nationalarchives.gov.uk/ukgwa/20160111174808/http:/www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm:77-215593.
49. This application also assumes that the constitution of the population has remained unchanged during the past ten years, which is not accurate (Page 14, Report of the MOH for LCC 1910).

2.8.1.2 Construction of Socioeconomic Dataset

Corresponding to Table 2.3.2, Table 2.8.3 lists the occupations classified as the "industrial" class in 1861 and 1871 Censuses. This variable proxies the lower tail of the socioeconomic distribution.

2.8.2 Statistical Analysis

Equation 2.8.1 is a statistical model estimating the effect of (the distribution of) socioeconomic conditions on the sigmoid dynamics of mortality rate:

$$\overline{d} = (\delta_l + \gamma_{\delta_l} S) + \frac{(\delta_u + \gamma_{\delta_u} S) - (\delta_l + \gamma_{\delta_l} S)}{1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}}, \tag{2.8.1}$$

where $S > 0$, $\delta_u + \gamma_{\delta_u} S > \delta_l + \gamma_{\delta_l} S$, and $\beta + \gamma_\beta S > 0$. The first-order derivative and the second-order derivative are:

$$\frac{d}{dt}\overline{d} = -\frac{\Big[(\delta_u + \gamma_{\delta_u} S) - (\delta_l + \gamma_{\delta_l} S)\Big] e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)} (\beta + \gamma_\beta S)}{\left[1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right]^2}; \tag{2.8.2}$$

$$\begin{aligned}
\frac{d^2}{dt^2}\overline{d} &= -\Big[(\delta_u + \gamma_{\delta_u} S) - (\delta_l + \gamma_{\delta_l} S)\Big] (\beta + \gamma_\beta S) \cdot \frac{d}{dt}\left[\frac{e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}}{\left[1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right]^2}\right] \\
&= -\Big[(\delta_u + \gamma_{\delta_u} S) - (\delta_l + \gamma_{\delta_l} S)\Big] (\beta + \gamma_\beta S) \\
&\quad \times \frac{\left[1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right]^2 e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)} (\beta + \gamma_\beta S)}{\left[1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right]^4} \\
&\quad \times \frac{- 2e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)} \left[1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right] e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)} (\beta + \gamma_\beta S)}{\left[1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right]^4} \\
&= -\Big[(\delta_u + \gamma_{\delta_u} S) - (\delta_l + \gamma_{\delta_l} S)\Big] (\beta + \gamma_\beta S)^2 \frac{e^{\Big((\beta+\gamma_\beta S_i)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)} \left(1 - e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right)}{\left[1 + e^{\Big((\beta+\gamma_\beta S)\big(t-(\tau_c+\gamma_{\tau_c} S)\big)\Big)}\right]^3}.
\end{aligned} \tag{2.8.3}$$

Table 2.8.3: Occupational classification of the industrial class, 1861 and 1871 Censuses

	Orders	Sub-orders
1	Persons engaged in art and mechanic productions, in which matters of various kinds are employed in combination	In books In musical instruments In prints and pictures In carving and figures In tackle for sports and games In designs, medals, and dies In watches and philosophical instruments In surgical instruments In arms In machines and tools In carriages In harness In ships In houses and buildings In furniture In implements In chemicals
2	Persons working and dealing in the textile fabrics and in dress	In wool and worsted In silk In cotton and flax In mixed materials In dress In hemp and other fibrous materials
3	Persons working and dealing in food and drinks	In animal food In vegetable food In drinks and stimulants
4	Persons working and dealing in animal substances	In grease, gut, bones, horn, ivory, and whalebone In skins, feathers, and quills In hair
5	Persons working and dealing in vegetable substances	In gums and resins In wood In bark In cane, rush and straw In paper
6	Persons working and dealing in minerals	In mining In coal In stone, clay In earthenware In glass In salt In water In gold, silver, and precious stones In copper In tin and quicksilver In zine In lead and antimony In brass and other mixed metals In iron and steel

[a] Source: Census of England & Wales Census, 1861. *Population Tables, Vol II, Part 1*, p. 12, Table 8, "Occupations of males and females under 20, and 20 years of age and upwards — in classes, orders, and sub-orders"; Census of England & Wales Census, 1871. *Population Abstracts, Vol III*, p. 12, Table 10, "Occupations of males and females under 20, and 20 years of age and upwards — in classes, orders, and sub-orders".

1. Given $S > 0$, we assume $\beta + \gamma_\beta S > 0$. Then $\lim_{t\to+\infty} \overline{d} = \delta_l + \gamma_{\delta_l} S$ and $\lim_{t\to-\infty} \overline{d} = \delta_u + \gamma_{\delta_u} S$. This means that the lower and upper asymptotes at the aggregate level are $\left(\delta_l + \gamma_{\delta_l}\overline{S}\right)$ and $\left(\delta_u + \gamma_{\delta_u}\overline{S}\right)$ respectively, where $\overline{S}$ is the average level of socioeconomic condition across all areas in the sample. Thus, γ_{δ_l} and γ_{δ_u} measure the marginal effects of the socioeconomic condition of area i on the lower and upper asymptotes of the mortality rate.
2. $\left(\tau_c + \gamma_{\tau_c}\overline{S}\right)$ has two properties:
   - It represents the year when the death rate lies halfway between the upper and lower asymptotes i.e., $\overline{d}\Big|_{t=\tau_c+\gamma_{\tau_c}\overline{S}} = \left(\left(\delta_l + \gamma_{\delta_l}\overline{S}\right) + \left(\delta_u + \gamma_{\delta_u}\overline{S}\right)\right)/2$;
   - It is the year of the inflection point at which the curvature changes sign. Based on the second-order derivative Equation 2.8.3, when $t < \tau_c + \gamma_{\tau_c}\overline{S}$, $e^{\left((\beta+\gamma_\beta S)\left(t-\left(\tau_c+\gamma_{\tau_c}S\right)\right)\right)} < 1 \implies 1 - e^{\left((\beta+\gamma_\beta S)\left(t-\left(\tau_c+\gamma_{\tau_c}S\right)\right)\right)} > 0$ so that $\frac{d^2}{dt^2}\overline{d}\Big|_{t<\tau_c+\gamma_{\tau_c}\overline{S}} < 0$. Similarly, $\frac{d^2}{dt^2}\overline{d}\Big|_{t=\tau_c+\gamma_{\tau_c}\overline{S}} = 0$ and $\frac{d^2}{dt^2}\overline{d}\Big|_{t>\tau_c+\gamma_{\tau_c}\overline{S}} > 0$. In other words, $\overline{d}(t)$ is concave when $t < \tau_c + \gamma_{\tau_c}\overline{S}$ and convex when $t > \tau_c + \gamma_{\tau_c}\overline{S}$. $\left(\tau_c + \gamma_{\tau_c}\overline{S}\right)$ is the inflection point at which the curvature of the curve changes sign at the aggregate-level and γ_{τ_c} measures the marginal effect of socioeconomic condition of area i on the inflection point.
   - $\frac{d^2}{dt^2}\overline{d}\Big|_{t=\tau_c+\gamma_{\tau_c}\overline{S}} = 0$ implies that the first-order derivative Equation 2.8.2 is maximised at $t = \tau_c + \gamma_{\tau_c}\overline{S}$, which means that the timing of the inflection point aligns with the maximum slope of the curve.
3. $\left(\beta + \gamma_\beta\overline{S}\right)$ shifts the slope of the trend in the death rate as Equation 2.8.2 illustrates. Since we assume $\delta_u + \gamma_{\delta_u} S > \delta_l + \gamma_{\delta_l} S$ and $\beta + \gamma_\beta S > 0$, the slope at the inflection point is negative. For example, at the inflection point where $t = \tau_c + \gamma_{\tau_c}\overline{S}$, the slope is:

$$\frac{d}{dt}\overline{d}\Big|_{t=\tau_c+\gamma_{\tau_c}\overline{S}} = -\frac{\left[\left(\delta_u + \gamma_{\delta_u} S\right) - \left(\delta_l + \gamma_{\delta_l} S\right)\right]\left(\beta + \gamma_\beta S\right)}{4}.$$

In addition, as described above, when the slope of the curve, $\frac{d}{dt}\overline{d}$ is maximal, $\frac{d^2}{dt^2}\overline{d} = 0$,

$$\begin{aligned}
&\frac{d^2}{dt^2}\overline{d} = 0 \\
\implies &-\left[(\delta_u + \gamma_{\delta_u} S) - (\delta_l + \gamma_{\delta_l} S)\right]\left(\beta + \gamma_\beta S\right)^2 \\
&\times \frac{e^{\left((\beta+\gamma_\beta S)\left(t-(\tau_c+\gamma_{\tau_c} S)\right)\right)}\left(1 - e^{\left((\beta+\gamma_\beta S)\left(t-(\tau_c+\gamma_{\tau_c} S)\right)\right)}\right)}{\left[1 + e^{\left((\beta+\gamma_\beta S)\left(t-(\tau_c+\gamma_{\tau_c} S)\right)\right)}\right]^3} = 0 \\
\implies &1 - e^{\left((\beta+\gamma_\beta S)\left(t-(\tau_c+\gamma_{\tau_c} S)\right)\right)} = 0 \\
\implies &t = \tau_c + \gamma_{\tau_c}\overline{S}, \text{ i.e., } \left.\frac{d}{dt}\overline{d}\right|_{t=\tau_c+\gamma_{\tau_c}\overline{S}} = 0.
\end{aligned} \tag{2.8.4}$$

Therefore, the maximum slope is reached at the inflection point when $t = \tau_c + \gamma_{\tau_c}\overline{S}$, which is $-\frac{\left[(\delta_u+\gamma_{\delta_u} S)-(\delta_l+\gamma_{\delta_l} S)\right](\beta+\gamma_\beta S)}{4}$. β and γ_β play important roles in determining the magnitude of steepness.

Then for each mortality measure j, we estimate the model in Equation 2.8.1 by applying Non-Linear Least Squares (NLS):

$$d_{i,t} = (\delta_l + \gamma_{\delta_l} S_i) + \frac{(\delta_u + \gamma_{\delta_u} S_i) - (\delta_l + \gamma_{\delta_l} S_i)}{1 + e^{\left((\beta+\gamma_\beta S_i)\left(t-(\tau_c+\gamma_{\tau_c} S_i)\right)\right)}} + \nu_{i,t}. \tag{2.8.5a}$$

where $\nu_{i,t}$ is the error term measuring the discrepancy between the actual value and the fitted curve. We show results from NLS estimation where $\nu_{i,t}$ is assumed to be independently distributed across area and time. $\nu_{i,t}$ includes area-specific random shocks $u_{i,t}$ which for each geography i, is serially correlated i.e., $\exists\ t, s\ (t \neq s) : \mathbb{E}\left[u_{i,t}, u_{i,s}\right] \neq 0$ and homoskedastic. We assume cross-sectional independence i.e., $\mathbb{E}\left[u_{i,t}, u_{k,t}\right] = 0$ for all areas $i \neq k$.

To take into account the serial correlation, we model the error term in Equation 2.8.5a as

$$u_{i,t} = \rho u_{i,t-1} + \epsilon_{i,t},$$

where $\epsilon_{i,t}$ is assumed to be independently distributed across areas and time. Then the statistical model of the sigmoid trend taking into account the serially correlated random shocks is

$$\begin{aligned} d_{i,t} = \rho d_{i,t-1} + \left[(\delta_l + \gamma_{\delta_l} S_i) + \frac{(\delta_u + \gamma_{\delta_u} S_i) - (\delta_l + \gamma_{\delta_l} S_i)}{1 + e^{\Big(\big(\beta + \gamma_\beta S_i\big) \big(t - \big(\tau_c + \gamma_{\tau_c} S_i \big) \big) \Big)}} \right] \\ - \rho \left[(\delta_l + \gamma_{\delta_l} S_i) + \frac{(\delta_u + \gamma_{\delta_u} S_i) - (\delta_l + \gamma_{\delta_l} S_i)}{1 + e^{\Big(\big(\beta + \gamma_\beta S_i\big) \big(t - 1 - \big(\tau_c + \gamma_{\tau_c} S_i \big) \big) \Big)}} \right] + \epsilon_{i,t}, \end{aligned} \tag{2.8.5b}$$

where the error term $\epsilon_{i,t}$ is independently distributed and heteroskedastic across time for each geography i.[50]

To estimate Equation 2.8.5b using the Gauss-Newton method, we apply the algorithm below.

Algorithm 3. *solving the diffusion model*

1. Start with initial guesses for the parameters
   $\boldsymbol{\Theta_0} = (\rho_0, \delta_{l,0}, \gamma_{\delta_l,0}, \delta_{u,0}, \gamma_{\delta_u,0}, \beta_0, \gamma_{\beta,0}, \tau_{c,0}, \gamma_{\tau_c,0})$.
2. Evaluate the predicted value at the guessed parameters and compute the in-sample prediction errors (residuals):

   $$\boldsymbol{r_{i,k}} = \boldsymbol{d_{i,t}} - \boldsymbol{\hat{d}_{i,t,k}},$$

   where $\boldsymbol{d_{i,t}}$ is the observed value, $\boldsymbol{\hat{d}_{i,t,k}}$ is the predicted value based on guesses of parameters in iteration k, $\boldsymbol{r_{i,k}}$ is the vector of residuals in iteration k.
3. Calculate the Jacobian matrix $\boldsymbol{J_{i,k}}$ containing the partial derivatives of the function with respect to $\boldsymbol{\Theta_k}$.
4. Update the parameter estimates using the Gauss-Newton formula:

   $$\boldsymbol{\Theta_{k+1}} = \boldsymbol{\Theta_k} + \left(\boldsymbol{J_{i,k}^T J_{i,k}} \right)^{-1} \boldsymbol{J_{i,k}^T r_{i,k}}.$$

50. See Appendix 1.5.2 in Chapter 1 for discussions on heteroskedasticity.

5. Check for convergence. If $\left|\boldsymbol{\Theta_{k+1}} - \boldsymbol{\Theta_k}\right| < \varepsilon$, terminate the iteration, otherwise go back to Step 2 with the updated parameters $\boldsymbol{\Theta_{k+1}}$.

Note that $\boldsymbol{r_{i,k}}$ denotes the (temporary) estimation discrepancy in iteration k and $\epsilon_{i,t}$ in Equation 2.8.5b is the error term after convergence is achieved.

As discussed in the previous section, we confirm the uniqueness of the local estimates provided by **Algorithm 3** by implementing a grid search method.

Algorithm 4. *ensuring the local uniqueness of estimates of the diffusion model*

1. Set initial guesses of parameters $\boldsymbol{\Theta_0}$ based on the empirical values — set $\delta_{l,0}$ and $\delta_{u,0}$ as the average values of all areas' minimal and maximal death rates in the sample i.e., $\delta_{l,0} = \frac{1}{N}\sum_{i=1}^{N}\bar{\delta}_{i,l}$, $\delta_{u,0} = \frac{1}{N}\sum_{i=1}^{N}\bar{\delta}_{i,u}$ where $\bar{\delta}_{i,l}$ and $\bar{\delta}_{i,u}$ are the minimal and maximal values of death rates of geography i across all sampled time periods, which have been used in Step 1 of **Algorithm 2** in Chapter 1; Set $\tau_{c,0}$ as the midpoint of the sample period i.e., $\tau_{c,0} = (t_{max} - t_{min})/2$, and β_0 as 0.1. Set $\rho_0 = 0.5$. Apply **Algorithm 3** to identify the benchmark estimates $\boldsymbol{\Theta^*}$.
2. Define sensible parameter space for each parameter

$$\boldsymbol{\Theta_{min}} = (\delta_{l,min}, \gamma_{\delta_l,min}, \delta_{u,min}, \gamma_{\delta_u,min}, \beta_{min}, \gamma_{\beta,min}, \tau_{c,min}, \gamma_{\tau_c,min}) \text{ and}$$
$$\boldsymbol{\Theta_{max}} = (\delta_{l,max}, \gamma_{\delta_l,max}, \delta_{u,max}, \gamma_{\delta_u,max}, \beta_{max}, \gamma_{\beta,max}, \tau_{c,max}, \gamma_{\tau_c,max}):$$

$$\begin{aligned}
&\rho_{min} = 0.4, && \rho_{max} = 0.9\\
&\delta_{l,min} = \lfloor\bar{\delta}_l\rfloor, && \delta_{l,max} = \lceil\bar{\delta}_l\rceil,\\
&\delta_{u,min} = \lfloor\bar{\delta}_u\rfloor, && \delta_{u,max} = \lceil\bar{\delta}_u\rceil,\\
&\beta_{min} = \beta^* - 2\ \text{s.e.}\left(\beta^*\right), && \beta_{max} = \beta^* + 2\ \text{s.e.}\left(\beta^*\right),\\
&\tau_{c,min} = 1870, && \tau_{c,max} = 1925\\
&\gamma_{j,min} = \gamma_j^* - \ \text{s.e.}\left(\gamma_j^*\right), && \gamma_{j,max} = \gamma_j^* + \ \text{s.e.}\left(\gamma_j^*\right) \text{ for } j = \delta_u, \delta_l, \beta, \tau_c.
\end{aligned}$$

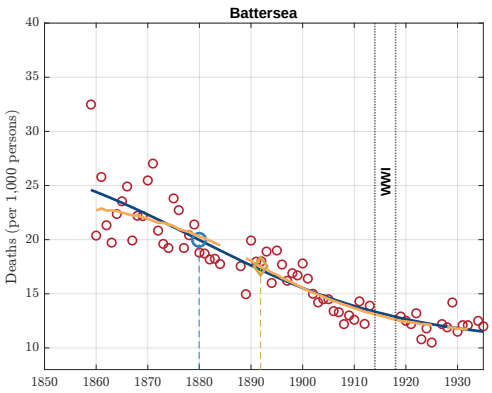
3. Create evenly-spaced grid for each parameter $\boldsymbol{\mathcal{G}_\Theta} \in [\boldsymbol{\Theta_{min}}, \boldsymbol{\Theta_{max}}]$. The step between adjacent grid points is set as 5 for δ_l, δ_u and τ_c, as 0.1 for ρ and as 0.01 for $\beta, \gamma_j, j = \delta_u, \delta_l, \beta, \tau_c$.

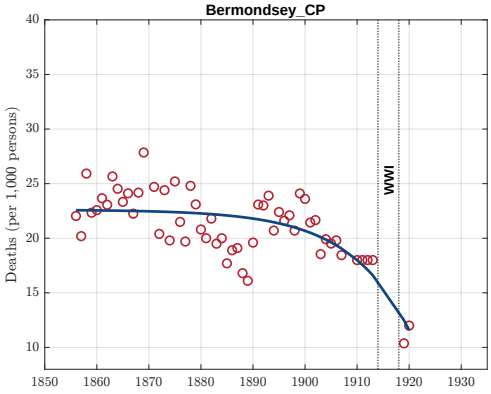
4. Take one point/parameter combination in the grid as the initial guess $\boldsymbol{\Theta_0}$ and apply **Algorithm 3** to get the resulted estimates $\tilde{\boldsymbol{\Theta}}$. If $\left|\tilde{\boldsymbol{\Theta}} - \boldsymbol{\Theta}^*\right| < \varepsilon$, take the next point in the grid and repeat this step, otherwise store the resulted estimates.

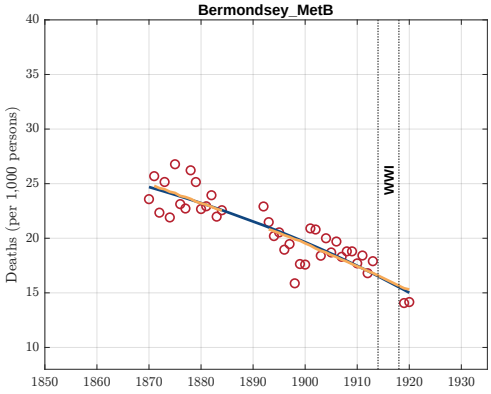
2.8.3 Additional Results for Mortality Dynamics

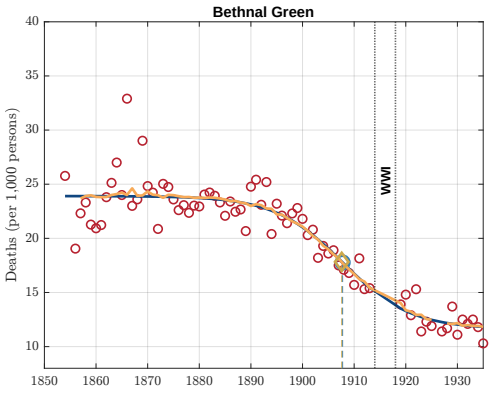
2.8.3.1 Dynamics of Crude Mortality

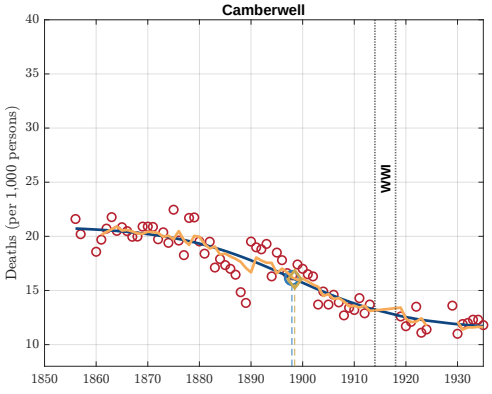
Figure 2.8.2 and Figure 2.8.3 plot the crude mortality and trends estimated by Equation 1.2.2a (blue curves) and Equation 1.2.2b (yellow curves) for all of 48 areas.

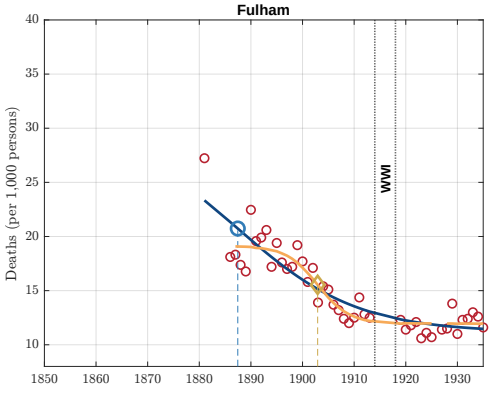

Battersea
Deaths (per 1,000 persons)
WWI

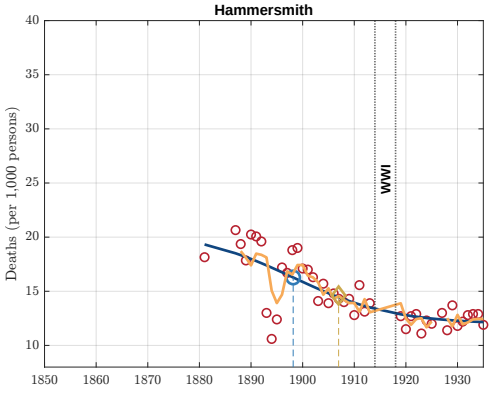

Bermondsey_CP
Deaths (per 1,000 persons)
WWI

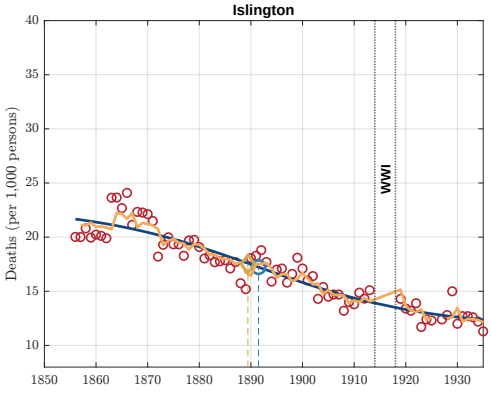

Bermondsey_MetB
Deaths (per 1,000 persons)
WWI

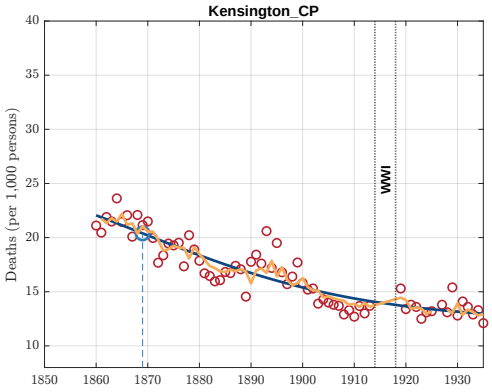

Bethnal Green
Deaths (per 1,000 persons)
WWI

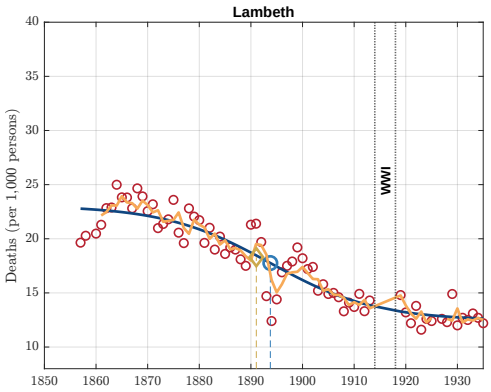

Camberwell
Deaths (per 1,000 persons)
WWI

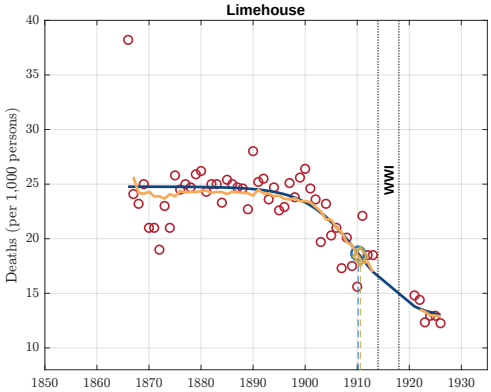

Fulham
Deaths (per 1,000 persons)
WWI

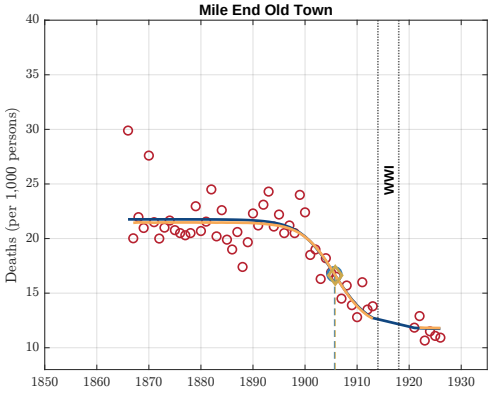

Hammersmith
Deaths (per 1,000 persons)
WWI

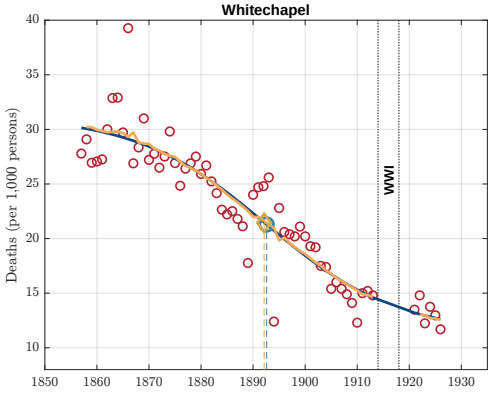

Islington
Deaths (per 1,000 persons)
WWI

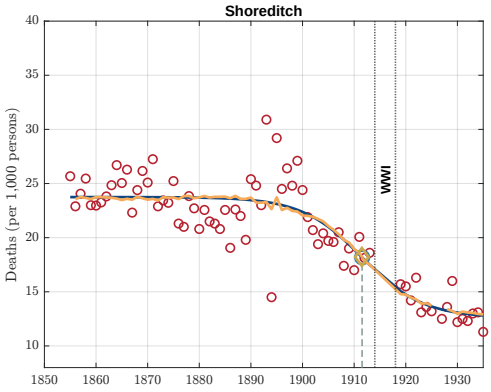

Kensington_CP
Deaths (per 1,000 persons)
WWI

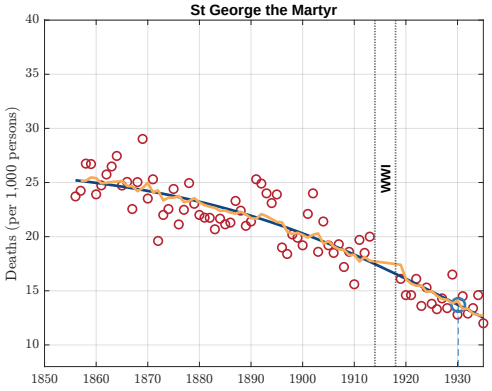

Lambeth
Deaths (per 1,000 persons)
WWI

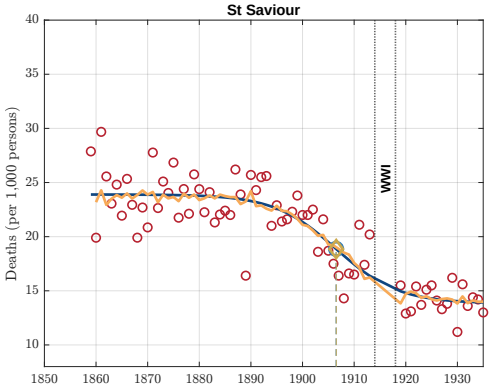

Limehouse
Deaths (per 1,000 persons)
WWI

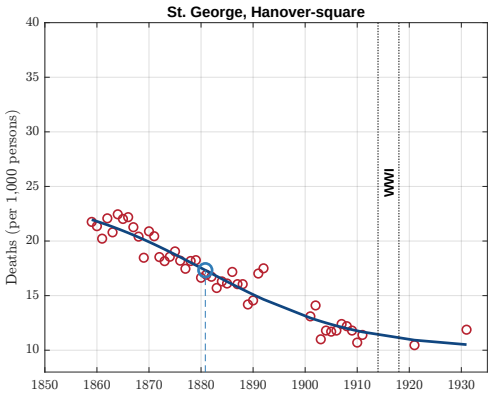

Mile End Old Town
Deaths (per 1,000 persons)
WWI

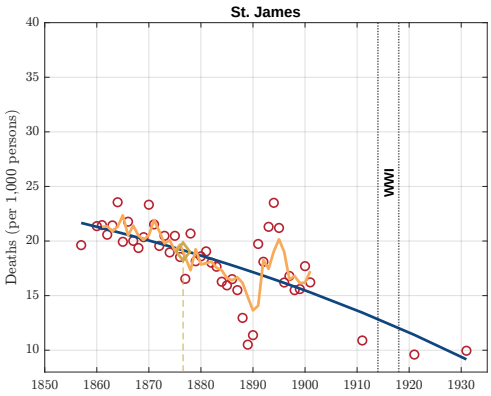

Whitechapel
Deaths (per 1,000 persons)
WWI

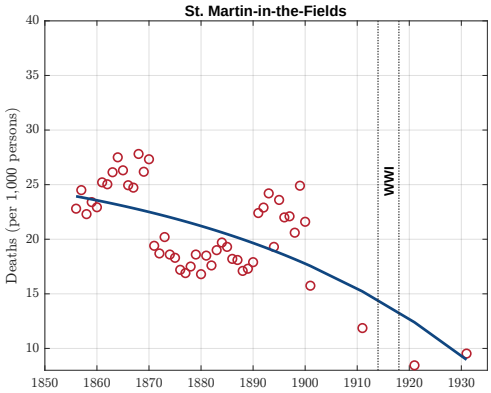

Shoreditch
Deaths (per 1,000 persons)
WWI

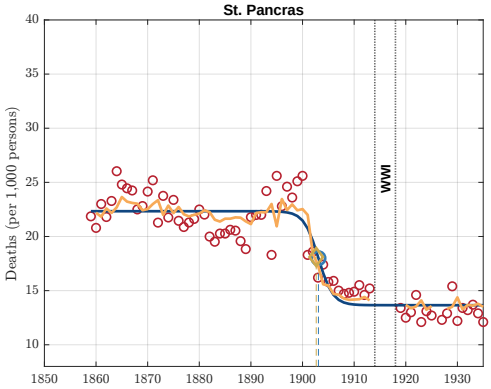

St George the Martyr
Deaths (per 1,000 persons)
WWI

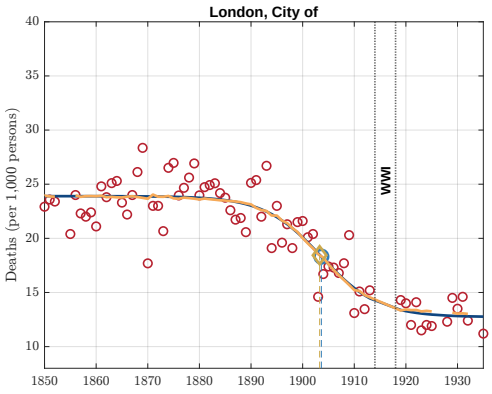

St Saviour
Deaths (per 1,000 persons)
WWI

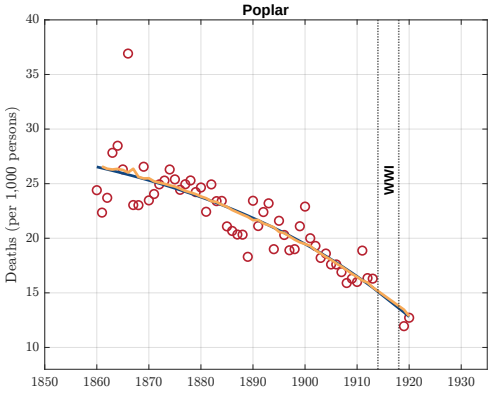

St. George, Hanover-square
Deaths (per 1,000 persons)
WWI

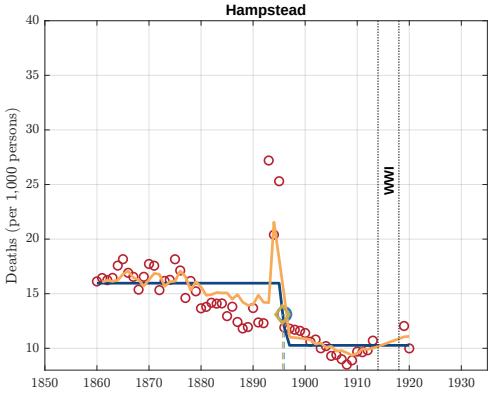

St. James
Deaths (per 1,000 persons)
WWI

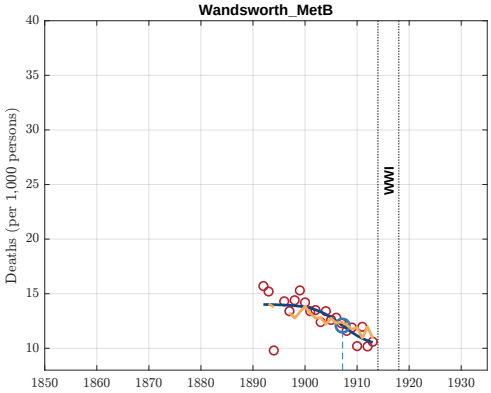

St. Martin-in-the-Fields
Deaths (per 1,000 persons)
WWI

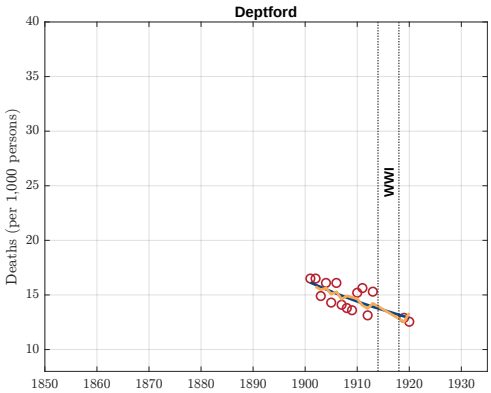

St. Pancras
Deaths (per 1,000 persons)
WWI


London, City of
Deaths (per 1,000 persons)
WWI


Poplar
Deaths (per 1,000 persons)
WWI


Hampstead
Deaths (per 1,000 persons)
WWI


Wandsworth_MetB
Deaths (per 1,000 persons)
WWI


Deptford
Deaths (per 1,000 persons)
WWI
40
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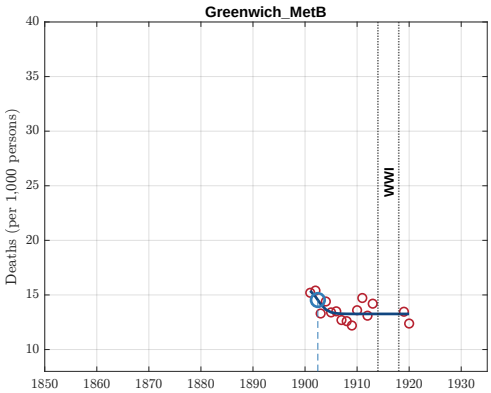
1850
1860
1870
1880
1890
1900
1910
1920
1930


Greenwich_MetB
Deaths (per 1,000 persons)
WWI
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1850
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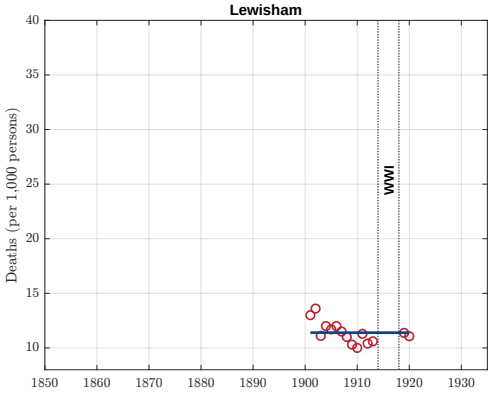
Lewisham
Deaths (per 1,000 persons)
WWI
40
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1850
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1890
1900
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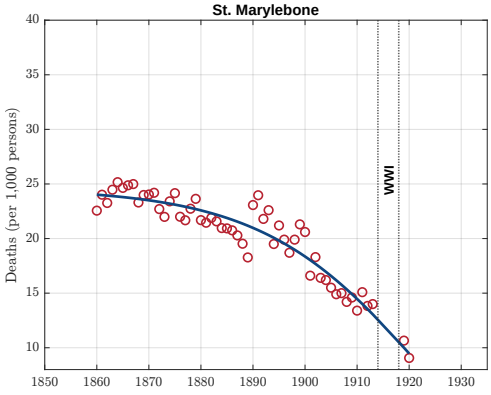
1920
1930


St. Marylebone
Deaths (per 1,000 persons)
WWI
40
35
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1850
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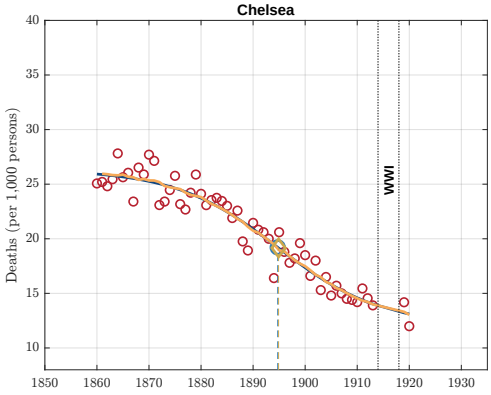
1900
1910
1920
1930


Chelsea
Deaths (per 1,000 persons)
WWI
40
35
30
25
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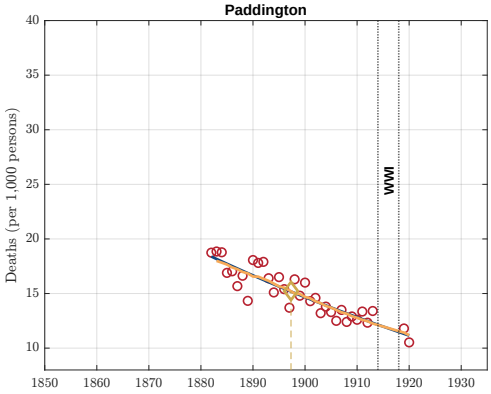
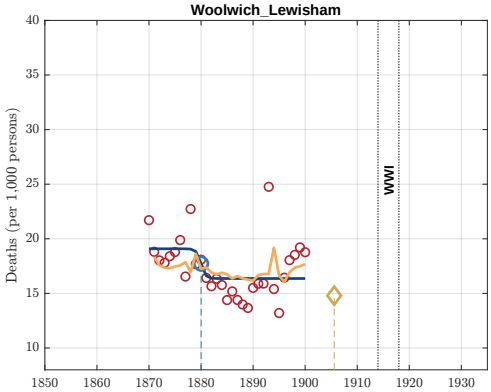
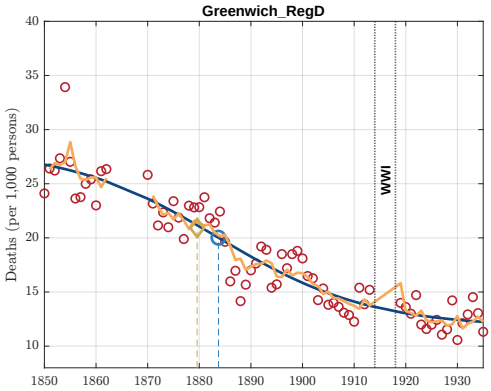
15
10
1850
1860
1870
1880
1890
1900
1910
1920
1930


Paddington
Deaths (per 1,000 persons)
WWI
40
35
30
25
20
15
10
1850
1860
1870
1880
1890
1900
1910
1920
1930


Woolwich_Lewisham
Deaths (per 1,000 persons)
WWI
40
35
30
25
20
15
10
1850
1860
1870
1880
1890
1900
1910
1920
1930


Greenwich_RegD
Deaths (per 1,000 persons)
WWI
40
35
30
25
20
15
10
1850
1860
1870
1880
1890
1900
1910
1920
1930

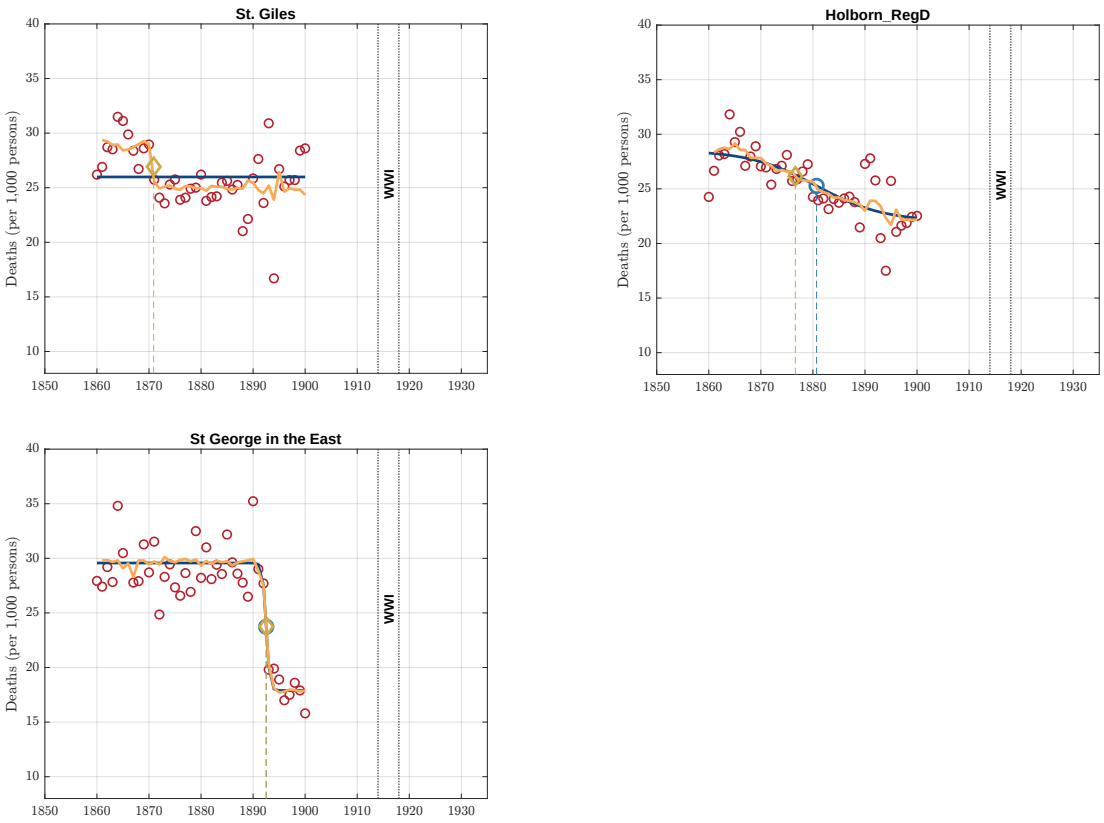
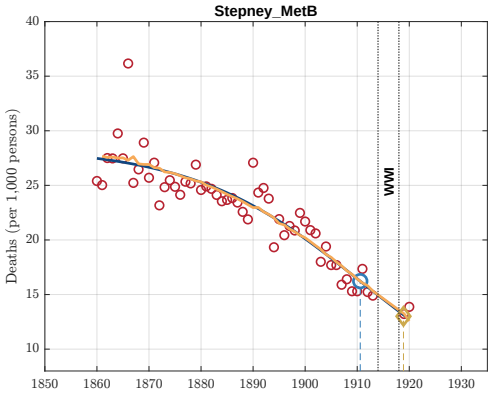


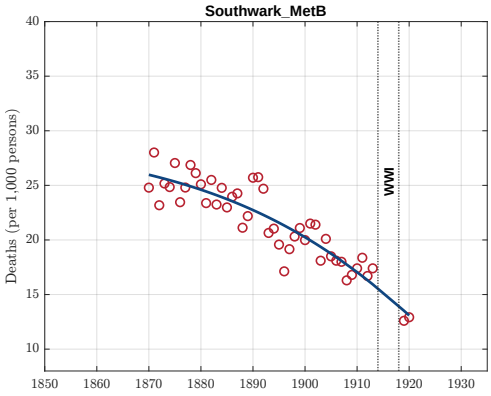
Figure 2.8.2: **Crude mortality per 1,000 persons, areas in London 1850–1935.**

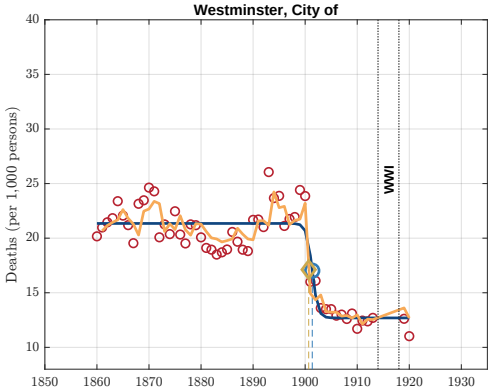
2.8.3.2 Dynamics of Corrected Death Rates

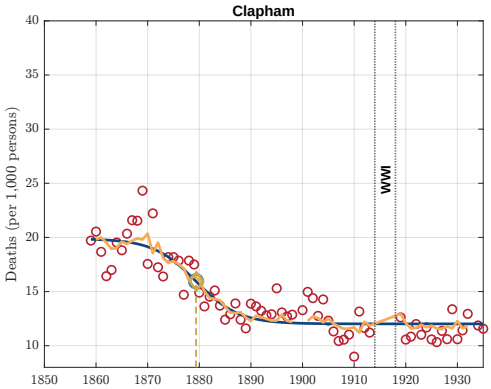
Figure 2.8.9 plots the crude mortality and corrected death rates for all available areas in London. The data exhibit highly similar trends and strong correlations across all areas.[51] While corrected death rates are available for 48 areas, many, such as Battersea, Bermondsey, and Rotherhithe, do not exhibit consistent trends over a relatively long time series (see Figure 2.8.9).

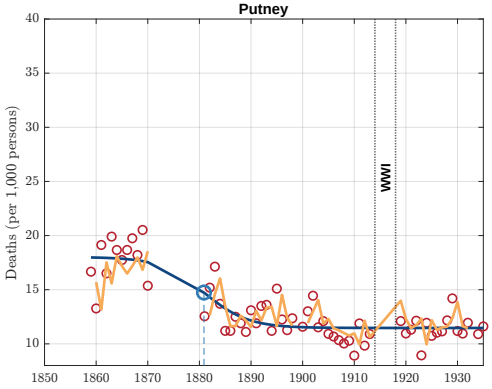
51. Two additional areas, Stoke Newington and Hackney, have mortality data available, but due to the lack of socioeconomic data, their trends are not shown.

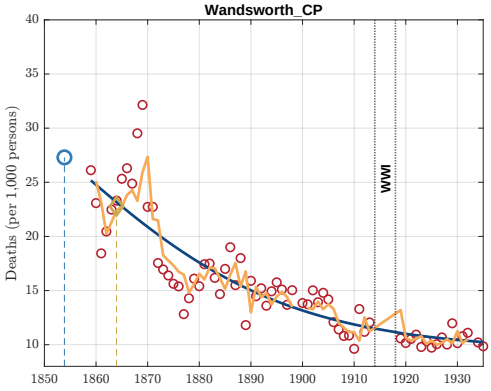

Stepney_MetB
Deaths (per 1,000 persons)
WWI

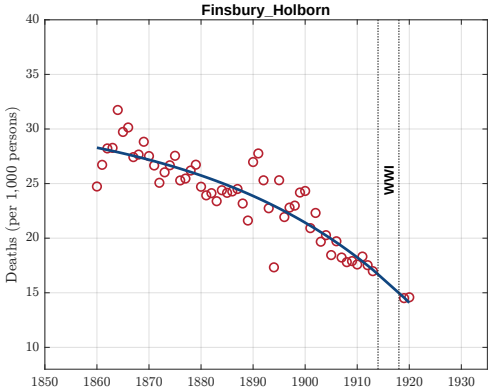

Southwark_MetB
Deaths (per 1,000 persons)
WWI

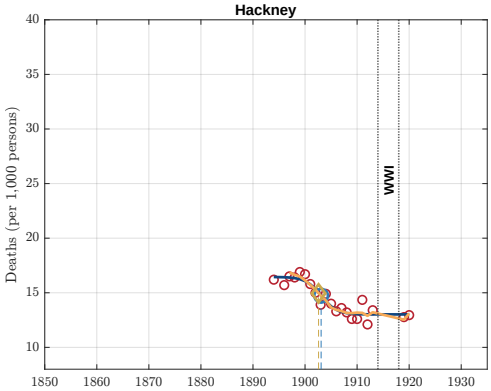

Westminster, City of
Deaths (per 1,000 persons)
WWI


Clapham
Deaths (per 1,000 persons)
WWI


Putney
Deaths (per 1,000 persons)
WWI


Wandsworth_CP
Deaths (per 1,000 persons)
WWI


Finsbury_Holborn
Deaths (per 1,000 persons)
WWI


Hackney
Deaths (per 1,000 persons)
WWI

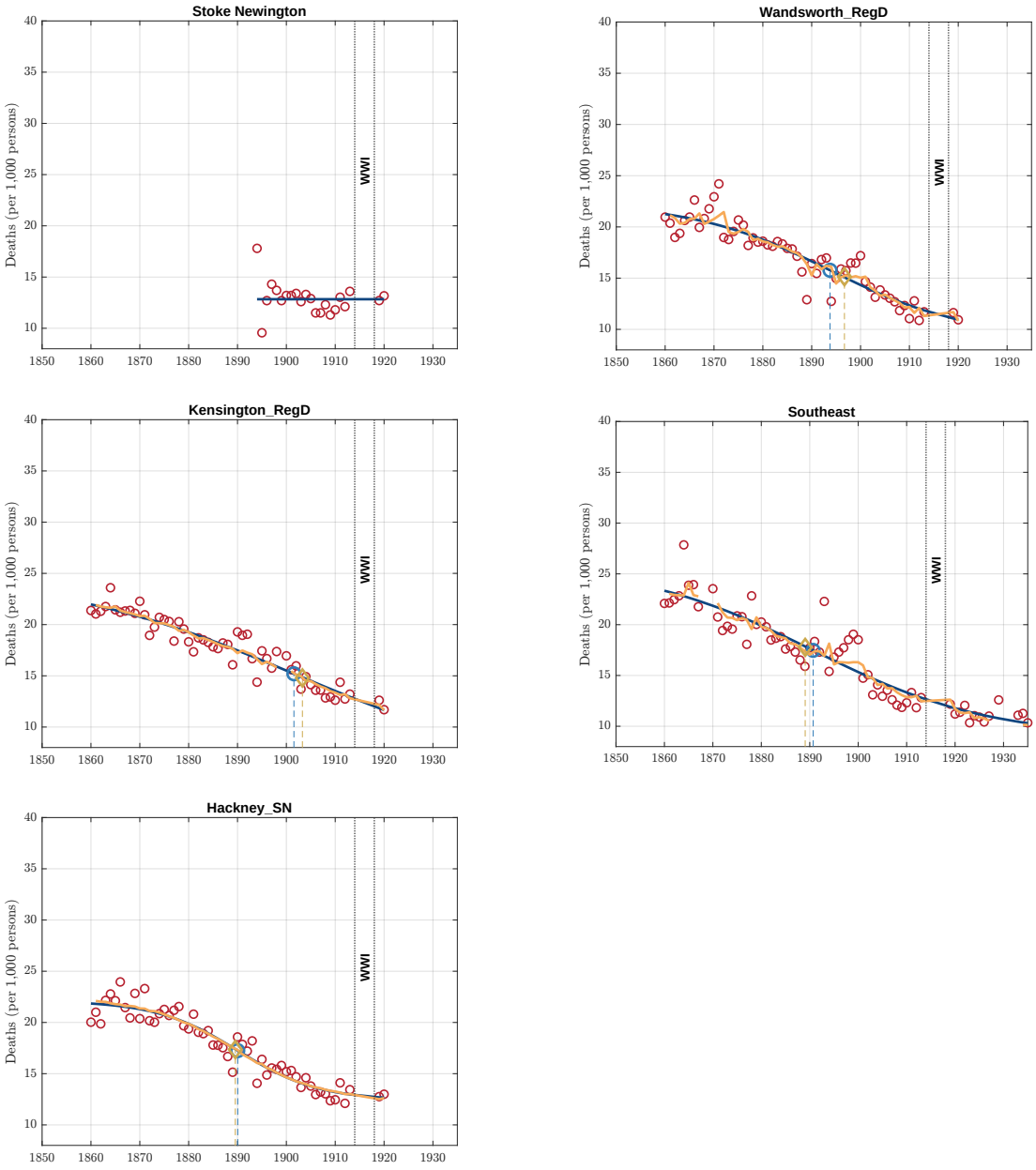
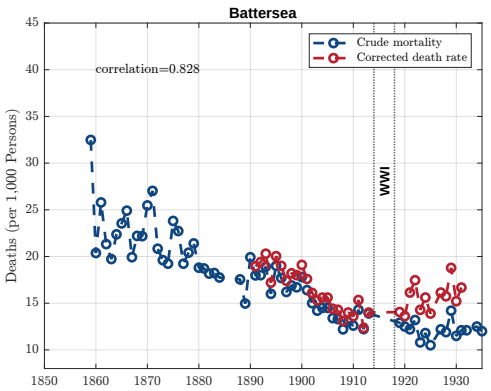
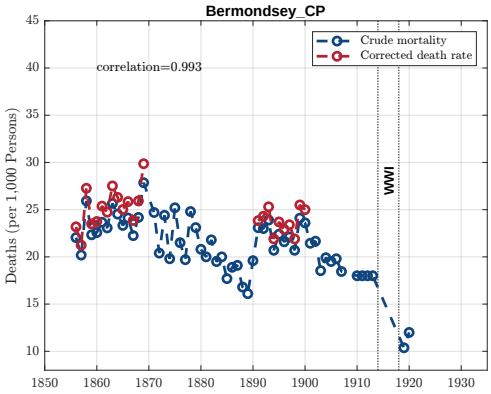
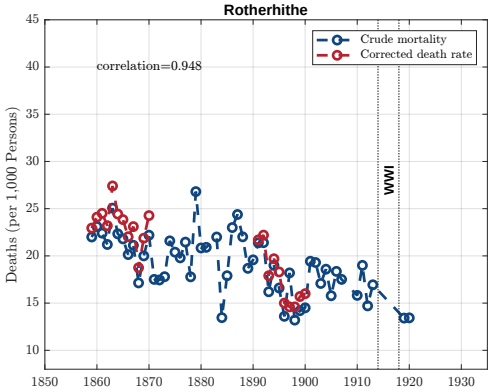
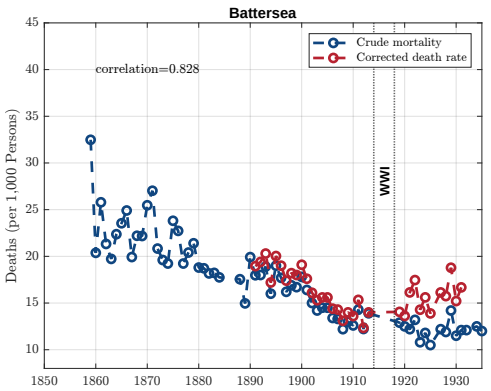


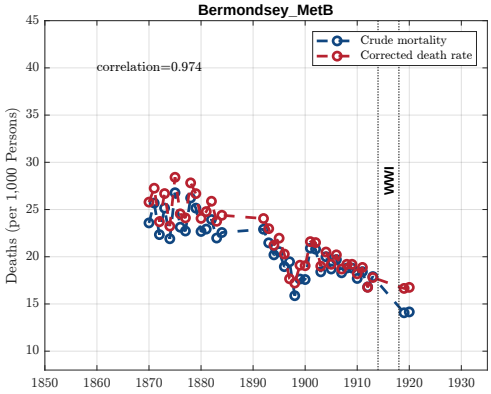
Figure 2.8.3: **Crude mortality per 1,000 persons, areas in London which are not included in the panel regressions, 1850–1935.**

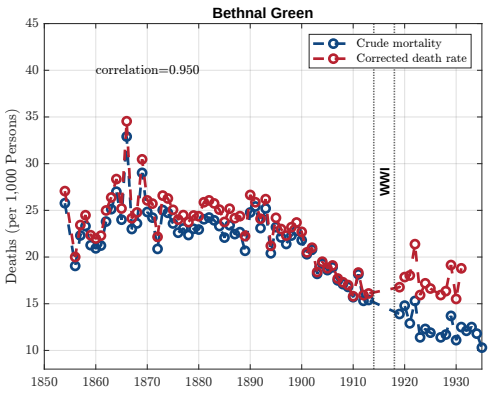

Battersea
correlation=0.828
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

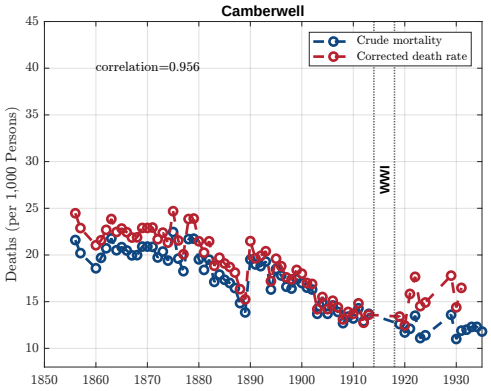

Bermondsey_CP
correlation=0.993
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

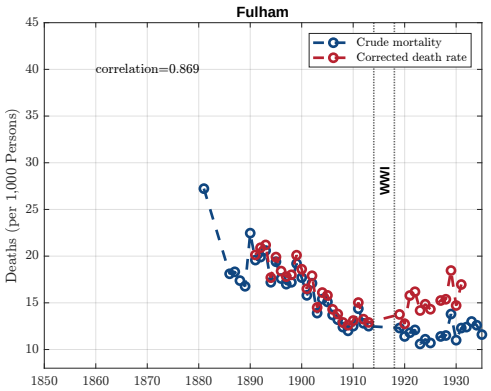

Rotherhithe
correlation=0.948
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

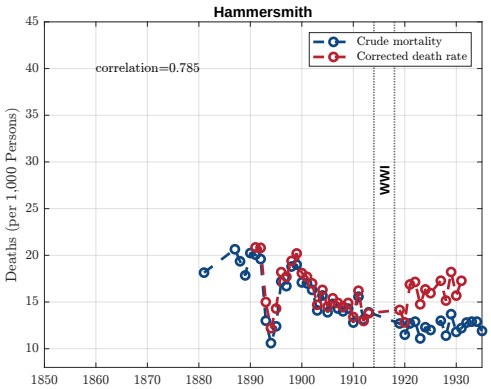

Battersea
correlation=0.828
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

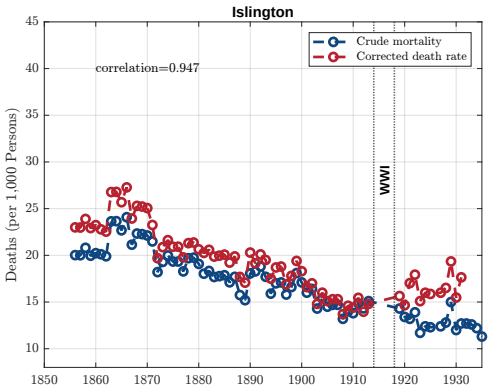

Bermondsey_MetB
correlation=0.974
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

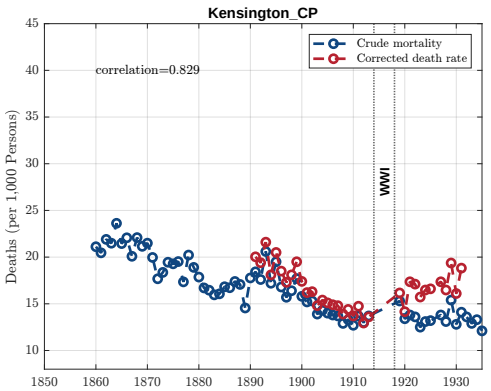

Bethnal Green
correlation=0.950
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

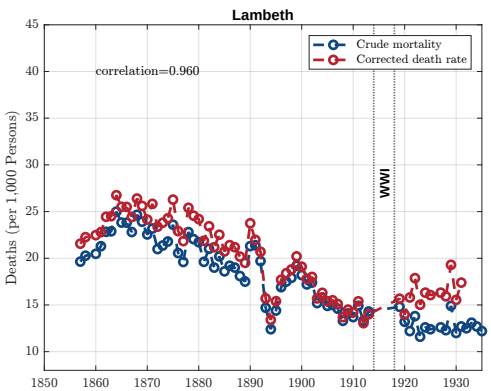

Camberwell
correlation=0.956
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

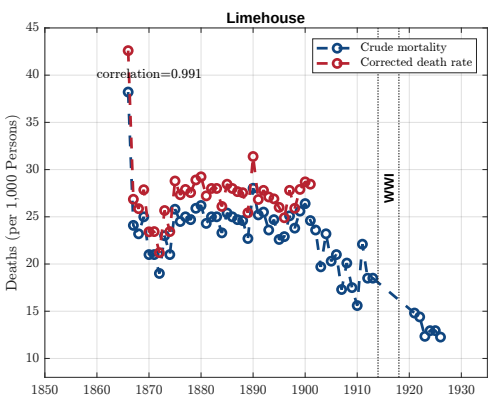

Fulham
correlation=0.869
Crude mortality
Corrected death rate
WWI
Deaths (per 1,000 Persons)

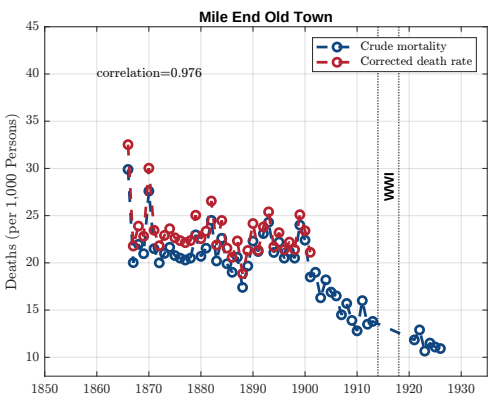

Hammersmith
Crude mortality
Corrected death rate
correlation=0.785
WWI
Deaths (per 1,000 Persons)

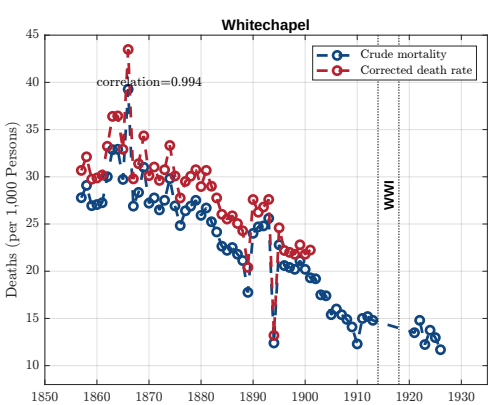

Islington
Crude mortality
Corrected death rate
correlation=0.947
WWI
Deaths (per 1,000 Persons)

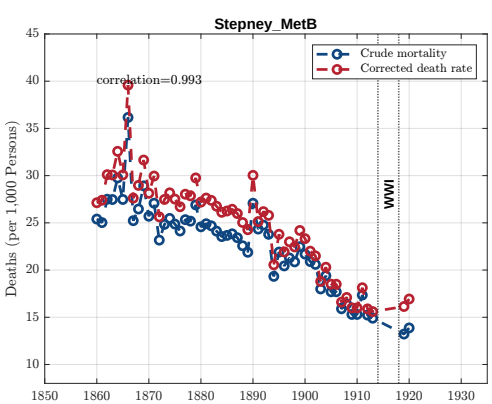

Kensington_CP
Crude mortality
Corrected death rate
correlation=0.829
WWI
Deaths (per 1,000 Persons)

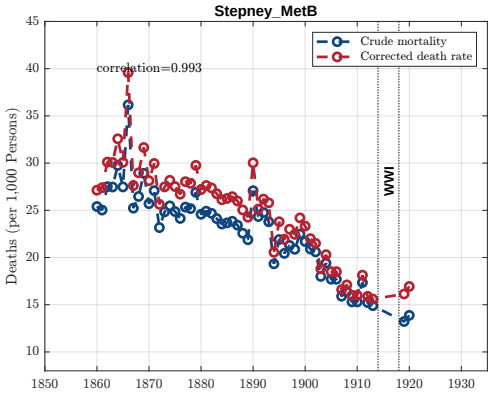

Lambeth
Crude mortality
Corrected death rate
correlation=0.960
WWI
Deaths (per 1,000 Persons)

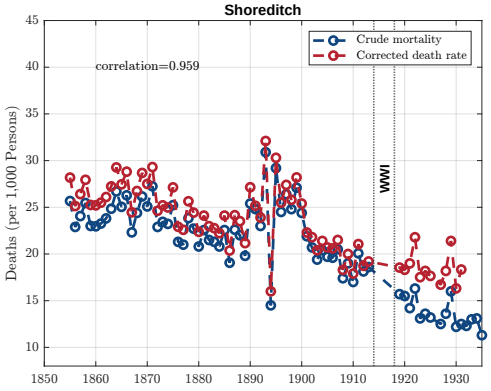

Limehouse
Crude mortality
Corrected death rate
correlation=0.991
WWI
Deaths (per 1,000 Persons)

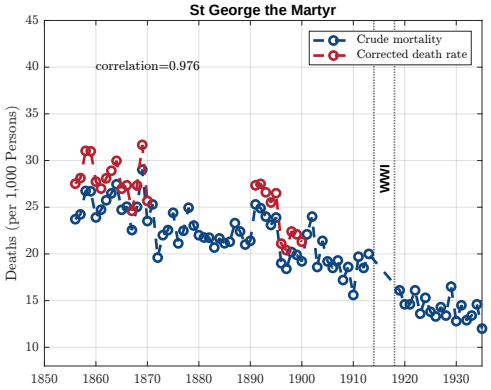

Mile End Old Town
Crude mortality
Corrected death rate
correlation=0.976
WWI
Deaths (per 1,000 Persons)

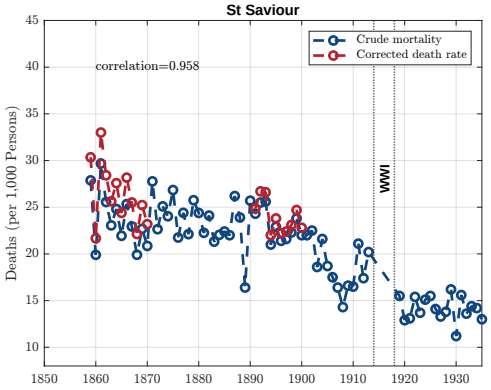

Whitechapel
Crude mortality
Corrected death rate
correlation=0.994
WWI
Deaths (per 1,000 Persons)

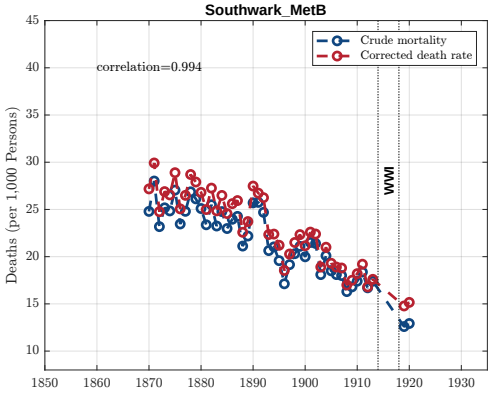

Stepney_MetB
Crude mortality
Corrected death rate
correlation=0.993
WWI
Deaths (per 1,000 Persons)

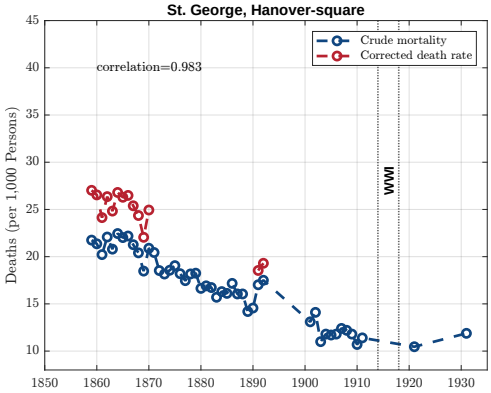

Stepney_MetB
Crude mortality
Corrected death rate
correlation=0.993
WWI
Deaths (per 1,000 Persons)

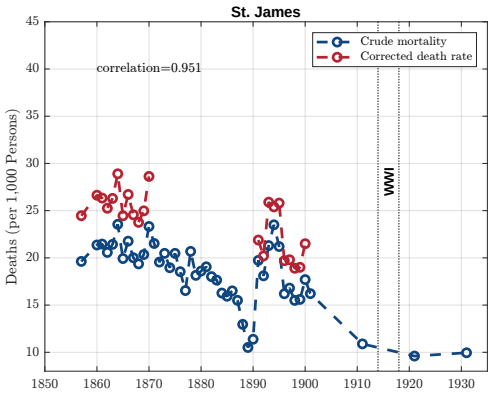

Shoreditch
Crude mortality
Corrected death rate
correlation=0.959
WWI
Deaths (per 1,000 Persons)

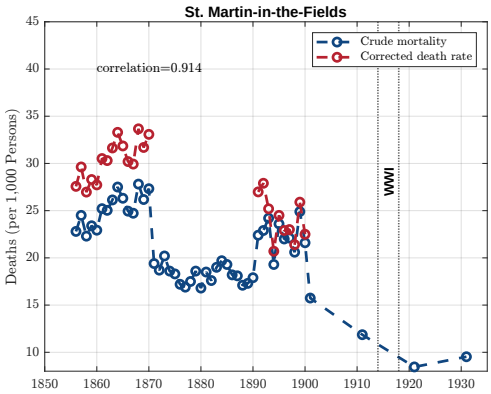

St George the Martyr
Crude mortality
Corrected death rate
correlation=0.976
WWI
Deaths (per 1,000 Persons)

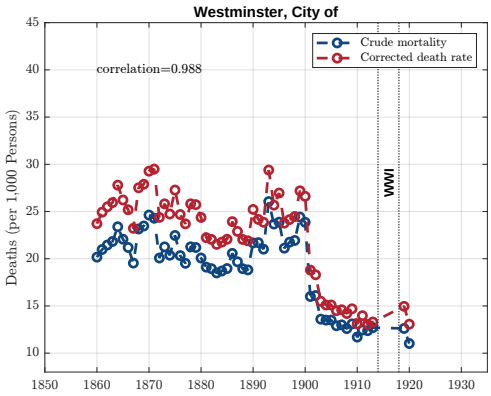

St Saviour
Crude mortality
Corrected death rate
correlation=0.958
WWI
Deaths (per 1,000 Persons)

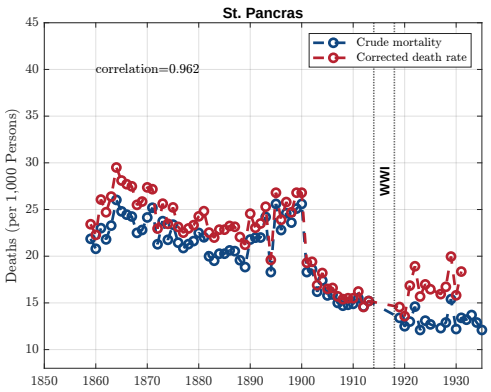

Southwark_MetB
Crude mortality
Corrected death rate
correlation=0.994
WWI
Deaths (per 1,000 Persons)

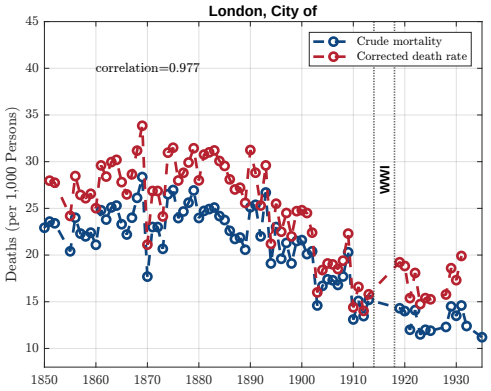

St. George, Hanover-square
Crude mortality
Corrected death rate
correlation=0.983
WWI
Deaths (per 1,000 Persons)

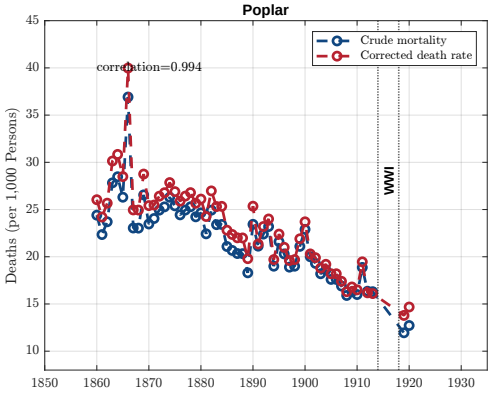

St. James
Crude mortality
Corrected death rate
correlation=0.951
WWI
Deaths (per 1,000 Persons)

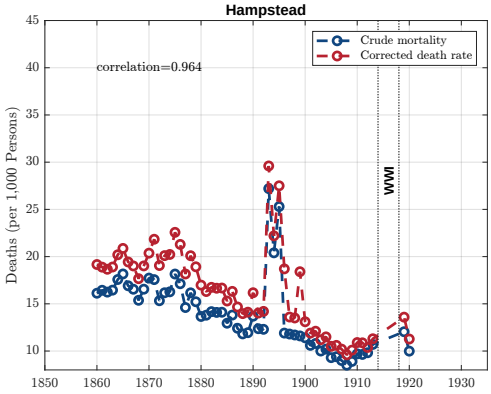

St. Martin-in-the-Fields
Crude mortality
Corrected death rate
correlation=0.914
WWI
Deaths (per 1,000 Persons)

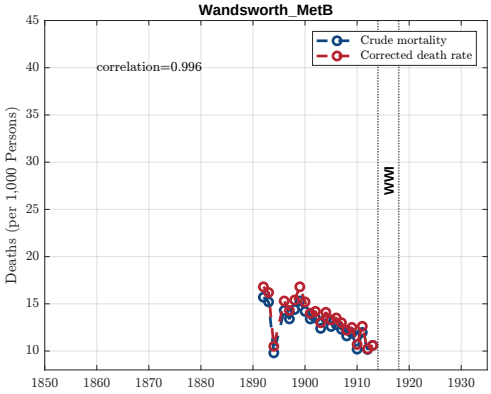

Westminster, City of
Crude mortality
Corrected death rate
correlation=0.988
WWI
Deaths (per 1,000 Persons)

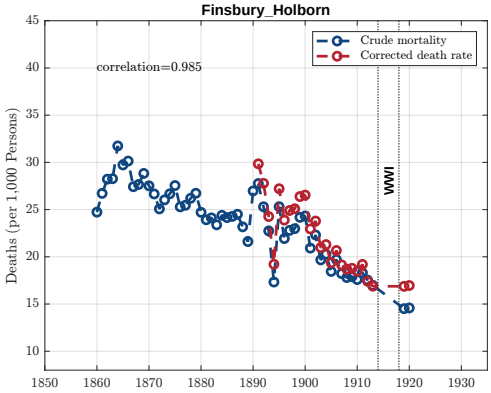

St. Pancras
Crude mortality
Corrected death rate
correlation=0.962
WWI
Deaths (per 1,000 Persons)

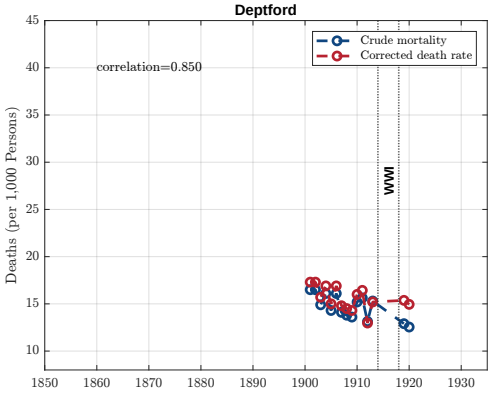

London, City of
Crude mortality
Corrected death rate
correlation=0.977
WWI
Deaths (per 1,000 Persons)

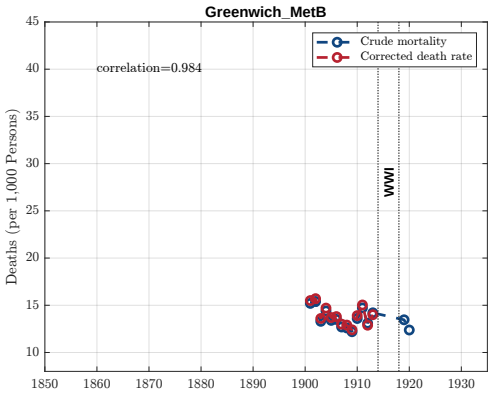

Poplar
Crude mortality
Corrected death rate
correlation=0.994
WWI
Deaths (per 1,000 Persons)

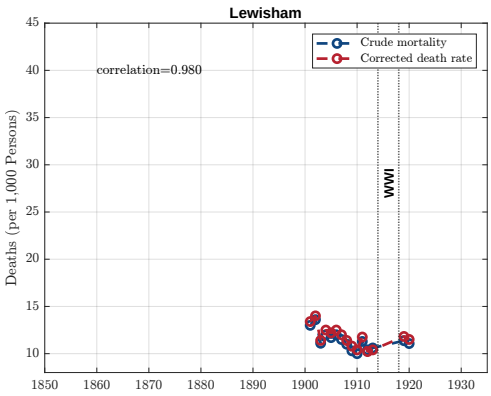

Hampstead
Crude mortality
Corrected death rate
correlation=0.964
WWI
Deaths (per 1,000 Persons)

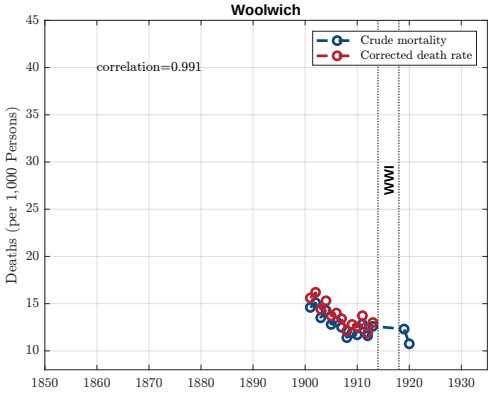

Wandsworth_MetB
Crude mortality
Corrected death rate
correlation=0.996
WWI
Deaths (per 1,000 Persons)

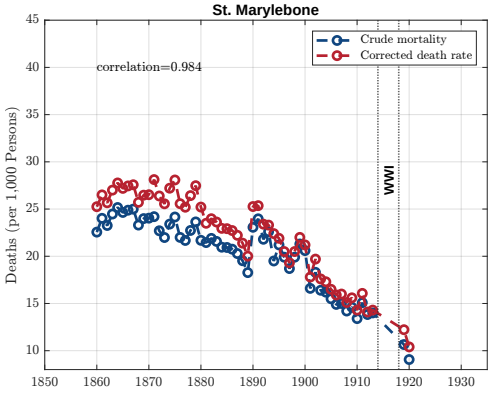

Finsbury_Holborn
Crude mortality
Corrected death rate
correlation=0.985
WWI
Deaths (per 1,000 Persons)

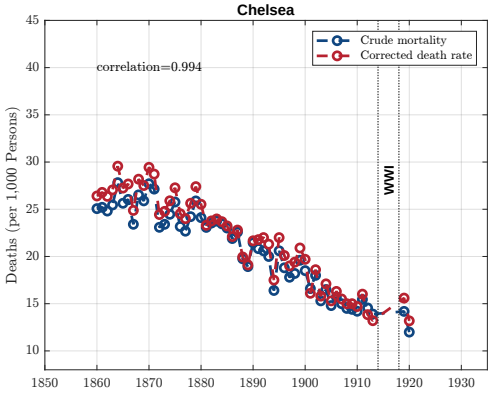

Deptford
Crude mortality
Corrected death rate
correlation=0.850
WWI
Deaths (per 1,000 Persons)

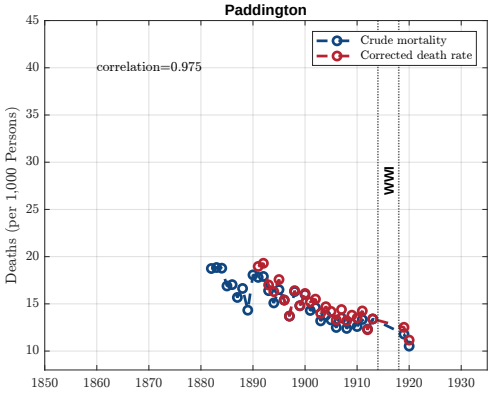

Greenwich_MetB
Crude mortality
Corrected death rate
correlation=0.984
WWI
Deaths (per 1,000 Persons)

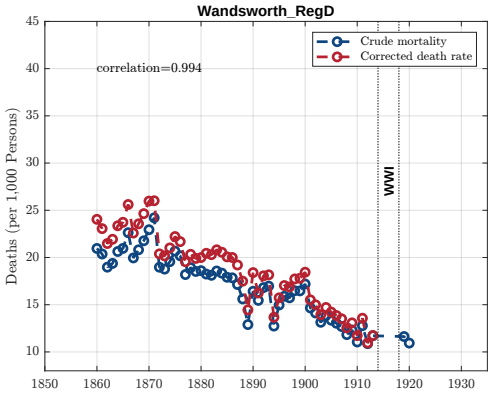

Lewisham
Crude mortality
Corrected death rate
correlation=0.980
WWI
Deaths (per 1,000 Persons)

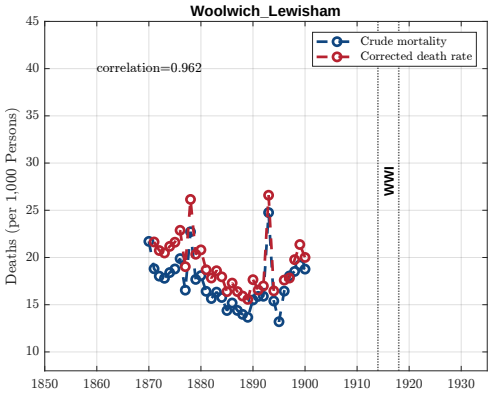

Woolwich
Crude mortality
Corrected death rate
correlation=0.991
WWI
Deaths (per 1,000 Persons)


St. Marylebone
Crude mortality
Corrected death rate
correlation=0.984
WWI
Deaths (per 1,000 Persons)


Chelsea
Crude mortality
Corrected death rate
correlation=0.994
WWI
Deaths (per 1,000 Persons)


Paddington
Crude mortality
Corrected death rate
correlation=0.975
WWI
Deaths (per 1,000 Persons)


Wandsworth_RegD
Crude mortality
Corrected death rate
correlation=0.994
WWI
Deaths (per 1,000 Persons)


Woolwich_Lewisham
Crude mortality
Corrected death rate
correlation=0.962
WWI
Deaths (per 1,000 Persons)

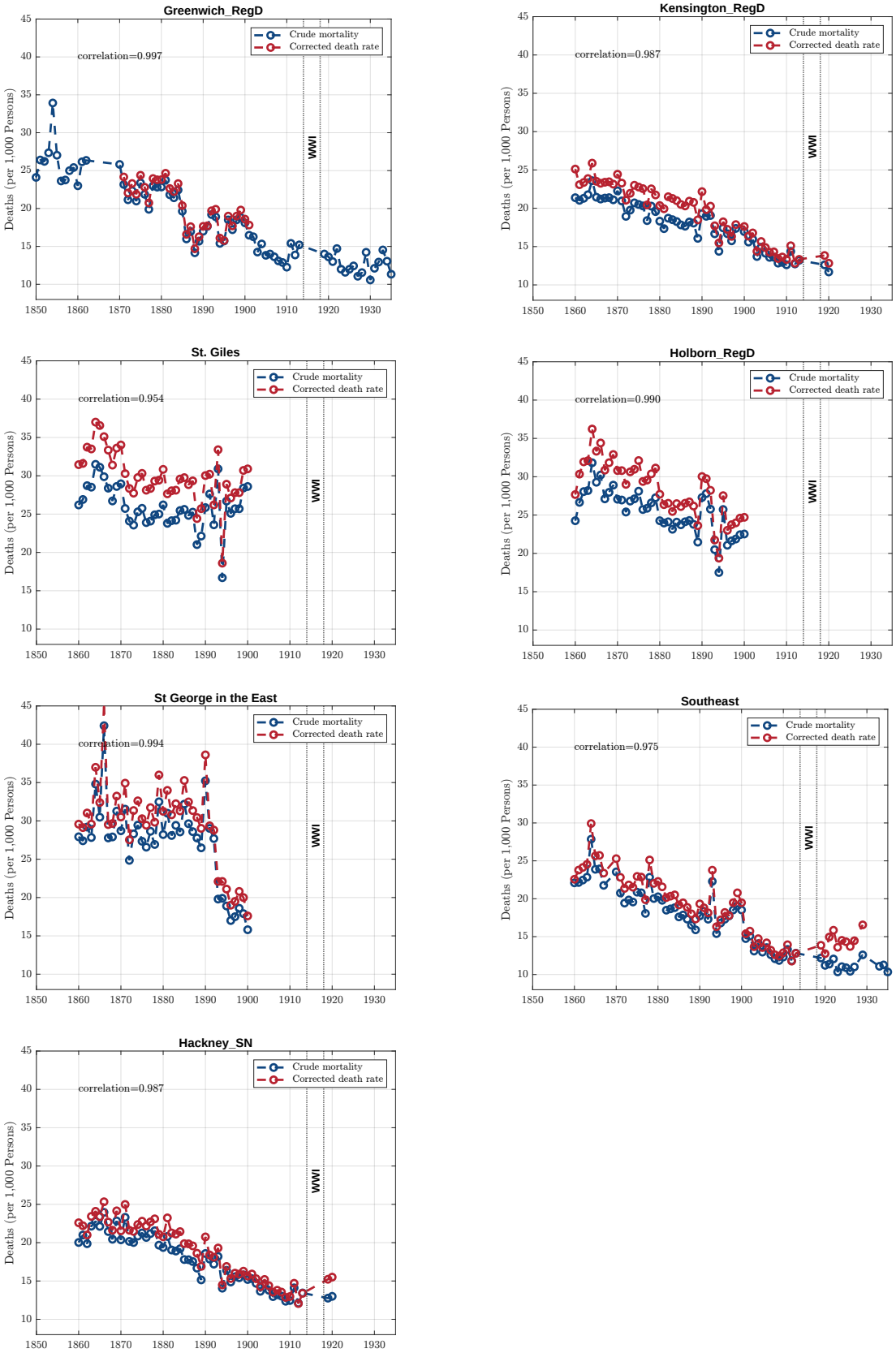


Figure 2.8.9: **Comparative trends of crude mortality and corrected death rates, areas in London.**

2.8.4 Additional Results for Mortality Inequality

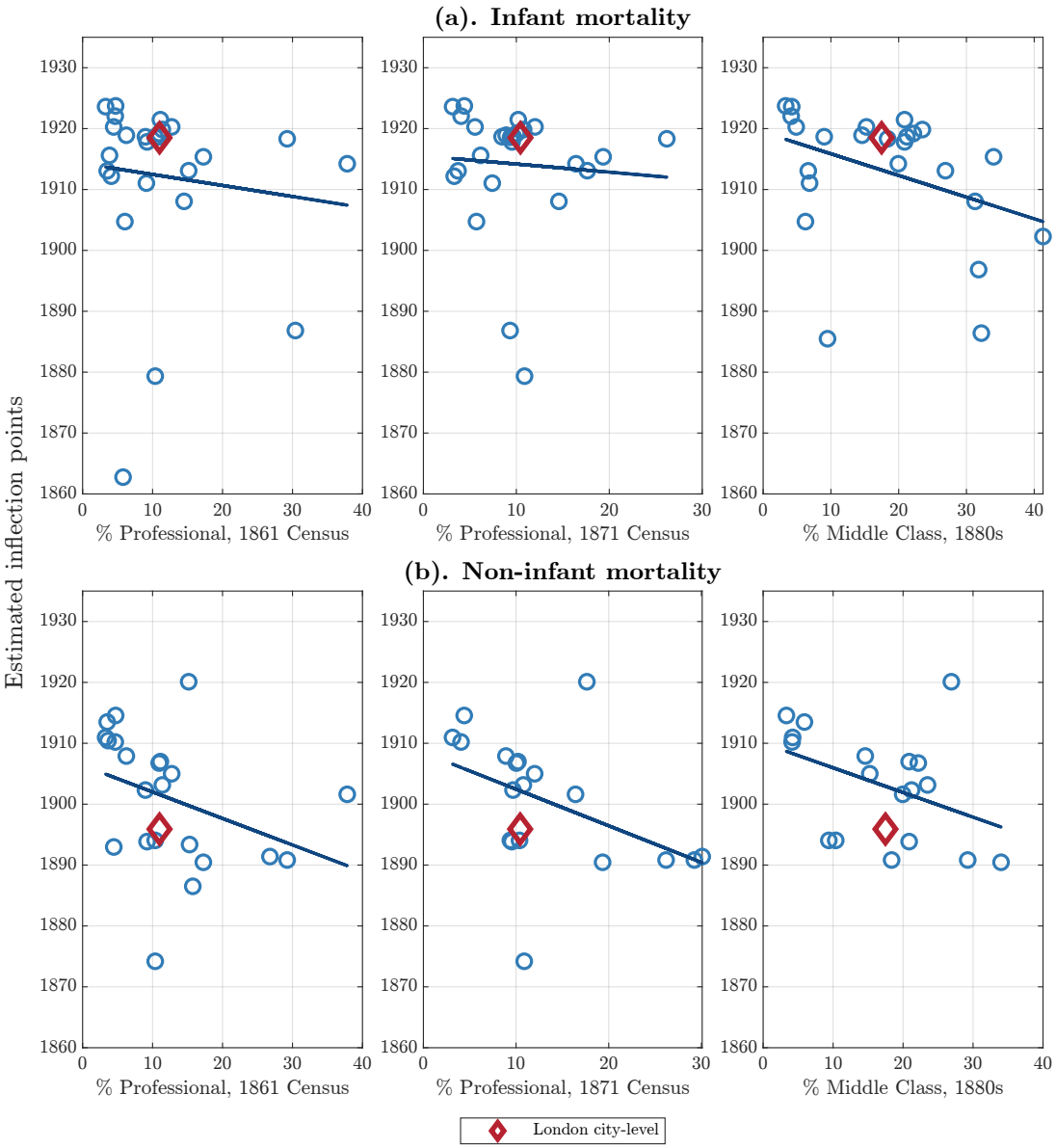


Figure 2.8.10: **Socioeconomic conditions vs. estimated inflection points of infant and non-infant mortality dynamics.** This figure plots the negative relationship between the socioeconomic conditions and the estimated inflection points estimated from the infant and non-infant mortality dynamics for each small area. Areas for which the corresponding estimated inflection points fall out of the sample (either before 1850 or after 1935) are considered invalid and are excluded. The red diamonds mark the London city-level using dataset introduced in Section 1.2.3 in Chapter 1.[33]

Chapter 3

Education- and Income-Based Inequalities in Physical Activity

Abstract

Physical activity is a fundamental contributor to overall health. Insufficient physical activity represents a significant health risk, as it substantially increases the likelihood of developing non-communicable diseases, such as cardiovascular diseases, colon and breast cancer, diabetes, and depression. This chapter investigates educational and income inequalities in physical activity among working-age adults in the UK.

Physical inactivity and its associated health detriments are not only widespread but also unequally distributed across socioeconomic groups. This study conducts a systematic analysis of inequalities in physical activity (PA) across the UK using Understand Society, a nationally representative panel dataset which provides high-quality measurements and covers the full socioeconomic distribution.

I employ an ordered logit model to estimate the nonlinear relationships between two key socioeconomic indicators—education and income—and three levels of PA: zero physical activity, engagement in at least some physical activity, and meeting WHO-recommended guidelines. I emphasise the joint analysis of education in conjunction with household disposable income to disentangle purchasing power effects from broader social class measures. The results reveal that, first, while more educated and higher income individuals are more likely to engage in physical activities and meet recommended PA guidelines, the income effects are modest. Educational interventions may therefore serve as a more effective policy lever than financial incentives for promoting physical activity. Second, the effects of total household disposable income are double those of total household gross labour income on PA, underscoring the importance of choice of income measure. Third, inequalities in physical activities manifest differently by gender, with educational attainment showing stronger and more significant effects on women's physical activity levels than among men.

3.1 Introduction

The WHO and UK health guidelines for physical activity (PA) recommend that adults should engage in at least 150 minutes of moderate-intensity physical activity, 75 minutes of vigorous-intensity physical activity, or an equivalent combination per week, encompassing activities such as leisure, transportation, work, and household chores (WHO, 2010; NHS, 2024). However, a significant portion of the global population falls short of meeting these recommendations—insufficient physical activity is prevalent worldwide (Lee et al., 2012). In fact, 31.1% of adults globally were considered physically inactive in 2011 (this figure became over a quarter by 2016 (Guthold et al., 2018)), with rates ranging from 17.0% in Southeast Asia to approximately 43.0% in the Americas and Eastern Mediterranean regions (Hallal et al., 2012). In the UK, the overall levels of physical activity among adults are notably low even compared to their counterparts in similar European nations (Sawyer et al., 2017). Recent data indicates that physical inactivity affects 34% of men and 42% of women in the UK (Office for Health Improvement and Disparities, 2022).

Lack of physical activity signals a major risk, as inadequate levels of PA significantly elevate the risk of various non-communicable diseases, particularly cardiovascular diseases, colon and breast cancer, diabetes, and depression (Lee et al., 2012; Bauman et al., 2012; Webster, 2015). Mokdad et al. (2004, 2005) estimated that poor diet and physical inactivity had become the second leading preventable causes of death, accounting for 15% of all mortality in the US in 2000. Analysis of 3,038 diabetic participants from the Health Survey for England and the Scottish Health Surveys revealed that those engaging in some physical activity, even below the recommended threshold, had a 26% lower risk of all-cause mortality compared to inactive individuals. Those who achieved the recommended levels of activity had a 35% lower risk of all-cause mortality (Sadarangani et al., 2014). On the contrary, a daily 30-minute walk, five times a week, could potentially extend one's lifespan by approximately 1.5 years, while more vigorous exercise might amplify that benefit (Franco et al., 2005).

The uneven distribution of health-related behaviours across socio-demographic groups is known to significantly contribute to health disparities (Wagstaff, 1986; Balia and Jones, 2008; Pampel et al., 2010; Stringhini et al., 2010; Lynch et al., 1997). Notably, physical activity levels demonstrate substantial differences across population subgroups. The general consensus is that females, elders, and those with lower socioeconomic status are less physically active, although exceptions exist in certain contexts (see e.g., Troiano et al., 2008; Meltzer and Jena, 2010; Pontin et al., 2021; Cawley and Ruhm, 2011, p. 103).[1] Among these socio-demographic factors, our analysis centres on how education and income shape physical activity patterns.

In particular, I examine how education and income influence the probabilities of zero physical activity, engaging in at least some physical activity, and meeting WHO-recommended guidelines. These two socioeconomic factors warrant separate analysis, as they shape physical activity through distinct mechanisms. Education fundamentally captures psychological and cognitive factors, while also serving as a proxy for broader social class and family background characteristics. In contrast, household disposable income—which UnSoc allows us to distinguish from gross income—directly measures purchasing power. In addition, understanding their relative impacts is crucial for policy design, as interventions targeting education versus income require different approaches (Chevalier et al., 2013).

This study makes three key contributions. First, examine the nonlinear effects of both education and income on physical activity. Despite numerous studies exploring the inequalities in PA, few have thoroughly modelled the non-linearity in PA comprehensively across three levels: zero physical activity, engagement in at least some physical activity, and meeting WHO-recommended guidelines. To conduct an empirical analysis of education- and income-based inequalities in physical activity across the UK, I estimated ordered logit models of physical activity based on a categorical variable with three levels: physically inactive, partially active (below recommended guidelines), and sufficiently active (meets recommended guidelines). The implications of examining these three levels are discussed in Section 3.5.

1. See e.g., Richmond et al. (2006) for racial/ethnic disparities in physical activity.

The second contribution lies in distinguishing the effects of various income measures on physical activity, as each measure may operate through a different mechanism and yield varying effect sizes. Previous research has demonstrated such distinct mechanisms in the context of body weight—according to (Lakdawalla and Philipson, 2002; Lakdawalla et al., 2005), earned income affects weight through occupational pathways (e.g., more skilled, sedentary jobs), while unearned income influences weight through changing the demand for thinness. However, to my knowledge, prior studies have not systematically examined how different income measures relate to physical activity patterns. In addition, disposable household income, which accounts for tax and transfers, better reflects the actual resources available (Blundell and Etheridge, 2010; Belfield et al., 2017), so the comparison between gross and disposable income should provide insights for policy implications.

Third, while disentangling the effects of education and income, I examine continuous income measures rather than categorical income groups—a departure from most empirical studies which rely on discrete income brackets—providing more nuanced insights for policy design. In particular, estimating the marginal effect of absolute income levels informs direct income support policies that aim to promote physical activity, enabling policymakers to evaluate the relative cost-effectiveness of income-based interventions compared to educational initiatives targeting similar health outcomes.

The empirical analysis is based on the *Understanding Society* (UnSoc) dataset. The UnSoc survey is a comprehensive and nationally representative sample of UK adults aged 16 and over, encompassing approximately 40,000 households in Wave 1 (Buck and McFall, 2012). The analysis presented here focuses on working-age adults between 25 and 60 years old, as this demographic has typically completed their education (Cutler and Lleras-Muney, 2010) and primarily relies on labour market earnings as their main source of income. The large sample size enables detailed analysis across various population subgroups (e.g., gender) (Buck and McFall, 2012). More critically, as a nationally representative dataset, UnSoc captures the complete spectrum of income distribution, allowing us to examine how physical activity patterns are linked to the macro-level income inequality in the UK, which would be methodologically impossible with small samples (see e.g., HM Treasury, 2020; Ray-Chaudhuri et al., 2023; Booker et al., 2017, for inequality research using the

UnSoc). Additionally, Understanding Society provides high-quality income measures that include detailed components of income sources. The derived income distributions and trends closely match those of the Households Below Average Income series (HBAI)—the official UK data source widely considered the gold standard for cross-sectional income data (Fisher and Hussein, 2023).

The rest of the chapter is structured as follows: Section 3.2 establishes the foundation by defining and discussing how physical activity is measured. To motivate this study, it then examines existing research on how socioeconomic factors influence health behaviours broadly, before focusing specifically on the relationship between education, income, and physical activity. The empirical analysis begins with Sections 3.3.1 and 3.3.2, which describe the dataset and define the key variables used in the analysis; Section 3.3.4 presents the ordered logit model developed; Section 3.4 displays the estimated results, analyses the findings, and discusses their policy implications. Finally, Section 3.5 synthesises the key results and their significance.

3.2 Background and Related Literature

3.2.1 Background

This section establishes the conceptual foundation by situating physical activity within health behaviours broadly, then reviews its various definitions and measurement approaches in the literature.

Health behaviours are actions or habits aimed at maintaining, restoring, and improving health and quality of life (Conner and Norman, 2017). These can involve either engaging in health-enhancing behaviours (e.g., exercise) or avoiding health-compromising behaviours (e.g., smoking) (Conner and Sparks, 2005, p. 18). In a broad sense, health behaviours encompass a wide range of activities, including but not limited to smoking, diet/exercise, alcohol use, illegal drug use, sexual behaviours, automobile safety, physician visits, medication adherence, screening, vaccination, household safety, preventive care, and management of chronic diseases such as diabetes or hypertension (Cutler and Lleras-Muney, 2010; Conner and Norman, 2017).

Physical activity (PA) is defined as "any bodily movement produced by skeletal muscles that requires energy expenditure" (WHO, 2010). This broad definition includes a diverse range of activities, from conventional sports to everyday tasks like walking, gardening, and household chores (Pontin et al., 2021).[2] Current research and standardised assessment tools like the International Physical Activity Questionnaire and the Global Physical Activity Questionnaire classify PA into three primary domains based on purpose: a) for work (paid or unpaid); b) for transport to get to and from places; c) during leisure time i.e., sports and active recreation (Scholes and Bann, 2018; Guthold et al., 2018).[3] This categorisation provides a comprehensive framework for understanding the various inequalities in PA. Van Lenthe et al. (2005) distinguished the time allocation for various PA among individuals residing in 78 neighbourhoods in Eindhoven, the Netherlands—the lowest quartile was more likely to engage in walking or cycling for utilitarian purposes, such as commuting to shops or work, but participated less in leisure-time walking, cycling, gardening, and sports activities. This probably explains why high-income Western countries recorded the highest levels of insufficient physical activity in 2016, as these regions tend to have more sedentary jobs and greater reliance on personal motorised transportation (Guthold et al., 2018). For example, Scandinavian studies elucidated differences in the direction of inequalities across various domains of PA—higher social classes were more active during leisure time, whereas lower socioeconomic groups were more engaged in PA through their occupations (Beenackers et al., 2012).[4] This chapter primarily examines leisure-time physical activity (LTPA)—a crucial and predominant contributor to total PA (Bensley et al., 2011), especially in "post-industrial" countries (Brownson et al., 2005).

Research on physical activity often begins with the challenge of accurately quantifying PA levels. Three prominent methods have been used to gather PA information:

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2. Plonczynski (2003) distinguished the term *physical activity* to broadly indicate any activity that moves the body and increases energy expenditure, and used *exercise* to specify PA that is "planned, structured, and repetitious for the intention of increasing some parameter of physical fitness".
3. Cawley (2004) proposed the SLOTH (sleep, leisure-time, occupation, transportation, and home-based activities) model to capture domains of PA.
4. See Drygas et al. (2009); Widyastari et al. (2022) for similar research in Poland and Thailand.

- Motion sensors, such as accelerometers, heart rate monitors (HRM), combined heart rate and accelerometry devices and pedometers (see e.g., Gorman et al., 2014; Doherty et al., 2017; Skender et al., 2016). While these tools can provide primary data, their high cost often restricts studies to small sample sizes and short durations, typically less than seven days (McCormack and Shiell, 2011). Additionally, individuals from higher socioeconomic strata are more likely to participate in such studies (Waters et al., 2011).
- Smartphone-based PA apps, like Garmin, Fitbit, and Apple watches. These have been questioned for potential selection bias and gaps in activity recording (Lin et al., 2018; Hicks et al., 2019).[5]
- Self-reported/questionnaire-assessed PA. This traditional method has been criticised for, for example, memory recall issues and social desirability bias, which can lead to the overestimation of PA levels (van Hees, 2012; Sylvia et al., 2014; Janevic et al., 2012). Nonetheless, incorporated into large-scale surveys, this method can effectively capture a representative sample.[6]

I employ data obtained through the last method, as I utilise survey data and concentrate on LTPA.

As Beenackers et al. (2012) discussed in their review, studies examining inequalities in PA typically measure socioeconomic positions, referred to here as socioeconomic status (SES), using indicators including (net or gross) individual/household income, education, occupation-based social class, as well as other measures such as neighbourhood mean/median income and home ownership status.

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5. See Pontin et al. (2021) for detailed reviews and another potential secondary data source from a commercial app.
6. Some studies combined/compared the questionnaires and motion sensors for the sake of more complementary and comprehensive data (see e.g., Prince et al., 2008; Skender et al., 2016; Sabia et al., 2014). These studies suggest that each method offers valuable insights into different perspectives of PA.

3.2.2 Mechanisms Underlying Inequalities in PA

To contextualise the investigation of education- and income-related inequalities in physical activity, I first review the fundamental mechanisms that produce disparities in physical activity behaviour.

Through the lens of ecological models, inequalities in PA stem from two domains of factors.[7] The first domain, individual factors, encompasses interpersonal variables—variables within individuals such as biological, psychological, and cognitive factors (Bauman et al., 2002).[8] For example, high-SES individuals are more likely to prioritise long-term gains in longevity over short-term pleasures of unhealthy behaviour (Pampel et al., 2010), and higher expected future income serves as a disincentive for more educated individuals to engage in unhealthy behaviour that could shorten their working lives (Cowell, 2006). Conversely, the deprived facing chronic stressors such as financial instability, unemployment, and feelings of powerlessness may turn to overeating and inactivity as coping mechanisms to regulate their mood (McNeill et al., 2006; Lantz et al., 2005). However, low-SES individuals are not always disadvantaged on the PA distribution. Higher-income individuals are more prone to time poverty, which is associated with physical inactivity (Kalenkoski and Hamrick, 2013). Indeed, in Canada, Spinney and Millward (2010) has shown that time poverty even surpasses income poverty as a barrier of PA. Although more educated people have better access to health and exercise information and tend to adopt new knowledge more quickly (Glied and Lleras-Muney, 2008), the awareness about the importance of adequate exercise has been widespread (Pampel et al., 2010). Additionally, schooling's effects on health behaviour remain, even controlling for knowledge (Kenkel, 1991). The second domain refers to environmental correlates within an ecological framework. In particular, lower-SES groups often face reduced access to PA-promoting infrastructure such as walking trails, safe roads, and public open spaces (Gordon-Larsen et al., 2006; Amuzu et al., 2009; Brownson et al., 2000; Evans et al., 2012). These neighbourhoods also suffer from unfavourable aesthetics and safety concerns, further discouraging physical activity (Kamphuis et al., 2009; Van Lenthe et al., 2005). The effects of environmental factors can

7. An ecological model integrates both individual and social environmental factors of individual behaviours (McLeroy et al., 1988).
8. See e.g., Cutler and Glaeser (2005) for the impact of genetics on health behaviours.

be long-lasting. For example, students attending a school with inadequate sports facilities are less likely to engage in PA during adulthood (Black et al., 2019). Collectively, these unequal distributions of environmental resources dilute PA opportunities for economically disadvantaged populations.

In contrast to work based on ecological models, the economic literature has extended the neoclassical perspective and developed the income-leisure trade-off models, in which "time" and "market goods" are resources to be allocated; leisure is the dual of work (Becker, 1965; Downward and Riordan, 2007). Research employing variations of this framework has explored diverse aspects of PA, including the influence of social capital (Downward and Riordan, 2007), individual preferences, and the factors determining PA frequency (among employed individuals) (Brown and Roberts, 2011) as well as participation decision in terms of duration (Humphreys and Ruseski, 2010), etc.

3.2.3 Effects of SES on Health Behaviours

I next synthesise existing evidence on how various socioeconomic indicators influence health behaviours broadly, providing an important context for understanding education- and income-related disparities in physical activity.

Differences in socioeconomic conditions among groups lead to variations in lifestyle, which in turn partly account for health disparities (Wagstaff, 1986; Balia and Jones, 2008; Pampel et al., 2010; Stringhini et al., 2010). Since the late 1980s, the concept of a "health lifestyle" has been promoted as a key framework for understanding health-related inequalities (Cockerham et al., 1988; Abel, 1991). Although the original Whitehall studies (Marmot et al., 1978, 2013) and subsequent research (see e.g., Lynch et al., 1997; Lantz et al., 1998) did not view behaviours as a pivotal driver of better health and longer life expectancy of individuals with higher socioeconomic status (SES), Contoyannis and Jones (2004); Balia and Jones (2008) demonstrated that accounting for endogeneity in behavioural choices—such as a higher probability of quitting smoking after a cancer diagnosis—

enhances the estimated impact of behaviours on health outcomes (Cawley and Ruhm, 2011). Therefore, more research on the patterns of health behaviours across socioeconomic divisions is needed to shape interventions aimed at reducing health inequalities (Mackenbach et al., 2003, p. 26).

Table 3.2.1: Effects of SES on health behaviours

	Income	**Education**	**Other SES indicators**
PA	(*See Table 3.2.2*)	Harper and Lynch (2007); Cutler and Lleras-Muney (2010); Adams (2009); Cleland et al. (2012); Droomers et al. (1998); Farrell and Shields (2002); Farrell et al. (2014)	Cleland et al. (2012); Farrahi et al. (2020); Farrell and Shields (2002); Amuzu et al. (2009); Sawyer et al. (2017); Chaudhury and Shelton (2010)
Other health behaviours	Apouey and Clark (2015); Cerdá et al. (2011); Birch et al. (2000); Pomerleau et al. (1997); James et al. (1997); Huisman et al. (2005)	Harper and Lynch (2007); Cutler and Lleras-Muney (2010); Adams (2009); Laaksonen et al. (2003); Fu et al. (2022); Hiscock et al. (2012); Probst et al. (2020); Schaap et al. (2009); Huisman et al. (2005)	Amuzu et al. (2009); Laaksonen et al. (2003); Amos et al. (2009); Hiscock et al. (2012); Probst et al. (2020); Duncan et al. (1999); Syed et al. (2013)

Extensive studies have since established the relationship between health behaviours and socioeconomic conditions using a variety of measures, datasets, and research methods (see Table 3.2.1). Several studies across different populations have identified residential location as a significant predictor of smoking habits, particularly in the UK and Netherlands (Amuzu et al., 2009). In England, factors such as housing tenure, car ownership, and lone parenting were also associated with higher smoking rates (Amos et al., 2011). From 1993 to 1999, smoking, infrequent vegetable consumption, and the use of saturated fat on bread were more commonly observed among Finnish adult men with lower SES, as defined by individual income, household income, education, and occupational class, in which the latter two dominated (Laaksonen et al., 2003). Access to healthcare services

has also shown socioeconomic patterns, with stronger social networks facilitating greater healthcare utilisation (Derose and Varda, 2009), while lack of transportation has been linked to significantly reduced preventive care visits, as demonstrated by a 51% decrease in the US (Syed et al., 2013).

Educational attainment consistently demonstrates a strong relationship with health behaviours across populations and contexts. For example, studies in the US show that better-educated people are more likely to participate in preventive and risk control behaviours, such as regular mammograms, Pap smears, colorectal cancer screenings, influenza vaccination, and wearing seatbelts, even when controlling for receipt of health insurance (Cutler and Lleras-Muney, 2010). Each additional year of education is linked to a 3% decrease in the likelihood of smoking in the UK (Cutler and Lleras-Muney, 2010).[9] Fu et al. (2022) employed the college enrollment expansion in China beginning in 1999, where enrollment rates in each province grew according to predetermined capacity, as an instrumental variable for schooling years, and found that each additional year of education reduces the probabilities of smoking and drinking by 2.3% and 1.2% respectively. For additional related research, see review papers Hiscock et al. (2012); Probst et al. (2020).[10]

Income—another key component of SES—independently shapes health behaviours through distinct patterns. In the US, individuals from households with a stable low income ($11,000–$20,000) over a 30-year period had 1.57 times higher odds of alcohol abstinence and 2.14 times higher odds of heavy drinking in adulthood (Cerdá et al., 2011). Using lottery winnings as a proxy for positive income shocks, Apouey and Clark (2015) discovered a positive relationship between increased income and smoking as well as social drinking. Schmeiser (2009) looked into the heterogeneous generosity of the federal Earned Income Tax Credit (EITC) program and found that the increase in real family income from 1990 to 2002 explains 23%–29% of sample women's increased obesity prevalence. In Québec, Canada, during 1992–1993, 38% of lower-income individuals were smokers compared to 31% in the higher income group (Birch et al., 2000). Among Ontario adults, those with lower house-

9. This estimate is based on data from the National Health Interview Survey (NHIS). The same analysis using the Health and Retirement Study establishes a decrease of 2%.
10. Also see Schaap et al. (2009); Huisman et al. (2005) for opposite effects of SES on tobacco use in some developing countries such as India, Thailand, and Malaysia where the high cost of smoking is a limiting factor.

hold income were also more likely to engage in unhealthy behaviours, including smoking and high-fat diet consumption, while alcohol intake presented an exception, showing a positive relationship with household income status (Pomerleau et al., 1997). Moreover, secondary school students entitled to free school meals (i.e., from low-income families) are more likely to be regular smokers (Amos et al., 2009, p. 37). The British sample illustrates that the low-income groups consumed more milk, meat and meat products, fats, sugar and preserves, potatoes, and cereals, but their intake of fresh vegetables, fruit, and high-fibre products was nearly 50% lower compared to higher-income groups (James et al., 1997).

While socioeconomic disparities are evident across various health behaviours, I now focus specifically on PA. Numerous studies have documented significant variations in self-reported PA across different socioeconomic strata (Brownson et al., 2001).[11] For example, partner's education, home ownership (Cleland et al., 2012), health insurance coverage, family background (Cutler and Lleras-Muney, 2010), and employment status (Droomers et al., 1998), showed positive correlations with the likelihood of engaging in sufficient PA. Unemployed elderly individuals in England showed markedly lower engagement in specific activities such as walking and swimming compared to the employed, which might be attributed to the fact that employment provides income for gym memberships, access to swimming pools, and sports facilities among those over 60 (Chaudhury and Shelton, 2010). Among 23 British towns, the number of women aged 60–79 years exercising for less than two hours per day exhibited a clear socioeconomic gradient, with progressively higher numbers observed from the most affluent to the most disadvantaged quintiles of neighbourhood deprivation (Amuzu et al., 2009). Similarly, 5,923 adult participants living in economically disadvantaged communities in Glasgow exhibited physical inactivity (i.e., $<$ 30 minutes/week of moderate physical activity) and inadequate walking levels (Sawyer et al., 2017). Conversely, population density and the number of housing units in row houses, which measures residential density, were associated with less time of light/moderate-to-vigorous PA (Farrahi et al., 2020). In summary, most studies reveal lower levels of PA among groups with lower SES.

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11. The articles reviewed below are all about leisure-time PA unless specified.

3.2.4 Effects of Education and Income on PA

Having examined broader socioeconomic influences on health behaviours, I now turn to the central focus of this study, namely how education and income influence physical activity patterns.

Strong educational gradients in physical activity have been documented across different countries and time periods. In the Netherlands in 1991, Droomers et al. (1998) found a clear educational gradient where physical inactivity in leisure time increased substantially as educational attainment decreased from university level to primary schooling. This pattern was echoed in England, where Farrell and Shields (2002) observed that individuals with degree-level qualifications were 21.4% more likely to participate in sports compared to those without qualifications in 1997. Similarly, in the 1990s UK, an additional year of schooling was associated with a 3.9%/3.7% higher likelihood of engaging in vigorous/moderate activity respectively, and passing A-level increased the probability of regular exercise by 6.3%–9.1% (Cutler and Lleras-Muney, 2010). In the US, Harper and Lynch (2007) found that physical inactivity was more prevalent among less-educated individuals across 31 states, with this disparity widening between 1990 and 2004, becoming one of the most pronounced inequalities in health behaviours. The first wave of the English Longitudinal Study of Ageing further supported these findings, showing that individuals who remained in full-time education longer were more likely to be physically active (Adams, 2009). More recent evidence from 2005 to 2011 showed that the difference between the least and most educated is in the order of ten percentage points in the probability of physical inactivity (Farrell et al., 2014). In Melbourne, Australia, medium-educated women had 33% lower odds and low-educated women had 50% lower odds of achieving sufficient leisure-time physical activity compared to highly educated women (Cleland et al., 2012).[12]

12. Additional evidence of educational inequalities in leisure-time PA across other European countries can be found in Demarest et al. (2014).

While most studies examining educational gradients have focused primarily on physical activity participation/physical inactivity, as illustrated above, the literature on income-related inequalities in PA reveals patterns across three distinct dimensions—activity duration/frequency, participation rates, and compliance with recommended guidelines. My specific focus on income measures and these three dimensions offers a comprehensive view of the pronounced income-related gradient in physical activity (Pontin et al., 2021).

Regarding activity duration and frequency, the majority of research points to a positive association with income.[13] In Brazil, adults from high-income households invested 1.06 times more time in leisure-time PA compared to those from low-income households (Manta et al., 2020). Across 52 high- and low-middle-income countries, adolescents from higher-wealth households demonstrated 0.5 to 0.6 days/week higher levels of moderate and vigorous physical activity (MVPA) outside of school, as well as greater activity frequency, compared to their peers from lower-wealth households (Bann et al., 2019).[14] In Finland, a 2011 study revealed that a one-unit increase in income level corresponded to a 0.11-unit increase in the (leisure-time) physical activity index, a 1% increase in pedometer-based total steps, and a 4% increase in continuous walking $\geq$ 10 min without interruption at a pace of $>$ 60 steps/min (Kari et al., 2015).[15] These findings align with earlier research conducted in Finland during 1995–1996, which also confirmed a positive relationship between income and PA (Arinen, 1998). However, another nationwide study in Finland conducted from 1993 to 1999 revealed a contrasting trend, observing a modest decrease in physical activity as income increased (Laaksonen et al., 2003). See more research in the review article Beenackers et al. (2012).

Income also determines the *participation* of PA (Humphreys and Ruseski, 2010; Scholes and Mindell, 2020) because participation in PA might require individuals to attain an income threshold to afford (Downward, 2007). The 2008 data from the National Health Interview Survey indicated that people from families earning at least $75,000 were less likely to be "physically inactive", i.e., engage in MVPA or strength training less than

13. Humphreys and Ruseski (2010); Scholes and Mindell (2020) referred to this aspect of PA as *volume*—"How much time should I spend participating in sport?".
14. Also see Guthold et al. (2020); Scholes and Mindell (2021).
15. Physical Activity Index (PAI) is a summary of five variables that captures the frequency and the intensity of physical activity.

once per week, compared to those from families with incomes under $35,000 (Cawley and Ruhm, 2011, p. 106), with this disparity reaching up to a 3-fold difference (Brownson et al., 2001). In England, the probability of engaging in PA not only increased as income rose but did so at an accelerating rate in 1997 (Farrell and Shields, 2002). The Swiss ageing population (comprised of individuals above 50) corroborates this relationship for moderate sports/exercise and vigorous sports/exercise, but an inverse relationship was found between income and habitual PA (e.g., walking, cycling, household chores, etc.) (Meyer et al., 2005). Among 147 neighbourhoods of Eindhoven and surrounding areas in the Netherlands, participation in recreational walking at minimal levels (less than 10 minutes per week) was more commonly reported by higher income groups, but the effects of neighbourhood aesthetics and individual cognitions were more pronounced in this context (Kamphuis et al., 2009). Analysing the BHPS from 1996/7 to 2002/3, Popham and Mitchell (2006) claimed no strong independent association between household income and leisure-time exercise frequency.

This stream of research generally employed self-reported frequency of PA or transformed continuous data into categorical or binary variables. This approach often results in the loss of valuable information and may mask both the lower and upper ends of the distribution (Scholes and Mindell, 2020, 2021). According to Humphreys and Ruseski (2010), cross-sectional studies based on single equation models will always identify a positive relationship between income and participation of PA. Recent studies have begun introducing models previously used for analysing other data with a high proportion of zeros and a positive skew, applying them to PA data, which provide insights into duration and participation rates of PA simultaneously.[16] For example, using the Cragg hurdle model, Scholes and Mindell (2020) found that English adults in high-income households were more likely to participate in MVPA than adults in low-income households. In addition, among those doing any sports/exercise, high-income households spent on average 1.3/1.0 more hours per week in sports/exercise. Regarding walking, Scholes and Mindell (2020) confirmed the higher participation probability of high-income families but noted no difference in the average walking hours per week. Another study for the UK, while acknowledging the

16. See economic papers Cragg (1971); Duan et al. (1983); Manning et al. (1987).

higher participation rate in sports activities among high-income households, suggested that conditional on participation, income did not influence the number of days in which the designated activity was practised (for at least 30 minutes) within the four-week period prior to interview (Buraimo et al., 2010). In Canada, higher income was associated with an increased likelihood of participation in swimming, golfing, weight lifting, and running, but had no effect on engagement in walking, home exercise, or cycling. Time spent on walking, home exercise, golfing, weight lifting and running decreases with income, while income had no impact on time allocated to cycling and swimming (Humphreys and Ruseski, 2010). As an alternative to double-hurdle models, Meltzer and Jena (2010) performed logarithm transformation on exercise data and discovered that individuals in the highest income group were 31% more likely to exercise than those with an annual family income below $20,000. Among those exercising, couples earning over $75,000 devoted nearly 27% more time to physical activity than couples earning below $20,000.

The third dimension of PA inequality focuses on adherence to recommended guidelines, which is conceptualised as the minimum level of activity required to achieve general health benefits, such as a lowered relative risk of cardiovascular disease (Chaudhury and Shelton, 2010), as expressed in e.g., WHO guidelines. In Canada, income raised the probability of meeting the guidelines for MVPA. The compliance rate of the lowest income group exceeded that of the second-lowest income group, primarily due to their reliance on walking as a mode of transportation (Bryan and Katzmarzyk, 2009a,b). In addition, guideline compliance was significantly correlated with higher household income among adolescents in the 100 largest US cities in 2005 (Butcher et al., 2008). This association was also observed among lower-income adults (Parks et al., 2003). Shuval et al. (2017) disentangled the relationship between household income and MVPA intensity, alongside the relationship between household income and meeting PA guidelines, over both a 2-day and a 7-day period. Measured via the accelerometers, (whole-sample) individuals with an income of $\geq 75,000$ engaged in 4.6/2.4 more daily minutes of MVPA/continuous MVPA lasting 10 minutes or more in comparison to those with annual income $< 20,000$. They were also 1.6/1.9 times more likely to meet PA guidelines—defined as 150 minutes of MVPA per week—over a 2- and 7-day period. Cleland et al. (2012) categorised walking, moderate- and vigorous-intensity leisure-time PA (LTPA) as zero minutes/week (none),

1–149 minutes/week (insufficient), and ≥ 150 minutes/week (sufficient). Their findings revealed that women in Melbourne earning less than $299 per week (gross income) were less likely to achieve either insufficient or sufficient LTPA compared to those with weekly earnings of $700 or more.

As enumerated in Table 3.2.2, these studies utilised income measures with varying degrees of precision.[17] While some researchers employed more meticulous income measures (see e.g., Laaksonen et al., 2003; Downward, 2007; Kamphuis et al., 2009), others relied on the data at hand and contributed in other aspects (see e.g., Buraimo et al., 2010; Meyer et al., 2005). However, to our knowledge, no study has compared results across different income measures. In other words, there remains a gap in understanding whether different income measures play distinct roles in shaping the three dimensions of PA discussed above. The most relevant study is (Laaksonen et al., 2003), which observed a modest decline in PA with increasing total taxable individual income and monthly disposable household income, using a sample of 19,982 Finnish adults from 1993 to 1999. As mentioned in Laaksonen et al. (2003), Arinen (1998) reported an opposite relationship when considering gross household disposable income per consumption unit.[18]

In summary, the literature synthesis in Table 3.2.2 categorises existing research on the income-physical activity relationship by distinguishing between three dimensions of physical activity and different income measurements employed. This systematic review reveals a notable research gap: the absence of studies examining the joint effects of education and income, particularly in differentiating between physical activity participation and guideline compliance. This limitation primarily stems from the infrequent use of house-

17. Table 3.2.2 categories income measures into gross and net income. Household net income refers to total income from all sources (wages, investments, transfers, etc.) after tax deductions and social security contributions. Household disposable income, on the contrary, includes social transfers and pensions, so disposable income is a broader measure of household economic resources, capturing both monetary and non-monetary benefits. The statistical analysis in this study uses household disposable income. See Section 3.3.2.2.

18. Different income measures also matter for other health behaviours. For example, according to (Lakdawalla and Philipson, 2002; Lakdawalla et al., 2005), earned income, generated through the labour market, impacts weight through occupational pathways, such as more skilled, sedentary jobs, while unearned income may lead to a higher demand for thinness. In addition, one possible explanation of the positive relationship between disposable income and smoking rate in China is that cigarette consumption accounts for a large share of an individual's disposable income in low-income countries, which would be masked if using gross income (Wang et al., 2018).

Table 3.2.2: Effects of income on physical activities

	Dimensions of PA			Household income measure		
	Duration/frequency	Participation	Guideline compliance	Gross income	Net income	Unspecified[d]
Manta et al. (2020)	✓					✓
Bann et al. (2019)[a]	✓					
Kari et al. (2015)[b]	✓					✓
Laaksonen et al. (2003)[b,c]	✓				✓	✓
Arinen (1998)	✓			✓		
Cawley and Ruhm (2011)		✓				
Popham and Mitchell (2006)		✓		✓		
Farrell and Shields (2002)		✓				✓
Meyer et al. (2005)		✓				✓
Kamphuis et al. (2009)[c]		✓			✓	
Farrell et al. (2014)		✓				✓
Kim and So (2014)[c]		✓				✓
Parks et al. (2003)			✓			✓
Butcher et al. (2008)			✓			✓
Bryan and Katzmarzyk (2009a)			✓			✓
Scholes and Mindell (2021)	✓	✓		✓		
Scholes and Mindell (2020)	✓	✓		✓		
Buraimo et al. (2010)	✓	✓				✓
Humphreys and Ruseski (2010)[b]	✓	✓				✓
Downward and Riordan (2007)	✓	✓		✓		
Meltzer and Jena (2010)	✓	✓				✓
Shuval et al. (2017)	✓		✓			✓
Cleland et al. (2012)	✓	✓	✓	✓		

[a] Bann et al. (2019) compared adolescents' activity levels among 52 countries, and within a country, students top- versus bottom-wealth quintile were compared.

[b] Also used individual income besides household income.

[c] Based on monthly rather than annual household income.

[d] "Unspecified" indicates gross/net income specification was not reported in text. Other income-related methodological details (e.g., household equivalisation, income categorisation) may be discussed according to each paper's focus.

[e] All of the papers in this table used discredited income groups (divided into quintiles) instead of continuous income for several reasons. First, annual household income in surveys is often recorded in a grouped format. Second, grouped income categories enable more straightforward cross-regional comparisons (see e.g., Bann et al., 2019) and comparison with previous papers (see e.g., Shuval et al., 2017). Third, categorical groups simplify interpretation by allowing relative comparisons, such as "compared to the lowest/highest income quintile".

hold net income or disposable income measures, which would effectively proxy purchasing power and enable the isolation of education's distinct mechanisms operating through cognitive capital, intertemporal preferences, social identity, and class-based behavioural norms.

3.3 Data and Methods

Having reviewed the broader relationship between socioeconomic status and health behaviours, I narrow my focus to the specific effects of education and income on physical activity, the primary interest of this study. This section introduces the nationally representative dataset employed in this study and describes both the key variables from the dataset and the variables I developed for the regression analyses.

3.3.1 Analysed Datasets

I conduct empirical analysis using the UK Household Longitudinal Study, also known as *Understanding Society* (UnSoc) (University of Essex, ISER, 2023). While health research often relies on cross-sectional data or accelerometer measurements (see Section 3.2 for details), I utilise UnSoc for four key advantages. First, the (main survey of) UnSoc collects data from a nationally representative sample of UK adults aged 16 and over, ensuring strong generalisability to the UK population (Buck and McFall, 2012). Second, with Wave 1 (January 2009–January 2011) encompassing approximately 40,000 households, this large sample size enables high-resolution analysis at both regional and sub-regional levels (Buck and McFall, 2012). Third, the longitudinal design allows us to track the same individuals and households over time, enabling the study of individual life transitions and family dynamics while controlling for time-invariant effects that might otherwise bias results (Hedeker and Gibbons, 2006). Fourth, the study's comprehensive coverage of multiple disciplines—including health, work, education, income, family, and social life—enables researchers to effectively isolate the effects of our key variables of interest. In this study, it enables precise differentiation between education levels, gross labour income, and disposable income, allowing for the identification of distinct socioeconomic mechanisms and relevant income measures.

The physical activity data (see Section 3.3.2.1 for details) was collected in Waves 7 and 9, which limits our main analysis to these two waves spanning from January 2015 to May 2019. The year of each respondent's personal interview `intdaty_dv' serves as the time variable in our panel regressions. Although the periods of waves overlap, every individual is interviewed at approximately 12-month intervals, so no respondent is interviewed twice within a wave or a calendar year (Institute for Social and Economic Research, 2023). See Institute for Social and Economic Research (2023) for detailed study information and sampling methodology. I also restrict our sample to individuals between the ages of 25 and 60, as this age range typically represents people who have completed their education, maintain physical capability for regular activity, and can make independent financial decisions (Cutler and Lleras-Muney, 2010). See the detailed sample selection procedure in Section 3.3.3.

3.3.2 Definitions of Key Variables

3.3.2.1 Definition of Physical Activities

The main dependent variable is the proportion of time allocated to physical activity (PA) per week. This measure encompasses two intensity levels—vigorous and moderate activities. The survey defines vigorous activities as those that "make you breathe much harder than normal and may include heavy lifting, digging, aerobics, or fast bicycling", and moderate activities are defined as those that "make you breathe somewhat harder than normal and may include carrying light loads, bicycling at a regular pace, or doubles tennis". See the complete list of questions in Appendix 3.6.1.1.

I generated the physical activity variable through the following steps. First, participants indicate whether they performed each type of activity for a minimum of 10 minutes in the last 7 days. For those reporting at least one day of activity, the survey then elicits the time spent on the activity during a typical day. I calculated the weekly time (in minutes) individuals spend on vigorous and moderate activities as the product of activity days

and typical daily duration.[19] Second, I standardised the weekly minutes of vigorous and moderate activities by dividing them by 4,200 minutes (equivalent to 10 hours daily for 7 consecutive days). This method assumes that an individual's maximum available leisure time, excluding physiological needs and work, is 10 hours per day.[20] This method yields a proportion of available time allocated to physical activity per week, ranging from 0 to 1. Observations with standardised values exceeding 1 which likely represent measurement error or reporting inconsistencies were dropped.[21]

Finally, I computed the weighted average of standardised vigorous and moderate activities, using Metabolic Equivalent of Task (MET) values as weights, which estimate energy expenditure.[22] In summary, I constructed a continuous variable that quantifies the proportion of available weekly time dedicated to either vigorous or moderate activities, ranging from 0 to 1.

Moreover, to investigate nonlinear relationships between income and PA, I categorised the continuous PA variable into three groups—zero PA, some PA below the guideline, and PA above the guideline. This categorisation allowed us to examine how income differently affects the probability of engaging in any PA and the likelihood of meeting recommended guidelines.[23]

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19. In cases where participants are unable to specify hours and minutes for either activity type, they are asked to provide a direct estimate of their total weekly time spent on vigorous and moderate activities. I estimate our models both with and without the inclusion of observations where physical activity is reported as aggregate weekly time rather than derived from daily measures. The results are very similar.
20. Gronau (1977) found that Israeli married women had approximately 5 hours of daily leisure time. According to Aguiar and Hurst (2007), individuals in the US had nearly 120 hours of free time per week between 1965 and 2003. Although I cannot quantify the available time, I conduct robustness checks using multiple thresholds and truncated upper bounds. See Appendix 3.6.3.3.
21. 69 outliers were removed from the 39,535 observations.
22. One MET equals the energy or oxygen used while sitting quietly. Physical activity that burns 6.0 METs or more/3.0 to 5.9 METs is vigorous/moderate intensity (Centers for Disease Control and Prevention, 2023). I used weights 7.0 and 5.0 respectively for vigorous and moderate activities.
23. Chaudhury and Shelton (2010) categorised the levels of reported PA based on the frequency of 30-minute moderate or vigorous activity sessions during the previous four weeks: "high" (20 or more occasions), "medium" (4–19 occasions), and "low" (up to 3 occasions).

3.3.2.2 Definition of Income and Education

I calculated and employed monthly gross household labour income and total monthly household disposable income as the primary independent variables. The UnSoc survey provides a) gross individual monthly labour income `fimnlabgrs_dv' earned from current job, second job, and self-employment earnings. b) net individual monthly total income `fimnnet_dv'—net of taxes on earnings and national insurance contributions (NIC). Besides labour income, b) also includes net-of-tax miscellaneous income, private benefit income, investment income, pension income, and social benefit income (see Institute for Social and Economic Research, 2023, p. 43–44, for detailed explanations of each component).[24] In other words, gross labour income refers to the total earnings from employment, while net total income can be interpreted as disposable income.[25]

I constructed equivalised household income measures by aggregating individual-level gross labour income and disposable income across household members, then dividing by the modified OECD equivalence scale `ieqmoecd_dv'.[26] This equivalisation procedure adjusts for household size and composition, enabling meaningful cross-household comparisons. I also omitted observations where household income is negative (see Step 5 in Table 3.3.3),[27] trimmed the top and bottom 0.5% of values in every year, deflated income values using the annual Consumer Price Deflator for the UK (2015 = 100) (Office for National Statistics, 2023), and took logarithms of the two income measures.

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24. I calculated total net household income as the sum of six components (`fimnlabnet_dv', `fimnmisc_dv', `fimnprben_dv', `fimninvnet_dv', `fimnpen_dv', and `fimnsben_dv') as stated in Institute for Social and Economic Research (2023). While the survey provides a total net income variable (`fimnnet_dv'), I found discrepancies between our calculated sum and the provided values for 431 observations (less than 1% of the sample). Given these potential recording errors, I used our calculated total in the main analyses. Robustness checks using the survey-provided total income variable yielded similar results.
25. `fimnnet_dv' is described as "total net personal income" in UnSoc, but since it also includes non-monetary benefits, I interpret it as disposable income.
26. `ieqmoecd_dv' assigns a weight of 1 to the first adult (person aged 14 or older) in the household, a weight of 0.5 to each additional adult, and a weight of 0.3 to each child (person aged 0–13).
27. Zero values in household gross labour income and household total disposable income, which includes transfers, were considered reporting errors.

Our analysis considers both households' absolute income levels and relative income positions. To approximate their positions in the income distribution, I classified households into five quintiles, following previous literature enumerated in Table 3.2.2 in Section 3.2.4. Table 3.3.1 summarises the key statistics of gross household labour income and total disposable household income.

To account for the influences of education on the patterns of physical activities independently of income effects, I employed the highest educational or vocational qualification (`w_hiqual_dv'). This is a categorical variable with 6 categories: "Degree", "Other higher degree", "A-level, etc", "GCSE, etc", "Other qualification", and "No qualification" (see Table 3.3.2 for descriptive statistics).

Table 3.3.1: Summary statistics of income measures

Income states	*N*	Mean	Median	Std. Dev.	Skewness	Min	Max
Panel A: monthly household gross labour equivalised income							
s_{q_1}	6,910	57.19	0.00	101.43	1.46	0.00	326.09
s_{q_2}	7,503	660.24	663.22	193.93	-0.01	326.80	999.93
s_{q_3}	7,652	1,323.57	1,320.70	195.24	0.03	1,000.00	1,666.67
s_{q_4}	7,980	2,073.01	2,061.47	253.34	0.13	1,666.67	2,552.59
s_{q_5}	8,875	3,705.12	3,398.67	1,040.55	1.39	2,552.89	11,110.67
Pooled	38,920	1,667.59	1,430.85	1,391.53	1.05	0.00	11,110.67
Panel B: monthly household disposable equivalised income							
s_{q_1}	6,566	447.83	500.00	218.74	-0.68	0.00	733.72
s_{q_2}	7,121	918.20	923.58	99.92	-0.08	733.89	1,087.88
s_{q_3}	7,900	1,283.78	1,283.34	113.96	0.04	1,088.10	1,485.92
s_{q_4}	8,253	1,760.43	1,750.42	169.65	0.14	1,486.03	2,075.99
s_{q_5}	9,096	2,895.88	2,649.58	774.36	1.48	2,076.11	7,346.72
Pooled	38,936	1,553.59	1,371.77	939.95	1.18	0.00	7,346.72

[a] This table presents summary statistics for the two income measures across quintiles, where s_{q_1} is the lowest quintile and s_{q_5} is the highest.
[b] The sample includes respondents aged 25–60 from Waves 7 and 9 of Understanding Society (2015–2019).

3.3.2.3 Additional Controls

To disentangle the impacts of education and household income, I included a set of control variables in the regressions that could potentially influence physical activities.

3.3.2.3.1 Health shock

The onset of a new disease can affect physical activity in two opposing ways—it may restrict one's ability to engage in exercise (Garthwaite, 2012), or it may inspire a stronger motivation to pursue better health (Zhang et al., 2022; Xiang, 2016).

I assume three states of health shocks: a) never had a severe illness, b) currently experiencing a health shock, and c) in the recovery period. An individual's situation is proxied via seven particular severe illnesses recorded, which are unlikely to be misreported so are free from justification bias (Gupta et al., 2015). In the UnSoc, respondents are asked about their existing health conditions to date in Wave 1 and whether any of the seven illnesses have been diagnosed since the last interview in the following waves. The selected exogenous health events include Congestive Heart Failure `w_hcondn3', Coronary Heart Disease `w_hcondn4', Heart attack or myocardial infarction `w_hcondn6', Stroke `w_hcondn7', Emphysema `w_hcondn8', Chronic Bronchitis `w_hcondn11', and Cancer or Malignancy `w_hcondn13'.[28] I define a current health shock (state b) as a new diagnosis in the current year with no prior history of any of these conditions i.e., any of the enumerated variables is equal to 1 in the current year and all variables are equal to 0 previously. All years following a diagnosis are classified as recovery period (state c). Note that the entire history prior to the first wave is investigated in Wave 1 by asking if the respondent had experienced any of the diseases, so I cannot distinguish between recent diagnoses (state b) and recovery period (state c). Therefore, all individuals with pre-existing conditions in Wave 1 are categorised as being in the recovery period.[29]

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28. The selected major health shocks are likely to be unanticipated and exogenous (Cheng et al., 2019).
29. Variables capturing the entire history in Wave 1 are `w_hcond3', `w_hcond4', `w_hcond6', `w_hcond7', `w_hcond8', `w_hcond11' and `w_hcond13'.

Table 3.3.2: Summary statistics of education and additional controls

Panel A: Categorical measures		
	N	Share (%)
Education[b]	38,774	
Degree		34.389
Other higher degree		13.102
A-level, etc.		20.034
GCSE, etc.		19.928
Other qualification		7.232
No qualification		5.315
Gender	39,782	
Male		43.248
Female		56.752
Urban/rural	39,761	
Urban		78.592
Rural		21.408
Ethnicity	39,756	
British/Irish		74.349
Mixed		5.672
Others		19.979
Marital status	39,640	
Single/never partnered		28.027
Currently in a partnership		59.516
Previously partnered		12.457
Region	39,761	
North East		3.521
North West		10.533
Yorkshire and the Humber		8.710
East Midlands		6.874
West Midlands		8.649
East of England		7.932
England		14.637
South East		11.597
South West		7.477
Wales		5.976
Scotland		8.015
Northern Ireland		6.079
Health shock	38,638	
Never had health shocks		95.629
Experiencing health shocks		0.805
Experienced health shocks before		3.566
Journey to work	25,813	
Inactive commute		86.054
Active commute (walk/cycle)		13.946
Physicality of job	26,098	
Non-physical job		37.183
Fairly physical job		12.944
Physical job		49.874

Panel B: Continuous measures				
Variable	*N*	Mean	Std. Dev.	Skewness
Age	34,193	43.378	9.834	-0.134
Household size	34,193	3.2685	1.4825	0.9830

[a] This table presents summary statistics for all control variables. The sample includes respondents aged 25–60 from Waves 7 and 9 of Understanding Society (2015–2019).
[b] In terms of education, "Degree" encompasses University Higher Degrees (e.g., MSc, PhD), first degree level qualifications including foundation degrees, graduate membership of a professional Institute, and PGCE, while "Other higher degree" comprises Diploma in higher education, Teaching qualification (excluding PGCE), Nursing or other medical qualification not yet mentioned, and other higher degree.

3.3.2.3.2 Commuting and Job Physicality

Our physical activity (PA) data includes "*...the activities you do at work, as part of your house and gardening, to get from place to place, and in your spare time for recreation, exercise or sport*". To better understand how income and education influence different aspects of PA, I incorporated two additional measures. First, following Guthold et al. (2018); Scholes and Mindell (2021), I created a binary commuting mode variable (`worktrav`) where 1 indicates active commuting (walking or cycling) and 0 indicates motorised transport. Second, I examined job physicality, which was recorded in Waves 2 and 5 of UnSoc, where respondents indicated whether their jobs were physically demanding. I initially created a dummy where 1 represented "very physically active" or "fairly physically active" jobs, and 0 represented "not very physically active" or "not at all physically active" jobs. To estimate job physicality for the waves I focus on, I calculated each individual's average score across Waves 2 and 5, resulting in three categories: 0 for "non-physical" jobs, 0.5 (recoded to 1) for "fairly physical" jobs, and 1.0 (recoded to 2) for "physical" jobs.

See additional demographic controls in Appendix 3.6.1.2.

3.3.3 Sample Selection

As shown in Table 3.3.3, the analysis excludes observations with missing person identifiers (`` `pidp' ``), restricts the age range to working-age adults between 25 and 60 years, eliminates duplicate observations within waves, and removes observations with missing physical activity measures or negative household (equivalised) income.

Table 3.3.3: Sample selection

Selection step	Number of individuals
Whole sample	410,649
1. Drop if person identifier is missing	409,863
2. Keep if the individual is between 25 and 60	243,081
3. Drop duplicated observations[b]	236,475
4. Drop if physical activity measure is missing[c]	39,493
5. Drop if total household (equivalised) income is negative	39,466[c]
Average number of individuals per year	3,993.20

[a] Data for different waves are merged in a `long` format, implying that observations in separate waves for one individual are recognised as different sample objectives.
[b] Although no respondent is interviewed twice within a wave or a calendar year, duplicate observations occur when individuals transition between households, resulting in unchanged personal identifiers but altered household identifiers. We drop one of these duplicates when performing individual-level panel regressions.
[c] As the primary independent variable, household income is measured through monthly gross household labour income and monthly household disposable income (see Section 3.3.2.2). Additional income measures, computed through alternative calculation methods, serve as robustness checks. As an example, after dropping negative values, 39,466 observations remain when using total household disposable income. Other income measures yield similar sample sizes.

3.3.4 Econometric Specification

This section presents the ordered logit model used to examine how education and income influence the likelihood of individuals being categorised into three hierarchical physical activity levels: physical inactivity, below-guideline activity, and guideline-adherent activity.

Based on the PA variables constructed in Section 3.3.2.1, let $y^c_{i,t} \in [0,1]$ denotes the continuous variable measuring the proportion of available weekly time dedicated to either vigorous or moderate activities. I transform this continuous measure into a categorical variable $y_{i,t} \in \{0,1,2\}$ defined as:

$$y_{i,t} = \begin{cases} 0 & \text{if } y^c_{i,t} = \tau_1 \quad \text{(Inactive)} \\ 1 & \text{if } \tau_1 < y^c_{i,t} < \tau_2 \quad \text{(Below guidelines)} \\ 2 & \text{if } y^c_{i,t} \geq \tau_2 \quad \text{(Meet guidelines)}, \end{cases} \tag{3.3.1}$$

where $\tau_1 = 0$[30] and τ_2 denotes the recommended guideline of PA—at least 150 minutes of moderate-intensity physical activity, 75 minutes of vigorous-intensity physical activity, or an equivalent combination per week (Shuval et al., 2017; WHO, 2010).[31]

Figure 3.3.1 presents the distribution of $y^c_{i,t}$ and visualises the cutoff points determining $y_{i,t}$. Table 3.3.4 summarises the key statistics of the continuous and categorical measures of PA.

Table 3.3.4: Summary statistics of physical activity measures

Continuous measure: $y^c_{i,t}$		
Mean		0.040
Standard deviation		0.075
Minimum		0.000
Maximum		0.857
Skewness		3.553
Share of zeros		37.853%
Categorical measure: $y_{i,t}$		
Category	Definition	Share (%)
0 (Inactive)	$y^c_{i,t} = 0$	37.853
1 (Below guidelines)	$0 < y^c_{i,t} < \tau_2$	27.071
2 (Meets guidelines)	$y^c_{i,t} \geq \tau_2$	35.077
N		39,733

30. As specified in Table 3.6.1 in Appendix 3.6.1.1, respondents are asked to report vigorous/moderate activities lasting for at least 10 minutes. This is to ensure the intensity of each activity (also see Shuval et al., 2017). Therefore, technically, $y_{i,t} = 0$ if $0 \leq y^c_{i,t} < 9.92\times10^{-4}$ and $y_{i,t} = 1$ if $9.92\times10^{-4} \leq y^c_{i,t} < \tau_2$, where $9.92 \times 10^{-4} = \frac{0+(10/4{,}200\times5)}{7+5}$, representing those who performed less than 10 minutes of moderate activity in the past 7 days. Since this number is very close to 0, I simplify by using 0 in the equations.

31. $\tau_2 = \frac{(75/4{,}200\times7)+(150/4{,}200\times5)}{7+5} = 0.0253$, where 7 and 5 are the weights assigned to vigorous and moderate activities respectively based on their MET values, and 4,200 minutes is the maximum available time in a week.

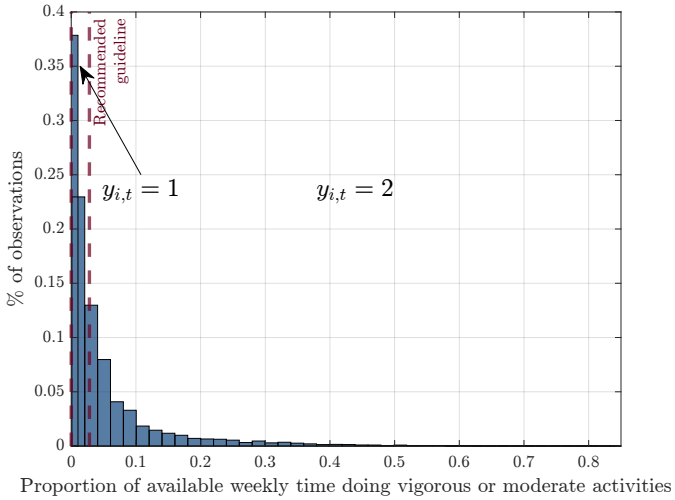

Figure 3.3.1: **Probability density function of weekly time of PA.** This figure plots the density histogram of the proportion of available weekly time doing vigorous or moderate activities of the whole sample. The vertical red lines mark the cutoff points that convert the continuous variable into three discrete categories.

Let $y^*_{i,t}$ denote the latent continuous propensity for physical activity, which represents an individual's underlying tendency to engage in physical activity based on their health consciousness and energy expenditure preferences. The ordered logit model is specified as:

$$y^*_{i,t} = \beta_0 + \beta_1 \ln\left(s_{i,t}\right) + \beta_2 w_{i,t} + \mathbf{X}'_{i,t}\boldsymbol{\gamma} + u_i + \epsilon_{i,t}, \tag{3.3.2}$$

$$y_{i,t} = \begin{cases} 0 & \text{if } y^*_{i,t} \leq \mu_1 \\ 1 & \text{if } \mu_1 < y^*_{i,t} \leq \mu_2 \\ 2 & \text{if } y^*_{i,t} > \mu_2. \end{cases}$$

For individual i in year t, $s_{i,t}$ is the monthly household income (gross labour or disposable income), $w_{i,t}$ is the highest education attainment level, $\mathbf{X}_{i,t}$ includes a series of controls. The threshold parameters μ_1 and μ_2 determine the cutoff points on the latent propensity scale that correspond to the observed physical activity categories.[32]

32. I assume constant thresholds for all individuals.

The error term consists of u_i and $\epsilon_{i,t}$, where u_i is the individual-specific time-invariant component (random effect) and $\epsilon_{i,t}$ is the individual and time-varying disturbance term.[33] I assume that $u_i \sim N(0, \sigma_u^2)$ and $\epsilon_{i,t}$ follows a logistic distribution with mean 0 and variance $\pi^2/3$. In addition, the two error components are assumed to be independent: $\text{Cov}\left(u_i, \epsilon_{i,t}\right) = 0$, and they are independent of all explanatory variables. The estimation of Equation 3.3.2 also includes year fixed effects to account for the yearly changes that are the same for all individuals, such as the 2012 Olympics.

3.4 Empirical Results

3.4.1 Estimated Coefficients

Table 3.4.1 and Table 3.4.2 present the estimated coefficients of Equation 3.3.2. Both tables show that monthly household income and education level have positive and statistically significant associations with physical activity levels.[34]

The results also reveal several demographic and socioeconomic factors associated with lower physical activity levels, which align with existing literature: being female, living in urban areas, being in a partnership, belonging to minority ethnic groups (compared to British/Irish), residing in Northern Ireland (compared to Londoners), experiencing health shocks (current or past),[35] using motorised transport for commuting, and working in non-physical occupations (Adams, 2009; Sawyer et al., 2017; Farrell and Shields, 2002; Demarest et al., 2014; Laaksonen et al., 2003).

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33. Option `fe` for fixed effect is not allowed for ordered logit models in Stata because of the Incidental Parameters Problem.
34. The magnitudes of estimated coefficients are not straightforwardly interpretable. For example, a one-unit increase in log gross household labour income is associated with a 0.0959 increase in the ordered log-odds of being in a higher physical activity category (from inactive → below guidelines → meets guidelines).
35. Therefore, in our sample, health shocks appear to reduce people's physical ability to exercise, outweighing any increased motivation to be physically active following health problems.

Table 3.4.1: Effects of monthly household gross labour income on PA, ordered logit coefficients

Variable	(1)	(2)	(3)	(4)	(5)
Log of household gross labour income ($\ln(s_{i,t})$)	0.1704***	0.0429*	0.0418*	0.0554*	0.0959**
	(0.0175)	(0.0177)	(0.0178)	(0.0267)	(0.0315)
Education *(ref: Degree)*					
Other higher degree	-0.0674	-0.0703	-0.0625	-0.0800	-0.1417*
	(0.0482)	(0.0480)	(0.0483)	(0.0544)	(0.0615)
A-level, etc.	0.0508	-0.0987*	-0.0927*	-0.0763	-0.1113*
	(0.0430)	(0.0425)	(0.0428)	(0.0486)	(0.0549)
GCSE, etc.	-0.0899	-0.1958***	-0.1843***	-0.1463**	-0.2231***
	(0.0461)	(0.0458)	(0.0461)	(0.0539)	(0.0606)
Other qualification	-0.2204**	-0.2601***	-0.2422***	-0.1776*	-0.3122**
	(0.0704)	(0.0699)	(0.0703)	(0.0848)	(0.0969)
No qualification	-0.9275***	-0.7564***	-0.7352***	-0.4981***	-0.6537***
	(0.0975)	(0.0967)	(0.0972)	(0.1302)	(0.1522)
Gender *(ref: Male)*					
Female		-0.8955***	-0.8982***	-0.8713***	-0.8932***
		(0.0326)	(0.0328)	(0.0375)	(0.0422)
Age (squared)		-0.0001**	-0.0001***	-0.0001**	-0.0001*
		(0.0000)	(0.0000)	(0.0000)	(0.0000)
Urban/rural *(ref: Urban)*					
Rural		0.2420***	0.2415***	0.2009***	0.2067***
		(0.0388)	(0.0390)	(0.0440)	(0.0478)
Ethnicity *(ref: British/Irish)*					
Mixed		-0.1207	-0.1103	-0.1159	-0.0838
		(0.0749)	(0.0755)	(0.0871)	(0.1055)
Others		-0.7703***	-0.7562***	-0.6630***	-0.7253***
		(0.0478)	(0.0485)	(0.0573)	(0.0720)
Marital status *(ref: Single/never partnered)*					
Currently in a partnership		-0.1211**	-0.1226**	-0.1285**	-0.1096
		(0.0410)	(0.0412)	(0.0471)	(0.0566)
Previously partnered		-0.0603	-0.0596	-0.0382	-0.0085
		(0.0575)	(0.0578)	(0.0646)	(0.0717)
Region *(ref: London)*					
North East		-0.2744**	-0.2845**	-0.0846	-0.0785
		(0.0954)	(0.0960)	(0.1087)	(0.1217)
North West		-0.2148**	-0.2269***	-0.1536*	-0.1278
		(0.0658)	(0.0662)	(0.0774)	(0.0918)
Yorkshire and the Humber		-0.1972**	-0.2106**	-0.1442	-0.1682
		(0.0713)	(0.0716)	(0.0842)	(0.1010)
East Midlands		0.0522	0.0412	0.0504	0.0129
		(0.0746)	(0.0751)	(0.0852)	(0.0999)
West Midlands		0.0406	0.0345	-0.0072	0.0216
		(0.0693)	(0.0697)	(0.0811)	(0.0979)

Continued on next page

Table 3.4.1 continued

Variable	(1)	(2)	(3)	(4)	(5)
East of England		-0.0036	-0.0129	0.0048	0.0266
		(0.0699)	(0.0704)	(0.0802)	(0.0940)
South East		0.1433*	0.1381*	0.1439	0.1244
		(0.0631)	(0.0635)	(0.0738)	(0.0879)
South West		0.1803*	0.1723*	0.1389	0.1916*
		(0.0725)	(0.0729)	(0.0842)	(0.0974)
Wales		-0.1206	-0.1484	-0.0706	-0.1189
		(0.0823)	(0.0829)	(0.0942)	(0.1077)
Scotland		0.0140	-0.0039	0.0210	0.0332
		(0.0745)	(0.0750)	(0.0860)	(0.0990)
Northern Ireland		-0.4353***	-0.4831***	-0.3428***	-0.3014**
		(0.0835)	(0.0846)	(0.0958)	(0.1100)
Log of household size		-0.1028**	-0.1029**	-0.0109	0.0111
		(0.0364)	(0.0366)	(0.0425)	(0.0505)
Health shock *(ref: Never had health shocks)*					
Experiencing health shocks		-0.7875***	-0.7823***	-0.7814***	-0.7178**
		(0.1806)	(0.1825)	(0.2183)	(0.2428)
Experienced health shocks before		-0.2194*	-0.2423*	-0.0230	-0.0639
		(0.0952)	(0.0959)	(0.1120)	(0.1192)
Journey to work *(ref: Inactive commute)*					
Active commute (walk/cycle)				0.4355***	0.4888***
				(0.0511)	(0.0591)
Physicality of job *(ref: Non-physical job)*[c]					
Fairly physical job					0.2109***
					(0.0607)
Physical job					0.5585***
					(0.0453)
Year FE	No	No	Yes	Yes	Yes
N	33,422	32,333	32,333	24,200	19,226

[a] The table shows the ordered logit/latent variable coefficients i.e., the effects of monthly household gross labour income on the *latent* level of energy expenditure, which underlies the observed three-category physical activity measure (inactive, below guidelines, meets guidelines). Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] Data on job physicality is available in Waves 2 and 5 (2010–2015). Since our main analysis uses Waves 7 and 9 (2015–2019), we construct a time-invariant measure of job physicality for each individual by averaging their reported job physicality scores from Waves 2 and 5 (See Section 3.3.2.3.2 for details).

Table 3.4.2: Effects of monthly household disposable income on PA, ordered logit coefficients

Variable	(1)	(2)	(3)	(4)	(5)
Log of household disposable income ($\ln(s_{i,t})$)	0.2173***	0.1110***	0.1130***	0.0852*	0.1322**
	(0.0202)	(0.0197)	(0.0197)	(0.0351)	(0.0406)
Education *(ref: Degree)*					
Other higher degree	-0.1271**	-0.1001*	-0.0914	-0.0803	-0.1441*
	(0.0468)	(0.0465)	(0.0468)	(0.0542)	(0.0614)
A-level, etc.	-0.0558	-0.1682***	-0.1620***	-0.0768	-0.1145*
	(0.0416)	(0.0413)	(0.0415)	(0.0482)	(0.0546)
GCSE, etc.	-0.2744***	-0.3220***	-0.3095***	-0.1454**	-0.2246***
	(0.0435)	(0.0434)	(0.0436)	(0.0534)	(0.0602)
Other qualification	-0.5134***	-0.4855***	-0.4691***	-0.1757*	-0.3122**
	(0.0649)	(0.0644)	(0.0647)	(0.0846)	(0.0967)
No qualification	-1.4143***	-1.2217***	-1.2018***	-0.4991***	-0.6575***
	(0.0806)	(0.0814)	(0.0818)	(0.1296)	(0.1518)
Gender *(ref: Male)*					
Female		-0.8541***	-0.8563***	-0.8725***	-0.8959***
		(0.0313)	(0.0315)	(0.0374)	(0.0421)
Age (squared)		-0.0001***	-0.0001***	-0.0001**	-0.0001*
		(0.0000)	(0.0000)	(0.0000)	(0.0000)
Urban/rural *(ref: Urban)*					
Rural		0.2552***	0.2555***	0.1996***	0.2053***
		(0.0376)	(0.0378)	(0.0441)	(0.0478)
Ethnicity *(ref: British/Irish)*					
Mixed		-0.1133	-0.1014	-0.1157	-0.0837
		(0.0720)	(0.0725)	(0.0872)	(0.1056)
Others		-0.7570***	-0.7405***	-0.6632***	-0.7294***
		(0.0457)	(0.0464)	(0.0571)	(0.0718)
Marital status *(ref: Single/never partnered)*					
Currently in a partnership		-0.0187	-0.0192	-0.1289**	-0.1075
		(0.0390)	(0.0392)	(0.0468)	(0.0564)
Previously partnered		-0.0583	-0.0561	-0.0426	-0.0151
		(0.0533)	(0.0535)	(0.0645)	(0.0717)
Region *(ref: London)*					
North East		-0.3033***	-0.3131***	-0.0763	-0.0704
		(0.0910)	(0.0915)	(0.1086)	(0.1217)
North West		-0.2136***	-0.2219***	-0.1509	-0.1269
		(0.0631)	(0.0635)	(0.0774)	(0.0919)
Yorkshire and the Humber		-0.2102**	-0.2220**	-0.1417	-0.1669
		(0.0685)	(0.0688)	(0.0842)	(0.1011)
East Midlands		0.0645	0.0555	0.0537	0.0164
		(0.0717)	(0.0721)	(0.0853)	(0.1001)
West Midlands		0.0928	0.0891	-0.0051	0.0227
		(0.0666)	(0.0670)	(0.0811)	(0.0979)

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Table 3.4.2 continued

Variable	(1)	(2)	(3)	(4)	(5)
East of England		0.0208	0.0145	0.0078	0.0297
		(0.0677)	(0.0681)	(0.0802)	(0.0941)
South East		0.1718**	0.1670**	0.1440	0.1232
		(0.0612)	(0.0616)	(0.0738)	(0.0879)
South West		0.1536*	0.1465*	0.1390	0.1887
		(0.0702)	(0.0706)	(0.0841)	(0.0973)
Wales		-0.1410	-0.1672*	-0.0662	-0.1161
		(0.0791)	(0.0797)	(0.0943)	(0.1078)
Scotland		-0.0535	-0.0703	0.0252	0.0372
		(0.0720)	(0.0724)	(0.0860)	(0.0991)
Northern Ireland		-0.5101***	-0.5575***	-0.3397***	-0.2988**
		(0.0799)	(0.0810)	(0.0957)	(0.1100)
Log of household size		-0.0115	-0.0116	-0.0088	0.0108
		(0.0339)	(0.0340)	(0.0422)	(0.0503)
Health shock *(ref: Never had health shocks)*					
Experiencing health shocks		-0.7398***	-0.7414***	-0.7830***	-0.7194**
		(0.1594)	(0.1608)	(0.2184)	(0.2430)
Experienced health shocks before		-0.4461***	-0.4629***	-0.0249	-0.0670
		(0.0850)	(0.0855)	(0.1120)	(0.1193)
Journey to work *(ref: Inactive commute)*					
Active commute (walk/cycle)				0.4358***	0.4881***
				(0.0510)	(0.0590)
Physicality of job *(ref: Non-physical job)*[c]					
Fairly physical job					0.2116***
					(0.0607)
Physical job					0.5579***
					(0.0452)
Year FE	No	No	Yes	Yes	Yes
N	37,820	36,609	36,609	24,199	19,225

[a] The table shows the ordered logit/latent variable coefficients i.e., the effects of total monthly household disposable income on the *latent* level of energy expenditure, which underlies the observed three-category physical activity measure (inactive, below guidelines, meets guidelines). Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] Data on job physicality is available in Waves 2 and 5 (2010–2015). Since our main analysis uses Waves 7 and 9 (2015–2019), we construct a time-invariant measure of job physicality for each individual by averaging their reported job physicality scores from Waves 2 and 5 (See Section 3.3.2.3.2 for details).

3.4.2 Odds Ratios

When analysing logistic regression models, researchers commonly express effect sizes in terms of odds—how much more (or less) likely one outcome is compared to the alternative (see e.g., Droomers et al., 1998; Shuval et al., 2017; Cleland et al., 2012; Salmon et al., 2000). In line with this convention, this section shows and explains the odds ratios of education and household income, as well as the policy implications.

Mathematically, odds are calculated by dividing the probability of an event occurring by the probability of it not occurring. In our context, for example, the odds of meeting guidelines is calculated as the probability of meeting physical activity guidelines divided by the probability of being inactive or below guidelines. See Appendix 3.6.2 for the derivation of odds in the ordered logit model. Table 3.4.3 presents odds ratios of income and education for the whole sample (Table 3.4.3a), the male sample (Table 3.4.3b), and the female sample (Table 3.4.3c).

3.4.2.1 Odds Ratios of Education

First, compared to those with degrees, individuals with GCSE-level education have their odds of being in a higher physical activity category multiplied by 0.73–0.86 (representing a 14%–27% decrease).[36] Those with no qualifications show a further reduction, with odds multiplied by over 0.50 relative to degree holders, demonstrating a clear gradient where physical activity odds decrease with lower educational attainment. Comparing *Panel A* with *Panel B* in Table 3.4.3a, the association between education and physical activity is stronger when controlling for disposable income compared to when controlling for gross labour income.

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36. The ordered logit model assumes proportional odds/parallel regression—the effect of a predictor on the odds is identical across all possible binary comparisons of the ordinal outcome. For example, the effect of education level (comparing GCSE to degree-level education) on the odds remains constant whether comparing above "inactive" versus "inactive" or comparing "meeting guidelines" versus below "meeting guidelines".

Comparing Table 3.4.3b and Table 3.4.3b, the education gradient in physical activity is primarily observed among females. For males, after controlling for household income, the education-activity relationship loses statistical significance and shows small, inconsistent, and sometimes counter-intuitive patterns: compared to degree holders, GCSE-level educated males show a 6%–10% increase in odds of higher physical activity when controlling for gross labour income, but a less than 10% decrease in odds when controlling for disposable income.

While direct comparisons with existing literature are not straightforward due to different educational systems across countries, our findings generally align with the established education gradient in physical activity. Several studies from different countries, using diverse physical activity measures and statistical approaches, corroborate this relationship. In the Netherlands, Droomers et al. (1998) found that individuals with intermediate vocational or secondary schooling had more than double the odds of physical inactivity compared to those with higher vocational schooling or university education. Similarly, in Melbourne, Cleland et al. (2012) reported that women without formal qualifications had half the odds of achieving insufficient and sufficient (versus none) leisure-time physical activity (LTPA) compared to those with university degrees. In England, Farrell and Shields (2002) demonstrated that having a degree-level qualification increased sports participation probability by 0.214 compared to those without qualifications. However, findings regarding gender differences in the education-physical activity relationship remain inconsistent. While Demarest et al. (2014) observed more pronounced educational inequalities in leisure-time physical activity among men in 15 European countries other than the UK, Droomers et al. (1998) found no significant gender differences in the Netherlands.

3.4.2.2 Odds Ratios of Income

Fixing the education levels, Table 3.4.3 presents the effects of household income on physical activity levels, with gross household labour income in *Panel A* and household disposable income in *Panel B*. A one-unit increase in $\ln\left(s_{i,t}\right)$ (logarithm of gross household labour income) is associated with 4%–18% higher odds (odds ratio: 1.04–1.18) of being in a higher physical activity category, holding other variables constant and conditioning

on the individual-specific effect u_i. The impact of disposable income is stronger, with a one-unit increase in its logarithm associated with 9%–24% higher odds (odds ratio: 1.09–1.24) of being in a higher physical activity category. This difference is noteworthy since disposable income represents disposable income that eventually benefits the family, suggesting that existing studies using gross income listed in Table 3.2.2 may underestimate the income effects. The stronger effect of disposable income also highlights the potential role of redistribution policies—tax and transfer systems that increase households' disposable income may be more effective in promoting physical activity than policies targeting gross labour income alone.

To facilitate comparison with existing research (see Table 3.2.2 and Section 3.2.4), which primarily analyses income through categorical measures (groups, classes, or quintiles), I present additional results using income quintiles in Table 3.6.4 in Appendix 3.6.3.1. Both monthly gross household labour income and household disposable income were divided into five quintiles, ranging from s_{q_1} (lowest) to s_{q_5} (highest). Our categorical analysis enables direct comparison with studies like Shuval et al. (2017), which found that for US individuals, the odds of meeting physical activity guidelines for those earning over \$75,000 annually (gross) were 1.6 and 1.9 times the odds for those earning less than \$20,000, measured over 2 and 7-day periods respectively. Among women in Melbourne, compared to those earning $\geq$ \$700 per week (gross income), women earning $<$ \$299 per week had 0.48 times the odds of insufficient leisure-time physical activity (LTPA) versus no LTPA, and 0.69 times the odds of sufficient LTPA versus no LTPA (Cleland et al., 2012). Our results show similar patterns: individuals in the highest quintile of household gross labour income (monthly income $\geq$ £2,553) have 1.3–2.0 times the odds of being in a higher physical activity category compared to those in the lowest quintile (monthly income $\leq$ £326).

However, our main analysis focuses on continuous income measures as they provide more precise insights for policy evaluation—policymakers need to know how incremental changes in household income through various interventions (such as tax reforms or income support programs) might affect physical activity levels. Therefore, I translate our continuous income estimates into more interpretable measures by examining the effects of percentage changes in income, rather than changes in log units.

The second row of each subtable in Table 3.4.3 shows how a 10% increase in absolute income ($\Delta_{10\%} s_{i,t}$) affects the odds of higher physical activity levels. To interpret these results, consider an odds ratio of 1.1006: a 10% increase in $s_{i,t}$ multiplies the odds by $\exp\left[\ln\left(\exp\left(\beta_1\right)\right)\ln\left(1+\frac{p}{100}\right)\right] = \exp\left[\ln\left(1.1006\right)\times\ln\left(1.1\right)\right] = 1.0092$ (see Appendix 3.6.2, Equation 3.6.2 and Equation 3.6.3 for detailed derivations). In other words, a 10% increase in gross labour income is associated with only 0.4%–0.9% higher odds of being in a higher physical activity category. The effect of disposable income is more pronounced, particularly by gender: a 10% increase raises the odds by 1.1%–2.2% for males but only about 0.7% for females.

To better understand the magnitude of these effects, I add an additional row calculating the income increase needed to reach the effect of upgrading from GCSE-level to degree-level education on the odds of higher physical activity in Table 3.4.3a. Consider the specification in Column (5): upgrading from GCSE-level to degree-level education multiplies the odds by $1/0.8 = 1.25$. To achieve an equivalent effect through income alone would require a 924.57% increase in gross household income, an enormous increase that highlights the substantial impact of education relative to income.[37] For disposable income, which is more relevant when considering redistributive policies, a 446.54% increase

37. Based on Equation 3.6.3, if $\exp\left[\ln\left(\exp\left(\beta_1\right)\right)\ln\left(1+\frac{p}{100}\right)\right] = \exp\left[\ln\left(1.1006\right)\times\ln\left(1+\frac{p}{100}\right)\right] = 1.25$, $p = 924.57$.

would be required, which is also substantial—it is equivalent to moving a median earner of the lowest income quintile to the highest. Such dramatic increases suggest that using income redistribution alone to promote physical activity would require near-complete income equalisation across all families.[38]

The income effects reveal notable gender differences.[39] Among males, household income shows a strong, statistically significant impact on physical activity levels (odds ratio: 1.11–1.26). For comparability with the broader literature, analysis using income quintiles revealed that males in the highest income category were 2.34–3.68 times more likely to engage in higher levels of physical activity. In contrast, females' physical activity levels appear unaffected by either household gross labour income or disposable income after accounting for commuting and occupational physical demands. In terms of magnitude, the income effects for males were consistently two to three times larger than those observed among females. The empirical evidence on gender differences in income effects on physical activity remains limited. While our findings show distinct income gradients between men and women, comparable analyses are scarce in the literature. Instead, some papers have focused on broader socioeconomic status differences, but with mixed results. For instance, Burton and Turrell (2000) found no relationship between working hours and insufficient activity levels among females. Conversely, Salmon et al. (2000) reported that after accounting for occupational and household physical activity, socioeconomic gradients persisted for women but weakened for men in eight Australian capital cities in 1989.

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38. The average tuition of £9,000–£9,250 per year in England (2015–2019) amounts to £46,250 total government expenditure (which takes a 5-year period, take £9,250 as an example) (Murphy et al., 2019). Distributing this sum instead as direct payments between ages 25–60 provides annual support of £1,321.43. While degree funding multiplies outcome odds by $1/0.8 = 1.25$, the direct payment yields only a 22.02% increase in disposable income (£110.12 monthly relative to £500 baseline, £500 is the disposable income of a median earner in the lowest income group, see Table 3.3.1), multiplying odds by 1.02—substantially below the education benefits.

39. I analyse male and female samples separately rather than using gender-household income interaction terms, as prior research shows gender-specific patterns in how demographic factors affect physical activity. For instance, Farrell and Shields (2002) found that men's sports participation decreases more steeply with age than women's. Also see Brown and Roberts (2011).

Table 3.4.3: Effects of monthly household income on PA, odds ratios

(a) Pooled sample

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $\boldsymbol{s_{i,t}}$—**household gross labour income**					
$\ln(s_{i,t})$	1.1858***	1.0438*	1.0427*	1.0569*	1.1006**
	(0.0208)	(0.0185)	(0.0186)	(0.0282)	(0.0346)
$\Delta_{10\%} s_{i,t}$	1.0164	1.0041	1.0040	1.0053	1.0092
$\Delta s_{i,t}$ equiv. to GCSE → Deg. (%)	69.5232	9,514.6152	8,113.5217	1,303.8083	924.5735
Education *(ref: Degree)*					
Other higher degree	0.9044*	0.9048*	0.9125	0.8926*	0.8355**
	(0.0436)	(0.0436)	(0.0442)	(0.0488)	(0.0519)
A-level, etc.	1.0522	0.9060*	0.9115*	0.9266	0.8947*
	(0.0452)	(0.0385)	(0.0390)	(0.0450)	(0.0491)
GCSE, etc.	0.9140	0.8222***	0.8317***	0.8639**	0.8000***
	(0.0421)	(0.0377)	(0.0384)	(0.0465)	(0.0485)
Other qualification	0.8022**	0.7710***	0.7849***	0.8372*	0.7318**
	(0.0564)	(0.0539)	(0.0552)	(0.0710)	(0.0709)
No qualification	0.3955***	0.4694***	0.4794***	0.6077***	0.5201***
	(0.0386)	(0.0454)	(0.0466)	(0.0791)	(0.0792)
N	33,422	32,333	32,333	24,200	19,226
Panel B: $\boldsymbol{s_{i,t}}$—**household disposable income**					
$\ln(s_{i,t})$	1.2428***	1.1174***	1.1196***	1.0889*	1.1414**
	(0.0251)	(0.0220)	(0.0221)	(0.0382)	(0.0463)
$\Delta_{10\%} s_{i,t}$	1.0209	1.0106	1.0108	1.0082	1.0127
$\Delta s_{i,t}$ equiv. to GCSE → Deg. (%)	253.4481	1,719.9970	1,448.9729	451.5635	446.5411
Education *(ref: Degree)*					
Other higher degree	0.8542***	0.8796**	0.8876*	0.8910*	0.8334**
	(0.0399)	(0.0410)	(0.0416)	(0.0485)	(0.0517)
A-level, etc.	0.9458	0.8451***	0.8504***	0.9260	0.8918*
	(0.0394)	(0.0349)	(0.0353)	(0.0446)	(0.0487)
GCSE, etc.	0.7600***	0.7247***	0.7338***	0.8646**	0.7989***
	(0.0331)	(0.0314)	(0.0320)	(0.0462)	(0.0481)
Other qualification	0.5985***	0.6154***	0.6256***	0.8389*	0.7319**
	(0.0388)	(0.0396)	(0.0405)	(0.0709)	(0.0708)
No qualification	0.2431***	0.2947***	0.3007***	0.6071***	0.5181***
	(0.0196)	(0.0240)	(0.0246)	(0.0787)	(0.0786)
N	37,820	36,609	36,609	24,199	19,225
Year FE	No	No	Yes	Yes	Yes

(b) Male sample

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $s_{i,t}$—**household gross labour income**					
$\ln(s_{i,t})$	1.1734***	1.0706*	1.0683*	1.1078*	1.2279***
	(0.0340)	(0.0323)	(0.0327)	(0.0541)	(0.0714)
$\Delta_{10\%} s_{i,t}$	1.0154	1.0065	1.0063	1.0098	1.0198
Education *(ref: Degree)*					
Other higher degree	1.0300	0.9604	0.9649	1.0030	0.9192
	(0.0816)	(0.0774)	(0.0783)	(0.0905)	(0.0947)
A-level, etc.	1.2664***	1.0831	1.0843	1.1452	0.9976
	(0.0839)	(0.0730)	(0.0737)	(0.0878)	(0.0851)
GCSE, etc.	1.1937*	1.0610	1.0757	1.1130	0.9297
	(0.0854)	(0.0779)	(0.0796)	(0.0952)	(0.0907)
Other qualification	0.8793	0.8998	0.9115	1.0422	0.7576
	(0.0951)	(0.0988)	(0.1008)	(0.1382)	(0.1157)
No qualification	0.4812***	0.5805***	0.5954***	0.8229	0.5681*
	(0.0727)	(0.0886)	(0.0916)	(0.1608)	(0.1300)
N	14,881	14,281	14,281	10,599	8,435
Panel B: $s_{i,t}$—**household disposable income**					
$\ln(s_{i,t})$	1.2548***	1.1840***	1.1860***	1.1232	1.2604**
	(0.0425)	(0.0408)	(0.0410)	(0.0690)	(0.0916)
$\Delta_{10\%} s_{i,t}$	1.0219	1.0162	1.0164	1.0111	1.0223
Education *(ref: Degree)*					
Other higher degree	0.9797	0.9379	0.9432	0.9969	0.9115
	(0.0763)	(0.0741)	(0.0749)	(0.0898)	(0.0938)
A-level, etc.	1.1277	0.9969	0.9990	1.1349	0.9833
	(0.0734)	(0.0659)	(0.0665)	(0.0865)	(0.0836)
GCSE, etc.	0.9807	0.9188	0.9325	1.1026	0.9158
	(0.0680)	(0.0653)	(0.0666)	(0.0933)	(0.0888)
Other qualification	0.6791***	0.7291**	0.7395**	1.0325	0.7478
	(0.0697)	(0.0756)	(0.0771)	(0.1367)	(0.1142)
No qualification	0.2614***	0.3211***	0.3275***	0.8026	0.5475**
	(0.0339)	(0.0426)	(0.0438)	(0.1554)	(0.1248)
N	16,449	15,795	15,795	10,598	8,434
Year FE	No	No	Yes	Yes	Yes

(c) Female sample

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $s_{i,t}$—**household gross labour income**					
$\ln(s_{i,t})$	1.0895***	1.0201	1.0199	1.0218	1.0352
	(0.0227)	(0.0222)	(0.0223)	(0.0324)	(0.0386)
$\Delta_{10\%} s_{i,t}$	1.0082	1.0019	1.0019	1.0021	1.0033
Education *(ref: Degree)*					
Other higher degree	0.8545**	0.8528**	0.8618*	0.8209**	0.7775**
	(0.0502)	(0.0509)	(0.0518)	(0.0561)	(0.0605)
A-level, etc.	0.8146***	0.7839***	0.7892***	0.7851***	0.7854***
	(0.0444)	(0.0428)	(0.0433)	(0.0492)	(0.0567)
GCSE, etc.	0.6975***	0.6751***	0.6818***	0.7201***	0.6935***
	(0.0406)	(0.0398)	(0.0404)	(0.0501)	(0.0540)
Other qualification	0.6541***	0.6710***	0.6840***	0.7007**	0.6876**
	(0.0583)	(0.0603)	(0.0619)	(0.0767)	(0.0852)
No qualification	0.3169***	0.3969***	0.4036***	0.4906***	0.4834***
	(0.0390)	(0.0497)	(0.0508)	(0.0869)	(0.0991)
N	18,541	18,052	18,052	13,601	10,791
Panel B: $s_{i,t}$—**household disposable income**					
$\ln(s_{i,t})$	1.1447***	1.0833***	1.0858***	1.0615	1.0749
	(0.0272)	(0.0257)	(0.0259)	(0.0450)	(0.0523)
$\Delta_{10\%} s_{i,t}$	1.0130	1.0077	1.0079	1.0057	1.0069
Education *(ref: Degree)*					
Other higher degree	0.8174***	0.8314**	0.8405**	0.8235**	0.7801**
	(0.0463)	(0.0477)	(0.0485)	(0.0561)	(0.0605)
A-level, etc.	0.7561***	0.7477***	0.7526***	0.7908***	0.7897***
	(0.0397)	(0.0394)	(0.0399)	(0.0491)	(0.0565)
GCSE, etc.	0.6185***	0.6146***	0.6210***	0.7269***	0.6986***
	(0.0335)	(0.0337)	(0.0342)	(0.0501)	(0.0540)
Other qualification	0.5034***	0.5392***	0.5481***	0.7088**	0.6935**
	(0.0407)	(0.0439)	(0.0449)	(0.0773)	(0.0857)
No qualification	0.2303***	0.2810***	0.2858***	0.4982***	0.4898***
	(0.0229)	(0.0287)	(0.0294)	(0.0880)	(0.1001)
N	21,371	20,814	20,814	13,601	10,791
Year FE	No	No	Yes	Yes	Yes

[a] The table shows the odds ratios of education and monthly household income (household gross labour income in *Panel A* and household disposable income in *Panel B*) for the whole sample (Table 3.4.3a), the male sample (Table 3.4.3b), and the female sample (Table 3.4.3c). A value greater than 1 indicates higher odds of being in a higher physical activity category (from inactive → below guidelines → meets guidelines), while a value less than 1 indicates higher odds of being in a lower category.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] The corresponding estimated coefficients are in Table 3.4.1 and Table 3.4.2. Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, $p < 0.05$, * $p < 0.01$.

3.4.2.3 Policy Implications

These findings have crucial implications for policy design. First, although disposable income shows stronger effects than gross income on physical activity, income support policies that boost disposable income (like benefits and direct transfers) would have limited impact. The small magnitudes of odds ratios in Table 3.4.3 suggest what truly matters is not short-term income fluctuations, but rather a family's persistent social position—as evidenced by the large and significant effects of income quintiles in Table 3.6.4 in Appendix 3.6.3.1). In other words, temporary income support may be insufficient to shape physical activity behaviours. This finding aligns with research indicating that while income influences health outcomes, social position—linked to workplace autonomy and social participation—is a more powerful predictor of health behaviours. One example is that Black Americans who, despite having four times the purchasing power of men in Costa Rica, experience an eight-year shorter life expectancy (Marmot, 2003).

In contrast, education emerges as a powerful lever for promoting health equity. Even after controlling for income, university education demonstrates tremendous effects on physical activity levels. As Farrell et al. (2014) illustrated, education and income are separately associated with physical inactivity. According to Marmot (2003), the relationship between income and mortality is considerably lessened when researchers analyse income and education simultaneously as mortality predictors. This suggests the effects of psychological and cognitive factors surpass absolute income as determinants of PA. Therefore, while education may not directly address income inequality, it could be a more effective tool for reducing health disparities. Educational interventions may therefore offer a more promising path than income support for policymakers seeking to promote healthier lifestyles across socioeconomic groups.

3.4.3 Robustness Checks

I performed several robustness checks to validate our findings. Our main findings remain robust to both alternative household income measures (Appendix 3.6.3.2) and different specifications of physical activity (Appendix 3.6.3.3), consistently showing a modest yet significant effect of income alongside a strong educational impact on physical activity.

3.5 Discussions and Conclusions

This chapter examines how education and household (gross/disposable) income influence three physical activity (PA) levels among British adults: zero physical activity, engagement in at least some physical activity, and meeting WHO-recommended guidelines.

Understanding these distinct levels is crucial for three reasons. First, each level independently influences health outcomes. As Healy et al. (2007) demonstrated, even small increases in light-intensity PA were associated with improved (decreased) 2-hour plasma glucose levels and reduced risk of type 2 diabetes and cardiovascular disease, independent of moderate-to-vigorous PA levels. In addition, Matthews et al. (2012) clarified that high levels of MVPA may not fully counteract the detrimental effects of prolonged sedentary behaviour, as these factors shape health outcomes independently. Given the medical advice that health outcomes are dependent on achieving specific PA thresholds, understanding how income affects the attainment of these thresholds provides insight into a key mechanism through which health inequalities are generated, moving beyond simple income-health correlations to examine specific behavioural pathways. Second, different policy interventions target different dimensions of PA, necessitating a comprehensive understanding of each. Small-scale interventions primarily influence MVPA duration—for instance, motivational materials and tailored feedback have been shown to increase minutes/week of MVPA (Marcus et al., 2013), while community resource counselling showed modest effects on PA levels (Keyserling et al., 2008).[40] On the other hand, national-level policies like encouraging non-motorised modes of transportation and improving public facilities

40. See Michie et al. (2009) for a review of interventions which actively involve adults living in the community in cognition and behaviour change sessions.

(e.g., streets, public open spaces) mainly affect PA initiation but have limited impact on meeting PA guidelines (Guthold et al., 2018; Cerin and Leslie, 2008).[41] The inequitable distribution of these facilities may further exacerbate socioeconomic disparities in PA (Macintyre et al., 1993). Third, different socio-demographic groups respond variably across these dimensions. For example, women may engage in longer daily activity durations while being less likely to meet recommended guidelines (Pontin et al., 2021). Understanding which dimensions are most affected by socioeconomic inequality and to what extent within specific socioeconomic groups is therefore crucial for designing effective and targeted interventions.

Employing an ordered logit model, the analysis confirms that both education and income positively affect PA and the effects are statistically significant—more educated and affluent individuals are more likely to engage in physical activities and meet recommended PA guidelines. The education gradient is particularly pronounced. Individuals with GCSE-level education have odds of being in a higher PA category multiplied by 0.73–0.86 (a 14%–27% decrease) compared to those with degrees. Those without qualifications face steeper declines, with odds multiplied by over 0.50 relative to degree holders.

While confirming the general consensus that higher income and education correlate with increased PA, this study makes three distinct contributions. First, while the continuous income effects are statistically significant, their practical implications are modest given that a one-unit increase in the logarithm of total disposable income represents a substantial change that far exceeds typical income support policy interventions. To achieve the equivalent PA benefit of upgrading from GCSE to degree-level education would require a 446.54% increase in household disposable income—effectively eliminating income inequality, approximately equivalent to £133,962 over a 5-year period for a median earner in the lowest income quintile (the standard period required to complete high school/sixth form education and an undergraduate degree). Second, household disposable income demonstrates double the impact of gross income on PA—a one-unit increase in log disposable income is associated with 9%–24% higher odds of being in a higher PA category. This underscores both the importance of redistributive policies in shaping physical activity

41. Public education and sensitisation are the additional focuses for developing countries (Lachat et al., 2013).

behaviours and the critical role of income measure selection in empirical analysis. Third, gender heterogeneity emerges in physical activity disparities, with educational gradients exhibiting greater magnitude and statistical significance for females relative to males, whereas income effects manifest more prominently among males.

Therefore, while our analysis using income quintiles aligns with the broader literature, the continuous income provides deeper insights—highlighting both the importance of precise income measurement and demonstrating that education may be a more effective policy lever than financial incentives for promoting physical activity. Moreover, the stronger and more significant effect of education on women's physical activity, and their overall lower engagement in physical activity, suggest that education-focused policies should prioritise women when aiming to promote population-wide physical activity.

3.6 Appendix

3.6.1 Data Appendix

3.6.1.1 PA-Related Survey Questions

Table 3.6.1 enumerated all questions in the exercise module of Waves 7 and 9 of the UnSoc, which are used to construct the PA variable in Section 3.3.2.1.[42] These questions are based on the introduction—"*Think about the activities you do at work, as part of your house and gardening, to get from place to place, and in your spare time for recreation, exercise or sport*".

Table 3.6.1: PA-related survey questions (Waves 7 & 9)

Q#	Survey question	Range	Next question
Vigorous activity			
1	Now, think about all the vigorous activities which take hard physical effort that you did in the last 7 days. Vigorous activities make you breathe much harder than normal and may include heavy lifting, digging, aerobics, or fast bicycling. Think only about those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities?	$[0, 7]$	If $0 \rightarrow$ Q4 If $> 0 \rightarrow$ Q2
2	How much time did you usually spend doing vigorous physical activities on one of those days? (provide hours and minutes)	$[0, 15]$; $[0, 59]$	If answered $\rightarrow$ Q4 If not answered $\rightarrow$ Q3
3	How much time in total did you spend over the last 7 days doing vigorous physical activities? (provide hours and minutes)	$[0, 50]$; $[0, 59]$	Q4
Moderate activity			
4	Now think about activities which take moderate physical effort that you did in the last 7 days. Moderate physical activities make you breathe somewhat harder than normal and may include carrying light loads, bicycling at a regular pace, or doubles tennis. **Do not include walking.** Again, think only about those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities?	$[0, 7]$	If $0 \rightarrow$ Q7 If $> 0 \rightarrow$ Q5

Continued on next page

42. Source: https://www.understandingsociety.ac.uk/documentation/mainstage/questionnaire-modules/exercise_w7/.

Table 3.6.1 continued from previous page

Q#	Survey question	Range	Next question
5	How much time did you usually spend doing moderate physical activities on one of those days? (provide hours and minutes)	[0, 16]; [0, 59]	If answered → Q7 If not answered → Q6
6	How much time in total did you spend over the last 7 days doing moderate physical activities? (provide hours and minutes)	[0, 30]; [0, 50]	Q7
Walking			
7	Now think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure. During the last 7 days, on how many days did you walk for at least 10 minutes at a time?	[0, 7]	If 0 → Next section If > 0 → Q8
8	How much time did you usually spend walking on one of those days? (provide hours and minutes)	[0, 16]; [0, 59]	If answered → Next section If not answered → Q9
9	How much time in total did you spend over the last 7 days walking? (provide hours and minutes)	[0, 30]; [0, 50]	Next section

3.6.1.2 Demographic Controls

A set of demographic factors, which also influence physical activity, were included as control variables in the regressions.[43]

1. `` `w_age_dv' ``: age of the respondent squared at the time of the interview.
2. `` `w_sex_dv' ``: sex of the respondent.
3. `` `w_marstat' ``: marital status of the respondent.
4. `` `w_urban_dv' ``: a dummy for either urban or rural areas.
5. `` `w_gor_dv' ``: government office region (derived from the household's postcode).
6. `` `w_ethn_dv' ``: indicators for ethnic group of the respondent.[44]
7. `` `w_hhsize' ``: size of households.

43. The prefix `` `w' `` denotes different waves.
44. I reclassified this variable into three categories—British/Irish, Mixed, and Others.

3.6.2 Derivation of Odds Ratios in the Ordered Logit Model

Based on Equation 3.3.2, the probability of observing outcome k for individual i at year t is

$$\begin{aligned}\Pr\left(y_{i,t}=k|s_{i,t},w_{i,t},\mathbf{X}_{i,t};u_i\right)=&\Lambda\left(\mu_{k+1}-\beta_0-\beta_1\ln\left(s_{i,t}\right)-\beta_2 w_{i,t}-\mathbf{X}'_{i,t}\boldsymbol{\gamma}-u_i\right)\\&-\Lambda\left(\mu_k-\beta_0-\beta_1\ln\left(s_{i,t}\right)-\beta_2 w_{i,t}-\mathbf{X}'_{i,t}\boldsymbol{\gamma}-u_i\right),\end{aligned}$$

where $\Lambda(\cdot)$ is the cumulative logistic distribution function, $\mu_0=-\infty$, and $\mu_3=\infty$. $\beta_j, j=0,1,2$ are estimated coefficients (so $\exp(\beta_1)$ is the odds ratio).

Therefore, the odds of individual i in year t having a $y_{i,t}$ above category k relative to below or equal to k is

$$\begin{aligned}\text{Odds}\left(k;s_{i,t},w_{i,t},\mathbf{X}_{i,t};u_i\right)&\equiv\frac{\Pr\left(y_{i,t}>k|s_{i,t},w_{i,t},\mathbf{X}_{i,t};u_i\right)}{\Pr\left(y_{i,t}\leq k|s_{i,t},w_{i,t},\mathbf{X}_{i,t};u_i\right)}\\&=\exp\left(\beta_0+\beta_1\ln\left(s_{i,t}\right)+\beta_2 w_{i,t}+\mathbf{X}'_{i,t}\boldsymbol{\gamma}+u_i-\mu_k\right).\end{aligned}\tag{3.6.1}$$

Then, the odds ratio for two random values of income $s^1_{i,t}$ and $s^2_{i,t}$ is

$$\begin{aligned}\frac{\text{Odds}\left(k;s^2_{i,t},w_{i,t},\mathbf{X}_{i,t};u_i\right)}{\text{Odds}\left(k;s^1_{i,t},w_{i,t},\mathbf{X}_{i,t};u_i\right)}&=\frac{\exp\left(\beta_0+\beta_1\ln\left(s^2_{i,t}\right)+\beta_2 w_{i,t}+\mathbf{X}'_{i,t}\boldsymbol{\gamma}+u_i-\mu_k\right)}{\exp\left(\beta_0+\beta_1\ln\left(s^1_{i,t}\right)+\beta_2 w_{i,t}+\mathbf{X}'_{i,t}\boldsymbol{\gamma}+u_i-\mu_k\right)}\\&=\exp\left[\beta_1\left(\ln\left(s^2_{i,t}\right)-\ln\left(s^1_{i,t}\right)\right)\right]\\&=\exp\left[\beta_1\ln\left(\frac{s^2_{i,t}}{s^1_{i,t}}\right)\right].\end{aligned}\tag{3.6.2}$$

Therefore, when $\ln\left(s^2_{i,t}\right)-\ln\left(s^1_{i,t}\right)=1$ (one-unit increase in log income), the odds ratio is $\exp(\beta_1)$.

If $s^1_{i,t}$ increases by $p\%$ to $s^2_{i,t}$, $s^2_{i,t} = s^1_{i,t} + \frac{p}{100} s^1_{i,t} = s^1_{i,t}\left(1 + \frac{p}{100}\right)$, so $\frac{s^2_{i,t}}{s^1_{i,t}} = 1 + \frac{p}{100}$. Therefore,

$$\begin{aligned}
\frac{\text{Odds}\left(k; s^2_{i,t}, w_{i,t}, \mathbf{X}_{i,t}; u_i\right)}{\text{Odds}\left(k; s^1_{i,t}, w_{i,t}, \mathbf{X}_{i,t}; u_i\right)} &= \exp\left[\beta_1 \ln\left(\frac{s^2_{i,t}}{s^1_{i,t}}\right)\right] \\
&= \exp\left[\beta_1 \ln\left(1 + \frac{p}{100}\right)\right] \\
&= \exp\left[\ln\left(\exp\left(\beta_1\right)\right) \ln\left(1 + \frac{p}{100}\right)\right], \qquad (3.6.3)
\end{aligned}$$

where $\exp(\beta_1)$ is the odds ratio provided by Stata.

3.6.3 Additional Empirical Results

3.6.3.1 Effects of Discrete Income States

Our primary analysis employs continuous income measures to facilitate direct policy implications. However, to compare with the existing literature (see Table 3.2.2 and Section 3.2.4), which predominantly uses categorical income measures (such as groups, classes, or quintiles), I also present results using discrete income classifications. The continuous income variables were transformed into quintiles of equal size (Table 3.6.4).

Table 3.6.2: Effects of monthly household gross labour income quintiles on PA, ordered logit coefficients

Variable	(1)	(2)	(3)	(4)	(5)
Household gross labour income quintiles *(ref: s_{q_1})*					
s_{q_2}	0.6411***	0.6155***	0.6209***	0.1643	0.1718
	(0.0486)	(0.0493)	(0.0496)	(0.1036)	(0.1276)
s_{q_3}	0.7681***	0.6172***	0.6241***	0.1561	0.1407
	(0.0488)	(0.0493)	(0.0496)	(0.1033)	(0.1269)
s_{q_4}	0.8674***	0.6420***	0.6483***	0.1963	0.2048
	(0.0481)	(0.0491)	(0.0493)	(0.1036)	(0.1269)
s_{q_5}	0.9570***	0.6738***	0.6780***	0.2347*	0.3086*
	(0.0485)	(0.0503)	(0.0506)	(0.1060)	(0.1296)
Education *(ref: Degree)*					
Other higher degree	-0.0760	-0.0703	-0.0618	-0.0750	-0.1349*
	(0.0467)	(0.0465)	(0.0468)	(0.0544)	(0.0616)
A-level, etc.	-0.0080	-0.1439***	-0.1381***	-0.0732	-0.1062
	(0.0417)	(0.0415)	(0.0417)	(0.0486)	(0.0551)
GCSE, etc.	-0.1740***	-0.2618***	-0.2495***	-0.1430**	-0.2180***
	(0.0438)	(0.0439)	(0.0442)	(0.0539)	(0.0607)
Other qualification	-0.3790***	-0.4049***	-0.3886***	-0.1760*	-0.3101**
	(0.0642)	(0.0641)	(0.0645)	(0.0846)	(0.0967)
No qualification	-1.1324***	-1.0257***	-1.0054***	-0.4885***	-0.6500***
	(0.0802)	(0.0811)	(0.0816)	(0.1298)	(0.1516)
Gender *(ref: Male)*					
Female		-0.8280***	-0.8302***	-0.8720***	-0.8978***
		(0.0310)	(0.0312)	(0.0376)	(0.0423)
Age (squared)		-0.0001***	-0.0001***	-0.0001**	-0.0001*
		(0.0000)	(0.0000)	(0.0000)	(0.0000)
Urban/rural *(ref: Urban)*					
Rural		0.2564***	0.2574***	0.2056***	0.2099***
		(0.0373)	(0.0375)	(0.0441)	(0.0479)
Ethnicity *(ref: British/Irish)*					
Mixed		-0.1007	-0.0894	-0.1125	-0.0768
		(0.0713)	(0.0719)	(0.0871)	(0.1057)
Others		-0.7203***	-0.7048***	-0.6626***	-0.7299***
		(0.0456)	(0.0462)	(0.0573)	(0.0722)
Marital status *(ref: Single/never partnered)*					
Currently in a partnership		-0.0642	-0.0647	-0.1320**	-0.1138*
		(0.0391)	(0.0394)	(0.0472)	(0.0568)
Previously partnered		-0.0536	-0.0519	-0.0436	-0.0183
		(0.0528)	(0.0531)	(0.0648)	(0.0719)
Region *(ref: London)*					
North East		-0.3108***	-0.3201***	-0.0899	-0.0837
		(0.0899)	(0.0904)	(0.1086)	(0.1218)
North West		-0.2258***	-0.2343***	-0.1590*	-0.1360
		(0.0628)	(0.0632)	(0.0776)	(0.0921)

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Table 3.6.2 continued

Variable	(1)	(2)	(3)	(4)	(5)
Yorkshire and the Humber		-0.2217**	-0.2346***	-0.1427	-0.1701
		(0.0680)	(0.0683)	(0.0843)	(0.1013)
East Midlands		0.0408	0.0317	0.0515	0.0125
		(0.0714)	(0.0718)	(0.0854)	(0.1002)
West Midlands		0.0904	0.0867	0.0083	0.0359
		(0.0664)	(0.0668)	(0.0814)	(0.0984)
East of England		-0.0025	-0.0090	0.0045	0.0217
		(0.0672)	(0.0676)	(0.0803)	(0.0944)
South East		0.1552*	0.1501*	0.1478*	0.1240
		(0.0610)	(0.0614)	(0.0740)	(0.0883)
South West		0.1378*	0.1314	0.1423	0.1938*
		(0.0698)	(0.0702)	(0.0845)	(0.0978)
Wales		-0.1683*	-0.1937*	-0.0693	-0.1196
		(0.0783)	(0.0789)	(0.0943)	(0.1079)
Scotland		-0.0719	-0.0878	0.0194	0.0275
		(0.0715)	(0.0720)	(0.0861)	(0.0993)
Northern Ireland		-0.5044***	-0.5490***	-0.3432***	-0.3031**
		(0.0794)	(0.0806)	(0.0959)	(0.1104)
Log of household size		-0.0267	-0.0274	-0.0105	0.0113
		(0.0341)	(0.0343)	(0.0425)	(0.0505)
Health shock *(ref: Never had health shocks)*					
Experiencing health shocks		-0.6895***	-0.6930***	-0.7840***	-0.7216**
		(0.1578)	(0.1593)	(0.2197)	(0.2444)
Experienced health shocks before		-0.3808***	-0.3983***	-0.0244	-0.0694
		(0.0834)	(0.0840)	(0.1123)	(0.1195)
Journey to work *(ref: Inactive commute)*					
Active commute (walk/cycle)				0.4372***	0.4863***
				(0.0512)	(0.0592)
Physicality of job *(ref: Non-physical job)*[c]					
Fairly physical job					0.2158***
					(0.0609)
Physical job					0.5637***
					(0.0453)
Year FE	No	No	Yes	Yes	Yes
N	37,932	36,707	36,707	24,069	19,107

[a] The table shows the ordered logit/latent variable coefficients i.e., the effects of monthly household gross labour income quintiles on the *latent* level of energy expenditure, which underlies the observed three-category physical activity measure (inactive, below guidelines, meets guidelines). Household income is divided into five quintiles, from s_{q_1} (lowest) to s_{q_5} (highest). See Section 3.3.2.2 for details. Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] Data on job physicality is available in Waves 2 and 5 (2010–2015). Since our main analysis uses Waves 7 and 9 (2015–2019), we construct a time-invariant measure of job physicality for each individual by averaging their reported job physicality scores from Waves 2 and 5 (See Section 3.3.2.3.2 for details).

Table 3.6.3: Effects of monthly household disposable income quintiles on PA, ordered logit coefficients

Variable	(1)	(2)	(3)	(4)	(5)
Total household disposable income quintiles *(ref: s_{q_1})*					
s_{q_2}	0.1655***	0.0871	0.0936	0.0615	-0.0049
	(0.0472)	(0.0475)	(0.0478)	(0.0702)	(0.0829)
s_{q_3}	0.2757***	0.1092*	0.1187*	0.0240	-0.0173
	(0.0466)	(0.0471)	(0.0474)	(0.0677)	(0.0797)
s_{q_4}	0.4614***	0.2379***	0.2422***	0.0850	0.0413
	(0.0461)	(0.0469)	(0.0472)	(0.0673)	(0.0788)
s_{q_5}	0.5857***	0.3104***	0.3136***	0.1396*	0.1567
	(0.0465)	(0.0483)	(0.0485)	(0.0696)	(0.0816)
Education *(ref: Degree)*					
Other higher degree	-0.0950*	-0.0762	-0.0684	-0.0712	-0.1330*
	(0.0468)	(0.0466)	(0.0469)	(0.0542)	(0.0614)
A-level, etc.	-0.0285	-0.1524***	-0.1473***	-0.0707	-0.1081*
	(0.0418)	(0.0416)	(0.0418)	(0.0483)	(0.0547)
GCSE, etc.	-0.2240***	-0.2933***	-0.2819***	-0.1385**	-0.2187***
	(0.0439)	(0.0440)	(0.0442)	(0.0535)	(0.0602)
Other qualification	-0.4546***	-0.4502***	-0.4347***	-0.1693*	-0.3093**
	(0.0649)	(0.0647)	(0.0650)	(0.0844)	(0.0965)
No qualification	-1.3465***	-1.1913***	-1.1725***	-0.4989***	-0.6655***
	(0.0803)	(0.0814)	(0.0818)	(0.1295)	(0.1516)
Gender *(ref: Male)*					
Female		-0.8469***	-0.8488***	-0.8755***	-0.9051***
		(0.0313)	(0.0315)	(0.0375)	(0.0422)
Age (squared)		-0.0001***	-0.0001***	-0.0001**	-0.0001*
		(0.0000)	(0.0000)	(0.0000)	(0.0000)
Urban/rural *(ref: Urban)*					
Rural		0.2511***	0.2521***	0.2032***	0.2095***
		(0.0377)	(0.0379)	(0.0441)	(0.0479)
Ethnicity *(ref: British/Irish)*					
Mixed		-0.1034	-0.0918	-0.1116	-0.0798
		(0.0720)	(0.0725)	(0.0874)	(0.1059)
Others		-0.7399***	-0.7223***	-0.6630***	-0.7306***
		(0.0459)	(0.0466)	(0.0572)	(0.0721)
Marital status *(ref: Single/never partnered)*					
Currently in a partnership		-0.0357	-0.0357	-0.1307**	-0.1088
		(0.0392)	(0.0394)	(0.0469)	(0.0565)
Previously partnered		-0.0551	-0.0533	-0.0454	-0.0203
		(0.0533)	(0.0536)	(0.0648)	(0.0719)
Region *(ref: London)*					
North East		-0.2843**	-0.2935**	-0.0714	-0.0626
		(0.0909)	(0.0914)	(0.1087)	(0.1218)
North West		-0.2024**	-0.2105***	-0.1521	-0.1276
		(0.0632)	(0.0636)	(0.0777)	(0.0922)

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Table 3.6.3 continued

Variable	(1)	(2)	(3)	(4)	(5)
Yorkshire and the Humber		-0.1977**	-0.2093**	-0.1333	-0.1530
		(0.0686)	(0.0689)	(0.0843)	(0.1014)
East Midlands		0.0810	0.0722	0.0606	0.0262
		(0.0720)	(0.0724)	(0.0855)	(0.1004)
West Midlands		0.1109	0.1076	0.0083	0.0398
		(0.0668)	(0.0671)	(0.0814)	(0.0984)
East of England		0.0299	0.0238	0.0125	0.0368
		(0.0678)	(0.0681)	(0.0803)	(0.0944)
South East		0.1798**	0.1753**	0.1520*	0.1327
		(0.0615)	(0.0619)	(0.0741)	(0.0884)
South West		0.1640*	0.1578*	0.1463	0.2021*
		(0.0706)	(0.0710)	(0.0846)	(0.0979)
Wales		-0.1283	-0.1546	-0.0585	-0.1053
		(0.0794)	(0.0800)	(0.0946)	(0.1082)
Scotland		-0.0394	-0.0553	0.0266	0.0405
		(0.0722)	(0.0726)	(0.0862)	(0.0994)
Northern Ireland		-0.4944***	-0.5409***	-0.3377***	-0.2927**
		(0.0801)	(0.0813)	(0.0958)	(0.1103)
Log of household size		0.0190	0.0182	-0.0078	0.0098
		(0.0343)	(0.0345)	(0.0423)	(0.0504)
Health shock *(ref: Never had health shocks)*					
Experiencing health shocks		-0.7530***	-0.7562***	-0.8037***	-0.7471**
		(0.1577)	(0.1591)	(0.2190)	(0.2437)
Experienced health shocks before		-0.4510***	-0.4683***	-0.0335	-0.0781
		(0.0850)	(0.0855)	(0.1124)	(0.1197)
Journey to work *(ref: Inactive commute)*					
Active commute (walk/cycle)				0.4359***	0.4848***
				(0.0511)	(0.0592)
Physicality of job *(ref: Non-physical job)*[c]					
Fairly physical job					0.2207***
					(0.0608)
Physical job					0.5605***
					(0.0452)
Year FE	No	No	Yes	Yes	Yes
N	37,950	36,719	36,719	24,094	19,132

[a] The table shows the ordered logit/latent variable coefficients i.e., the effects of monthly household disposable income quintiles on the *latent* level of energy expenditure, which underlies the observed three-category physical activity measure (inactive, below guidelines, meets guidelines). Household income is divided into five quintiles, from s_{q_1} (lowest) to s_{q_5} (highest). See Section 3.3.2.2 for details. Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] Data on job physicality is available in Waves 2 and 5 (2010–2015). Since our main analysis uses Waves 7 and 9 (2015–2019), we construct a time-invariant measure of job physicality for each individual by averaging their reported job physicality scores from Waves 2 and 5 (See Section 3.3.2.3.2 for details).

Table 3.6.4: Effects of monthly household income quintiles on PA, odds ratios

(a) Pooled sample

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $s_{i,t}$—**household gross labour income**					
Household gross labour income quintiles *(ref:* s_{q_1}*)*					
s_{q_2}	1.8986***	1.8505***	1.8606***	1.1785	1.1874
	(0.0922)	(0.0913)	(0.0923)	(0.1221)	(0.1515)
s_{q_3}	2.1557***	1.8537***	1.8665***	1.1690	1.1511
	(0.1052)	(0.0915)	(0.0926)	(0.1208)	(0.1460)
s_{q_4}	2.3808***	1.9003***	1.9123***	1.2169	1.2273
	(0.1145)	(0.0933)	(0.0944)	(0.1261)	(0.1557)
s_{q_5}	2.6039***	1.9616***	1.9698***	1.2645*	1.3615*
	(0.1264)	(0.0987)	(0.0996)	(0.1340)	(0.1765)
Education *(ref: Degree)*					
Other higher degree	0.8985*	0.9069*	0.9150	0.8987	0.8435**
	(0.0419)	(0.0423)	(0.0430)	(0.0491)	(0.0525)
A-level, etc.	0.9921	0.8659***	0.8710***	0.9294	0.8993
	(0.0413)	(0.0359)	(0.0363)	(0.0452)	(0.0495)
GCSE, etc.	0.8403***	0.7697***	0.7792***	0.8668**	0.8042***
	(0.0368)	(0.0338)	(0.0344)	(0.0468)	(0.0488)
Other qualification	0.6845***	0.6671***	0.6780***	0.8386*	0.7334**
	(0.0439)	(0.0428)	(0.0437)	(0.0710)	(0.0709)
No qualification	0.3223***	0.3585***	0.3659***	0.6135***	0.5220***
	(0.0259)	(0.0291)	(0.0299)	(0.0796)	(0.0791)
N	37,932	36,707	36,707	24,069	19,107
Panel B: $s_{i,t}$—**household disposable income**					
Household disposable income quintiles *(ref:* s_{q_1}*)*					
s_{q_2}	1.1799***	1.0910	1.0981	1.0634	0.9951
	(0.0557)	(0.0518)	(0.0525)	(0.0746)	(0.0825)
s_{q_3}	1.3175***	1.1154*	1.1260*	1.0243	0.9828
	(0.0614)	(0.0525)	(0.0533)	(0.0694)	(0.0784)
s_{q_4}	1.5863***	1.2685***	1.2741***	1.0887	1.0421
	(0.0731)	(0.0595)	(0.0601)	(0.0733)	(0.0821)
s_{q_5}	1.7963***	1.3640***	1.3684***	1.1498*	1.1697
	(0.0835)	(0.0658)	(0.0664)	(0.0800)	(0.0955)
Education *(ref: Degree)*					
Other higher degree	0.8829**	0.9024*	0.9099*	0.9016	0.8456**
	(0.0412)	(0.0422)	(0.0428)	(0.0492)	(0.0525)
A-level, etc.	0.9719	0.8586***	0.8630***	0.9318	0.8975*
	(0.0406)	(0.0357)	(0.0361)	(0.0450)	(0.0491)
GCSE, etc.	0.7993***	0.7458***	0.7544***	0.8706**	0.8035***
	(0.0351)	(0.0328)	(0.0334)	(0.0465)	(0.0484)
Other qualification	0.6347***	0.6375***	0.6474***	0.8442*	0.7340**
	(0.0412)	(0.0412)	(0.0421)	(0.0713)	(0.0708)
No qualification	0.2602***	0.3038***	0.3096***	0.6072***	0.5140***
	(0.0209)	(0.0247)	(0.0253)	(0.0786)	(0.0779)
N	37,950	36,719	36,719	24,094	19,132
Year FE	No	No	Yes	Yes	Yes

(b) Male sample

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $\boldsymbol{s_{i,t}}$—**household gross labour income**					
Household gross labour income quintiles *(ref:* s_{q_1}*)*					
s_{q_2}	2.7815***	2.7299***	2.7515***	2.1653***	2.9495***
	(0.2339)	(0.2394)	(0.2428)	(0.4881)	(0.8532)
s_{q_3}	3.2097***	2.8976***	2.9423***	2.1952***	2.9003***
	(0.2637)	(0.2459)	(0.2512)	(0.4918)	(0.8327)
s_{q_4}	3.4205***	2.9265***	2.9527***	2.3443***	3.3004***
	(0.2721)	(0.2405)	(0.2441)	(0.5255)	(0.9476)
s_{q_5}	3.3946***	2.8527***	2.8660***	2.3448***	3.6796***
	(0.2713)	(0.2381)	(0.2405)	(0.5321)	(1.0682)
Education *(ref: Degree)*					
Other higher degree	0.8985*	0.9069*	0.9150	0.8987	0.8435**
	(0.0419)	(0.0423)	(0.0430)	(0.0491)	(0.0525)
A-level, etc.	1.1655*	1.0220	1.0231	1.1398	0.9976
	(0.0754)	(0.0674)	(0.0679)	(0.0877)	(0.0855)
GCSE, etc.	1.0532	0.9684	0.9814	1.1103	0.9272
	(0.0727)	(0.0688)	(0.0702)	(0.0952)	(0.0907)
Other qualification	0.7740*	0.7938*	0.8046*	1.0356	0.7584
	(0.0783)	(0.0816)	(0.0832)	(0.1372)	(0.1158)
No qualification	0.3839***	0.4352***	0.4441***	0.8663	0.5956*
	(0.0488)	(0.0568)	(0.0583)	(0.1696)	(0.1366)
N	16,459	15,796	15,796	10,526	8,366
Panel B: $\boldsymbol{s_{i,t}}$—**household disposable income**					
Household disposable income quintiles *(ref:* s_{q_1}*)*					
s_{q_2}	1.3150***	1.2566**	1.2710**	1.2325	1.1475
	(0.1066)	(0.1040)	(0.1058)	(0.1631)	(0.1876)
s_{q_3}	1.4985***	1.3275***	1.3514***	1.1686	1.1091
	(0.1193)	(0.1091)	(0.1118)	(0.1527)	(0.1774)
s_{q_4}	1.7276***	1.5001***	1.5125***	1.2250	1.2539
	(0.1340)	(0.1207)	(0.1225)	(0.1591)	(0.1995)
s_{q_5}	1.8849***	1.6288***	1.6343***	1.2952	1.4214*
	(0.1465)	(0.1340)	(0.1354)	(0.1725)	(0.2321)
Education *(ref: Degree)*					
Other higher degree	0.8829**	0.9024*	0.9099*	0.9016	0.8456**
	(0.0412)	(0.0422)	(0.0428)	(0.0492)	(0.0525)
A-level, etc.	1.1439*	1.0136	1.0139	1.1370	0.9868
	(0.0748)	(0.0675)	(0.0680)	(0.0870)	(0.0843)
GCSE, etc.	1.0172	0.9506	0.9623	1.1012	0.9161
	(0.0714)	(0.0685)	(0.0699)	(0.0937)	(0.0892)
Other qualification	0.7172**	0.7643*	0.7740*	1.0385	0.7534
	(0.0741)	(0.0800)	(0.0816)	(0.1375)	(0.1151)
No qualification	0.2849***	0.3440***	0.3501***	0.8035	0.5461**
	(0.0368)	(0.0457)	(0.0469)	(0.1560)	(0.1247)
N	16,476	15,808	15,808	10,549	8,392
Year FE	No	No	Yes	Yes	Yes

(c) Female sample

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $s_{i,t}$—**household gross labour income**					
Household gross labour income quintiles *(ref:* s_{q_1}*)*					
s_{q_2}	1.5003***	1.4975***	1.5030***	0.9687	0.9279
	(0.0868)	(0.0879)	(0.0887)	(0.1103)	(0.1283)
s_{q_3}	1.4874***	1.4050***	1.4108***	0.9395	0.8776
	(0.0880)	(0.0844)	(0.0853)	(0.1075)	(0.1215)
s_{q_4}	1.5832***	1.4360***	1.4432***	0.9473	0.8873
	(0.0940)	(0.0875)	(0.0885)	(0.1090)	(0.1232)
s_{q_5}	1.8234***	1.5668***	1.5738***	1.0316	1.0068
	(0.1097)	(0.0983)	(0.0992)	(0.1222)	(0.1437)
Education *(ref: Degree)*					
Other higher degree	0.8985*	0.9069*	0.9150	0.8987	0.8435**
	(0.0419)	(0.0423)	(0.0430)	(0.0491)	(0.0525)
A-level, etc.	0.7839***	0.7593***	0.7642***	0.7926***	0.7934**
	(0.0415)	(0.0404)	(0.0409)	(0.0497)	(0.0575)
GCSE, etc.	0.6649***	0.6420***	0.6488***	0.7242***	0.6977***
	(0.0366)	(0.0359)	(0.0365)	(0.0505)	(0.0545)
Other qualification	0.5500***	0.5690***	0.5785***	0.7042**	0.6903**
	(0.0444)	(0.0465)	(0.0475)	(0.0769)	(0.0854)
No qualification	0.2671***	0.3123***	0.3178***	0.4928***	0.4811***
	(0.0269)	(0.0322)	(0.0329)	(0.0870)	(0.0982)
N	21,473	20,911	20,911	13,543	10,741
Panel B: $s_{i,t}$—**household disposable income**					
Household disposable income quintiles *(ref:* s_{q_1}*)*					
s_{q_2}	1.0479	1.0161	1.0213	0.9873	0.9502
	(0.0595)	(0.0584)	(0.0591)	(0.0813)	(0.0910)
s_{q_3}	1.0935	1.0110	1.0182	0.9529	0.9261
	(0.0615)	(0.0578)	(0.0585)	(0.0754)	(0.0853)
s_{q_4}	1.2825***	1.1501*	1.1542*	1.0180	0.9408
	(0.0721)	(0.0662)	(0.0668)	(0.0800)	(0.0853)
s_{q_5}	1.4472***	1.2400***	1.2458***	1.0873	1.0670
	(0.0830)	(0.0739)	(0.0747)	(0.0890)	(0.1010)
Education *(ref: Degree)*					
Other higher degree	0.8829**	0.9024*	0.9099*	0.9016	0.8456**
	(0.0412)	(0.0422)	(0.0428)	(0.0492)	(0.0525)
A-level, etc.	0.7775***	0.7580***	0.7624***	0.7989***	0.7958**
	(0.0411)	(0.0403)	(0.0407)	(0.0497)	(0.0571)
GCSE, etc.	0.6451***	0.6281***	0.6343***	0.7356***	0.7050***
	(0.0353)	(0.0348)	(0.0354)	(0.0506)	(0.0544)
Other qualification	0.5254***	0.5511***	0.5599***	0.7124**	0.6924**
	(0.0424)	(0.0449)	(0.0459)	(0.0774)	(0.0852)
No qualification	0.2370***	0.2815***	0.2862***	0.4991***	0.4849***
	(0.0236)	(0.0288)	(0.0294)	(0.0879)	(0.0987)
N	21,474	20,911	20,911	13,545	10,740
Year FE	No	No	Yes	Yes	Yes

[a] The table shows the odds ratios of education and monthly household income quintiles (household gross labour income quintiles in *Panel A* and household disposable income quintiles in *Panel B*) for the whole sample (Table 3.6.4a), the male sample (Table 3.6.4b), and the female sample (Table 3.6.4c). Household income is divided into five quintiles, from s_{q_1} (lowest) to s_{q_5} (highest). See Section 3.3.2.2 for details. A value greater than 1 indicates higher odds of being in a higher physical activity category (from inactive → below guidelines → meets guidelines), while a value less than 1 indicates higher odds of being in a lower category.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from

3.6.3.2 Robustness to Household Income Measures

As illustrated in Section 3.3.2.2, I constructed total household disposable income by summing six components. This calculated measure shows minor discrepancies (affecting less than 1% of the sample) from the survey-provided variable `fimnnet_dv'. While our main analyses use our constructed measure, I verify robustness using the survey-provided variable. In addition, I test robustness using household income measures directly from the household response file (`hhresp'), rather than aggregating individual-level data. Specifically, I use total gross household labor income (`fihhmnlabgrs_dv') and total household disposable income without deducting transfers between family members (`fihhmnnet1_dv'), both equivalised using the modified OECD scale. See similar results in Table 3.6.5.

Table 3.6.5: Effects of monthly household income (provided by the UnSoc) on PA, odds ratios

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $s_{i,t}$—**household gross labour income**					
$\ln(s_{i,t})$	1.2321***	1.0993***	1.0951***	1.0944**	1.1331***
	(0.0236)	(0.0208)	(0.0208)	(0.0329)	(0.0407)
Education *(ref: Degree)*					
A-level, etc.	1.0350	0.9061*	0.9108*	0.9349	0.9079
	(0.0440)	(0.0380)	(0.0385)	(0.0454)	(0.0498)
GCSE, etc.	0.8887**	0.8207***	0.8298***	0.8672**	0.8040***
	(0.0402)	(0.0369)	(0.0375)	(0.0466)	(0.0487)
Other qualification	0.7380***	0.7382***	0.7501***	0.8546	0.7261***
	(0.0506)	(0.0501)	(0.0512)	(0.0722)	(0.0703)
No qualification	0.3442***	0.4282***	0.4357***	0.6062***	0.5206***
	(0.0316)	(0.0393)	(0.0402)	(0.0793)	(0.0795)
N	34,363	33,239	33,239	24,041	19,083
Panel B: $s_{i,t}$—**household disposable income**					
$\ln(s_{i,t})$	1.3603***	1.2307***	1.2291***	1.2238***	1.2962***
	(0.0370)	(0.0328)	(0.0328)	(0.0524)	(0.0640)
Education *(ref: Degree)*					
A-level, etc.	0.9597	0.8639***	0.8689***	0.9483	0.9214
	(0.0400)	(0.0357)	(0.0361)	(0.0457)	(0.0501)
GCSE, etc.	0.7688***	0.7416***	0.7503***	0.8838*	0.8211**
	(0.0335)	(0.0323)	(0.0329)	(0.0472)	(0.0494)
Other qualification	0.6064***	0.6352***	0.6447***	0.8703	0.7430**
	(0.0393)	(0.0409)	(0.0417)	(0.0734)	(0.0717)
No qualification	0.2448***	0.3044***	0.3102***	0.6196***	0.5345***
	(0.0196)	(0.0246)	(0.0252)	(0.0806)	(0.0812)
N	37,859	36,632	36,632	24,075	19,121
Year FE	No	No	Yes	Yes	Yes

[a] The table shows the odds ratios of education and monthly household income (household gross labour income in *Panel A* and household disposable income in *Panel B*) provided by the UnSoc (`hhresp`). A value greater than 1 indicates higher odds of being in a higher physical activity category (from inactive → below guidelines → meets guidelines), while a value less than 1 indicates higher odds of being in a lower category.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, $p < 0.05$, * $p < 0.01$.

3.6.3.3 Robustness to Physical Activity Measurement

In the main analysis, I assumed that an individual's maximum available leisure time, excluding physiological needs and work, is 10 hours per day (see Section 3.3.2.1). As a robustness check, I recalculate the physical activity measures using a more conservative estimate of 6 daily discretionary hours. The continuous variable remains a proportion ranging from 0 to 1, representing the share of available weekly time spent in vigorous or moderate activities. See results in Table 3.6.6.

In addition, our main analysis follows the WHO physical activity guidelines. I also conducted robustness check using an alternative threshold—at least 100 minutes of moderate-intensity physical activity, 50 minutes of vigorous-intensity physical activity, or an equivalent combination per week. See results in Table 3.6.7.

Table 3.6.6: Effects of monthly household income on PA (with alternative maximum available time), odds ratios

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $s_{i,t}$—**household gross labour income**					
$\ln(s_{i,t})$	1.1853***	1.0447*	1.0434*	1.0623*	1.0994**
	(0.0208)	(0.0187)	(0.0188)	(0.0285)	(0.0348)
Education *(ref: Degree)*					
A-level, etc.	1.0011	0.8705**	0.8758**	0.8913*	0.8711*
	(0.0430)	(0.0371)	(0.0376)	(0.0434)	(0.0480)
GCSE, etc.	0.8384***	0.7633***	0.7714***	0.7908***	0.7494***
	(0.0387)	(0.0352)	(0.0358)	(0.0429)	(0.0459)
Other qualification	0.7089***	0.6901***	0.7015***	0.7334***	0.6430***
	(0.0500)	(0.0487)	(0.0499)	(0.0630)	(0.0636)
No qualification	0.3612***	0.4300***	0.4385***	0.5723***	0.5019***
	(0.0353)	(0.0419)	(0.0430)	(0.0751)	(0.0770)
N	32,742	31,672	31,672	23,693	18,839
Panel B: $s_{i,t}$—**household disposable income**					
$\ln(s_{i,t})$	1.2419***	1.1195***	1.1215***	1.0940*	1.1408**
	(0.0249)	(0.0220)	(0.0221)	(0.0386)	(0.0466)
Education *(ref: Degree)*					
A-level, etc.	0.8989*	0.8112***	0.8162***	0.8901*	0.8686*
	(0.0372)	(0.0335)	(0.0339)	(0.0430)	(0.0476)
GCSE, etc.	0.7019***	0.6756***	0.6836***	0.7906***	0.7486***
	(0.0305)	(0.0294)	(0.0299)	(0.0425)	(0.0455)
Other qualification	0.5394***	0.5584***	0.5671***	0.7338***	0.6430***
	(0.0349)	(0.0361)	(0.0369)	(0.0629)	(0.0635)
No qualification	0.2284***	0.2769***	0.2822***	0.5705***	0.4998***
	(0.0183)	(0.0225)	(0.0230)	(0.0745)	(0.0765)
N	37,119	35,926	35,926	23,692	18,838
Year FE	No	No	Yes	Yes	Yes

[a] The table shows the odds ratios of education and monthly household income (household gross labour income in *Panel A* and household disposable income in *Panel B*). A value greater than 1 indicates higher odds of being in a higher physical activity category (from inactive → below guidelines → meets guidelines), while a value less than 1 indicates higher odds of being in a lower category. Instead of 10 hours per day, we assume that an individual's maximum available leisure time, excluding physiological needs and work, is 6 hours per day, above which observations are omitted, with observations above this threshold being omitted.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, $p < 0.05$, * $p < 0.01$.

Table 3.6.7: Effects of monthly household income on PA (with alternative recommended guideline), odds ratios

Variable	(1)	(2)	(3)	(4)	(5)
Panel A: $s_{i,t}$—**household gross labour income**					
$\ln(s_{i,t})$	1.2023***	1.0519**	1.0508**	1.0698*	1.1139***
	(0.0219)	(0.0195)	(0.0195)	(0.0297)	(0.0364)
Education *(ref: Degree)*					
A-level, etc.	0.9816	0.8424***	0.8468***	0.8646**	0.8642*
	(0.0440)	(0.0377)	(0.0380)	(0.0440)	(0.0498)
GCSE, etc.	0.8029***	0.7230***	0.7301***	0.7517***	0.7216***
	(0.0379)	(0.0341)	(0.0346)	(0.0414)	(0.0448)
Other qualification	0.7026***	0.6717***	0.6819***	0.7212***	0.6352***
	(0.0499)	(0.0475)	(0.0485)	(0.0614)	(0.0623)
No qualification	0.3456***	0.4107***	0.4189***	0.5269***	0.4757***
	(0.0337)	(0.0399)	(0.0408)	(0.0692)	(0.0728)
N	33,405	32,315	32,315	24,189	19,269
Panel B: $s_{i,t}$—**household disposable income**					
$\ln(s_{i,t})$	1.2724***	1.1380***	1.1392***	1.1198**	1.1842***
	(0.0262)	(0.0228)	(0.0229)	(0.0404)	(0.0493)
Education *(ref: Degree)*					
A-level, etc.	0.8864**	0.7895***	0.7938***	0.8661**	0.8652*
	(0.0386)	(0.0343)	(0.0346)	(0.0437)	(0.0496)
GCSE, etc.	0.6692***	0.6392***	0.6462***	0.7547***	0.7247***
	(0.0299)	(0.0286)	(0.0290)	(0.0411)	(0.0446)
Other qualification	0.5268***	0.5391***	0.5470***	0.7254***	0.6396***
	(0.0346)	(0.0352)	(0.0359)	(0.0615)	(0.0625)
No qualification	0.2118***	0.2569***	0.2617***	0.5289***	0.4780***
	(0.0171)	(0.0210)	(0.0215)	(0.0691)	(0.0728)
N	37,795	36,578	36,578	24,188	19,268
Year FE	No	No	Yes	Yes	Yes

[a] The table shows the odds ratios of education and monthly household income (household gross labour income in *Panel A* and household disposable income in *Panel B*). A value greater than 1 indicates higher odds of being in a higher physical activity category (from inactive → below guidelines → meets guidelines), while a value less than 1 indicates higher odds of being in a lower category. The highest category ("meets guidelines") is defined as engaging in at least 100 minutes of moderate-intensity physical activity, 50 minutes of vigorous-intensity physical activity, or an equivalent combination per week.

[b] Column (2) adds controls for demographic characteristics, health shocks, and education. Column (3) includes year fixed effects. Column (4) accounts for physical activity from commuting, and Column (5) accounts for physical activity from occupation, thus isolating the income effects specifically on leisure-time physical activity.

[c] Standard errors are clustered at the individual level and in parentheses; * $p < 0.10$, $p < 0.05$, * $p < 0.01$.

Conclusion

This thesis contributes to our understanding of health inequality. Chapters 1 and 2 reveal that the underlying socioeconomic inequality drove both the sigmoid pattern of mortality reduction during London's mortality transition period from the 1850s to the 1950s, and the pattern of mortality inequalities across London's constituent areas—specifically, the mortality transition saw a decades-long divergence in mortality rates across socioeconomic groups before eventual convergence, rather than immediate convergence. In the contemporary UK, Chapter 3 demonstrates systematic inequalities in physical activity between individuals of different educational attainments and household disposable income levels.

Chapter 1 models the unprecedented mortality decline in London from the 1850s to the 1950s and provides a possible explanation for the decline pattern. To systematically document the mortality dynamics in London across multiple measures—crude mortality, corrected death rate (adjusted for age and sex composition), infant mortality, and non-infant mortality—I constructed a novel dataset featuring annual, continuous data spanning over a century. The mortality observations reveal that London's mortality decline followed sigmoid dynamics. To characterise this pattern formally, I used a four-parameter logistic model. This model addresses a crucial gap in the literature by providing a mathematical description of the mortality declines during the period of mortality transitions, with the inflection point, around 1896, signalling the timing of the most rapid reduction. I then present a theoretical framework linking these sigmoid mortality dynamics to the diffusion of health technologies following the underlying log-normal socioeconomic distribution. In particular, the adoption pattern exhibits three phases: initial slow uptake among affluent groups in the upper tail of the socioeconomic distribution, accelerating adoption as technologies dispersed, and subsequent deceleration as most of the population had already adopted. This theoretical mechanism aligns with the observed mortality transition pat-

tern. The early phase corresponds to gradual mortality declines, the period of maximum adoption velocity coincides with the period around the inflection point in mortality reduction, after which both technology diffusion and mortality improvements decelerate until reaching a new equilibrium.

Chapter 2 extends the research of the mortality transition at the city level to constituent areas of London. I created a novel panel dataset by constructing consistent geographic boundaries over time, allowing me to track three mortality rates—crude mortality, infant mortality, and non-infant mortality—and measures of socioeconomic distribution across these areas. Having constructed sufficiently long time series, I demonstrate that a sigmoid pattern characterises the dynamics of mortality decline, which can be systematically captured by the logistic function developed in Chapter 1. This enables the identification of the heterogeneity in inflection points, revealing substantial variations in mortality dynamics across London's constituent areas—there can be a discrepancy of nearly forty years in the inflection points. The economic model of health progress diffusion presented in Chapter 1 explains these variations through differences in socioeconomic conditions. The model predicts that areas with higher initial socioeconomic conditions saw the most rapid falls in mortality earlier, probably due to earlier adoption of new health technologies. I then formally test this prediction by developing a statistical model that expresses the four mortality dynamic parameters as functions of socioeconomic inequality measures. The results yield negative and highly significant correlations between socioeconomic status and the timing of inflection points—for example, a 1% increase in the proportion of professionals within an area in 1861 is associated with a 1.473 years earlier occurrence of the inflection point—confirming the model's predictions. This finding reveals a "divergence-convergence" pattern of health inequality: areas with higher socioeconomic status experienced mortality improvements first, creating a temporary divergence that only closed once lower socioeconomic areas underwent their own mortality transitions.

Understanding the historical mortality transition in today's developed countries is crucial. First, it provides a valuable analytical framework for contemporary developing economies facing similar public health and infrastructural challenges (Banerjee and Duflo, 2007; Baisa et al., 2010; Galiani et al., 2009), particularly in their ongoing epidemiological

and demographic transitions (Omran, 1971). The historical experience of the developed areas suggests that developing economies may face heightened health inequalities during their transition periods, as differential access to emerging medical technologies and infrastructure improvements often correlates with socioeconomic status. Without concurrent policies addressing income disparities and healthcare access, these transitions risk amplifying existing health gradients. Second, it offers insights into how modern transformative technologies influence health and economic inequalities. The complex interplay between mortality reduction and economic growth observed historically illuminates how initial advantages in accessing health innovations can perpetuate and even exacerbate health disparities. Indeed, the substantial mortality improvements experienced during the historical transition, while broadly beneficial, appear to have contributed to persistent health inequalities throughout the twentieth century, as the benefits of medical and infrastructural advances were not uniformly distributed across socioeconomic strata.

In Chapter 3, I employed an ordered logit model to examine how education and household (gross/disposable) income influence physical activity (PA) levels among British working-age adults. First, while both education and income show positive, statistically significant effects on physical activity, their magnitudes differ substantially. Holding income constant, upgrading from GCSE-level to degree-level education multiplies the odds of being in a higher physical activity category by 1.25 (a 25% increase). However, to achieve the equivalent PA benefit while holding education constant would require a 446.54% rise in household disposable income—effectively eliminating income inequality. For a median earner in the lowest income quintile, this translates to approximately £133,962 over five years, the standard period required to complete high school/sixth form education and an undergraduate degree. This striking difference suggests the effects of psychological and cognitive factors surpass absolute income as determinants of PA. Therefore, while education may not directly address income inequality, it could be a more effective tool for reducing health disparities. Educational interventions may therefore offer a more promising path than income support for policymakers seeking to promote healthier lifestyles across socioeconomic groups. Second, disposable household income exhibits twice the magnitude of effect on physical activity compared to gross labour income, highlighting the

criticality of income measurement precision. Third, gender-specific disparities emerge in physical activity patterns, whereby education demonstrates more pronounced and statistically significant effects on females, and household income exhibits stronger effects on male physical activity.

There are a number of potential avenues for further research. The analysis of mortality transitions in Chapters 1 and 2 could be enriched by examining concurrent fertility dynamics, providing a more complete picture of demographic change during this period. In addition, to deepen our understanding of geographic influences on mortality, future work may investigate how specific public health interventions—such as sewage systems, clean water provision, and factories producing air pollution—affected the transmission of waterborne and airborne infectious diseases. Regarding Chapter 3, it is interesting to understand the role of income risk in generating health inequalities and thus the importance of social insurance policies to reduce health inequalities via risk mitigation. Future research may extend the analysis by computing and incorporating income shock measures, which would provide more granular insights into the dynamics of income uncertainty and health behaviours.

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