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Abstract

Poor housing conditions, and the negative effects of Household Air Pollution (HAP) in particular, remain one of the most pressing global public health challenges. While the association between poor housing and health has a long history, evidence of a direct link is lacking. In this paper, we examine a rare example of a public housing intervention in rural areas, namely the large-scale provision of high-quality housing in Ireland in the late 19th and early 20th centuries. We exploit a novel dataset of deaths-by-disease and deaths-by-age-and-sex over the period 1871–1919, to test the impact of the intervention on mortality. Our difference-in-difference estimates indicate that improved housing conditions reduced mortality by as much as 1 death per 1000. This effect is driven by reductions in deaths from respiratory diseases. We propose a likely mechanism that is consistent with the pattern of results we observe: a reduction in Household Air Pollution through improved housing quality and better ventilation. A cost-benefit analysis reveals that the scheme was a highly cost-effective intervention.

JEL Codes: N33, N93, Q53, O18, I14, J10
Keywords: Ireland, Labourers Acts, household air pollution, health transition, social housing, infectious disease

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1 Introduction

According to the World Health Organization (WHO), air pollution is one of the leading causes of premature deaths globally. The combination of Ambient (outdoor) Air Pollution and Household Air Pollution (HAP) is associated with almost 7 million premature deaths annually, with more than 3 million linked to HAP alone (World Health Organization 2024a; World Health Organization 2024b). Largely a product of indoor solid fuel burning for cooking and heating, inadequate ventilation and poor-quality buildings, HAP remains one of the most pressing global public health challenges in low and middle-income countries (World Health Organization 2024b). HAP is a leading risk factor for morbidity as well as mortality worldwide, with a recognized link to poor respiratory health and respiratory disease (Raju et al. 2020). Nearly half of all deaths from lower respiratory infection among children under five are due to the inhalation of particulate matter from HAP (World Health Organization 2024b). The health burden of HAP is striking, given these deaths are largely preventable and relatively simple interventions can be cost-effective (Das et al. 2021).

While the direct link between housing conditions and health – particularly via HAP – is a growing public health concern today, the association between poor housing and health has a long history. In the nineteenth century, there was near-unanimous agreement as to the need for better ventilation to remove 'foul air' from the indoor environment, improve health and fight disease (LaFay and Sampson 2024). Although largely based on miasma theory, a theory of disease transmission that would ultimately be replaced by modern germ theory, the focus on incorporating clean air and ventilation into building design on the grounds of public health persisted well into the twentieth century. Despite being based on a misunderstanding of the transmission mechanism of infectious disease, the focus on ventilation may have had unintended benefits.

A number of papers identify the detrimental effects of historical air pollution on health (e.g. Clay and Troesken 2011; Clay, Lewis, et al. 2018; Clay, Lewis, et al. 2024; Beach and Hanlon 2018; Hanlon 2020; Hanlon 2024; Schneider 2025). So far, this literature has examined cities, the industrial centers where pollution was most visible. However, in such settings, amid high levels of overall pollution, it becomes impossible to disentangle ambient from household air pollution. As such, we currently lack empirical evidence on how HAP affected health in the past. Meanwhile, recent work in development economics and public health has tested interventions, such as improved cookstoves, that lower HAP (e.g. Hanna et al. 2016; Bensch and Peters 2015; Mobarak et al. 2012). However, the relatively limited success of these interventions, especially over longer horizons, highlights both the limits of behavior-dependent household technologies and the need for structural solutions to ventilation and air quality.

In this paper, we leverage a unique historical policy intervention to identify the effect of improved housing quality on health over a long time horizon. Our setting is the large-scale

provision of social housing in Ireland under the *Labourers Acts* of the late nineteenth and early twentieth centuries. Aalen (1987) refers to this as the "first major public housing enterprise in the British Isles" while the US Bureau of Labor Statistics described it as "singularly effective" (Bureau of Labor Statistics 1915, p.337). By the end of the scheme, almost 50,000 cottages had been built for agricultural laborers in rural areas, providing roughly 250,000 of the poorest landless peasants (as much as 10% of the rural population) with significantly improved housing. The rural setting of this intervention has the unique advantage of isolating the effect of housing – and HAP – from other environmental factors. In an urban environment, health is subject to a profusion of other developments (e.g. electrification, industrial pollution, or waterworks) that are absent in this rural setting. As we document below, this plausibly exogenous shock to housing quality improved ventilation and sanitary conditions with the aim of improving population health. Indeed, these cottages were hailed by contemporary officials as a successful contributor to the fight against disease (Registrar General 1911; Registrar General 1912; Registrar General 1913; Registrar General 1914).

We assemble a new dataset of deaths by cause, district and year for Ireland's 'Poor Law Union' districts for the period 1871–1919. We also assemble series on deaths by age, sex, district and year. Combined with local government series and with detailed data on the construction of *Labourers Act* cottages, by district and year from 1883, this allows us to test the cottages' impact on mortality. We leverage variation in the timing and intensity of cottage construction across district-periods to estimate the causal effect of the improvements to housing quality. Specifically we utilize a difference-in-difference methodology to establish the effect on all-cause mortality. All-cause mortality decreased by as much as one death per 1000 in districts after the construction of cottages commenced. The richness of the deaths-by-cause and sex-age data allows us to illuminate the mechanism. We find that the impact of cottages is driven by a decline in deaths from communicable (infectious) diseases and in particular, respiratory diseases. Across the age-sex groups, the reduction in mortality is concentrated among children and older women. The pattern of deaths is consistent with the cottages significantly reducing ill-health through improved housing design, increased ventilation and a reduction in household air pollution from the burning of biomass (peat turf) in well-ventilated fire places. Together with the particularities of the historical setting, such as low ambient air pollution, lack of access to clean fuels, and household size being held constant, these results lend limited support to alternative causal channels. Last, the granularity of our deaths-by-age-sex data, paired with detailed life tables, enable us to calculate the quality adjusted life years (QALY) saved by the scheme. We estimate that each cottage saved 26 QALYs at a cost of £150 ($21,200 2020 USD). When benchmarked against contemporary GDP per capita figures (approximately £26 or $3,720 2020 USD), a cost per QALY of £5.75 ($813) makes this a highly cost-effective public health intervention according to commonly-applied WHO guidelines (Marseille et al. 2015). These findings

highlight the public health potential of structural housing reforms and demonstrate that investments in the built environment can generate durable population health benefits.

We contribute to a growing literature on the broader consequences of historical housing schemes, including work by Garside (2000), Morris (2001), and de Bromhead and Lyons (2023). Here, we give evidence on the health impacts of such schemes. The question of the importance of better housing conditions to improved health in the late nineteenth and early twentieth centuries links to a broader debate around the causes of the dramatic and sustained increase in life expectancy during this period. The mortality transition – the move from a high mortality to a low mortality regime – is one of the key stages of modern economic development. In higher-income countries, the most rapid phase of the health transition generally occurred in the late 19th and early 20th centuries. In the United States and the United Kingdom, life expectancy at birth rose from around 40 years in the mid 19th century, to around 50 by 1900 and around 60 by the 1920s (Arias and Xu 2020; ONS 2015). For many developing countries, the health transition has been relatively recent and rapid. In Eastern and South Eastern Asia, for example, life expectancy increased by 34 years between 1950 and 2021 (United Nations 2023). However, in other developing regions, progress has been modest and mortality remains relatively high.

Despite its importance, we are still uncertain as to how the mortality declines of the late nineteenth and early twentieth centuries were achieved, or how much can be attributed to public health interventions at the time, including housing regulations and provision (Costa 2015). Modern medical science was still in its infancy and can only have played a relatively minor role in driving the fall in mortality, notwithstanding some notable exceptions with respect to specific diseases (Szreter 1988; Ager, Hansen, et al. 2023). Other explanations include improved health and disease resistance resulting from better nutrition and rising incomes (McKeown 1976), public health interventions through training and information (Egedesø et al. 2020; Hoehn-Velasco 2018; Anderson et al. 2022), increased contact with medical professionals (Saaritsa et al. 2024; Bauernschuster et al. 2020) or large-scale infrastructure programs, such as water filtration and sewerage systems (Cutler and Miller 2005; Beach, Ferrie, et al. 2016; Alsan and Goldin 2019).

The growing literature assessing the impact of investments in public health infrastructure has focused on urban areas. The "urban penalty" of higher mortality rates and its decline over the late nineteenth and early twentieth centuries, is well documented and it has prompted the study of large-scale (and high-cost) interventions, especially water and sanitary schemes (Shaw-Taylor 2020; Gallardo-Albarrán 2020).[1] Although improvements in housing are noted, the effect of housing is not seldom identified directly (Ager, Feigenbaum, et al. 2024). This is despite the fact that in the mid-nineteenth century airborne diseases

[1] A useful recent survey of the 'Global Sanitary Revolution' is provided by Gallardo-Albarrán (2024). Recent research, however, casts some doubt as to the true extent of the urban penalty decline over this period and indeed the efficacy of some of these interventions (Feigenbaum et al. 2020; Anderson et al. 2022).

accounted for around 60 *per cent* of all deaths in England and Wales, being about twice as significant as water and food-borne diseases (Burnett 1991). While the focus on urban areas is understandable, in almost all countries at this point, including the United States, the majority of the population still lived outside of cities (Hoehn-Velasco 2019).[2] In recent research, and building on the earlier work of Higgs (1973), Hoehn-Velasco (2018; 2019) has directly examined the rural mortality decline in the United States in the early twentieth century. She finds that the rural mortality decline was driven by a fall in child mortality and that public health interventions, in the form of the establishment of County Health Departments, cannot explain overall mortality declines.

The results of our study indicate that large-scale capital interventions to improve public health can be effective in a rural setting, not just an urban one. It adds to our understanding of the drivers of the health transition and the importance of the link between poor housing conditions and health outcomes. Ager, Feigenbaum, et al. (2024) emphasis this link in a historical setting by leveraging reduced overcrowding in early-20th century US cities. However, in contrast to their study, we examine improvements to housing quality directly rather than a reductions in overcrowding.

This latter point has contemporary relevance for housing policy in countries of all income levels, where the link between housing conditions and health, and particularly the damaging effects of HAP, has been comprehensively set out by the World Health Organisation (World Health Organization 2018; World Health Organization 2021) in pursuit of UN Sustainable Development Goals (SDGs).[3] Here we contribute to the broad contemporary literature on the link between housing improvements and health outcomes (e.g. Krieger and Higgins 2002; Thomson et al. 2013; Rolfe et al. 2020). Our contribution to this literature relates to the long-term perspective our setting brings, as well as to a natural experiment separating housing improvements from higher income, which often confounds the analysis.

Our work is related to a range of studies on interventions designed to reduce pollution. In a literature that spans development economics and the health sciences, evidence on the efficacy of interventions to reduce household air pollution through stove usage is limited. Improved "non-traditional" stoves can lower particulate matter in the home but such benefits are often transient and do not scale (Hanna et al. 2016; McCracken et al. 2007; Mobarak et al. 2012). In randomized control trials (RCTs) in countries such as India and Senegal, initial reductions do not last, with little improvement in respiratory health (Hanna et al. 2016; Bensch and Peters 2015; Rosa et al. 2014). It appears that demand for stoves is low and price-sensitive, with adoption shaped by preferences, peer learning

[2] Only a small number of industrialised European countries, including the UK, Belgium and the Netherlands, had majority urban populations by 1900 (Ritchie et al. 2024).

[3] The World Health Organization (2021) links the issue of health and housing to several of the UN's SDGs, including SDGs 3 (Good health and well-being), 10 (Reduced inequality) and 11 (Sustainable cities and communities).

and framing (Mobarak et al. 2012; Bensch and Peters 2015; Miller and Mobarak 2015). Berkouwer and Dean (2026) highlight that reducing peak exposure may yield improvements but high ambient pollution dilutes the benefits. Central constraints include compliance and monitoring, based on lessons from national programs and bundle trials (Rosa et al. 2014; Nagel et al. 2016). In sum, the modern literature points to a challenge involving technology quality, consistent use and maintenance. This highlights the value of analysis using historical, observational contexts – and with a focus on structural housing features, rather than stand-alone devices. Our findings provides causal evidence on the long-term effect of improved household air quality at a time when regulations surrounding the prohibition of solid-fuel stoves in newly-built houses are the focus of public policy debate in many developed countries.

Our paper proceeds as follows. The next section provides the historical context to the housing scheme and describes the overall disease environment in Ireland over the late nineteenth and early twentieth centuries. Section 3 describes the data and the sources used to construct the dataset. Section 4 outlines our empirical approach. Section 5 reports our baseline results before discerning the mechanisms through which housing affected health outcomes. Section 6 tests the robustness of our results. The final section concludes and indicates some avenues for future research.

2 Historical Context

2.1 Disease and Mortality in Ireland

Mortality rates in Ireland in the late nineteenth and early twentieth century, as elsewhere, were high by modern standards. In the 1870s, Ireland faced a crude death rate of 18 per thousand, around three times the rate seen in the early 21st century (Pringle 2012). Comparatively however, Ireland's mortality rate was lower than other parts of the UK, reflecting in particular a combination of industrial under-development and, related, a less urbanized population (see Figure 1).[4] High rates of overall mortality coincided with high rates of infant mortality, particularly in urban areas. Infant mortality rates were around 100 per thousand in the 1890s, with infant deaths accounting for 21% of all deaths. The cities of Dublin and Belfast were as unhealthy as any in the United Kingdom at the turn of the twentieth century, with high infant mortality rates reflecting the well-recognized unsanitary conditions of urban environments (Walsh 2017).

In terms of cause of death, infectious diseases accounted for the majority of deaths over the period, with respiratory diseases such as tuberculosis (TB) and bronchitis among

[4] Walsh (2017) suggests another reason: less comprehensive registration of deaths (as well as births), meaning the true difference between Ireland and Britain would be smaller than measured. However the case for the use of these statistics and their reliability has been recently made by McLaughlin and Whelehan (2024).

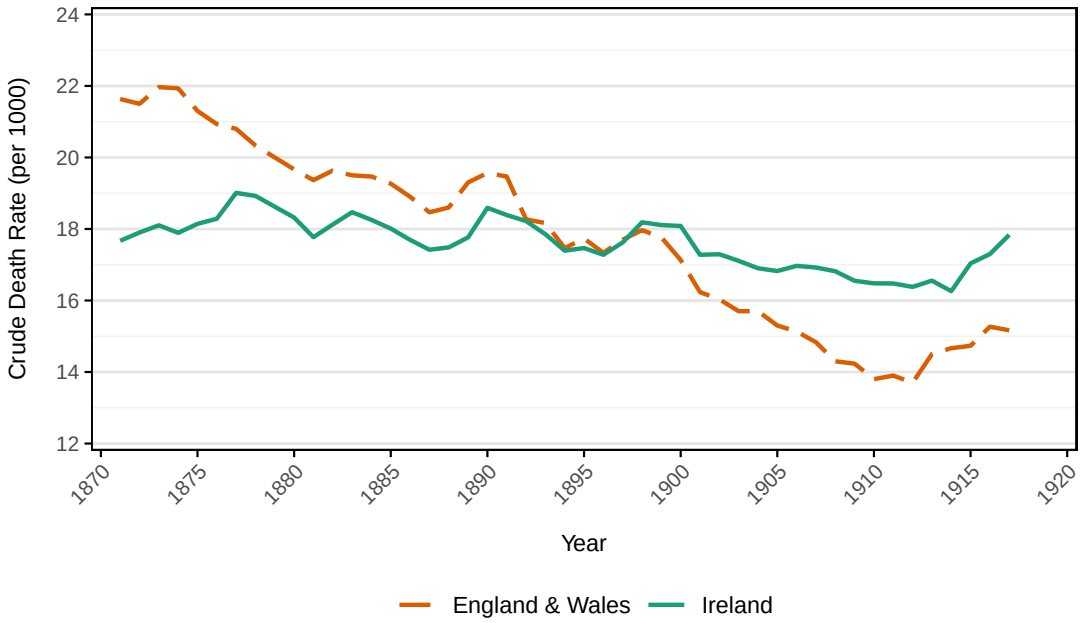


Figure 1: Crude Death Rates in Ireland vs. England and Wales.
Sources: 3-year moving average of crude death rate. *Sources:* England and Wales: Murphy and Dyson (1986). Ireland: Registrar General Reports (various years).

the leading causes of death (see Figure 2) (Pringle 2012). Unlike the rest of the UK however, Ireland's TB mortality rate failed to decline as rapidly as England and Scotland in the late nineteenth century, leaving it with a markedly higher rate of TB deaths by 1914: 2.1 per 1000 versus 1.4 and 1.6 per 1000 respectively (Geary 1930; Registrar General 1915). Indeed, the lack of a clear downward trend in all-cause mortality in Ireland in the final decades of the nineteenth century is in contrast to England and Wales, where mortality fell steadily from the 1860s until the First World War (Office of National Statistics 2021). It was not until the end of the twentieth century that mortality rates from infectious diseases in Ireland converged to those of England and Wales (Hall 2013).

Within Ireland, deaths also had a noticeable spatial pattern over this period. As elsewhere, the existence of an urban mortality penalty was apparent. 'Civic unions' (districts with towns of 10,000 or more) had death rates in the 1890s of 22 per thousand, compared to 17 in rural districts (Registrar General 1904). The gap was most pronounced for infectious and respiratory diseases in particular: 32% of all deaths in urban districts were attributed to TB and other respiratory diseases, compared to 25% in rural areas. Deaths due to bronchitis alone accounted for over 10% of all deaths in Ireland 1891-1900 (Registrar General 1904). Conversely, mortality rates from influenza – a category with an "alarming increase" towards the end of the nineteenth century – tended to be higher in rural districts than in urban areas (Registrar General 1904, p. 30).

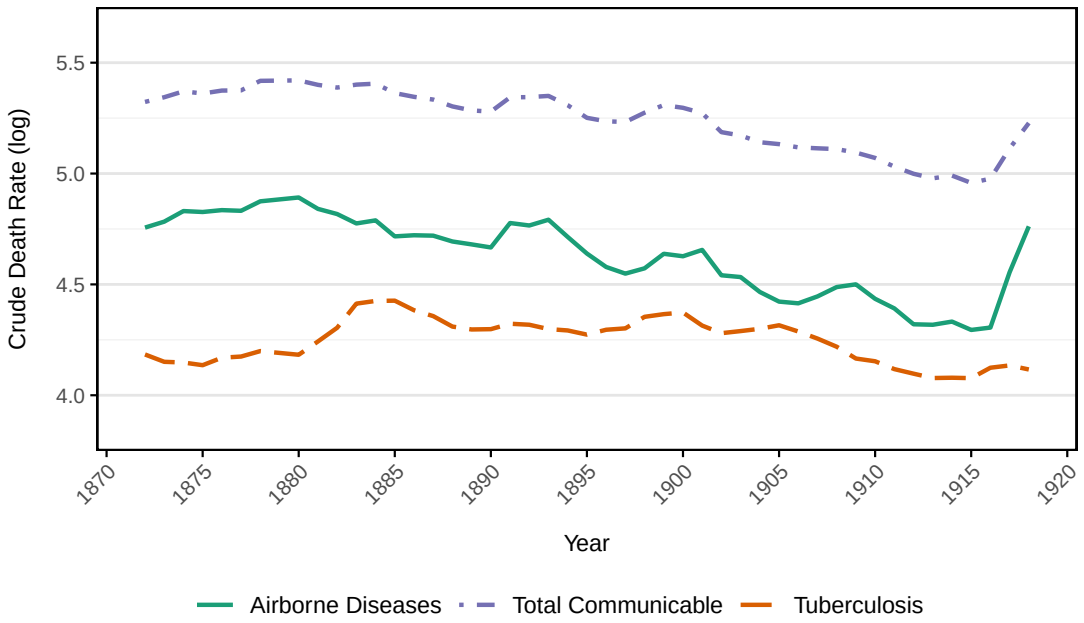


Figure 2: Crude Death Rates by Disease Category in Ireland.
Notes: Diseases are aggregated according to the classification described Table 1. Airborne diseases are reported without Tuberculosis. 3-year moving average of crude death rate. *Source:* Registrar General Reports (various years).

2.2 Housing Policy and The *Labourers Acts*

The roots of the *Labourers Acts* lie in the unique political and economic context of land and housing in Ireland in the late 19th century. Ireland and Britain entered a full political and economic union in 1801. For much of the 19th century, both before and after the Great Famine of the late 1840s, the quality of housing of most of rural Ireland was among the worst in Europe (Fraser 1996). The Great Famine had a dramatic effect on rural Ireland, not only through its immediate effect but also by starting a long period of population decline due to emigration. Ireland's rural population fell from roughly 7 million in 1841 to roughly 3 million by 1901. This was driven both by internal migration, to cities such as Dublin and Belfast, but in particular by international migration, especially to the United States or Britain. Landless laborers were among those most likely to emigrate.

Roughly three quarters of Ireland's population lived in rural areas at the turn of the 20th century (Fraser 1996). But emigration, in particular of the poorest, changed the prevalence of lowest-quality housing. In the 1841 Census, 44% of rural housing was classified as one room cabins made of sod or turf. By 1881, that share had fallen to 7%. Still, this meant that approximately 60,000 rural laborers and their families lived in the worst-quality housing. While the quality of urban housing was a more prevalent issue (Aalen 1987), it was politically less salient. By the late 19th century, The Irish Parliamentary Party (IPP)

emerged in the late 19th century as the dominant force of Irish nationalism and was committed to securing Home Rule (political autonomy) for Ireland. The IPP successfully used its position in parliament to extract concessions from the London-based government, including on issues relating to land and housing. Successive *Land Acts* financed the transfer of agricultural land in Ireland from landlords to their tenants: while only 3% of Irish farmers owned their land in 1870, by 1914 that share had grown to more than half (Fraser 1996; Guinnane and Miller 1997).

While tenant farmers were politically prominent, a large class of landless laborers also existed, making up roughly one quarter of rural workers in the 1880s. As they did not have farms, they could not benefit from the *Land Acts* and their situation may have been exacerbated by those Acts, as often landlords provided housing for laborers. Thus, a number of *Labourers Acts* followed the *Land Acts*, with similarly increasing ambition over time. The IPP did not control government and the willingness of the UK government to implement exclusively Irish schemes reflects the political context. In particular, the *Labourers Acts* were part of successive policies collectively referred to as 'constructive unionism', which aimed to reduce the demand for political autonomy by improving Ireland's economic and social condition (Aalen 1993). While the poor housing of rural labourers in parts of England and Wales was also recognised, the striking disparity between how the Irish rural poor were treated by public policy and how the rural and urban poor elsewhere in the UK were treated reflects the political reality of the time (Fraser 1996).

The first *Labourers Act* was enacted in 1883 and, while pioneering, was somewhat limited: twelve or more rate payers could apply to the Board of Guardians of a Poor Law Union, the responsible unit of local government at the time, to create a rural housing scheme, which in turn would apply to central government for a loan. Cottages with two or three rooms, and half an acre for subsistence farming, could be built by local authorities, with maximum rents set at approximately 1 shilling (£0.05) per week. Amendments in 1885 and 1886 improved loan terms and broadened the definition of eligible agricultural laborers, bringing an increase in building, albeit with uneven geographic take-up (Fraser 1996). Nonetheless, local authorities' loan repayments had to be paid fully out of local taxation. Given landlords still controlled local government, this limited the appetite for the original scheme.

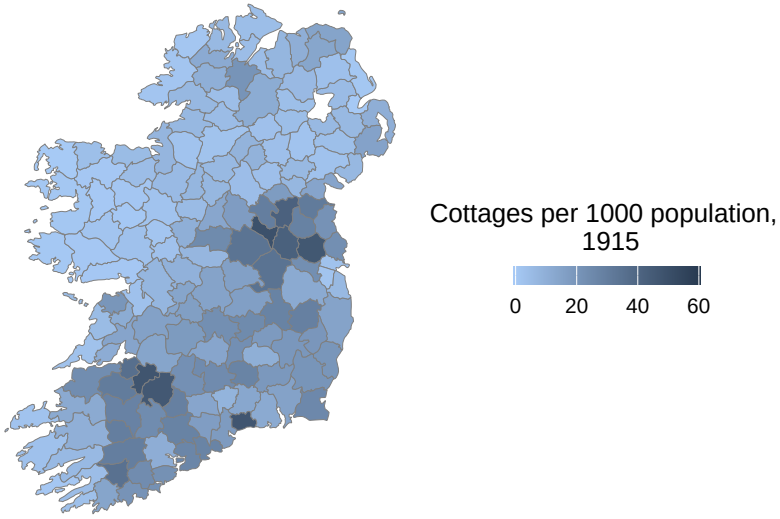


Figure 3: Map of Cottages Construction by 1915.

Source: Local Government Board Reports, UK Parliamentary Papers (various years).

The scheme grew over the 1890s and 1900s, due to three substantive changes. Firstly, direct subsidies for housing were provided, starting in 1891, in the *Land Act* of that year, albeit in a limited fashion. Secondly, the 1898 *Local Government Act* transformed the administration of local government in Ireland. Responsibility for administering the *Labourers Acts* now rested with newly-created Rural District Councils, whose members were elected by a significantly extended franchise that included women and laborers themselves. Together, these changes reduced, but did not eliminate, potential fiscal and electoral obstacles to take-up of the scheme.

The third major change happened with the 1906 *Labourers Act*, following IPP pressure on central government to match terms in the 1903 *Land Act*. In addition to broadening the definition of those eligible, the 1906 Act reduced the financial burden on Councils considerably, through two changes. Firstly, the monthly cost of interest was lowered, by reducing and extending the debt, from rate of interest of 4.5% over 50 years to 3.25% over 68.5 years. Secondly, central government would meet 36% of loan repayments. Cottages in Tipperary with a construction cost of £160 would, following the Act, see their cost to Councils fall from £7.20 annually to just £3.33 (Bureau of Labor Statistics 1915). In weekly terms, the new cost ($1s$ $3.4d$) was below the rent Councils could charge ($1s$ $7d$). The typical *Labourers Act* cottage rent of 1 shilling in 1911 was roughly half that prevailing in the open market, for a poorer quality home (Department of Agriculture and Technical Instruction 1911). Given agricultural laborer wages per a year of £25 to £30 in many counties, the share of income set aside for rent would have been as low as 10%. Given the new financial arrangements, building of cottages accelerated markedly after 1906, with over 26,000

cottages being built after 1906, compared to a little over 20,000 between 1884 and 1906 (McKay 1992; de Bromhead and Lyons 2023).

Improving the living conditions of agricultural workers was a primary aim of the policy. Although the intensity of building changed across subsequent schemes, the aim and the content of the policy remained the same. Despite falling in number since the 1840s in the wake of the Great Famine, the living conditions of the agricultural labourers that remained was viewed as a serious public health problem in the late 19th century. In the early 1890s, *The Royal Commission on Labour* examined the condition of Irish agricultural labourers specifically. Their inquiry, based on reports of conditions in Poor Law Unions across the country, painted a grim picture of the housing conditions of a typical agricultural labourer. While praising the early examples of newly built cottages under the Labourers Acts, general housing condition was deemed to be very poor. A medical officer commenting on the situation in the 1870s, recorded:

> The house accommodation of the labourers is simply wretched. Their houses are seldom fit to keep out rain, the floors are damp, the windows do not deserve the name, mere holes frequently stopped with rags, they have very rarely built chimney braces, and in nine cases out of 10 are filled with smoke every time a fire is put on. (Royal Commission on Labour 1893, p. 11)

Improving sanitary conditions drove the architectural design of the Laborers Act cottages. Indeed from the outset, a condition for providing new cottages was that existing cottages were deemed unfit for human habitation due to "the want of light, air, ventilation, or proper conveniences" (Local Government Board 1887, p. 110). Although there was some variation in design over time and space, the cottages were relatively practical, solidly built and of a recognizable design. Initially, the Local Government Board gave few requirements but called for a minimum of 3-4 rooms, a ratio of window size of one-twelfth of the floor area and the provision of an outdoor WC at least ten feet from the dwelling. Generally these cottages had a kitchen/living room with a central hearth, and bedrooms that connected onto this central room (Fraser 1996). By the early 1900s, cottage design had been more formally standardized. To this end, the LGB held an architectural competition, which provided to set of designs being distributed to local districts (Aalen 1986). A Local Government Board memorandum from 1908 provided the rationale for the designs, with ventilation central to combating disease (Local Government Board 1908, p.269):

> Ventilation is of the greatest importance, specifically in small houses, and markedly affects the incidence of some of the diseases above noted [pulmonary tuberculosis, bronchitis and other diseases of the respiratory organs]. A fireplace should therefore be provided in each room, not only for its primary purpose, but also because it is one of the most satisfactory means of ventilation.

Windows and doors too were carefully specified to provide adequate ventilation but also to "secure a good chimney draught". Even the most most modern ventilation technology was recommended with the goal of improving household air quality.[5]

3 Data

To investigate the relationship between cottage building and mortality, we construct a new dataset covering 158 Poor Law Unions across Ireland over the period 1871-1919. Poor Law Unions (PLUs) were an administrative division established in the 1830s with local responsibilities in health, housing, sanitation and poor relief. Following local government reforms in 1898, housing and public health related duties were transferred to newly-establish rural and urban district councils, which were nested within existing Poor Law Unions. For the purposes of general registration of births, deaths and marriages, which was established in Ireland in 1864, the country was divided into Superintendent Registrar's Districts (SRDs), which were consistent with Poor Law boundaries. To allow for consistent reporting, we employ PLUs as our unit of analysis throughout the period. Our data begin in the census year 1871 and continue until 1919, just prior to Irish independence, after which reporting units for vital statistics became less disaggregated. In this section, we describe in more detail the data employed in our analysis: firstly our treatment variable of interest, the *Labourers Act* cottages; secondly our outcomes of interest, death rates by cause (or sex-age group), district and year; and thirdly, our control variables.

3.1 Cottage Construction

Information on the number of cottages built in each district in each year is taken from all relevant years of the *Annual Report of the Local Government Board for Ireland* (LGB) and similar returns contained in British Parliamentary papers. Cottages are recorded from 1887, after the enactment of legislation in 1883, and end in 1915. The spatial patterns of cottage construction can be seen in Figure 3. We construct two treatment variables. The first is a continuous variable of the cumulative number of cottages constructed in each district. In our analysis, we standardize this variable to study the effect of cottages constructed per 1000 people. Additionally we use an absorbing treatment indicator that turns from zero to one in the year where the first cottages were constructed in a district.[6]

[5] The inclusion of Tobin tubes and Sheringham valves, early inlet ventilators, were also suggested by the Local Government Board.

[6] In Table A1 we demonstrate that our results are robust to alternative treatment variables, such as cottages constructed normalized by land area.

3.2 Mortality data

Mortality data are taken from tabulations of death certificates in the *Annual Report on Marriages, Births and Deaths in Ireland* by the Registrar General for Ireland. At the most disaggregated level, this source consistently reports annual total deaths, deaths by age and sex, and annual deaths for several causes in each PLU.[7] In addition to these mortality data, we also transcribed other relevant vital statistics – births by sex and marriages – by PLU and year.

Deaths by Cause At the PLU level, not all causes of death are reported consistently across the study period. In particular, more consistent series exist for cause of death due to communicable diseases. In the earliest years, the only non-communicable cause included is childbirth. However, following changes to the cause of death classification scheme in 1881, more non-communicable causes are included. More generally, however, the classifications within both communicable and non-communicable causes changes throughout, as medical understanding improved. To ensure consistent categories of disease classification over time, some aggregation of cause of death data was required. Table 1 classifies the causes of death we can identify consistently for most of our study period, and the higher levels of aggregation we use in our analysis. Our outcomes of interest, at the cause level, are death rates per 1000 population.

Deaths by Age and Sex Tabulations of deaths by age group and sex are available annually at the PLU level for our entire period, 1871–1919. We construct age-specific death rates for both sexes across each of seven age groups under-1, 1-4, 5-14, 15-24, 25-44, 45-64, and over 65. To build these outcomes of interest, we use age and sex structure at the district level and annual interpolated population from the census to calculate the number of people in each age-sex group.[8] Death rates for 1-4 year-olds are not reported separately but we exploit the reporting of both infant (under-1) and under-5 deaths. Specifically, 1-4 mortality is calculated mechanically by subtracting deaths under-1 from deaths under-5, and by subtracting those surviving to age 1 from the 0-4 projected group size.

3.3 Other Control Variables

Other information is also collected from LGB, such as overall receipts and expenditure at the district level, expenditure on medical officers and administration, and spending on poor relief. LGB reports are also a source for population and district valuation at the

[7] Henceforth we will use 'district' when when referring to PLUs and concurrent SRDs

[8] We use the 1901 age-sex structure since earlier age-sex structure has not been digitised at the PLU level. While a stationary age structure is a strong assumption, our results are robust to using the 1911 age-sex structure, or to using crude deaths by age-sex group. As such, local changes in the age structure are unlikely to color our results.

Table 1: Disease Aggregation

Communicable				
Airborne			***Waterborne***	***Other***
Respiratory[a]	*Tuberculosis*	*Certain Infectious*		
Unspecified Respiratory	Pulmonary TB	Whooping Cough	Cholera	Typhus
Bronchitis & Pneumonia	Other TB	Diphtheria	Diarrhea/Dysentery	
Influenza		Measles	Enteric	
		Scarlet Fever		
		Smallpox[b]		

Non-Communicable	**All Other**[c]
Cancer	Other Local Disease
Childbirth	Other Diseases
Local Urinary Diseases	Causes Ill-defined
Local Circulatory Diseases[c]	
Local Nervous Diseases[c]	
Local Digestive Diseases[c]	

Notes: Diseases were aggregated to have the largest number of consistently defined groups. The source for cause of death data is the tabulation "Deaths from Zymotic (Communicable) Disease and some other Causes" from the *Annual Report of the Registrar General for Ireland (1870-1919)*. As such we have more consistent data for communicable diseases (starting 1870) than for non-communicable disease (starting 1882).
[a] Throughout *unspecified respiratory* and *bronchitis & pneumonia* are combined to form one category to account for changes in registration.
[b] Smallpox is not included in the aggregate categories since it stopped being consistently reported after it was mostly eradicated in 1880s.
[c] These disease categories are not included in the aggregate category since they are not consistently available after 1882. Instead they are included in the **All Other** aggregate category.

PLU level. Population at the PLU level is available in the decennial census. We interpolate intervening years to obtain death rates. Further, to get a clearer picture of the pre-treatment standard of housing, a cross-section of data at the PLU level on the quality of housing, average household size, and the number of families per dwelling were collected from the 1881 census. Descriptive statistics for the main variables of our dataset are shown in Table 2.

4 Empirical Strategy

We begin our analysis by examining aggregate death rates and the evolution of cottage-building over time. Local medical officers, and other contemporaries noted the positive effect the construction of cottages, and the ensuing improvements to the housing conditions of the poor, had on the health of those benefiting (Registrar General 1912; House of Commons 1919). These anecdotes are supported by raw mortality trends. Figure 4 plots

Table 2: Summary Statistics

	Mean	SD	Min	Max
Death rate (All Cause)	16.32	3.08	6.22	44.23
Death rate (Communicable)	5.41	1.60	1.36	15.28
Death rate (Non-communicable)	1.43	0.52	0.20	3.85
Any Cottage (1/0)	0.47	0.50	0.00	1.00
Cottages Rate *per 1000*	4.37	7.84	0.00	49.47
Population share under-24	0.50	0.03	0.44	0.60
Population share 25–44	0.24	0.02	0.19	0.29
Population share over-45	0.26	0.02	0.15	0.30
Population (*000s*)	30.09	33.49	3.65	408.55
HH Size	5.37	0.55	3.38	8.30
Very-poor housing (share)	0.04	0.03	0.00	0.18
Poor housing (share)	0.42	0.16	0.02	0.86
Agricultural housing (share)	0.75	0.17	0.05	0.97
Population Density (*per* km^2)	73.94	172.29	11.38	2172.65
Distance to Dublin (*km*)	152.78	66.75	4.56	307.48
PLU Valuation (*000£*)	91.43	118.80	10.66	1740.00
PLU Spending (*000£*)	8.01	10.14	1.04	100.71
Medical Officers (*per 10,000*)	2.01	0.81	0.39	24.47
Dispensaries (*per 10,000*)	3.74	1.70	0.24	11.13
Workhouse Poor Relief (*per 10,000*)	824.78	713.21	43.70	7544.23
Number of Districts	158			
Number of Periods	17			

Notes: Table reports summary statistics for the full sample of districts across 17 periods. The first and last period are 2 years, encompassing 1871–1872 and 1918–1919 respectively, all other periods are 3 years. Death rates are expressed per 1,000 population. Death rates are the average annual rate across the period. Cottages are defined as the stock of cottages at the beginning of the period. Cottages Rate is scaled per 1,000 population. District population is reported in thousands and interpolated linearly for non-census years. PLU Valuation and Spending are reported in thousands of pounds. Other variables are defined as indicated in the text.

the average mortality rate for all causes, communicable diseases, non-communicable diseases and violent deaths across two groups of district: those that got their first cottage before 1900 and those that got their first cottage later.

The top-left panel shows that, compared to districts that got their first cottage later, early-treated districts had higher mortality rates in the 1870s. This gap persisted until the 1880s, shrinking thereafter with overall mortality rates by the final period effectively the same across early- and late-treated districts. This differential trend across early and late adopters is driven entirely by communicable diseases: as the top-right panel shows, a significant gap existed in the 1870s but had closed by the turn of the century. No similar trends are obvious in the two bottompanels, for deaths due to non-communicable and violent causes. The higher initial level is consistent with the known motivation of the program – areas of greatest need of cottages were likely to be the poorest and least healthy

among rural districts – while the closing of the gap represent initial evidence in favor of the hypothesis that the cottages had an impact on death rates.

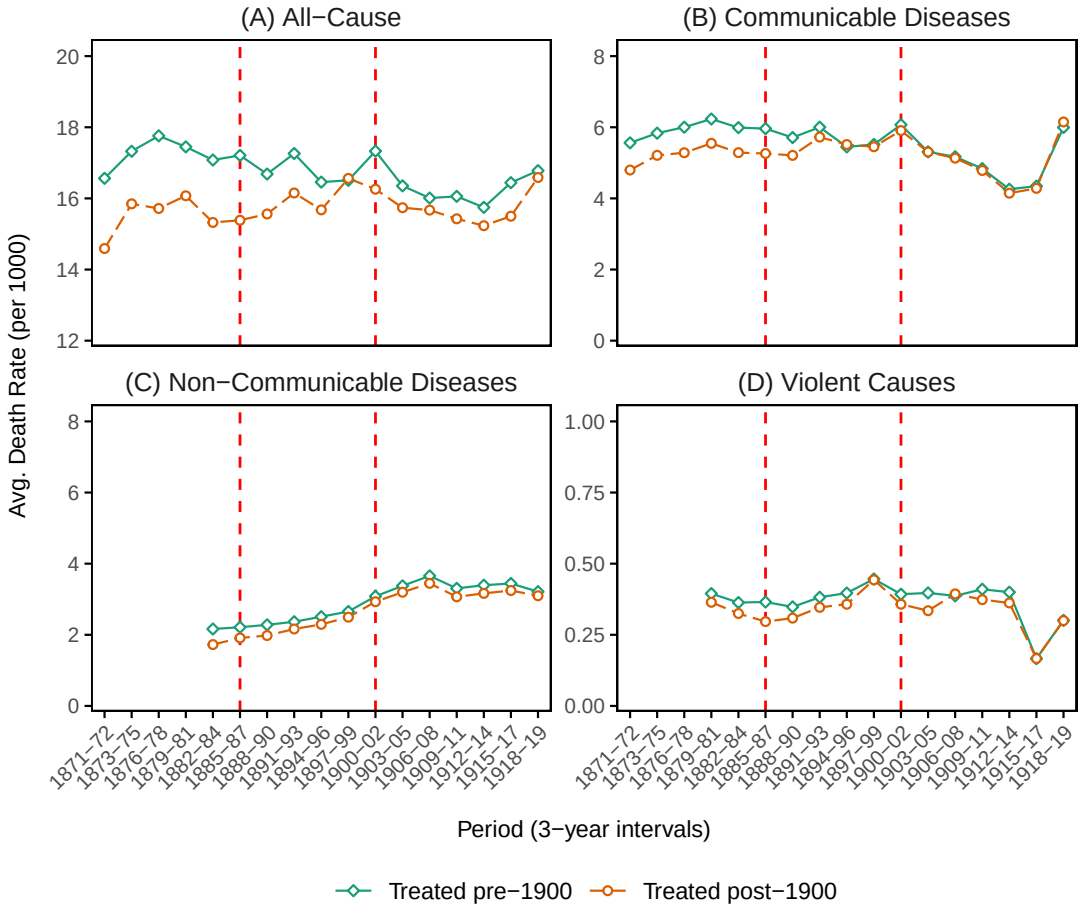


Figure 4: Average Mortality Rates across Districts by Treatment Timing.

Source: Registrar General Reports (various years).
Notes: This figure plots the average annual death rate from different cause-of-death groups – communicable, non-communicable disease, all-cause, and violent causes – for two groups of districts: early-treated districts where the first cottage was constructed prior to 1900 and late-treated districts that only built cottages after 1900.

Visual inspection of mortality trends, in other words, supports the hypothesis that the construction of cottages had positive health outcomes. Districts experiencing worst mortality from communicable diseases received cottages earlier and were subsequently able to converge to the remaining districts. Below, we describe the empirical strategy that we use to test whether these descriptive trends are reflective of more formal tests of causal effects of better housing quality – via the construction of cottages – on health. We leverage variation in the *timing* and the *intensity* of cottage construction to causally identify this effect, using two-way fixed effects (TWFE), heterogeneity robust DiD estimators (DID_l)

and triple difference specifications.

As described in section 3, our dataset is at the district (i) year (t) level. To account for noise in mortality outcomes, we collapse the data to a panel of three-year periods.[9]. For age and communicable causes, we have data for 17 periods (1871–1919), while for non-communicable causes we observe 13 periods (1882-1919). The treatment variable is calculated as the cottages constructed at the beginning of each period.[10] Throughout, our broad goal remains the same: we investigate whether mortality rates changed following the construction of cottages within districts.

4.1 Difference-in-Difference Estimation

In our baseline, we estimate the effect of cottage construction on mortality using identifying variation in the timing and intensity of treatment. We compare, first, districts where any cottages were built to those where none were built (*timing*) and, second, the extent of cottage building across districts over time (*intensity*). The first model estimates an *intention-to-treat* effect in a DiD model with staggered treatment timing. In the second model, when we leverage variation in the *intensity* of construction, we estimate the average marginal effect of building an additional cottage.[11] We estimate both models using the following two-way fixed-effects (TWFE) specification;

$$Y_{it} = \beta \cdot Cottages_{it} + \alpha_i + \pi_t + \gamma' \mathbf{X}_{it} + \epsilon_{it} \tag{1}$$

where Y_{it} is the mortality rate in district i and period t. Our baseline results investigate the effect of cottages on all-cause mortality. Subsequently we estimate the effect for component deaths rates, by cause-of-death j or age-sex s group. We use $Cottages_{it}$ to refer to, as appropriate, our binary absorbing treatment variable $AnyCottage_{it}$ or the continuous measure of cumulative construction $CottagesRate_{it}$. In all specifications, we include district α_i and period π_t fixed effects to account for unobserved heterogeneity. In the baseline, we take a minimalist approach and add no additional controls; however, our results are robust to including a vector of controls $\mathbf{X}_{it}$ that includes district finances, poor relief and medical provision, as well as spatial controls. We cluster standard errors at the district level.[12]

Under constant treatment effects estimating Equation 1 using OLS identifies the ATT. However, if the effect of cottage construction varies by period and district – which it likely does – TWFE is biased for staggered and/or continuous treatments (Goodman-Bacon

[9] All results are robust to using alternative periodization, to using annual data, and to the use of moving averages of the outcome variables. See Table A2.

[10] Our results replicate when using the average or the maximum number of cottages during a period. See Table A1.

[11] The two parameters should be related such that $ITT \approx mean(CottageRate) \times AME$.

[12] In the robustness section we demonstrate that our results are consistent across different levels of clustering.

2021; Chaisemartin and d'Haultfoeuille 2020; Chaisemartin and d'Haultfoeuille 2022). To account for this, our preferred estimation approach utilizes the heterogeneity-robust estimator DID_l as introduced in Chaisemartin and d'Haultfoeuille (2024). While most heterogeneity-robust estimators can deal with staggered treatment, DID_l is among the few that also allows for the non-binary staggered treatment in our setting.

Although DID_l is not estimated using regression, a representative regression equation is helpful to establishing the causal parameters we identify (Chaisemartin and d'Haultfoeuille 2024). We include the same fixed effect structure as in the TWFE model and standard errors are again clustered at the district level.

$$Y_{it} = \sum_{\tau=-l}^{n} \delta_\tau \cdot Cottages_{it} + \alpha_i + \pi_t + \epsilon_{it} \tag{2}$$

The estimator DID_l is constructed in a stepwise manner, beginning with comparisons at the group-period level and building up to estimates of average treatment effects. The starting point is $\delta_{i,\tau}$, the change in outcomes for district i that receives treatment at time k, compared to a set of control districts that had the same treatment status at time $k-1$ but have not yet experienced the treatment. This comparison is made over τ periods following the treatment switch. These district-level comparisons are then aggregated to form δ_τ, analogous to an event-study coefficient in dynamic difference-in-differences models. Specifically, δ_τ represents a weighted average of all the $\delta_{i,\tau}$ for a given period τ. However, because these averages do not adjust for the size or intensity of the treatment, they lack a direct structural interpretation.

To address this, Chaisemartin and d'Haultfoeuille (2024) propose a refinement, δ^n_τ: a normalized event-study coefficient that explicitly accounts for variation in treatment intensity across units. It represents the effect at period τ of receiving a standardized one-unit treatment τ periods ago, thereby recovering a more interpretable dynamic treatment effect. Finally, δ is a weighted average across all $\delta_{i,\tau}$, summarizing the overall average treatment effect on the treated (ATT). In the context of a continuous treatment, such as the number of cottages constructed, δ can be interpreted as the average causal response (ACR) to the construction of one additional cottage. While our primary focus is on this overall effect, the dynamic coefficients δ_τ and δ^n_τ are valuable for testing the main identifying assumption and for understanding how treatment effects evolve over time.

Identifying Assumptions Both TWFE and DID_l share the same identifying assumptions of no anticipation and parallel trends. No anticipation means that mortality rates should not preempt the construction of cottages, i.e. residents start living in a more healthily manner, in anticipation of receiving better housing. We believe that there is no reason to think that anticipation induced behavioural changes. Further, we can test this assumption using pre-trends. Parallel trends imply that mortality rates in districts where cottages

were constructed should have developed similarly to those where cottages were not yet built, had no cottages been constructed. While impossible to test directly, an absence of pre-trends suggests that this assumption is reasonable. For TWFE, we run lead and lag event-study regressions to test this assumption. For DID_l, we use *placebo* event-study effects δ_τ^{pl}, which make the same comparison as δ_τ but backwards in time.

4.2 Triple Difference

One potential concern with regards to identification is that our econometric specifications do not account for time-varying confounders at the district level. Although there is no evidence for other policy changes that may have affected mortality rates and were concurrent with the construction of cottages, other confounding factors may be unobserved. For example, changes to the age structure or the population composition – perhaps due to patterns of migration in late 19th century Ireland – could potentially confound our results.

Variation in our data in deaths-by-cause, and deaths-by-age/sex, allows us to apply further restrictions to our empirical model. The health literature predicts that improvements in housing conditions among the poorest (where the burden of non-infectious diseases is low) should have a differential impact on certain causes of death and age-sex groups. This pattern – specifically between communicable and non-communicable causes – is also evident in Figure 4. We leverage these differential impacts in triple-difference specification. The advantage of this approach is that we can non-parametrically control for time-varying confounders at the district level that affect both cause-of-death (age-sex) groups equally.

We stack our data twice, first by different cause-of-death groups and second by sex. This results in two three-dimensional panels indexed by district i, period t, and cause-of-death j or sex s. We run the following triple-difference model for different outcome groups:

$$Y_{itj/s} = \beta \cdot Cottages_{it} \cdot \mathbf{1}\{j/s = J/S\} + \rho_{ij} + \varphi_{tj} + \vartheta_{it} + \epsilon_{ijt} \tag{3}$$

where $Y_{itj/s}$ is the cause-of-death or age-sex group specific mortality rate. $\mathbf{1}\{j/s = J/S\}$ is an indicator variable that switches on if cause-of-death j or sex s correspond to the outcome group J or S. Since we are now leveraging the difference across groups within a district, we can include district-disease fixed effects (ρ_{ij}), period-disease fixed effects at the national level (φ_{jt}), and district-period fixed effects ϑ_{it}. As before, standard errors are clustered at the district level. We estimate this model using OLS. Given the aforementioned concerns regarding heterogeneous treatment effects, we are cautious when interpreting the magnitude of the effects. However, given the difference between TWFE and and DID_l observed in our baseline specifications above, the triple-difference estimates are likely biased towards zero. Thus, we remain confident in interpreting their direction and significance.

5 Analysis

5.1 Baseline

Table 3 summarizes the baseline results of our analysis, where we leverage variation in both the timing and intensity of treatment to estimate the effect of cottage building on all-cause mortality. Columns (1) and (2) report ITT effects and use the timing of first cottages built per district as the treatment. Columns (3) and (4) use the continuous measure of treatment: the number of cottages constructed per 1000 population. Columns (3) and (4) are our preferred specifications since they utilize the full extent of the identifying variation and capture a more relevant causal parameter; the average marginal effect per cottage.

As per Equation 1 and Equation 2 we estimate both using OLS (TWFE) and DID_l. Due to the bias of TWFE under heterogeneous treatment effects we are careful about interpreting the magnitude of the coefficients in columns (1) and (3). Still, so long as negative weights from "problematic" treatment comparisons – such as late-treated vs early-treated – do not overwhelm positive weights in the average treatment effect we can interpret their direction and significance. Negative weights bias the coefficient in the opposite direction to the actual effect, as such, if they are insufficient for a *sign-reversal*, TWFE will be biased towards zero (Chaisemartin and d'Haultfoeuille 2022).[13]. Since DID_l accounts for these "problematic" treatment comparisons our preferred estimates are found in columns (2) and (4).

The TWFE and DID_l results are consistent with this pattern across all regressions. We identify a consistent negative relationship between the construction of cottages and all-cause mortality. Since the TWFE estimates are likely biased towards zero we will focus on the coefficients in columns (2) and (4). The ITT effect in column (2) implies that annual all-cause mortality decreased by 1 death per 1000 in districts after cottage building commenced. For reference the average all-cause death rate across our sample is 16.32. Turning to column (4), the average marginal effect of one additional cottage per 1000 is -0.145 deaths per 1000. The average district had 9.27 cottages per 1000 across the post period. If we rescale the ITT effect by the average treatment dose we recover a marginal effect per cottage of $-1.000 \,/\, 9.269 = -0.108$. This serves to cross-validate the magnitude of the effect in column (4) – that two different sources of identifying variation yield such similar results increases our confidence in the baseline effect. The construction of ten laborers cottages, and the resulting improvement to housing conditions, reduced all-cause mortality by approximately one death per year.

To test the identifying assumptions and understand the dynamics of the effect, Figure 5

[13] We can retrieve the sum of positive and negative weights using the `twowayfeweights` package (Chaisemartin and d'Haultfoeuille 2020). Over 75 % of individuals ATTs receive positive weights summing to 1.21, while the negative weights sum to -0.21.

Table 3: Impact of Cottages on Mortality

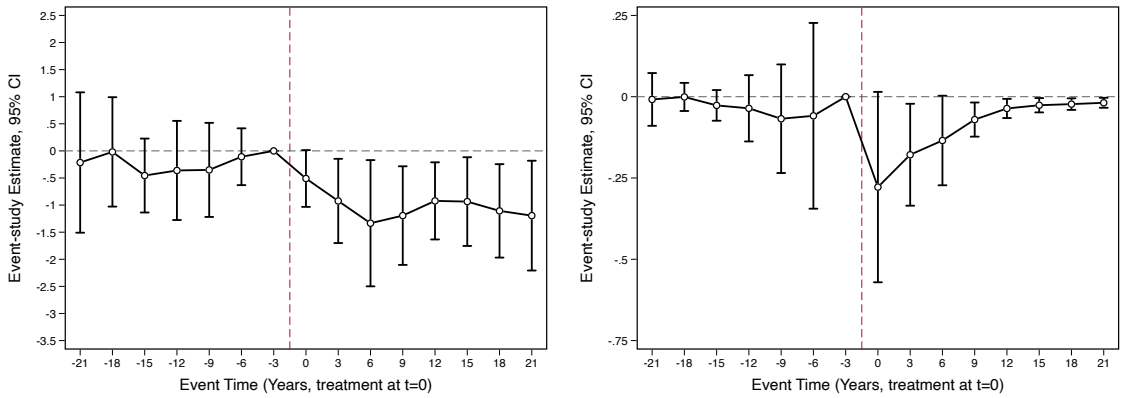
Dependent Variable:	Death Rate per 1000 (All-cause)			
Identifying Variation:	Timing		Timing & Intensity	
	(1) TWFE	(2) DID_L	(3) TWFE	(4) DID_L
β^{TWFE}: Cottages	-0.444***		-0.042***	
	(0.169)		(0.015)	
ATT: Cottages		-1.000***		-0.145***
		(0.369)		(0.054)
Observations	2653	2653	2653	2653
Estimation Groups		1468		1468
Estimation Switchers		1008		1008
Period FE	✓	✓	✓	✓
District FE	✓	✓	✓	✓
Clusters	Districts (158)			

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports our baseline DiD estimates for the effect of cottage construction on all-cause mortality. The unit of analysis are district-periods. The dependent variable is the average annual death rate per 1000 population from all-causes across the period. In column (1) and (2) the treatment variable is a indicator variable equal to one if any cottages were constructed in the district-period, these columns capture the *intention to treat effect* of any cottage construction. In columns (3) and (4) the treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period, capturing the average marginal effect of constructing an additional cottage. In columns (1) and (3) we estimate these effects using OLS in a canonical TWFE model, in columns (2) and (4) we use the heterogeneity robust estimator (DID_L) introduced by (Chaisemartin and d'Haultfoeuille 2024). DID_L is estimated over eight post periods. The reported effect from DID_L estimation is the *average total effect per treatment unit* equivalent to an ATT. Groups are the number of observations that are used in the estimation of the effect. Switchers are the number of treatment switches across these groups. All regressions include district and period fixed effects. The sample period is 1871-1919.

shows event study plots corresponding to the estimation in Column (4) of Table 3.[14] Panel (A) plots non-normalized event-study and placebo effects. The placebo effects (pre-periods) test the identifying assumptions of the DID_l estimator (Chaisemartin and d'Haultfoeuille 2024). All placebo effects are jointly equal to zero (*p-value* = 0.87) consistent with the identifying assumptions.

Turning to the effects dynamics, in panel (A) we see that the coefficient grows over the first six years post-treatment before stabilizing around -1. Panel (B) plots normalized event-study effects that are weighted by the treatment dose each district received up to period l. These can be interpreted as the contemporaneous effect in period l of having been

[14] we also estimate a leads-and-lags specification of the TWFE model in Column (1) of Table 3. The pre-treatment coefficients are jointly indistinguishable from zero, supporting the parallel trends assumption (see Figure A1).



(a) *Event-study effect* (b) *Normalized event-study effect*

Figure 5: Event-study effects: Cottages constructed per 1000 on all-cause mortality.

Notes: The figures plot event-study effects estimated using the heterogeneity robust DiD estimator DID_l by Chaisemartin and d'Haultfoeuille (2024). All estimates are relative to time period 0, also known as $t = -1$. The treatment variable is CottagesRate. Panel (a) plots non-normalized event-study effects δ and placebo effects δ^{pl}, whereas panel (b) plots normalized event-study effects δ_n. Normalized event study effect are weighted by the total treatment dose received up to period l. Both regressions use an balanced panel of districts across the period 1871–1919. Bars indicate 95% confidence intervals calculated using clustered standard errors at the district level.

treated τ periods ago. We observe that the construction of cottages has the greatest impact in the years immediately after treatment. This effect shrinks over time and approaches zero after 12 years. This implies that the decrease in all-cause mortality brought about by the cottages set in rapidly and improved the level of health in the district in a manner consistent with a one-time public investment yielding lasting but not indefinitely growing gains.

5.2 Mechanism

The baseline analysis in the previous section indicates that cottage building, and the corresponding improvements to housing quality, reduced all-cause mortality by as much as 6.3 *per cent* and that the impact of improved housing on health was relatively rapid. This result is consistent with improved housing reducing the negative health effects of poor housing conditions. To explore potential mechanisms in more detail, we exploit the richness of our data and split deaths from communicable (and non-communicable) disease into sub-categories based on mode of transmission, as shown in Table 1. As will become clear, our results are highly-supportive of a reduction in Household Air Pollution (HAP) being a key mechanism.

Household Air Pollution Before presenting empirical tests of the mechanism, it is worth setting out the forms of pollution relevant to improved housing quality in our setting. The combustion of fossil fuels releases a mix of tiny particles and toxic chemicals, with the three most important types of pollution in this context being sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and particulate matter (PM) (Meetham et al. 1984). NO_2 is emitted primarily from the combustion of gasoline, a fuel with little relevance in our context. With respect to SO_2 and PM; SO_2 is released as a byproduct of burning coal, while PM – suspended ash, soot, and carbon molecules – are released during the burning of all fossil fuels. The intensity of PM emission depends on the completeness of combustion, the fuel source used, and the site of combustion. The health impact of PM depends on the size of the particulates. At less than 10 μm (PM_{10}) particulate penetrates the lungs, placing a heavy burden on the respiratory system. Smaller particulates ($PM_{2.5}$) have even broader health impacts. They can enter the bloodstream, and hence affect other organ systems. Improved ventilation contributes to dispersing and removing pollutants from the household, reducing exposure to both PM_{10}, $PM_{2.5}$, and SO_2. Additionally improvements to chimneys and hearths would lead to more complete combustion, reducing the amount of PM emitted in the first place. Structural solutions to ventilation and air quality – such as the cottages built under the laborers acts – are a treatment package that significantly reduces household exposure to pollution.

Testing the Mechanism We next exploit the richness of our data to empirically investigate HAP as the principal mechanism through which housing improvements affected mortality. Table 4 presents the DID_l results, using the baseline specification in Table 3, for each sub-category of cause of death, with communicable disease divided into airborne, waterborne and typhus (insect-to-human) transmission.[15][16] The results reveal that the relationship between cottages and reduced mortality is driven almost entirely by a reduction in deaths from airborne disease. It is not linked with non-communicable, waterborne or insect-borne disease. This is strongly supportive of the hypothesis that improvements in living conditions within the house played the primary role, rather than improvements to water, sewerage or other sanitary conditions.

The detail contained in the Registrar General reports allows us to go further and look at more specific disease categories. Figure 6 shows ATT estimates, from DID_l, *within* the sub-categories in Table 4. Within airborne disease it is respiratory tract diseases, such as bronchitis and pneumonia, that are most strongly associated with cottage building.

[15]To avoid compositional bias, we use the `same_switchers` option and restrict estimation to districts for which all event-study coefficients can be estimated. This is particularly important for communicable diseases, where mortality is often spatially clustered in specific district-years. Using all groups in estimation has no significant effect on the pattern or magnitude of the results (see Table A3).

[16]We only report results for our preferred estimation method – heterogeneity robust DiD – but the pattern of the results replicate when using TWFE (unreported).

Table 4: Effect by Disease Category

	Disease Category specific Death Rate					
	Communicable				*Non-Com.*	*Other*
	Total	Airborne	Waterborne	Typhus		
	(1)	(2)	(3)	(4)	(5)	(6)
ATT: $CottagesRate_{it}$	-0.079***	-0.076***	-0.002	-0.002	0.001	-0.033*
	(0.020)	(0.020)	(0.002)	(0.002)	(0.006)	(0.018)
Mean DV	5.407	5.015	0.323	0.061	1.434	9.080
AME (%)	-1.469	-1.506	-0.572	-3.420	0.096	-0.358
Groups	1230	1230	1230	1230	1230	1230
Switchers	760	760	760	760	760	760
Period FE	✓	✓	✓	✓	✓	✓
District FE	✓	✓	✓	✓	✓	✓
Clusters	Districts (158)					
Years	1871-1919				1882-1919	1871-1919

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports DiD estimates for the effect of cottage construction on mortality from different disease categories. The unit of analysis are district-periods. The dependent variable is the average annual disease category specific death rate per 1000 population across the period. Columns (1) to (4) report the effects for different communicable diseases, starting with total deaths, followed by airborne, waterborne and insect-borne (i.e. Typhus). Column (5) reports the effect for non-communicable diseases, and column (6) for other cause-of-death categories. For more details on the disease contained in each category see Table 1.The treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period. All models are estimated using the heterogeneity robust estimator (DID_L) introduced by (Chaisemartin and d'Haultfoeuille 2024). DID_L is estimated over eight post periods. We control for compositional change using the `same_switchers` option. The average marginal effect (AME) is calculated at the mean and is reported as the % change for one unit of treatment. All regressions include district and period fixed effects. The sample period is 1871-1919 in columns (1-4) and (6), and 1882-1919 in column (5).

Although HAP is associated with a number of health conditions, respiratory conditions such as lower respiratory infections and chronic obstructive pulmonary disease (COPD) account for approximately half of all deaths attributable to HAP (Gordon et al. 2014; Simkovich et al. 2019). The *Certain Infectious* category, which contains a number of childhood diseases, is also negatively associated. Although diseases of this category are not caused by HAP, the susceptibility to and severity of diseases of this category has been linked to increased exposure to pollutants (Monoson et al. 2023). Together, these results are supportive of the hypothesis that reductions in HAP may have played a key role.

However, laborers cottages also increased housing quality along other dimensions; they increased the space in the home, improved sanitary conditions, and removed animals from the dwelling. Still, although we cannot rule out that these improvements are partial contributors to reduced mortality, lower HAP appears to outweigh them. We see a large effect for the cause-of-death category most clearly associated with HAP while we see little movement for diseases more readily associated with alternative mechanisms. For example,

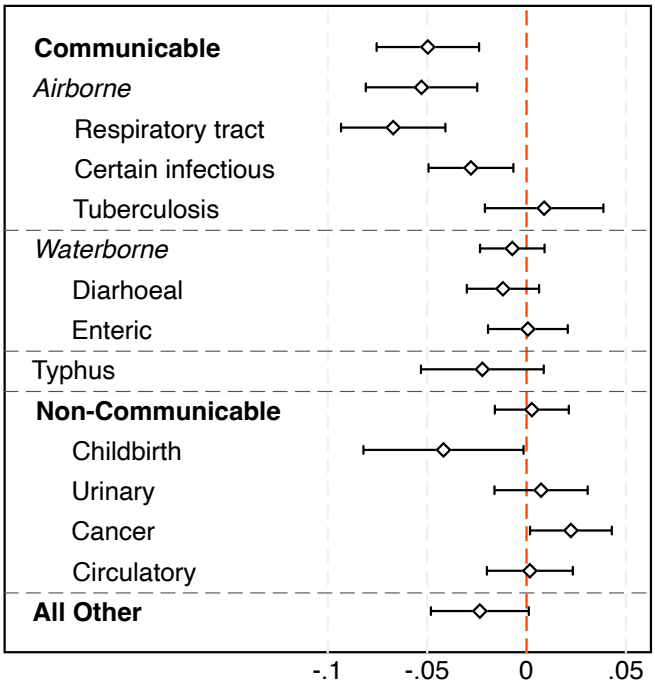


Figure 6: Effect of cottages construction by disease sub-category.

Notes: This figure reports DiD estimates for disease sub-groups. The unit of analysis are district-periods. The dependent variable is the average annual disease category specific death rate per 1000 population across the period. The treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period. All models are estimated using the heterogeneity robust estimator (DID_l) introduced by (Chaisemartin and d'Haultfoeuille 2024). DID_l is estimated over eight post periods. We control for compositional change using the `same_switchers` option. Bars indicate 95% confidence intervals calculated using clustered standard errors at the district level.

if removing animals were the most consequential improvement we would expect to see an effect for *zoonotic* diseases such as Typhus that are aggravated by close proximity to farm animals (Rees et al. 2021). Similarly, if sanitary improvements – such as washable floors and a WC at a minimum distance from the cottage – were the relevant component of the treatment package construction should lead to a reduction in deaths from waterborne diseases (Kyu et al. 2025).

Greater space, with parents and children sleeping in separate rooms, and the possibility of isolating sick family members, could affect the incidence and transmission of the diseases we observe. The significant effect for the *certain infectious* category lends support this mechanism. However, if overcrowding were the driver of the effect, we would expect significant coefficients for comparatively more infectious diseases such as Measles. Instead we see that the effect is concentrated in Scarlet Fever (see Table A5).

This pattern suggests that reduced contagious infection, was likely secondary to less-

ened HAP. High levels of HAP increase the susceptibility to and severity of airborne diseases. While it is unclear why this would affect scarlet fever specifically – point estimates for whooping cough, diphtheria, and measles are negative but insignificant – the absence of a strong significant effect for measles contradicts the overcrowding mechanism. Anecdotally, the extent of the housing improvements supports this interpretation.

In sum, although alternative mechanisms cannot be ruled out entirely, the pattern of cause-of-death results strongly supports the hypothesis that the effect of laborers cottages on mortality operated primarily via a reduction in HAP.

Next, to further explore the HAP mechanism, we estimate our baseline model for component death rates by age-sex groups.[17] The DID_l baseline results are presented in Table 5, with Panel A and B showing results for females and males respectively.[18] Looking across both panels reveals a clear pattern of results across the age distribution. We do not find a link between our treatment and deaths of infants or deaths among the working age population and our treatment. However, there is a clear link between cottage building and death rates of children and elderly women, in terms of both statistical significance and magnitude, as indicated by the Average Marginal Effects (AME). There is also evidence of a sex differential, with the relationship generally stronger for female mortality. This age-sex pattern offers further support to our proposed mechanism; many studies have identified the increased disease burden of HAP on women, children and the elderly due to greater exposure to pollutants through increased time spent in the home (Po et al. 2011; Bozzola et al. 2024; Sloyan and Maitre 2024).

Additionally, these age-sex results serve to validate the cause-of-death results. Prior research raises legitimate questions around the accuracy of death registration, and particularly disease classification, in the Irish records (Walsh 2017). Here the misclassifications of tuberculosis as other respiratory diseases could pose a challenge to the interpretation of the results in Figure 6 (Breathnach 2022). If this were the case we should see significant negative effects for the 15-24 and 25-44 age groups, where the incidence of TB was greatest. The age pattern we observe – with the effect concentrated among children (1-14) and the elderly (over-65) – is consistent with diseases of the respiratory tract being the most affected category.

Our argument on the mechanism is also supported by other factors, including fuel choice. Households in rural Ireland in the 19th and 20th century relied heavily on peat as their primary fuel for cooking and heating (Kennedy 2013). When burned in an open fire, this solid fossil fuel produces twice as much $PM_{2.5}$ as coal and more than three times as much as wood (Maher et al. 2021). It is likely, therefore, that there were significant negative

[17] Again, we normalize to rates of death per 1000 population in each age group. While deaths by cause and deaths by age and sex are reported, deaths by age, sex *and* cause are, unfortunately, not reported at the district level.

[18] We control for compositional change in these regressions using the `same_switchers` option. Our results are robust to including all groups. See Table A4.

Table 5: Effect by Age-Sex Group

	Age Group specific Death Rate						
	(1) under-1	(2) 1-4	(3) 5-14	(4) 15-24	(5) 25-44	(6) 45-64	(7) over-65
Panel A: Female							
ATT: $CottagesRate_{it}$	0.224	-0.241**	-0.078**	-0.008	-0.024	0.030	-0.906**
	(0.270)	(0.113)	(0.032)	(0.040)	(0.054)	(0.085)	(0.400)
Mean DV	73.46	15.57	4.04	5.41	8.08	16.18	96.10
AME (%)	0.31	-1.55	-1.94	-0.14	-0.30	0.19	-0.94
Panel B: Male							
ATT: $CottagesRate_{it}$	0.202	-0.172*	-0.067***	-0.000	0.022	0.021	-0.414
	(0.317)	(0.100)	(0.026)	(0.026)	(0.037)	(0.098)	(0.416)
Mean DV	88.88	15.19	3.29	5.21	7.47	16.93	88.53
AME (%)	0.23	-1.13	-2.05	-0.00	0.30	0.13	-0.47
Period FE	✓	✓	✓	✓	✓	✓	✓
District FE	✓	✓	✓	✓	✓	✓	✓
Clusters	Districts (158)						

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports DiD estimates for the effect of cottage construction on mortality among different age-sex groups. The dependent variable is the average annual age-sex group specific death rate across the period. Population per age-sex are calculated based on the 1901 age structure and annual interpolated population. The death rate for under-1 is defined as deaths under-1 divided by births. All outcomes are scaled to 1000 age-sex population. Panel (A) reports the effect on female, and panel (B) for male death rates. The treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period. All models are estimated using the heterogeneity robust estimator (DID_L) introduced by (Chaisemartin and d'Haultfoeuille 2024). DID_L is estimated over eight post periods. We control for compositional change using the `same_switchers` option. The average marginal effect (AME) is calculated at the mean and is reported as the % change for one unit of treatment. All regressions include district and period fixed effects. The sample period is 1871-1919.

health effects of burning peat in open fires in the low-quality housing that the *Labourer's Act* cottages replaced. Moreover, the Victorian fixation on ventilation that motivated new cottage design likely delivered substantial benefits. Increasingly, ventilation is identified as a key factor in reducing HAP. In an important recent field experiment, households in London were randomly assigned real-time information on $PM_{2.5}$ concentrations. Being provided with this information resulted in a 17 *per cent* reduction in HAP. Most notably, through a follow-up survey, the authors were able to identify the key mechanism – more natural ventilation from treated households opening windows and doors (Metcalfe and Roth 2025). In a historical context too – specifically 1930s Britain – indications are that good ventilation was an important factor related to child health, development and longevity (Hatton and Martin 2010; Frijters et al. 2010).

Summarizing our results so far, we observe that cottage building is associated with

reductions in overall mortality. This result is driven by a decline in deaths from communicable diseases in general and by a decline in deaths from respiratory disease in particular. Cottage building reduced mortality among children and the elderly, with in general a greater impact on women than men. Based on similar evidence from contemporary studies of developing countries, this distinctive pattern of results suggests a plausible primary mechanism through which cottages led to health improvements: a reduction in Household Air Pollution, in particular $PM_{2.5}$, due to improved fireplaces, chimneys, windows and general ventilation.

Alternative Mechanisms Although our results strongly imply that improvements to housing quality – and specifically reduced HAP due to better ventilation – explain the effect of cottage building on mortality, it is necessary to evaluate alternative mechanisms unrelated to housing quality.

As discussed in subsection 2.2 cottages were let at around half the market rate of a cheap dwelling. Aside from the improvement in housing quality, families that received a cottage also experienced an increase in disposable income. McKeown famously argued that the primary driver of mortality decline was improved nutrition due to higher incomes (McKeown 1976). Aside a large literature calling into question the McKeown thesis (Szreter 1988; Szreter 2000; Colgrove 2002) the pattern of the cause-of-death and age-sex results do not point to an income effect, with the decline of TB playing a central role in McKeown's work.

Further, by leveraging detailed data on the quality of housing pre-treatment, we can show that the efficacy of cottage building is closely tied to housing quality. First, we construct a variable that captures the stock of low quality housing (3rd and 4th class) in the district.[19] By replacing the treatment variable in Equation 1 we find that the stock of low quality housing is positively and significantly associated with the all-cause death rate (see Columns (1) of Table A7). A one standard deviation decrease in the stock of poor quality housing is associated with 2.47 fewer deaths per 1000. Second, we identify heterogeneity across the extent of the scheme (see Columns (2) of Table A7). We construct a variable that measures the number of low quality houses (3rd and 4th class) remaining after the scheme ended. By including non-parametric controls for the initial stock of poor-quality housing, and interacting this variable with the treatment we find that the effect of cottage building was greatest were most low quality housing was replaced. This implies that the scheme could have had an even greater impact if it replaced more of the 3rd class housing that was also prevalent among social groups, such as tenant farmers, not targeted by the policy.

Although we cannot entirely rule out that alternative causal channels such as an income

[19]We construct this variable by subtracting the cottages built at the beginning of each period from the sum of all 3rd and 4th class housing in the district in 1881. The variable is normalized by population, to mirror our main treatment variable.

effect contributed to the decline in mortality we observe, the weight of our results highlight the relevance of improvements to housing quality in general, and a reduction in HAP specifically.

5.3 Alternative Identification

While the baseline results above indicate a strong and consistent association between cottage building and reductions in mortality, a key concern is whether these findings reflect true causal effects or are confounded by unobserved trends or omitted variables. One potential threat to identification is that the timing of cottage construction may coincide with broader public health or sanitation improvements that independently reduce mortality. Additionally, if local authorities selectively placed cottages in areas already experiencing declining mortality – or if more health-conscious districts were more likely to advocate for and receive housing investment – our estimates might reflect reverse causality or selection bias rather than the effect of improved housing conditions.

To address these concerns, Table 6 presents a series of triple-difference specifications that leverage variation across sex, as described above in section 4. We interact the measure of treatment with a female indicator, allowing us to explore whether mortality reductions were larger among groups more exposed to HAP in the home environment. Column (1) shows the overall association between cottage construction and mortality, and Column (2) interacts this effect with a female indicator. The estimates reveal that mortality reductions were disproportionately concentrated among women. As before, this heterogeneity in treatment effects aligns closely with contemporary evidence on the sex-specific health burden of household air pollution. The inclusion of district-period fixed effects means that we can non-parametrically control for confounders that should effect both sexes equally. Although this does not close all back door paths – many policy interventions have sex-biased effects, in one direction or another – it does increase the confidence in a causal interpretation of the effect. In particular, we can rule out confounders such as selective construction in districts with already falling mortality rates or more health-conscious districts since we would expect even effects across sex in these cases.

Estimating this triple-difference model across different configurations of cause-of-death groups can help rule out other confounders. By running a triple-difference across airborne and waterborne diseases we control for the effect of waterworks or other sanitation schemes that should affect both diseases groups. Here the coefficient captures the change in deaths from airborne diseases relative to waterborne disease deaths. The coefficient in column (3) of Table 6 reveals that the effect is concentrated in airborne diseases even when controlling for this set of confounders. Similarly, to account for changing age-structure we can run the model across airborne diseases of the respiratory system and certain infectious diseases since a changing age structure should affect both

Table 6: Triple Difference

	$DeathRate_{i,t,j/s}$				
Dataset=	Age-Sex		Cause-of-Death		
	(1)	(2)	(3)	(4)	(5)
J/S=	Any	Female	Airborne	Respiratory	Communicable
$Cottages_{it}$	-0.041***				
	(0.015)				
$Cottages_{it}$		-0.025***	-0.004***	-0.021***	-0.003***
$\times 1\{j/s = J/S\}$		(0.007)	(0.001)	(0.005)	(0.001)
Within Sample			Communicable	Airborne	All
Period × Disease	✓	✓	✓	✓	✓
District × Disease	✓	✓	✓	✓	✓
District × Period		✓	✓	✓	✓
Observations	5,366	5,366	26,830	18,781	37,246
Clusters			Districts (158)		

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports triple-difference estimates for the effect of cottage construction on mortality, allowing for heterogeneity by sex (S) and by disease category (J). The unit of analysis are disease-district-period (and where relevant sex) cells. The dependent variable is the average disease-specific death rate across the period, scaled to 1000 population. The treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period. $1\{j = J\}$ is an indicator equal to 1 if disease j belongs to category J. Column (1) reports the overall effect across all diseases. Column (2) reports the differential effect for females. Columns (3) through (5) report the differential effect for diseases of type J (Airborne, Respiratory, Communicable). Fixed effects included are as indicated in the panel. All models are estimated using OLS. The sample period is 1871–1919.

in a comparable manner. As column (4) reveals our results are robust to controlling for age-structure in this manner. Last, in column (5), we run the triple-differences across all communicable and non-communicable diseases. Here the district-period fixed effects control for any confounders that alter mortality across all diseases such as changes in the quality of death registration. Again our result is robust, with the effect concentrated in communicable diseases.

The persistence of this result across different empirical strategies, supports our hypothesis that the primary health benefits of the *Labourers Act* cottages stemmed from improvements in household environmental conditions, particularly ventilation and reductions in exposure to $PM_{2.5}$ from peat smoke. The robustness of our results to the inclusion of district-period fixed-effects bolsters the internal validity of our baseline results and reinforces their causal interpretation. Further, taken together, the results from Table 6 and Table 6 support our proposed mechanism. By demonstrating that mortality reductions are concentrated in specific diseases plausibly linked to HAP – and among subpopulations most exposed to that pollution – we rule out a range of confounding explanations. These findings, in conjunction with the event-study evidence showing no pre-trends and strong post-treatment effects, provide a coherent narrative: the *Labourers Act* cottages reduced

mortality primarily by improving the home environment, and specifically by reducing HAP.

5.4 Policy Implications

Our results demonstrate the effectiveness of improved housing quality as a public health intervention, but we are also interested in whether the policy was cost-efficient. At a total cost of £9 million (roughly $1.27 billion in 2020 USD) the laborers acts were a large-scale public investment project (Aalen 1986).[20]

While historical studies are often limited to estimating the cost per infant life saved, the richness of our deaths-by-age-sex results, together with life-tables, enables us to compute the preferred metric for evaluating the cost-effectiveness of health interventions; the cost per quality-adjusted life year (QALY) saved. QALYs are a generic measure that combines the extra life-years an intervention yields with the health related quality of life during these years measured using utility weights. There exists a multitude of methodologies for calculating these utility weights (Lancsar et al. 2020; Alabbad et al. 2025). Since improved housing conditions have no adverse effect on health related quality of life, our baseline approach ($QALY$) assigns an utility weight of one to all life years saved. Additionally we also calculate $QALY^{age}$ where utility weights vary in age, and $QALY^{3\%}$ where future QALYs are discounted at a rate of 3%.

Panel (A) of Table 7 reports the calculation we use to obtain the life-time QALYs gained per cottage per year post-construction. We use the significant coefficients for the different age-sex groups from Table 5. Since these coefficients capture the effect on the age-sex specific death rate we weight them by the population share, and multiply them by the average life-expectancy of that age-sex group to obtain the life years saved by each cottage per year after construction LY_t. In the base case, $\Delta QALY_t$ is equal to ΔLY_t, while the two other QALY measures adjust ΔLY_t according to age-specific utility weights or the present value of future LYs.[21] In Panel (B) we report different estimates for the total QALYs per Cottage based on different time horizons. Since we estimate all effects over a 24 year period this is also our preferred time horizon, implying that each cottage saved a total of 26.2-52.3 QALYs.

Beyond assessing the effect of an individual cottage, it is also worth quantifying the impact of the entire scheme. In total the scheme saw the construction of 45,631 cottages. Based on our estimation we can be confident that the effect of cottage building persisted

[20] This and and all subsequent calculations converting pound to 2020 USD are based on the measuring worth tool by Officer and Williamson (2025).

[21] Modern age-specific utility weights are based on large scale health survey such as EQ-5D-5L (Alabbad et al. 2025). In the absence of such surveys for 19th century Ireland we choose to draw from contemporary research for the UK, while increasing the gradient to account for the lower level general health in the 19th century (McNamara et al. 2023). We use the utility weights of 0.80, 0.85, and 0.6 for the age-groups 1-4, 5-14, and over 65. For discounted QALYs, the discount factor for age group x is calculated as $e_x^{-1} \int_0^{e_x} 1/1.03^t dt$.

Table 7: Cost-Effectiveness

Panel A: Annual QALY per Cottage

					$\Delta QALY_t$		
Sex	Age	β_s	$PopW_s$	e_s	Basic	Age-specific	Discounted
M	1–4	-0.172	4.8%	60.0	0.50	0.40	0.23
M	5–14	-0.067	10.9%	55.2	0.40	0.34	0.17
F	1–4	-0.241	4.7%	59.9	0.68	0.54	0.32
F	5–14	-0.078	10.1%	54.9	0.43	0.37	0.21
F	over 65	-0.906	3.4%	5.5	0.17	0.10	0.16
QALY per Cottage per Year:					2.18	1.75	1.09

Panel B: Total QALY per Cottage

Time Horizon (years)	5	10	15	**24**	30	35	40
$\sum QALY$	10.9	21.8	32.7	**52.3**	65.4	76.3	87.2
$\sum QALY^{age}$	8.8	17.5	26.3	**42.0**	52.5	61.3	70.0
$\sum QALY^{3\%}$	5.5	10.9	16.4	**26.2**	32.7	38.2	43.6

Notes: In this table we calculate life years (LY) and different types of quality-adjusted life years (QALY) saved by the construction of cottages. In Panel (A) we compute how many LY/QALY each cottage saved per year post construction. The coefficient β_s captures the average marginal effect of cottage construction on the death rate of age-sex group s. To obtain the effect at the population, instead of age-sex group, level we weight each coefficient by the population share of the group s $PopW_s$. We multiply these population-level mortality effects by the average life-expectancy e_s among group s to find the number of life years saved annually per cottage ΔLY_t due to the reduction in the mortality rate. Since both mortality rates and cottage counts are scaled per 1,000 population, the estimated effects correspond to population-level changes. Basic $\Delta QALY_t$ are equivalent to ΔLY_t. Age-specific $\Delta QALY_t$ assign lower utility weights to certain age groups, and discounted $\Delta QALY_t$ discount future QALYs at a rate of 3%. In panel (B) we consider total QALY each cottage saved if the effect persisted over different time horizons. Our estimation window is 8 periods, or 24 years, meaning that this is also the most realistic time horizon.

for 24 years, implying a total of 2.38 million life-years or 1.19 million discounted quality-adjusted life-years saved.

General World Health Organization guidelines for assessing cost-effectiveness are that interventions costing less than three times GDP per capita, per QALY, are considered cost-effective, while those costing less than one times GDP per capita are considered *highly*-cost-effective (Marseille et al. 2015). Based on these guidelines, and using a GDP per capita of £26.29 (Andersson and Lennard 2019), the threshold for cost-effectiveness in Ireland during these decades would be £78.9 ($11,200 USD 2020). The cost per cottage typically ranged from £100 to £150 in contemporary terms, equivalent to approximately $14,100–$21,200 in 2020 USD.

Even if we only allow for a conservative time horizon of 5 year, and use discounted QALYs, the cost per QALY is $150/5.45 = 27.52$ ($3,890 USD 2020) which falls well below the threshold for cost-effectiveness. A more realistic estimate of QALYs saved, such as 26.2, leads to a cost per QALY of $150/26.2 = 5.72$ ($813 USD 2020). Our results imply costs

per QALY that are markedly lower than the WHO cut-offs used widely in modern cost-effectiveness calculations. Further, if instead of using a GDP adjusted threshold, we apply a universal threshold such as $100,000-150,000 USD (Institute for Clinical and Economic Review 2023), the policy appears exceptionally cost-effective. Thus, this historical housing intervention would readily meet modern criteria for cost-effectiveness.[22]

Moreover, in 19th century Ireland the burden of non-communicable disease was low. In contemporary settings, characterized by a double disease burdens, the effect of improved housing quality is likely even larger. Contemporary studies point to the effect of HAP on cardiovascular, neoplastic diseases, and on mental health (Maher et al. 2021; Feeney and Kenny 2024). As such, these estimates necessarily represent a lower-bound for the impact of improved housing quality.

This cost-benefit analysis is strong evidence that the *Labourers Act* cottages were not only effective in improving health outcomes, but also clearly cost-effective by modern public health standards.

6 Robustness

Registration of Deaths As noted above, while some researchers have argued that official statistics are generally accurate (McLaughlin and Whelehan 2024), others such as Walsh (2017) argue that there was less comprehensive registration of deaths (as well as births) in the early years of the registration system. Since the district fixed effects demean mortality at the unit of death registration (i.e. SRDs) we need not be concerned about level differences in the quality of registration. However, if cottage-building was associated with trend-differences in the capacity of district authorities, this could also be reflected in more accurate or complete registration of deaths. Such an association would bias our estimates downward, as areas with more cottages would also register deaths more completely, potentially offsetting the reduction in mortality brought about by the cottages. Alternatively, the competency of local government could lead to more precise registration (rather than more complete registration), including a reassignment of deaths formerly categorized as due to a communicable disease ('zymotic') to one of the non-communicable diseases. While this would lead to an effect for communicable diseases in line with what we estimate, we would then expect a corresponding positive effect for the category to which deaths are reassigned.[23]

Although the pattern of results makes such a relationship between cottages and death

[22] It is important to acknowledge that the use of cost per QALY to decide on the implementation of public health policies and particularly the development/insurance coverage of medicines is controversial (Kirkdale et al. 2010). Still they constitute a useful metric for evaluating and comparing the cost-effectiveness of past policies.

[23] Reid et al. (2015) provide a thoughtful and detailed analysis of cause of death data for Scotland over this period examining 'old age' as an ill-defined category.

Table 8: Quality of Death Registration

	(1) Death from Fever per 1000	(2) Uncategorised Deaths per 1000
ATT: $CottagesRate_{it}$	0.002	-0.069
	(0.002)	(0.059)
Groups	1257	1270
Years	1882–1919	1871–1919
Period FE	✓	✓
District FE	✓	✓
Clusters	Districts (158)	

Clustered standard errors at the district level in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports DiD estimates of the effect of cottage construction on two indicators of death registration quality. Column (1) uses as the dependent variable the number of reported deaths from fever per 1,000 population. Column (2) uses the number of unreported deaths per 1,000 population, constructed as the difference between total deaths and deaths with a cause reported at the district level. The treatment variable is $CottagesRate$, the number of cottages constructed per 1,000 population at the beginning of the period. All regressions are estimated using the heterogeneity-robust estimator (DID_L) introduced by (Chaisemartin and d'Haultfoeuille 2024), with period and district fixed effects.

registration unlikely, we nonetheless investigate under-registration as a potential source of bias. To test for any association with the quality of death registration, we look at two proxies for the extent and quality of reporting. First, deaths categorized as 'fever', a symptom observed across a wide range of diseases and not a direct cause of death, proxy the quality of reporting. Deaths were assigned to fever when local registrars either lacked the know-how, information or diligence to correctly identify the underlying cause. Given the impossibility of directly observing mis- or under-registration of deaths, deaths from fever are our best proxy. The time-trend in deaths from fever reveals that, at an aggregate level, accuracy of registration improved in step with medical know-how. Deaths categorized as due to fever fell by two orders of magnitude, from 0.389 to just 0.005 per 1000 people during our period.

Second, the difference between total deaths and the sum of deaths with a reported cause-of-death at the district-year level, proxies the extent of the source tables. If there were a systematic relationship between how many deaths are assigned to the categories we observe, and the construction of cottages, this would bias the results presented in subsection 5.2.

We estimate Equation 1 for the death rate from fever in column (1) and the difference between total deaths and classified deaths in column (2) of Table 8. A significant negative coefficient for either outcome would be of concern, as this would suggest that reporting improved in places that received more cottages. Instead, we see insignificant point estimates for both proxies. This is reassuring evidence that the positive impact of cottage building on health is not driven by improvements in reporting.

Table 9: Robustness to Alternative Estimators

	ATT Estimate	Clustered SE	LB 95% CI	UP 95% CI
TWFE	-0.444***	0.169	-0.777	-0.112
DID_L	-1.000***	0.369	-1.724	-0.276
Borusyak, Jaravel, & Spiess (2023)[a]	-1.084***	0.224	-1.523	-0.645
Wooldridge (2021)[b]	-1.081***	0.230	-1.532	-0.629
Callaway & Sant'Anna (2021)[c]	-0.971***	0.299	-1.558	-0.385

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports average treatment effect on the treated (ATT) estimates from alternative heterogeneity-robust DiD estimators, alongside the baseline TWFE specification. The unit of analysis is the district-period, and the outcome is the annual all-cause death rate per 1,000 population. Since only Chaisemartin and d'Haultfoeuille (2024) can accommodate a continuous treatment, all other estimators use a binary indicator for whether any cottage has been built.
[a] Estimated using the `did_imputation` package of Borusyak et al. (2024).
[b] Estimated using the `xthdidregress` implementation of Wooldridge (2021).
[c] Estimated using the `xthdidregress` implementation of Callaway and SantAnna (2021), with not-yet-treated units as the control group.
The estimators of Wooldridge (2021) and Callaway and SantAnna (2021) first estimate cohort-specific event-study effects; an overall ATT is obtained using the `aggregation` option, which computes a weighted average across all cohort-time specific effects.

DiD Estimators Next, we consider the robustness of our results to alternative estimators designed to be account for heterogeneous treatment effects. Table 9 shows estimates of the ATT across a selection of estimators, where our treatment is a staggered binary treatment and the outcome is all-cause mortality rate, as we considered in Table 3. Reassuringly, the estimated treatment effect is similar in direction and magnitude across each of four estimators designed to allow for heterogeneous treatment effects, with a coefficient close to 1 in each case.

Table 10: Excluding Provinces

	$DeathRate_{it}$				
	(1)	(2)	(3)	(4)	(5)
Exclude:	None	Leinster	Ulster	Connaught	Munster
ATT: $CottagesRate_{it}$	-0.145***	-0.104**	-0.153**	-0.126***	-0.206*
	(0.054)	(0.050)	(0.064)	(0.048)	(0.107)
Groups	1468	1186	1003	1089	1029

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports robustness checks excluding one province at a time from the sample. The dependent variable is the average annual all-cause death rate per 1,000 population. The treatment variable is $CottagesRate$, defined as the number of cottages constructed per 1,000 population at the beginning of the period. All effects are estimated using the heterogeneity-robust DiD estimator (DID_L) of Chaisemartin and d'Haultfoeuille (2024), with district and period fixed effects.

Provinces Table 10 examines the sensitivity of our baseline DID_l result to the exclusion of each province in turn. Column (2), for example, reports the coefficient when the eastern province of Leinster (which contains Dublin and its hinterland) is excluded. While the coefficient is slightly smaller when any province is dropped, compared to the baseline of -0.145, suggesting that each province contributes to the overall identification, it is nonetheless negative, statistically significant and similar in magnitude in all four cases.

Table 11: Spatial Clustering

	$DeathRate_{it}$		
	(1) All Cause	(2) Communicable	(3) Non-Communicable
ATT: $CottagesRate_{it}$	-0.145***	-0.090**	-0.004
SE (Province, $N = 4$)	0.074*	0.053*	0.006
SE (County, $N = 33$)	0.052***	0.039**	0.004
SE (IDplu, $N = 158$)	0.052***	0.036**	0.004
Groups	1468	1508	1508

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports DiD estimates of the effect of cottage construction on mortality outcomes, estimated using the heterogeneity-robust estimator (DID_L) of Chaisemartin and d'Haultfoeuille (2024). The dependent variable is the average annual death rate per 1,000 population. Column (1) reports the effect for all-cause mortality, column (2) for communicable diseases, and column (3) for non-communicable diseases. To assess robustness to spatial correlation, standard errors are reported when clustering at different levels of aggregation: province ($N = 4$), county ($N = 33$), and district ($N = 158$). All specifications include district and period fixed effects.

Spatial Clusters An important recent literature has highlighted the possibility of spurious correlations and inflated t-statistics that can emerge when analyzing spatially correlated data (Becker et al. 2025; Conley and Kelly 2025). In recent work, Conley and Kelly (2025) identify two key characteristics that drive this effect: the ubiquity of spatial trends and other forms of large-scale spatial structure, and the strong autocorrelative properties of spatial data. The effect is that the number of useful observations is lower than it would appear. While spatial trend controls, such as a quadratic in longitude and latitude, can avoid spurious results due to spatial trends, they can not be relied on to eliminate spatial correlation in residuals. Instead, Conley and Kelly (2025) recommend large-cluster spatial inference. We present, in Table 11, the ATT where the outcome is deaths due to all causes, communicable and non-communicable diseases, for three additional levels of spatial clustering: provinces ($N = 4$), counties ($N = 33$), and PLUs ($N = 158$). Regardless of the level of spatial clustering, the pattern of results holds with a negative impact on all-cause

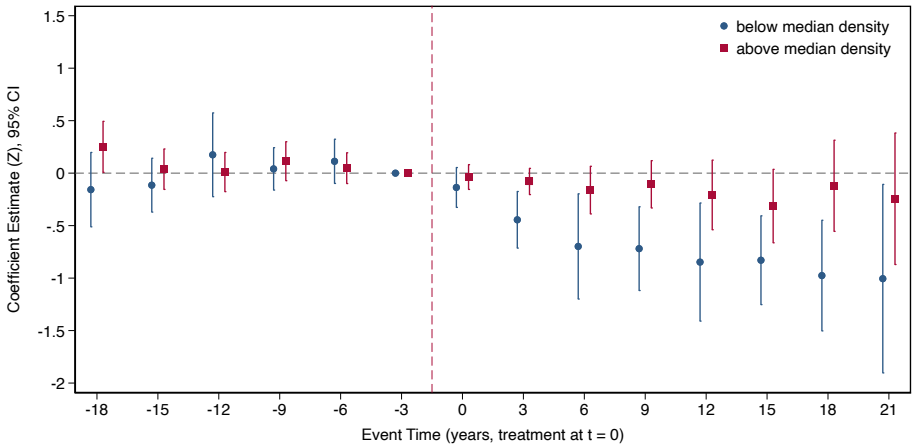


Figure 7: Event-study DID_l: Effect by Population density.

Notes: This figure plots event-study effects obtained using the heterogeneity-robust DID_l estimator of Chaisemartin and d'Haultfoeuille (2024). It splits the sample by population density reporting effects for districts with below and above median population density separately. The dependent variable is the annual all-cause death rate. To aid interpretability the outcomes are standardized. The treatment variable is $CottagesRate$, the number of cottages constructed per 1000 at the beginning of each period. District and period fixed are included. Bars denote 95% confidence intervals computed based on standard errors clustered at the district level.

mortality driven by communicable diseases.[24]

Additional Controls The richness of our data allows us to include a range of additional control variables, relating to local government and locally-provided healthcare. In Table 12, we show eight coefficients for the ATT, where the all-cause death rate is the outcome variable. The first column repeats our baseline result of the DID_l result from Table 3, using timing and intensity of cottages.In the following seven columns, we add sequentially different controls variables available at the district level and varying over time, including PLU spending and revenue, the assessed value of land, spending on workhouses and poor relief, and lastly the amount of medical expenditure at local level. None of these is driving the result and the estimated coefficient is at least as large when these are included individually. The eighth and final column reports the coefficient when all controls are included simultaneously. Again, the estimated coefficient is, if anything larger, when these local time-varying controls are added.

[24]We also implement the procedures described in Becker et al. (2025) and our results are robust to most of the transformations used to account for spatial autocorrelation (see Table A8). Some transformations remove important identifying variation, although the magnitude and direction of the point estimates are consistent, they are not significant at conventional thresholds.

Table 12: Robustness to Controls

	$DeathRate_{it}$ (All Cause)							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
ATT: $CottagesRate_{it}$	-0.145***	-0.126***	-0.136***	-0.148***	-0.132***	-0.134***	-0.134***	-0.137***
	(0.054)	(0.044)	(0.041)	(0.053)	(0.042)	(0.042)	(0.045)	(0.045)
Spending		✓						✓
Revenue			✓					✓
Valuation				✓				✓
Workhouse					✓			✓
Poor Relief						✓		✓
Medical Exp.							✓	✓

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Notes: This table reports robustness checks sequentially adding time-varying controls to our baseline specification. Panel A shows specifications (1)(4); Panel B shows specifications (5)(8). Each column adds a different set of district-level controls: per capita spending, revenue, property valuation, workhouse admissions, poor relief expenditure, and medical expenditure. Column (8) includes the full set of controls jointly. The treatment variable is $CottagesRate_{it}$, defined as the number of cottages constructed per 1,000 population at the beginning of the period. All models are estimated using the heterogeneity-robust estimator (DID_L) of Chaisemartin and d'Haultfoeuille (2024) and include district and period fixed effects.

Population Density Lastly, we examine whether our estimate effects differ across low- and high-population density districts. Rates of communicable disease were significantly higher in urban places, if a decrease in urban death rates drove our results, this would contradict our proposed mechanism. Figure 7 shows the event-study results for above and below median districts, based on population density: the bottom quarter (blue circles) and the top quarter (red squares). These results show that it is not the most densely population districts driving the overall results. Instead, the negative impact on mortality is clearly seen for those districts that are least densely populated. As with the results by age group, this is inconsistent with the hypothesis that the cottages broke chains of transmission of highly infectious disease. While not categorical proof, it is instead consistent with the hypothesis that cottages reduced exposure to Household Air Pollution.

7 Conclusion

This paper provides new evidence on the causal relationship between housing quality and health during the historical mortality transition. Using detailed district-level administrative data for Ireland between 1871 and 1919, we study the large-scale rollout of rural public housing under the *Labourers Acts* and show that this intervention significantly reduced mortality. Our results suggest that improved housing conditions - especially greater ventilation and better household environments - led to a meaningful decline in deaths from communicable diseases, particularly respiratory illnesses. These effects are concentrated among children and older women, consistent with differential exposure to Household Air Pollution. Together, the findings imply that the introduction of well-ventilated cottages played a central role in reducing the health burden of poor household environments in rural Ireland. Based on back-of-the-envelope calculations cottages construction under the *Labourers Acts* can account for as much as 66% of the decline in deaths from communicable diseases between 1880 and 1915.[25]

The inability to truly isolate the effects of housing on health from other associated factors is recognized in both contemporary and historical settings (Burnett 1991). Despite the use of detailed data and careful methods, it is not possible to entirely rule out the influence of concurrent, unobserved factors or selection into treatment. Moreover, while the triple-difference design helps isolate a plausible mechanism – reduced exposure to household air pollution – direct measures of ventilation or household fuel use are not available. Nonetheless, we find evidence that strongly indicates improved housing contributed materially to the rural mortality decline. Our context provides several useful

[25] By the end of the scheme the average district saw the construction of 14.06 cottages per 1000 population. Over the same period, the mortality rate from communicable diseases fell from 5.93 per 1000 to 4.32. Based on the coefficient for deaths from communicable diseases in column (1) of Table 4 the construction of cottages reduced the mortality rate by $0.076 \times 14.05 = 1.064$, accounting for $1.064/(5.93 - 4.32) = 0.66$ of the overall decline.

features that reduce potential confounding, such as the absence of ambient air pollution due to our rural setting, the inability to switch to clean energy such as electricity, or the fact that housing density and household size are held largely constant. Our results should therefore be interpreted as evidence of a causal impact of housing improvements on mortality, operating through a mechanism that is highly consistent with both the historical context and modern epidemiological understanding.

In terms of external validity, the Irish context differs from many contemporary settings: ambient pollution was minimal, fuels were largely homogeneous, and cottages were built to uniform designs. Nevertheless, these differences sharpen the interpretation of our findings by isolating the role of structural housing improvements, rather than confounding influences from urban density or energy substitution. While the magnitude of effects may vary, the underlying mechanism - reducing household exposure to smoke and improving ventilation - remains highly relevant to low- and middle-income countries today, where HAP is still a leading cause of preventable morbidity and mortality. Our findings complement modern evidence from improved cookstove trials, suggesting that durable health gains are more likely when interventions alter the built environment itself rather than relying solely on household behavior.

From a policy perspective, this historical case demonstrates that investments in housing infrastructure can be powerful public health tools, particularly when they target the environments in which women and children spend the greatest share of time. For todays policymakers in low- and middle-income countries, this suggests that housing and ventilation improvements deserve a central place in strategies to reduce the health burden of HAP, alongside cleaner fuels and improved stoves. More broadly, the results underscore that the health transition was not driven solely by medical advances or urban infrastructure, but also by rural interventions that reshaped the household environment.

For researchers, the study highlights that historical housing policies can yield new insights into the mechanisms underpinning mortality decline and help explain persistent health disparities shaped by structural and environmental conditions. The evidence from Ireland suggests that when public health policy tackles environmental exposures inside the home, it can accelerate the mortality transition in ways that remain relevant to current global health challenges.

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A Appendix

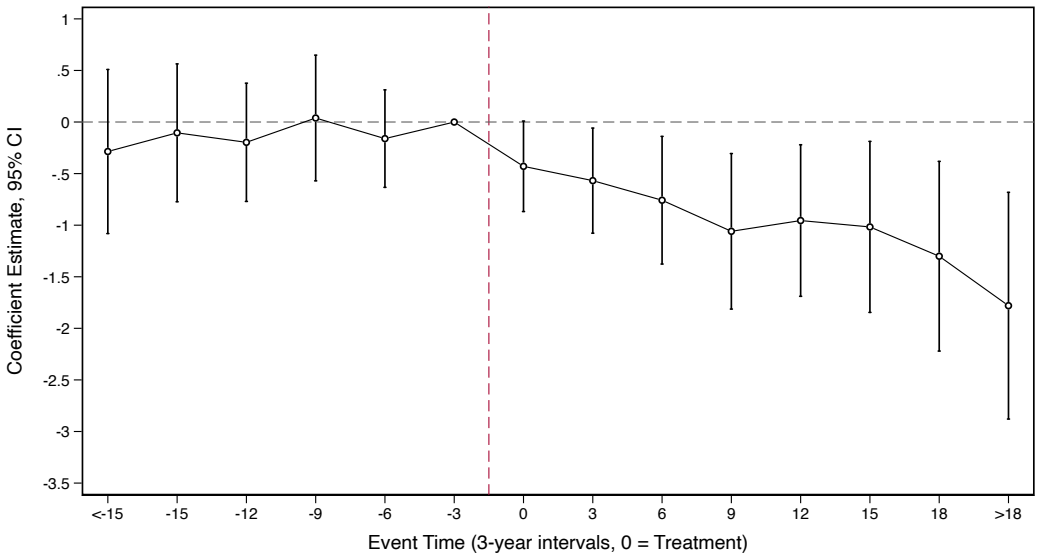

Figure A1: Event-study TWFE: Treatment is first cottage built.

Table A1: Different Treatment Variables

	$DeathRate_{it}$				
	(1)	(2)	(3)	(4)	(5)
$Var =$	$CottagesRate_{it}$	$CottagesMin_{it}$	$CottagesMean_{it}$	$CottagesMax_{it}$	$Cottages_km^2_{it}$
ATT: Var	-0.145***	-0.007***	-0.006**	-0.005**	-3.267***
	(0.054)	(0.003)	(0.002)	(0.002)	(1.206)
Groups	1468	1468	1565	1565	1468
Switchers	1008	1008	1062	1062	1008

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table tests the robustness of the baseline specification to alternative definitions of the treatment variable. Column (1) reports the baseline results using cottages per 1,000 population. Columns (2)(4) use the absolute number of cottages instead: column (2) uses the minimum (initial) number of cottages per period, column (3) the mean number, and column (4) the maximum (final) number. Column (5) scales the number of cottages by district land area (cottages per km^2) instead of by population. All specifications follow the baseline model reported in column (4) of Table 3.

Table A2: Robustness to Periodization

	$DeathRate_{it}$			
	(1) Baseline (3-year)	(2) Post-1881	(3) Annual	(4) Annual (moving avg.)
ATT: $CottagesRate_{it}$	-0.145***	-0.145***	-0.137*	-0.122***
	(0.054)	(0.054)	(0.074)	(0.039)
Groups	1468	1468	4502	4528
Switchers	1008	1008	3102	3118

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table assesses the robustness of the baseline results to alternative choices of periodization. Column (1) reproduces the baseline specification using three-year intervals. Column (2) restricts the sample to the post-1881 period. Column (3) estimates the model at the annual level, while column (4) smooths annual variation by using a centered three-year moving average. All specifications follow the baseline model and include district and period fixed effects.

Table A3: By Cause of Death without `same_switchers` option

	Disease Category-specific Death Rate					
	Communicable				*Non-comm.*	*All Other*
	Total (1)	Airborne (2)	Waterborne (3)	Typhus (4)	(5)	(6)
ATT: $CottagesRate_{it}$	-0.090**	-0.081**	-0.005*	-0.004	0.001	-0.010
	(0.036)	(0.034)	(0.003)	(0.004)	(0.004)	(0.029)
Mean DV	5.407	5.015	0.323	0.061	1.434	9.080
AME (%)	-1.658	-1.607	-1.432	-7.321	0.047	-0.108
Groups	1508	1508	1508	1508	1508	1508
Switchers	1038	1038	1038	1038	1038	1038
Period FE	✓	✓	✓	✓	✓	✓
District FE	✓	✓	✓	✓	✓	✓
Clusters	Districts (158)					
Years	1870-1919				1882-1919	1870-1919

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table reports DiD estimates for the effect of cottage construction on mortality from different disease categories, demonstrating robustness to not specifying the `same_switchers` option. The unit of analysis are district-periods. The dependent variable is the average annual disease category specific death rate per 1000 population across the period. Columns (1) to (4) report the effects for different communicable diseases, starting with total deaths, followed by airborne, waterborne and insect-borne (i.e. Typhus). Column (5) reports the effect for non-communicable diseases, and column (6) for other cause-of-death categories. For more details on the disease contained in each category see Table 1.The treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period. All models are estimated using the heterogeneity robust estimator (DID_L) introduced by (Chaisemartin and d'Haultfoeuille 2024). DID_L is estimated over eight post periods. The average marginal effect (AME) is calculated at the mean and is reported as the % change for one unit of treatment. All regressions include district and period fixed effects. The sample period is 1871-1919 in columns (1-4) and (6), and 1882-1919 in column (5).

Table A4: By Age-Sex Group without `same_switchers` option

	Age Group specific Death Rate						
	(1) under-1	(2) 1-4	(3) 5-14	(4) 15-24	(5) 25-44	(6) 45-64	(7) over-65
Panel A: Female							
ATT: $CottagesRate_{it}$	0.078	-0.160*	-0.066***	-0.075	-0.057	0.023	-1.431*
	(0.246)	(0.089)	(0.024)	(0.090)	(0.057)	(0.085)	(0.825)
Mean DV	73.46	15.57	4.04	5.41	8.08	16.18	96.10
AME (%)	0.11	-1.03	-1.63	-1.39	-0.71	0.14	-1.49
Mean DV	73.46	15.57	4.04	5.41	8.08	16.18	96.10
AME (%)	0.31	-1.55	-1.94	-0.14	-0.30	0.19	-0.94
Panel B: Male							
ATT: $CottagesRate_{it}$	0.389	-0.174*	-0.066***	-0.041	-0.002	-0.024	-0.391
	(0.341)	(0.100)	(0.023)	(0.039)	(0.035)	(0.109)	(0.383)
Mean DV	88.88	15.19	3.29	5.21	7.47	16.93	88.53
AME (%)	0.44	-1.14	-2.01	-0.78	-0.03	-0.14	-0.44
Period FE	✓	✓	✓	✓	✓	✓	✓
District FE	✓	✓	✓	✓	✓	✓	✓
Clusters	Districts (158)						

Standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Notes: This table reports DiD estimates for the effect of cottage construction on mortality among different age-sex groups, demonstrating robustness to not specifying the `same_switchers` option. The dependent variable is the average annual age-sex group specific death rate across the period. Population per age-sex are calculated based on the 1901 age structure and annual interpolated population. The death rate for under-1 is defined as deaths under-1 divided by births. All outcomes are scaled to 1000 age-sex population. Panel (A) reports the effect on female, and panel (B) for male death rates. The treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period. All models are estimated using the heterogeneity robust estimator (DID_L) introduced by (Chaisemartin and d'Haultfoeuille 2024). DID_L is estimated over eight post periods. The average marginal effect (AME) is calculated at the mean and is reported as the % change for one unit of treatment. All regressions include district and period fixed effects. The sample period is 1871-1919.

Table A5: By Cause of Death: Certain Infectious

	$DeathRate_{it}$			
	(1) Diphtheria	(2) Whooping Cough	(3) Scarlet Fever	(4) Measles
$CottagesRate_{it}$	-0.001	-0.003	-0.006**	-0.003
	(0.001)	(0.003)	(0.003)	(0.003)
Mean DV	0.059	0.215	0.146	0.113
AME (%)	-2.350	-1.580	-4.379	-2.512
Groups	1230	1230	1230	1230
Switchers	760	760	760	760

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. This table reports DiD estimates for the effect of cottage construction on mortality from disease of the certain infectious category. The unit of analysis are district-periods. The dependent variable is the average annual disease category specific death rate per 1000 population across the period. The treatment variable is $CottagesRate$, the number of cottages per 1000 constructed at the beginning of the period. All models are estimated using the heterogeneity robust estimator (DID_L) introduced by (Chaisemartin and d'Haultfoeuille 2024). DID_L is estimated over eight post periods. We control for compositional change using the `same_switchers` option. The average marginal effect (AME) is calculated at the mean and is reported as the % change for one unit of treatment. All regressions include district and period fixed effects. The sample period is 1871-1919 in columns (1-4) and (6), and 1882-1919 in column (5).

Table A6: Cottages and Medical Provision

Dependent Variable:	Medical Expenditure (1)	Dispensaries (2)	Medical Officers (3)	Midwifes (4)
$CottagesRate_{it}$	30.857	1.499	-0.425	0.145
	(29.999)	(1.554)	(0.394)	(0.172)
Mean DV	999.68	7.80	5.21	3.47
Observations	2053	158	2369	1826

Clustered standard errors at the district level (IDplu) in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table explores whether the construction of cottages coincided with improved medical provision. Each column reports a separate panel fixed-effects regression of the listed outcome on an absorbing treatment indicator equal to 1 if any cottage had been constructed by the start of the period. Columns (1), (3), and (4) include district and period fixed effects; column (2) includes period fixed effects only (dispensary counts are available only in a limited cross-section for some districts). All models are estimated using OLS, and standard errors are clustered by district.

Table A7: Cottages and Housing in 1881

Dependent Variable:	Death Rate per 1000 (All-cause)	
	(1)	(2)
$PoorHousingRate_{it}$	0.069***	
	(0.011)	
$CottagesRate_{it} \times PoorHousing1915[0-20th]$		-0.057***
		(0.018)
$CottagesRate_{it} \times PoorHousing1915[20-40th]$		-0.044**
		(0.020)
$CottagesRate_{it} \times PoorHousing1915[40-60th]$		-0.054**
		(0.021)
$CottagesRate_{it} \times PoorHousing1915[60-80th]$		-0.035
		(0.025)
$CottagesRate_{it} \times PoorHousing1915[80-100th]$		0.017
		(0.043)
Initial Poor Housing Stock FE		✓
Observations	2653	2653

Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Notes: This table examines the relationship between the stock of poor-quality housing and mortality, and how cottage construction affected this relationship. In column (1), the treatment variable is replaced with the share of poor-quality housing at the beginning of each period. This measure is constructed by subtracting newly built cottages from the number of third- and fourth-class houses recorded in 1881. In column (2), we construct a measure of the remaining poor-quality housing stock after the cottage scheme and interact indicators for each quintile of this distribution with the treatment variable. All specifications include district and period fixed effects; column (2) additionally controls for initial poor housing stock fixed effects. Models are estimated using OLS and follow the baseline specification in Equation 1.

Table A8: Spatial Unit Root Correction

	Death Rate$_{it}$				
	(1) Basic	(2) NN	(3) ISO	(4) Cluster	(5) LBM–GLS
$CottagesAny_{it}$	-0.444***	-0.430*	-0.490**	-0.330†	-0.360†
	(0.135)	(0.243)	(0.229)	(0.208)	(0.248)
Observations	2653	2653	2653	2653	2653

Robust standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$, † $p < 0.20$.
Notes: This table implements spatial autocorrelation corrections following Becker et al. (2025). The dependent variable is the average annual all-cause death rate per 1,000 population. Column (1) reports baseline results without correction. Columns (2)(5) apply alternative spatial HAC estimators: nearest-neighbor (NN), isotropic kernel (ISO), clustering on spatial contiguity groups (Cluster), and the local Bartlett kernel with GLS correction (LBM–GLS).

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