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Descriptive Finding

The effects of wealth, occupation, and immigration on epidemic mortality from selected infectious diseases and epidemics in Holyoke township, Massachusetts, 1850−1912

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Abstract

BACKGROUND
Previous research suggests individual-level socioeconomic circumstances and resources may be especially salient influences on mortality within the broader context of social, economic, and environmental factors affecting urban 19th century mortality.

OBJECTIVE
We sought to test individual-level socioeconomic effects on mortality from infectious and often epidemic diseases in the context of an emerging New England industrial mill town.

METHOD
We analyze mortality data from comprehensive death records and a sample of death records linked to census data, for an emergent industrial New England town, to analyze infectious mortality and model socioeconomic effects using Poisson rate regression.

RESULTS
Despite our expectations that individual resources might be especially salient in the harsh mortality setting of a crowded, rapidly growing, emergent, industrial mill town with high levels of impoverishment, infectious mortality was not significantly lowered by individual socio-economic status or resources.

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1. Introduction

Social, economic, and environmental factors all impacted mortality in 19th century Europe and the United States. The type of place (rural or urban, industrial or non-industrial area, stable or growing population) was a powerful predictor of mortality, especially for children, even when socioeconomic status (SES) is taken into account (e.g., Preston and Haines 1991; Vinovskis 1981; Preston and Van de Walle 1978). The effects of structural and social environmental mechanisms (sanitation, overcrowding, occupational structure, population stability) also varied within geographic regions (e.g., Preston and Van de Walle 1978; Melosi 1999). Yet individual-level socioeconomic circumstances influenced mortality across the wider disparities and inequalities of geographic circumstance in 19th century society (e.g., Condran 1995; Garrett et al. 2001; Preston and Haines 1991), and may have even been most salient where other circumstances were most dire (Reid 1997). We sought to expand on our own previous work in 19th century Massachusetts towns, by asking whether individual-level socioeconomic factors influenced mortality from infectious diseases characterized by epidemics in emerging urban, often stressful and harsh, environments.

Holyoke, Massachusetts is located on the Connecticut River in the western portion of the state. The township was a planned industrial community and experienced rapid growth from the 1850s through 1910s, fueled primarily by immigration from Ireland, Canada, and Eastern Europe. Structural issues related to sanitation and overcrowding were continual problems in Holyoke during the last half of the 19th century and led to numerous recorded epidemic outbreaks (Green 1939). The industrial structure of the township created a high level of economic and social inequality, with immigrants entering primarily to work in low-paying mill jobs. The constant influx of new workers further impacted the township's demography by maintaining a relatively young population (Leonard, Beemer, and Anderton 2012). Immigrants of Irish and Canadian origin then experienced an accumulation in both personal and real estate wealth at a fast rate, eventually bringing their level of wealth close to that of the native-born. We argue that this accumulation of wealth over time represents a maturation effect, whereby later immigrants from a specific country of origin benefit from established social connections and resources of prior co-nationals. Overall mortality was high during periods of rapid growth, and only recent immigration significantly lowered the likelihood of death.

2. Data and methods

We utilized two datasets for this analysis. The first dataset includes records of all deaths in Holyoke between 1850 and 1912. The second dataset is an urban-based geographic sample of Federal census records, linked to property tax information and death records in the year following the census, allowing us to analyze personal and family characteristics not available from the death records. Gutman (1956) demonstrates the extraordinary completeness of Massachusetts death reporting and previous research (e.g., Leonard, Beemer, and Anderton 2012) demonstrates representation of the impoverished within the tax-linked dataset. We looked at trends in mortality rates for the entire population using the first dataset, and modeled mortality rates for the sampled census population using Poisson rate regression. Cause-specific counts of deaths are the numerators of the rates, with interpolated census population counts as the denominators. The model presented here was developed in prior research and applied to overall adult and child mortality (Leonard, Beemer, and Anderton 2012). In this note we replicate the same model, limited to mortality from the important infectious diseases of the time in this place.

We focused on infectious diseases that caused significant epidemics in the 19th century United States (Condran and Murphy 2008) and were continually present in Holyoke between 1850 and 1912. The ten causes in our analysis include eight infectious diseases listed in the first edition of the International Classification of Disease: diphtheria and croup, influenza, measles, meningitis, scarlet fever, smallpox, typhoid, and whooping cough. We combine cholera nostra, dysentery, and diarrheal enteritis into a single category of diarrheal causes, and include tuberculosis because it is a contagious disease that was a consistent cause of excess mortality during this time.

We control for age effects on mortality by dividing our data into two broad age groups: less than 15 years of age (children) and 15 years and older (adults). The age cutoff was chosen for consistency with previous work and to allow robust analysis with small sample sizes.

Previous research (Leonard, Beemer, and Anderton 2012; Anderton, Beemer, and Leonard 2005) found that estimating wealth effects on general mortality in this population without an effort to control for healthy worker selection effects among more recently arrived immigrant groups can "confound selection effects from recently arriving immigrants with effects of later periods when the same immigrant group was a more stable residential population", resulting in longitudinally inconsistent and erroneous results (Leonard, Beemer, and Anderton 2012: 447). Since census data do not record actual individual-level time of arrival over most of the study period, we follow our prior research in using a nested interaction of ethnic group and periods of peak immigration as a proxy for a 'healthy migrant' effect on adult mortality. The interaction

is specified by three dichotomous categories: born in Ireland and observed before 1870 when Irish immigration declined, born in Canada and observed before 1880 when Canadian immigration declined, and born in Eastern Europe, which was substantial only in our last study periods. These categories roughly proxy likely recent foreign-born arrivals with significant selection effects. Conversely, the excluded category includes all those less likely to be recent arrivals from abroad than those observed during one of these three substantial immigration waves, including US-born internal immigrants, and less likely subject to the same substantial selection effects. For convenience we label this excluded category simply as "other residents and immigrants".

Two measures of wealth and one of income represent resources available within families. We created two dichotomous variables indicating any taxable real estate and any taxable personal estate of the family (as identified in the census). Previous research indicated that these forms of wealth can have different effects. To test the effect of income, we summed the IPUMS variable 'occscore' over the census family unit. Occscore is the median wage for specific occupations in 1950 (IPUMS-USA 2015). We use it as a relative ranking of income, not an absolute amount of income.

We considered several different variables to measure occupational effects of census family head. First we tested the broad industry categories from the 1880 census occupational classification system. These categories were not significant, most likely because classification is industry-based, grouping high and low income occupations together. We then identified occupations with the lowest median incomes within each 1880 industry category, defined as the bottom quartile of the value of occscore. These measures were also not significant. In our final model we include the dichotomous variable which has the most salience and combines the lowest scored occupations within "trade and transportation" and "manufacturing, mechanical, and mining" industries, compared to all other occupations.

One might also expect crowding within dwellings to have an effect on the spread and intensity of these infectious diseases. However, urban sample areas of Holyoke had many tenement houses with uniformly high density (individual mean dwelling size of 36.9 persons per dwelling and lower quartile range of 13 persons per dwelling). No significant relationships with dwelling density were found and it is not included in the models presented.

3. Results

3.1 Population-level trends

Figure 1 shows rates for the combined selected causes, combined infectious causes (tuberculosis excluded), and all other causes, with population. Mortality from the selected causes increased sharply in the late 1860s, peaking in 1872 at a rate of 23.36 deaths per 1,000. Mortality from infectious causes and tuberculosis started declining in the 1870s, despite the rapidly increasing population. The transition to predominately non-infectious causes occurred around 1890.

Figure 1: Combined selected cause, combined infectious and other cause mortality rates, per 1,000, and population

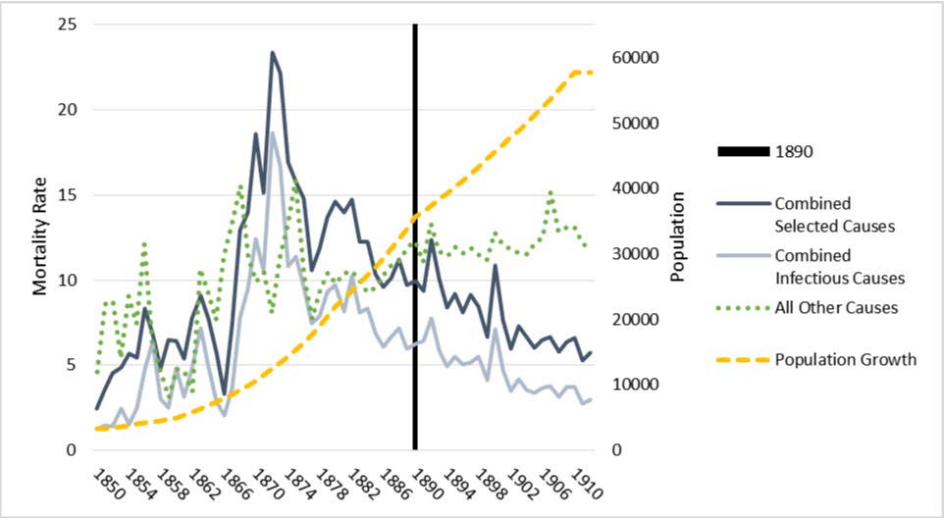

Figure 2 shows cause-specific rates of excess mortality, and highlights the concurrent nature of epidemic mortality from different causes. Two categories, tuberculosis and diarrheal causes, consistently make up the majority of the deaths. Outbreaks of diphtheria and croup, measles, scarlet fever, smallpox, and typhoid mainly occurred prior to 1890, and multiple infectious causes contributed to periods of high excess mortality.

Figure 2: Mortality rates for selected causes in the total population per 1,000, 1850−1912

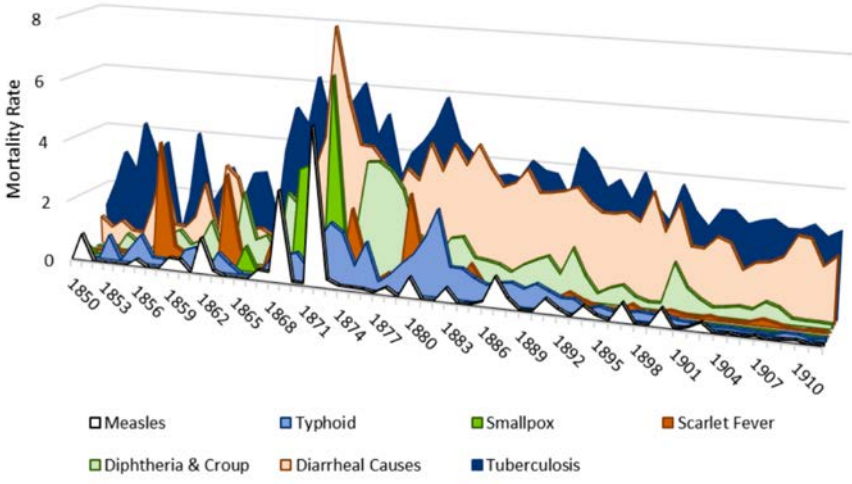

Table 1 shows cause-specific mortality by age group and period. Tuberculosis was the leading cause of death for adults in both time periods, with diarrheal causes the leading category for children. Mortality declined between periods for both age groups in all selected causes except meningitis and influenza.

Table 1: Cause-specific mortality by age group, per 1,000

	Pre-1890		1890 & Later	
	Children[1]	Adults	Children[1]	Adults
Diarrheal Causes	10.00	0.43	7.05	0.25
Diphtheria & Croup	4.50	0.02	1.60	0.01
Influenza	0.02	0.00	0.12	0.19
Measles	1.17	0.04	0.42	0.01
Meningitis	1.30	0.12	2.00	0.17
Scarlet Fever	1.72	0.02	0.25	0.00
Smallpox	0.91	0.18	0.00	0.00
Typhoid	0.56	0.93	0.04	0.24
Tuberculosis	2.60	4.03	2.25	3.09
Whooping Cough	0.87	0.00	0.41	0.00
Combined Selected Causes	23.65	5.78	14.15	3.99
All Other Causes	17.69	5.92	18.19	8.43

[1] Children are aged less than 15 years.

3.2 Poisson models

As in prior studies, we modeled the risk of mortality from the selected causes using Poisson rate regression. Table 2 presents the results, reported as incidence rate ratios. The linked dataset contains death data for the year following the decennial census, and thus captures peak epidemic periods only when they coincide with these years.. The geographic sampling frame of the linked dataset differs from the population-level data, but mortality trends observed in the datasets are similar.

Table 2: Incidence rate ratios of death from combined selected infectious mortality from the final Poisson models

	Under 15	15 & Older
Decade		
1860[1]		
1870	2.84 ***	1.67
1880	1.50	2.27 **
1900	2.08 *	1.21
1910	0.62	0.51 *
Wealth and Occupation		
Family wealth: any personal estate	0.61	1.31
Family wealth: any real estate	1.53 *	1.64 **
Family occupation score	1.00	0.99 *
Low SES T&T and MMM occupations[2]	1.83	0.20
Healthy Worker Proxy and Nativity		
Irish (1860-70)	-	0.22 *
Canadian (1860-80)	-	0.43 **
Eastern European (1860-1910)	-	0.19 *
Other residents and immigrants[1]		
Non-native	0.31 ***	-
US-born[1]		
Likelihood ratio χ^2	82.95 ***	106.25 ***
n of observations	7,613	15,908

*p <.05, **p<.01, ***p<.001; [1]Reference category; [2]SES = socio-economic status, T&T = trade and transportation occupations; MMM = manufacturing, mechanical, and mining occupations (as categorized in the US Census of 1880).

As expected, the early decades had higher mortality from the selected causes in both age groups. Similar to the population-level data, the model indicates that the largest period effect occurred during the decades corresponding to the highest combined selected cause mortality, 1865−1885. The significant effect for 1900 among children reflects the last distinct outbreak of diphtheria and croup in combination with a high rate from diarrheal causes, as seen in Figure 2.

Given our initial reason for this analysis, an unexpected finding was the lack of significance for personal wealth effects on mortality for both children and adults. Given the high level of economic inequality in Holyoke we expected personal wealth to have a beneficial effect for those who possessed it and increase mortality for those without, but our data indicates that even in places of high economic disparity, where salience could be expected to have been greatest, individual wealth may not offer an advantage for avoiding death from infectious diseases.

Largely because family real estate wealth increases with time since immigration into the town and with age, our previous research using all causes of death suggests that mortality advantages of healthy immigrants decline as real estate wealth increases. This is not causal: it is the general effect of the passage of time operating on both phenomena, and is not limited to international migrants. Thus, family real estate wealth significantly and substantially increased the likelihood of death while the three variables designed to assess the healthy migrant effect (country of birth and decade of peak migration) are all significant and reduce the likelihood of death from combined selected causes in Holyoke. Real estate wealth could also indicate residing away from the extensive company tenements in the heart of town, but again the expected mortality direction would be positive, not negative.

The work of Reid and colleagues (Reid 1997; Garrett et al. 2001) suggests socioeconomic effects were perhaps even larger in homogeneously heavy industrial areas of England and Wales. Based on this we expected that a higher family occupational score would be especially salient in lowering the likelihood of death in Holyoke, with its high concentration of industrial mills. However, the sum of family occupation scores, as a proxy for SES effects, was not significant in the model.

4. Conclusion

The period of rapid growth in the emergent mill town, with city-wide crowding, industrial mills, pervasive inequality, and lagging public health infrastructure saw significantly higher mortality overall, with frequent epidemics. Infectious mortality was not significantly lowered by individual socio-economic status measured through taxable personal or real estate wealth, occupation-based related measures, or proxies for family income. The only protective effect against infectious disease mortality was for recent immigrants. Over time, residents retained their group membership (through birthplace) but lost these protective effects. At the same time, real estate wealth grew among some of these stable residents, which in effect makes wealth accumulation a proxy for the loss of healthy migrant status among earlier in-migrants. These are similar results to those for our studies of overall mortality, and perhaps they are unsurprising, given the levels and etiology of infectious and epidemic mortality. Social and structural environmental factors at the community level impacted excess mortality from selected infectious causes, but individual resources appear to have offered little protection.

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