Epidemics in Motion. Exploring the Interaction between Childhood Diseases in a Norwegian City, 1863–1928

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Epidemics in Motion

Exploring the Interaction between Childhood Diseases in a Norwegian City, 1863–1928

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ABSTRACT

This article explores interactions between measles and whooping cough in Christiania (now Oslo) during the period 1863–1928, using annual morbidity, mortality, and fertility data. Drawing on the ecological interference model proposed by Rohani et al. (2003), we examine whether epidemic patterns shifted from so-called out-of-phase to in-phase dynamics as fertility declined and the pool of susceptibles decreased. Dividing the analysis into two periods based on crude birth rates, we find that during the high-fertility era, disease cycles were typically out of phase. Surprisingly, this dynamic persists even in a period with comparatively lower birth rates, contradicting theoretical expectations. We discuss potential explanations, including population size thresholds for transmission and limitations in the available data. Modest in scope but exploratory in spirit, the study contributes to ongoing efforts - such as those initiated by Angélique Janssens — to use historical health data to understand long-term epidemic dynamics and inter-disease relationships.

Keywords: Ecological interference, Measles, Whooping cough, Case fatality rates, Historical populations

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1 INTRODUCTION

A marked decrease in urban mortality was a key driver of the overall reduction in death rates in late 19th-century Norway. During the early stages of this transition — specifically before and around its onset, between 1860 and 1920 — Norway consistently recorded some of the lowest crude death and infant mortality rates in Europe. Airborne diseases emerged as major contributors to the decline, accounting for over one-third of the reduction observed in Christiania, the historical name of Norway's capital, now known as Oslo. Notably, nearly half of this decrease stemmed from reductions in mortality due to common childhood illnesses such as diphtheria, measles, scarlet fever, and whooping cough (Hubbard, 2002). This underscores the crucial role of declining childhood mortality in the broader urban mortality transition.

The historical interplay between infectious diseases has long shaped patterns of mortality, vulnerability, and survival. With the growing availability of digitized, individual-level cause-of-death and morbidity data, researchers are now in a position to revisit these dynamics with new tools and perspectives. This contribution explores how interactions between infectious diseases — so-called ecological interference — may have influenced epidemic trajectories in the past. We propose that historical time series on reported illness, cause-specific deaths, and births offer a compelling opportunity to explore how one pathogen can constrain or shape the trajectory of another.

This approach builds on recent insights from ecology and epidemiology, but also aligns closely with emerging directions in historical demography and the social history of medicine. It reflects a broader move away from studying diseases in isolation, toward understanding their interaction before, during and after the demographic and epidemiological transition.

This perspective also resonates with the scientific legacy of Angélique Janssens, whose work has significantly shaped the field of historical demography. Her research brought together gender, health, mortality, and the overall impact of infectious diseases (Janssens, 2016). Her leadership in major research projects such as Studying the history of health in port cities (SHiP) and Lifting the burden of disease exemplifies a dedication to interdisciplinary research. It is in this spirit that our study seeks to push methodological boundaries and examine mortality patterns through the lens of inter-disease dynamics.

2 ECOLOGICAL INTERFERENCE

We draw our inspiration from an exploratory exercise by ecology experts Rohani and colleagues, entitled Ecological interference between fatal diseases, published in *Nature* in 2003 (Rohani et al., 2003). In this letter, they present a technically elaborate analysis, but at its core lies the idea that specific infectious diseases interact or "compete". Measles, for example, typically occurs only once in a person's life, due to the combination of initial susceptibility at birth and lifelong immunity after infection. For measles to circulate, a continuous supply of susceptible children is needed. This so-called pool of susceptible children (the population at risk for catching measles) is replenished through births and depleted by measles infections or deaths prior to infection.

If some of these susceptible children are temporarily bedridden with another infection — Rohani et al. use whooping cough (*pertussis*) as an example — they are removed from active circulation. Their illness reduces their social contact, effectively shrinking the susceptible pool for measles. This, in turn, slows the spread of measles. Likewise, during a measles outbreak, whooping cough might spread more slowly for similar reasons. This phenomenon is referred to as ecological interference. Rohani et al. argue that this interaction is not purely theoretical: their modelling suggests that the effect should be detectable, and their data analysis provides empirical indications of its presence.

Building on this framework, our study investigates whether ecological interference between measles and whooping cough can be observed in a historical setting. Specifically, we test two hypotheses (this will be discussed in more detail in section 3):

Out-of-phase epidemic dynamics: During periods of high fertility, when the pool of susceptible individuals is rapidly replenished, measles outbreaks reduce the number of susceptibles available to whooping cough, resulting in alternating or negatively correlated epidemic cycles.

In-phase epidemic dynamics: During periods of low fertility, the slower replenishment of the susceptible pool allows measles and whooping cough outbreaks to occur simultaneously, resulting in positively correlated epidemic patterns.

A second, related line of inquiry draws from the more recent work of Mina and colleagues (Mina et al., 2015), who demonstrate that measles infection can suppress the immune system for up to several years, increasing vulnerability to other infections (see also Mercer, 1990). From this perspective, we might expect measles outbreaks to be followed by increased mortality from other infectious diseases, including whooping cough.

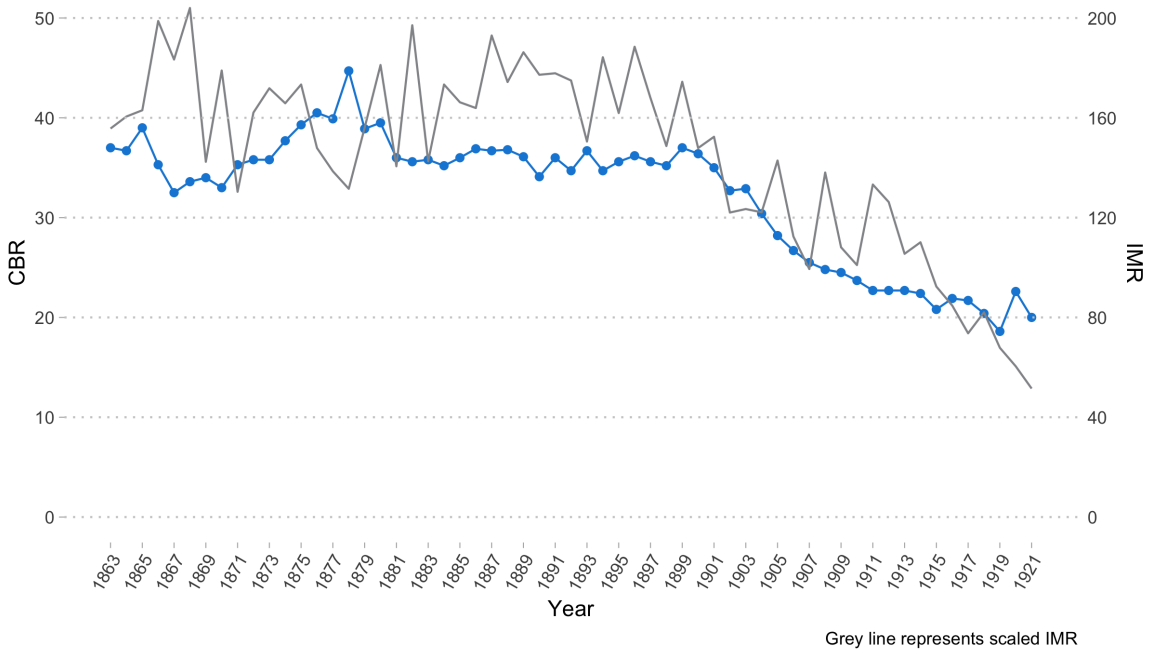
Ultimately, these types of diseases should not be studied in isolation, but in relation to each other. Like Rohani et al. and Mina et al., most authors in the medical sciences stress how rare it is to find data sources robust enough to properly test hypotheses like these. However, historical individual-level cause-of-death data, in combination with morbidity records, might offer opportunities to do so. By exploring these interactions in historical Christiania, we aim to contribute to a growing body of research that seeks to understand disease dynamics through the lens of ecological interaction.

3 CONCEPTS AND DATA

Like Rohani et al., our exploration is based on measles and whooping cough. As stated, measles provides lifelong immunity. Experiencing a measles infection also causes broader immune suppression, which can last for up to two years in those who survive the illness. People who have had whooping cough acquire some immunity to future whooping cough infections (Rohani et al., 2003). Other infectious diseases, such as scarlet fever, pose a slightly different challenge. Immunity after infection is not always lifelong, and the pathogen can circulate without clear clinical symptoms. It is plausible that children sick at home with scarlet fever might reduce measles transmission, but measles might not significantly influence scarlet fever dynamics in return.

We use data from Christiania to explore the ecological interference hypothesis. The dataset was specifically constructed for this study and includes annual counts of births, infant deaths, population figures, counts of measles and whooping cough cases, as well as deaths attributed to both diseases, covering the period 1863–1928.[1] On average, there were 4,591 births per year. The average population of Christiania during this period was approximately 175,000.

Figure 1 *Crude fertility rates and infant mortality rates, Christiania 1863–1928*



1 Data on infant mortality beyond 1921 are lacking.

Morbidity data were drawn from annual medical reports submitted by district physicians. Beginning in 1861, these medical officers were required to report cases of acute diseases with epidemic potential to the city's health commission on a monthly basis. The reporting form included details such as the patient's name, age, occupation, address, diagnosis, date of onset, suspected cause, and treatment administered.[2] This practice coincided with Norway's adoption of its first official nomenclature and cause-of-death certificates (Sommerseth, 2023).

Mortality data were derived from the same medical reports. Until 1874, death counts were based on priest burial registers; from 1875 onwards, they were drawn from doctor-issued death certificates. The medical reports also noted the coverage rate of these certificates, which ranged between 93% and 98% annually when compared to priest-reported deaths.

It is important to interpret these sources with caution. The number of reported infections is likely underestimated, which may result in an overestimation of the case fatality rate.

How can we conceptualize the pool of susceptible individuals in a historical urban setting? Measles has a very limited survival time outside the human body. It requires a continuous supply of new, non-immune individuals to sustain transmission. In populations with low birth rates and minimal immigration of non-immune individuals, the virus cannot maintain itself and will likely disappear before causing a major outbreak.

Following Rohani et al., we use crude birth rates to approximate the recruitment of new susceptibles. A key limitation of using crude rates is that infants must survive their first year of life to become part of the susceptible population: a factor that changed significantly over time. In Christiania, for example, the annual infant mortality rate (IMR) shows a clear downward trend beginning in the early 1900s. While the IMR exceeded 200 per 1,000 live births in the early 1860s and continued to fluctuate for decades, it began to decline more steadily in the twentieth century. By the early 1900s, the IMR consistently fell below 150, and after 1910 the decline became more pronounced, reaching approximately 50 per 1,000 by 1921 (see Figure 1). As a result, not all newborns entered the pool of susceptibles for infections such as measles and whooping cough.

Another crucial consideration is that even after surviving infancy, children remained at risk of dying from other infectious diseases, such as scarlet fever and diphtheria. These should be regarded as competing risks. As a result, estimating the number of children who lived long enough to become susceptible to infections like measles or whooping cough is highly challenging, particularly in contexts where a significant proportion of children succumbed to other diseases before reaching the typical age of infection. In such cases, arriving at a reliable estimate of the susceptible population may be difficult, if not impossible.

Despite relatively high infant mortality, the population of Christiania doubled from 112,000 to 221,000 between 1878 and 1898. This growth was driven by boundary expansion in 1877, a surplus of births over deaths, and the immigration of young people seeking employment. Fertility began to decline in the early 1890s (see Figure 1), leading to a noticeable slowdown in population growth toward the end of the period.

As suggested by Rohani et al., we introduce the concepts of *in-phase* and *out-of-phase* epidemic dynamics to describe the temporal relationship between two diseases competing for the same pool of susceptible individuals. In-phase refers to epidemics of two diseases occurring simultaneously, while out-of-phase indicates alternating outbreaks, presumed to be driven by ecological interference. This raises the question: how can interference be detected in our historical data, and are there signals in our data indicating trends of synchronized or staggered epidemics over time?

When birth rates were high, measles (being more contagious) tended to infect and remove susceptible individuals more quickly, thereby delaying the onset of whooping cough outbreaks. This dynamic resulted in alternating epidemic cycles, or out-of-phase patterns, typically reflected in a negative correlation between the case numbers (or case fatality rates) of the two diseases, as a function of fertility levels. In practical terms, one disease would peak while the other declined. In contrast, when birth rates were low and fewer new susceptibles entered the population, measles and whooping cough outbreaks tended to occur simultaneously (i.e., in-phase) with deaths from both diseases rising and falling together on an annual basis.

2 A pilot project at HistLab, UiT The Arctic University of Norway, is underway to digitize the sources and link them to the Historical Population Register of Norway.

4 EXPLORING ECOLOGICAL INTERFERENCE IN HISTORICAL CHRISTIANIA

Figure 2 shows the annual (squared) case fatality rates for measles and whooping cough in Christiania between 1863 and 1928. The graph illustrates how the two diseases fluctuated over time, with noticeable peaks and declines. While both exhibit epidemic cycles, their timing appears to vary, suggesting potential out-of-phase dynamics, particularly in the earlier period.

While a visual inspection of the epidemic curves suggests potential phase relationships between measles and whooping cough outbreaks, we complement this with a more systematic approach by calculating Pearson correlation coefficients. This allows us to quantify the strength and direction of the relationship between the two diseases across different fertility regimes.

To do this, the analysis is divided into two distinct periods, based on differing levels of crude birth rates: *Period 1*, marked by higher birth rates, and *Period 2*, characterized by comparatively lower birth rates. Period 1 spans from 1863 to 1901 and is characterized by relatively high and fluctuating fertility levels, averaging around 35 births per 1,000 inhabitants. Period 2, from 1902 to 1928, is marked by a sharp and sustained decline in fertility, dropping from 32 to 7.2 births per 1,000 inhabitants in less than three decades.

Within each period, crude birth rates are further subdivided into defined intervals (see Figure 3). For each interval, we calculate a correlation coefficient, representing the strength and direction of the relationship between the fatality rates of the two diseases. The symbols displayed in Figure 3 indicate the number of observations (i.e., case fatality rates) per interval, offering insight into sample size and the robustness of the correlation estimates.

We expect to observe a negative correlation between measles and whooping cough fatality rates during Period 1, when birth rates were high. This would reflect out-of-phase epidemic dynamics, in which one disease suppresses the other by depleting the shared pool of susceptible individuals. In contrast, during Period 2, we anticipate a shift toward a positive linear relationship, indicating in-phase behaviour as the recruitment of susceptibles declines.

Figure 2 *Square of Case Fatality Rates for Measles and Whooping Cough, Christiania 1863–1928*

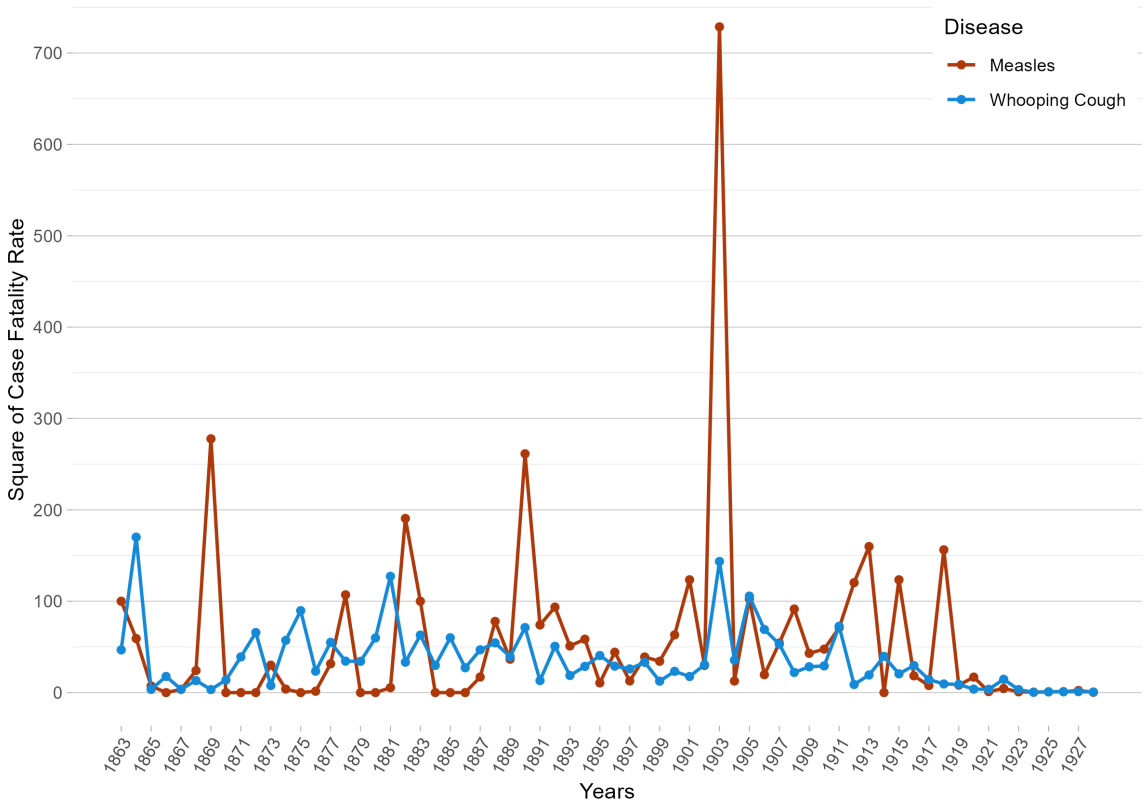
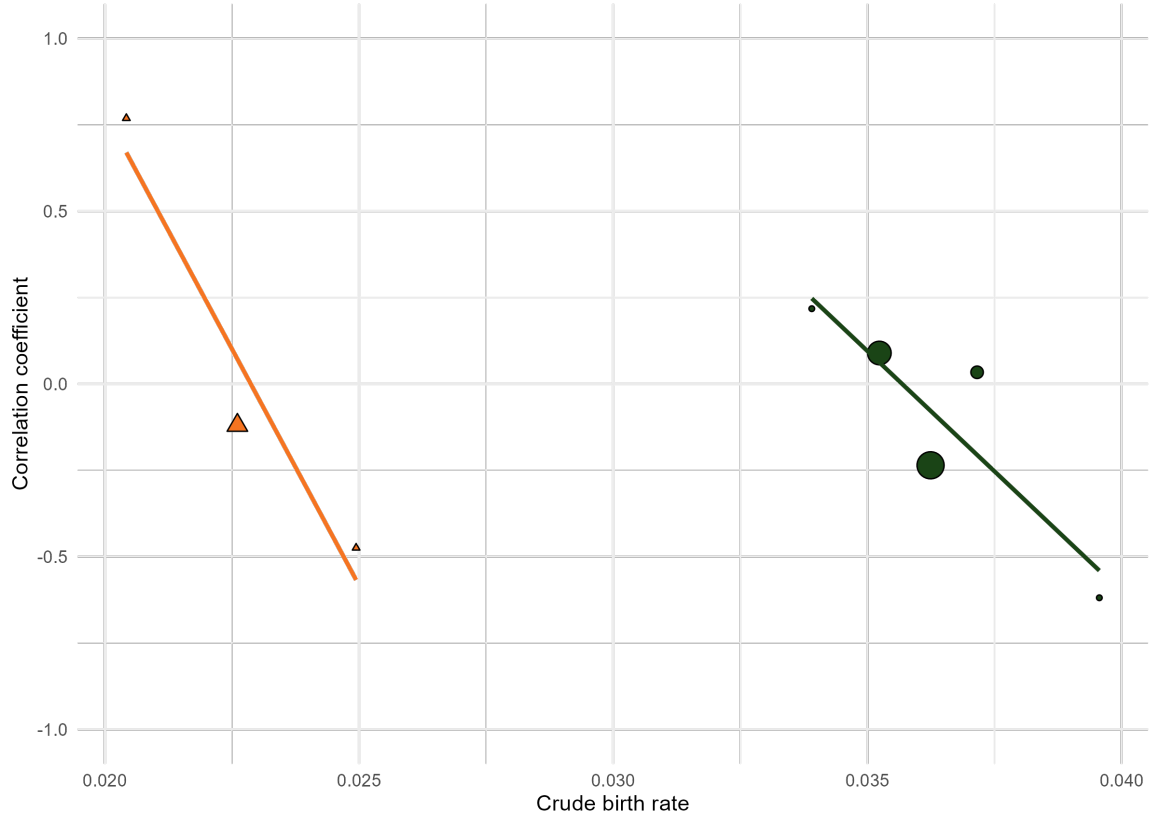


Figure 3 *Correlation coefficients between measles and whooping cough deaths as a function of the crude birth rates. Green symbols/lines refer to the 1863–1901 era; orange symbols/lines represent the period 1902–1928*



Our results show that during the first period — marked by high and fluctuating crude birth rates — the correlation between the two diseases tends to decline. This downward trend suggests that in a high-fertility context, an increase in measles mortality is often accompanied by a decrease in whooping cough mortality, and vice versa, indicating out-of-phase epidemic dynamics. In the second period, marked by a sharp and sustained decline in crude fertility, we observe a similar negative correlation. This is at odds with the findings of Rohani et al., who suggested that in low-fertility settings, epidemic patterns for measles and whooping cough tend to align annually, resulting in in-phase dynamics.

5 CONCLUSION AND DISCUSSION

This exploratory analysis set out to investigate the interaction between two major childhood infectious diseases in historical Christiania, with a particular focus on how fertility dynamics shaped their epidemic patterns. One of the central insights to emerge from this study is that examining infectious diseases in isolation may obscure the complex ecological relationships that govern epidemic behaviour.

Our findings support this view. In the first period (1863–1901), marked by relatively high and fluctuating crude birth rates, we observe a downward trend in the correlation coefficients between measles and whooping cough fatality rates. This suggests an out-of-phase dynamic, where increases in mortality from one disease tend to coincide with declines in the other. In the second period (1902–1928), characterized by a sharp and sustained decline in fertility, we observe a similar negative correlation, contrary to the expectations derived from the ecological interference model developed by Rohani et al. Their work predicts a shift toward in-phase dynamics in low-fertility settings, as both diseases would draw on the same limited pool of susceptibles at the same time.

One potential explanation for this discrepancy lies in the demographic context of Christiania. It is plausible that the city's total population was simply too small to sustain continuous endemic transmission. As Mercer (1990) has argued, measles may require a minimum population of around

7,000 individuals for persistence, and potentially 250,000 to support truly endemic transmission. Christiania's population may not have met this threshold, thereby limiting the ecological space in which a clear phase shift could emerge.

While the ecological interference model remains a powerful theoretical framework, our analysis does only represent a simplified approximation. A more nuanced understanding of epidemic dynamics would require morbidity data at a higher temporal resolution (preferably weekly or monthly), allowing for the inclusion of key epidemiological parameters such as contact rates, latency, infectious periods, and recovery times. Population structure also remains a critical factor in determining whether diseases persist or fade out.

Finally, our reliance on crude fertility rates introduces a methodological limitation. These rates encompass the entire population and do not account for age-specific fertility or infant mortality, both of which are essential to estimating the true number of children entering the susceptible pool. Future research should seek to incorporate age-specific fertility rates and survival adjustments in order to generate more realistic estimates of susceptibility. Moreover, the current dataset, while valuable, does not fully encompass a long enough time span to capture the full "arc" of fertility transition — from high to transitional to low fertility. Extending the temporal scope of the data would allow for a more nuanced analysis of how changes in population structure and birth dynamics shaped epidemic patterns over time. A refined demographic approach would contribute to a deeper and more accurate understanding of disease dynamics in historical urban populations.

This study, modest in scope and exploratory in spirit, aligns with the initiatives led by Angélique Janssens to continue the systematic collection of data on illness and causes of death.

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