The Rhythm of Death. Seasonality of Mortality in Amsterdam, 1812–1931

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The Rhythm of Death

Seasonality of Mortality in Amsterdam, 1812–1931

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ABSTRACT

Between 1812 and 1931, Amsterdam experienced profound demographic, social, and epidemiological changes that reshaped how, when, and why people died. By tracing seasonal mortality patterns over this time period, trends in the rhythm of death are explored in our study. Using monthly death counts from municipal yearbooks and the Amsterdam Cause-of-Death Database, and applying wavelet power spectrum analysis, we identify both persistent winter excess mortality and key disruptions caused by epidemics. For the period 1856–1891, for which continuous cause-specific data is available, our findings reveal that, although airborne infectious diseases largely shaped the winter mortality peak, excess winter deaths remained evident even after their removal. This suggests the important role of other causes-of-death, such as cardiovascular diseases, which are caused by other factors than seasonal viruses. Beyond these findings, it is argued that environmental exposures, such as temperature and reduced sunlight, alongside social inequalities in shaping seasonal vulnerability should be taken into account in future research.

Keywords: Mortality, Seasonality, Amsterdam

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1 INTRODUCTION

The diseases that people contract, and ultimately die from, reflect the conditions in which they lived. The same holds true for when, during the year, they pass away. Mortality is not distributed evenly across the calendar; instead, it follows a rhythm shaped by environmental pressures, economic hardships, cultural practices, and biological vulnerabilities. Studying the seasonality of death provides insights into how historical populations were affected by factors such as food scarcity, temperature extremes, epidemic outbreaks, and social customs related to work, migration, and caregiving. By examining mortality trends in Amsterdam in the 19th and early 20th century, our study seeks to explore these seasonal dimensions of death and their broader implications. In other words, our aim is to investigate how mortality rhythms changed over time and what they reveal about living conditions in the past. Were there consistent seasonal peaks in deaths, and if so, can we hypothesize what forces drove them?

Earlier research has demonstrated that in temperate climates such as Europe, mortality rates tend to peak during winter months, a phenomenon known as excess winter mortality (Fowler et al., 2015). Three main factors help explain why winter remains such a lethal season. First, many viruses, including influenza, follow a seasonal cycle and reach their peak during winter, accounting for a significant portion of these excess deaths. That many other excess winter deaths are from cardiovascular causes provides a second likely factor. Cold temperatures have biological effects that increase the risk of blood clots, coronary thrombosis, and respiratory infections. However, despite large temperature differences between Northern and Southern Europe, winter mortality rates are surprisingly similar. This suggests that people adapt to their local climates, meaning the relative impact of cold weather is felt similarly across regions (The Eurowinter Group, 1997). More recent comparative studies, including those involving very warm countries, suggest that temperature alone does not fully explain excess winter mortality. Reduced exposure to sunlight, and consequently lower Vitamin D production, may also play a related role. Finally, behavioral and environmental changes in winter, such as dietary shifts, decreased physical activity, air pollution, varying humidity, and even cultural events, may contribute as well to excess mortality, but are almost never studied from this perspective (Kinney et al., 2015).

Relatively few historical studies have focused specifically on these kinds of seasonality effects, despite the extensive body of studies examining mortality in the past (Ekamper et al, 2009; Kunst et al., 1991). While many adopt a descriptive approach (e.g., Lee & Son, 2012, on Korea; Williams, 1992, on Sheffield, UK), some employ statistical models, such as Cox proportional hazard models and time series techniques, to analyze seasonal mortality patterns. One key finding is that the seasonality of mortality is not necessarily stable over time. For instance, in Germany (1946–1995), winter mortality persisted but declined in intensity and sensitivity to cold (Lerchl, 1998). In Northern England, infant deaths shifted from winter peaks in the early 19th century to summer peaks by the century's end (Huck, 1997), whereas in 18th-century London, the reverse trend was observed (Landers & Mouzas, 1988). Others have therefore argued that changes in mortality seasonality can signal broader social, environmental, and public health transformations. The reduction of winter excess mortality over the 20th century is often linked to improved heating systems (Lerchl, 1998; Staddon et al., 2014), while the rise of summer infant mortality in the 19th century correlates with declining breastfeeding and increased waterborne infections (Huck, 1997). Public health measures, such as water sanitation improvements in the late 19th and early 20th centuries, likely weakened summer peaks in infant deaths (Macassa et al., 2006; Peltola & Saaritsa, 2019). Additionally, shifts in epidemic disease patterns, such as smallpox seasonality, have been associated with vaccination programs (Krylova & Earn, 2020). In short, seasonal mortality patterns are shaped by a complex interplay of biological, environmental, and societal factors, making their historical analysis crucial for understanding long-term trends in public health.

Our study contributes to our current understanding by taking a long-term perspective (covering the years between 1812 and 1931) to examine how the seasonality of all-cause mortality developed over time. This period was one of profound transformation, marked by industrialization, urbanization, medical advancements, and shifts in public health policies, all of which reshaped patterns of disease and mortality. In particular, Amsterdam underwent the epidemiological transition, shifting from a mortality regime dominated by infectious diseases to one increasingly driven by chronic conditions (Lammertink, 2023; Stalpers et al., 2021). These changes likely influenced not only how people died, but also the seasonal patterns of their vulnerability. Moreover, our study is able to take a closer look at how patterns may have changed during the period 1856–1891 by focusing on deaths due to specific causes such as respiratory diseases, diarrheal infections, or cholera and smallpox epidemics. It allows us to assess more closely whether the decline of infectious diseases altered the seasonal rhythm of death and whether certain causes of

death were more sensitive to seasonal influences than others. In doing so, we build on earlier observations that the effects of the assumed decline of infectious diseases and rise in non-infectious diseases as part of the epidemiological transition may require further revisions (Wolleswinkel-van den Bosch et al., 1997).

By examining these seasonal patterns of mortality, we not only explore how environmental, medical, and societal forces might have shaped life and death in Amsterdam but also contribute to a broader historiographical tradition that seeks to contextualize health and disease of ordinary people within their social fabric. It is in this spirit that we dedicate this article to Angélique Janssens, whose pioneering research over the past decades has greatly enriched our understanding of historical mortality and health. As she retires, her legacy continues to inspire scholars in historical demography, reminding us that the study of death is, at its core, a study of life.

2 SOURCES, DATA AND METHODS

For our study we use two types of sources. The first are the Statistical Yearbooks of Amsterdam in which the total number of annual and monthly deaths are reported. While the Statistical Yearbook 1905–1909 (Bureau van Statistiek der Gemeente, 1910, pp. 108–115) covers the information for the period 1812–1908, information about later years are coming from the following individual yearbooks. The second source is the Amsterdam Cause-of-death Database (ACD) (Janssens et al., 2023), which covers the period between 1854 and 1940. However, we limit ourselves to the period 1856–1891 because the analysis depends on a complete sequence of annual and monthly data. Missing information (1855 and from January 1892 to June 1894) therefore preclude the possibility of performing the analysis before or after the study period. Still, although shorter, this period still covers the start of the mortality decline in the 1880s. After removing stillbirths (N = 22,632), observations without address (N = 4,770)[1] and cause of death (N = 4), the remaining death count is 272,344.

To explore how mortality seasonality evolved in Amsterdam, the percentage of deaths in each month and the excess winter mortality is calculated first. The latter is done by calculating the excess winter death index (EWDI) and can be interpreted as the difference between the number of deaths in winter months (December to March) and the average number of deaths in non-winter months (average from August to November of the previous year and from April to July in the year of interest) expressed as a percentage of the latter (Healy, 2003). Next, wavelet analysis is used for examining the periodicity of mortality patterns over time. It allows us to break down a time series into its spectral components, identifying dominant cycles and how they change across different periods (Cazelles et al., 2007; Rösch & Schmidbauer, 2018). In essence, the technique measures the correlation between the mortality data and a set of wavelets: mathematical functions of varying widths that correspond to different periodicities. As these wavelets are moved along the time series, their correlation with the data fluctuates, revealing moments when certain seasonal patterns were more or less pronounced. The stronger the correlation between the death counts and a particular wavelet, the greater the wavelet power, meaning that a particular cycle was a significant feature of the mortality pattern at that point in time.

We apply wavelet power analysis to monthly death counts between 1812 and 1931, focusing first on overall mortality trends before turning to cause-specific patterns in the period 1856–1891. Our aim is to determine whether mortality exhibited clear seasonal rhythms, whether these rhythms remained stable or shifted over time, and how different causes of death contributed to any observed changes. To achieve this, we begin by analyzing all-cause mortality to establish whether seasonality was a consistent feature of Amsterdam's death patterns or if it varied over time. Once we confirm the presence of periodicity, we move step by step to isolate the influence of specific disease categories.[2] First, we remove deaths attributed to cholera and smallpox, two epidemic diseases that caused dramatic but generally irregular

1 Observations without addresses were left out because further analyses of poor, rather poor, rather wealthy and wealthy neighborhoods (not included in this article) required this information.

2 Before the analysis, the data was pre-processed as suggested by Thai et al. (2015). After adjustment for month length, counts which deviated more than 10% from the official statistics were linearly interpolated and transformed by taking the square root. Trends were modelled with local polynomial regression with span parameter 0.5 ("loess" package in R). Finally, the detrended time series were normalized to restrict the range of values between zero and one. The analysis was conducted using the "WaveletComp" library in R (Rösch & Schmidbauer, 2018). The significance tests are based on simulated (N = 15,000) white noise processes.

spikes in mortality. Next, we exclude deaths related to the respiratory system, followed by diarrheal diseases, and then endemic causes, each time observing how their removal alters the seasonal patterns in mortality. This process allows us to assess the contribution of different disease groups to overall seasonality, without conflating their effects. However, the approach is not cumulative: while cholera and smallpox remain excluded throughout, other causes of death are removed one at a time, ensuring that their individual impact on mortality rhythms can still be observed. Only in the final step, when all major infectious causes have been removed, do we analyze the seasonality of non-infectious deaths, to see what patterns persist once epidemic and communicable diseases are no longer a factor.

3 SEASONALITY OF MORTALITY IN AMSTERDAM 1812–1931

Throughout the 19th century, mortality in Amsterdam exhibited significant year-to-year fluctuations, largely driven by epidemics and endemic diseases. Only from the 1880s onwards a gradual decline in mortality began, which accelerated in the 1890s. Figure 1 visualizes how death was distributed across the months per year as a percentage from 1812 to 1931, using a color gradient where earlier years are represented in dark blue and later years in yellow. The difference in distributions becomes smaller and more stable over time (i.e. lines with a yellowish color referring to later years are closer to each other than lines referring to earlier years). However, a distinct seasonal pattern remains evident: in most years, mortality was higher in winter than in summer. This pattern is further confirmed in Figure 2, which illustrates the excess winter mortality index over time. The smoothed trend (dashed line) shows that excess winter deaths were particularly pronounced at both the beginning and the end of the study period, with somewhat lower values in between.

Interestingly, prior to the 1880s, some years saw mortality peaks in the summer instead. Several factors could explain this. For example, cholera outbreaks struck the Netherlands in 1833 and 1849, and similar epidemics may have contributed to summer mortality spikes in other years. Additionally, fluctuations in infant mortality could have played a role. Previous research indicates that excess winter deaths were less common among infants (Janssens & Riswick, 2023), meaning that changes in infant mortality patterns may have influenced the overall seasonal distribution of deaths. After 1900, summer mortality peaks disappear entirely, while winter peaks persist, largely due to seasonal viruses. Influenza and other respiratory illnesses tend to thrive in colder months, though the reasons for this remain a topic of ongoing debate (Huang et al., 2023; Nichols et al., 2021). Notably, influenza epidemics swept through Amsterdam in 1890 and again between 1918 and 1922, while a severe whooping cough outbreak claimed many lives in 1891.

Figure 1 *Percentage of total deaths per month in Amsterdam, 1812–1931*

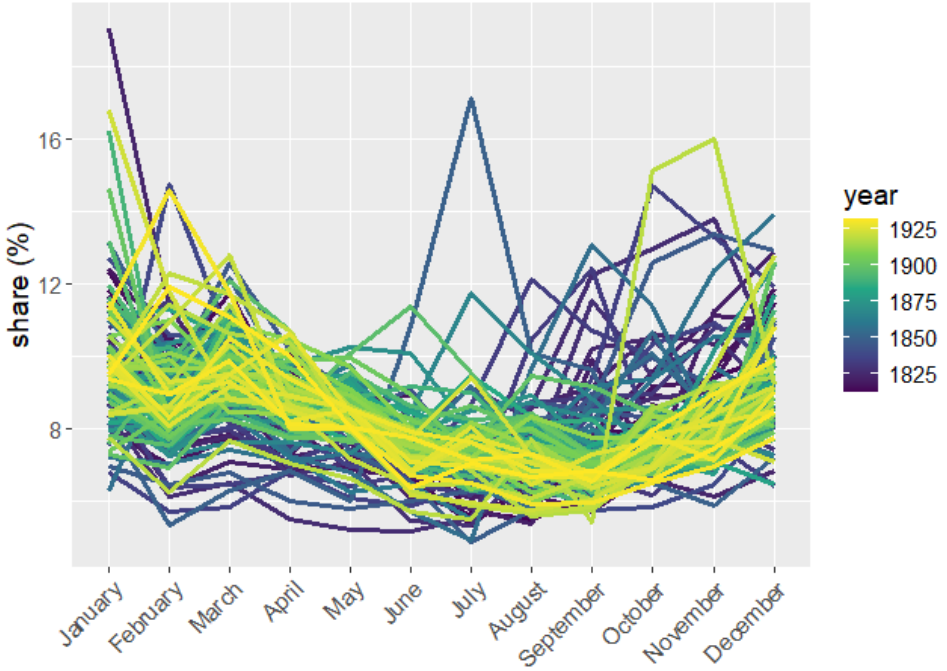
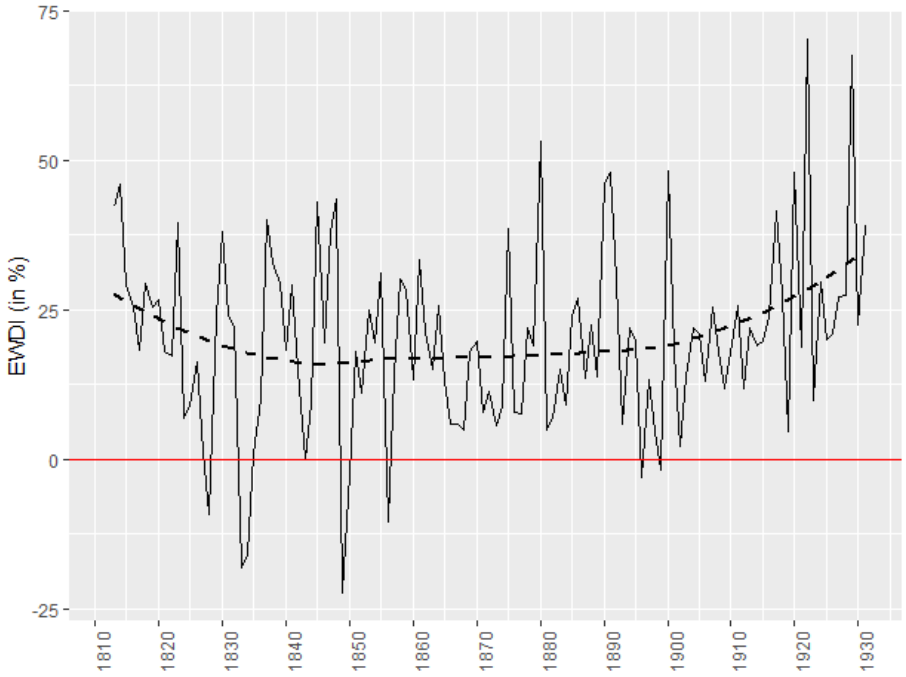
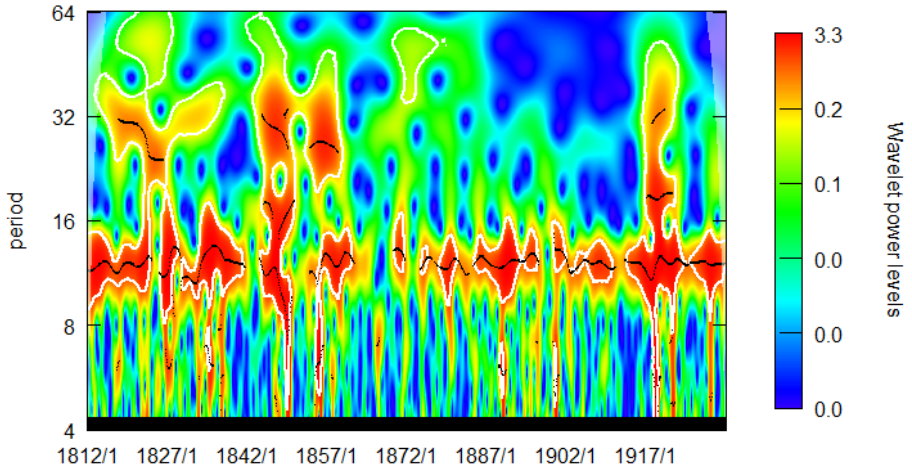


Figure 2 *Excess winter death index (EWDI) by year Amsterdam, 1812–1931*



Note: The dashed line represents smoothed values.

Figure 3 *All-cause mortality Wavelet power spectrum Amsterdam, 1812–1931*



To further explore seasonal mortality patterns in Amsterdam, Figure 3 presents a wavelet power spectrum analysis. The months of our study period (January 1812 to December 1931) are presented on the x-axis, the investigated wavelet lengths in months are on the y-axis. The color scheme reflects the power of the wavelets. Significant wavelet powers are marked with white contours. The black lines, called ridges, point out the periods with the highest power. We find the highest wavelet power (red areas) around month 12 throughout the study period. Although the wavelet power analysis is not able to show when this annual cycle of excess mortality takes place in a year, we know from the previous figures that it reflects a winter peak. However, the wavelet spectrum also reveals disruptions in this pattern. These breaks correspond to periods of lower excess winter death index (EWDI) values observed in Figure 2, suggesting that certain epidemics temporarily altered the typical seasonal mortality trends. Beyond this dominant 12-month cycle, the analysis identifies multi-year fluctuations in mortality. The presence of such multi-year cycles, visible in periodicities extending up to 32 months, might capture the presence of certain diseases that followed outbreak patterns spanning several years rather than adhering strictly to annual seasonal variation. In other words, these longer-term cycles point to the influence of endemic diseases with extended transmission patterns and/or affecting specific age groups. For instance, measles and whooping cough affected mainly children, which likely contributed to variations in mortality over time.

The same is true for intensity of seasonal mortality patterns within the 12-month cycle, which fluctuate across the study period. When patterns deviate from the typical annual cycle this indicates the presence of additional seasonal peaks. Instead of following a strict 12-month periodicity, the wavelet power spectrum reveals shorter cycles of approximately six months, suggesting that mortality peaked not only in winter but also in summer during certain years. This pattern is particularly evident during major epidemic outbreaks. For instance, cholera epidemics in 1833 and 1849 most likely introduced significant summer mortality peaks, disrupting the usual winter-dominated trend. These findings may indicate that while mortality was generally influenced by seasonal factors, severe disease outbreaks could override this structure, leading to increased deaths at different points throughout the year. Still, these seasonal mortality peaks are not really weakening after 1900.

These findings suggest that there may only be a very gradual reduction in the seasonality of mortality over time, which may partly explain why the winter excess mortality is still observed nowadays. Improvements in public health, sanitation, housing, and medical advancements, which could have mitigated the impact of seasonally driven diseases, particularly respiratory infections, seem therefore to have limited effects in our study period. The clearest exception is the influenza pandemic of 1918–1922 which led to mortality surges outside of the expected seasonal pattern, contributing to a breakdown of the standard yearly rhythm. In other words, when taking a long-term perspective we observe that Amsterdam's mortality patterns were highly seasonal, but also shaped by epidemic outbreaks and longer-term disease cycles. Winter peaks dominated throughout the 19th century, major epidemics periodically disrupted these patterns, and seasonality appeared to remain in the early 20th- century, despite advancements in disease prevention and medical care. Yet, to be able to give more detailed answers, in the next section specific diseases are taken into account.

4 SEASONALITY OF CAUSE-SPECIFIC MORTALITY IN AMSTERDAM 1856–1891

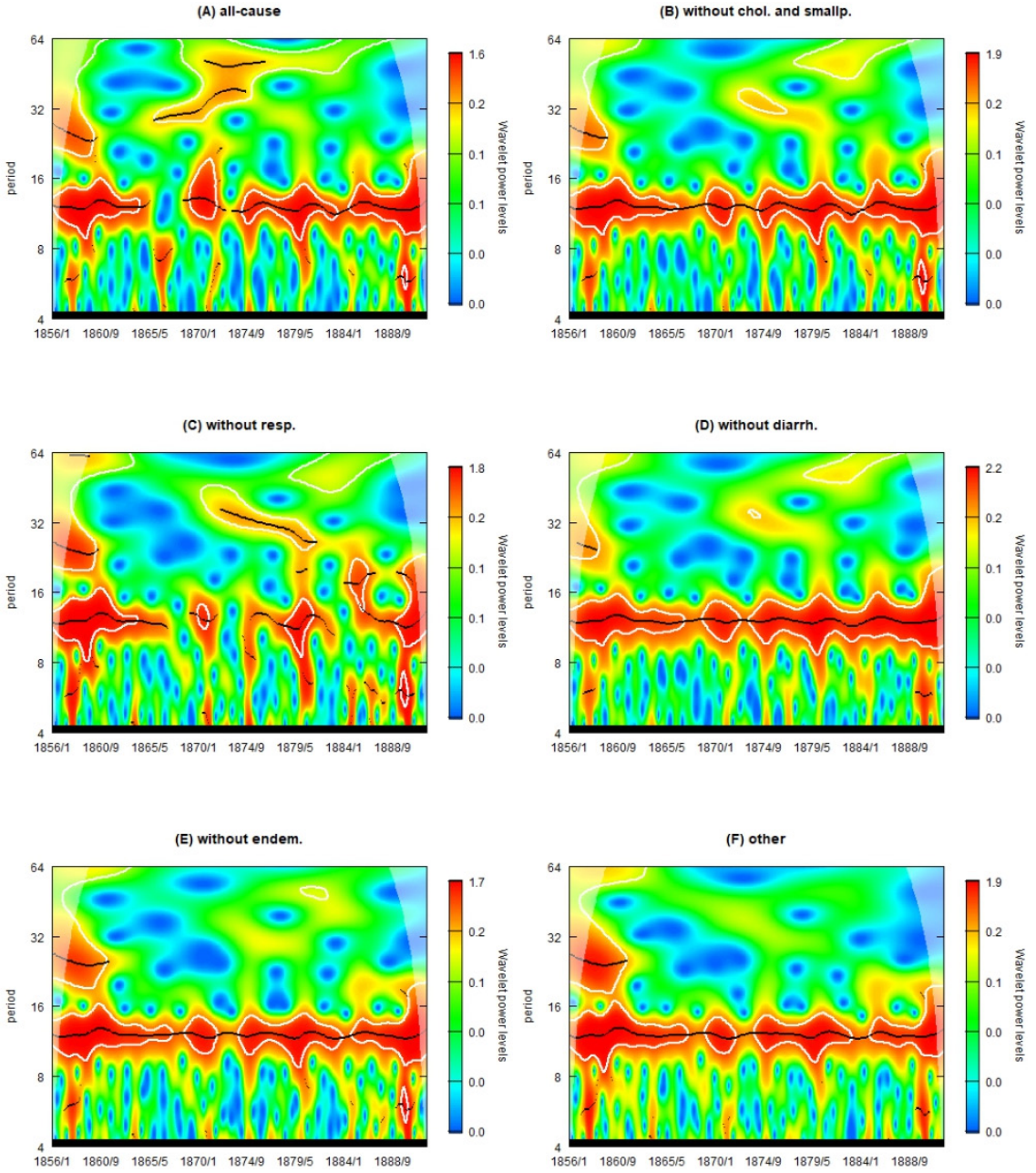
When examining all-cause mortality for the period 1856–1891 in Figure 4 (Panel A), the timeframe for which cause-specific mortality data is also available, earlier conclusions are reinforced. The analysis confirms a strong 12-month periodicity, with deaths consistently peaking in winter.[3] However, with cause-specific data, it becomes possible to identify which diseases were responsible for disrupting this annual pattern in certain years (see Figure 5 which shows the seasonality for specific causes of death). For example, significant breaks in the 12-month cycle are evident during the cholera epidemic of 1866 and the smallpox epidemic between 1870 and 1872. Since both of these outbreaks peaked in summer and had high mortality rates, their disruptive effect on the usual winter mortality pattern is unsurprising.

To further isolate the effects of individual causes of death, we progressively remove specific categories of disease from the analysis. When cholera and smallpox deaths are excluded (Panel B), the annual periodicity becomes more stable, though power remains weak around the epidemic years. This likely reflects the fact that removing individuals who died in an epidemic does not account for the competing risks of diseases: some may have died from other causes later, altering seasonal mortality distributions. These considerations highlight inherent limitations in our approach.

The removal of respiratory diseases (Panel C), a typical winter disease group, further disrupts the dominant 12-month cycle, as the proportion of deaths from non-winter-seasonal causes, such as diarrheal diseases, increases. While a seasonal ridge remains, it is weaker and frequently interrupted. Interestingly, vertical bands of high wavelet power emerge, indicating short-lived mortality spikes across multiple frequencies. These likely correspond to epidemic outbreaks, as seen in measles surges in December 1874 and early 1880, and a cluster of outbreaks in the early 1880s involving scarlet fever, diphtheria, and whooping cough. This suggests that, once respiratory deaths are removed, endemic diseases become the primary drivers of the highly variable seasonal mortality patterns.

3 This latter conclusion cannot be drawn from the wavelet power plots, we performed additional analysis to confirm the excess winter death.

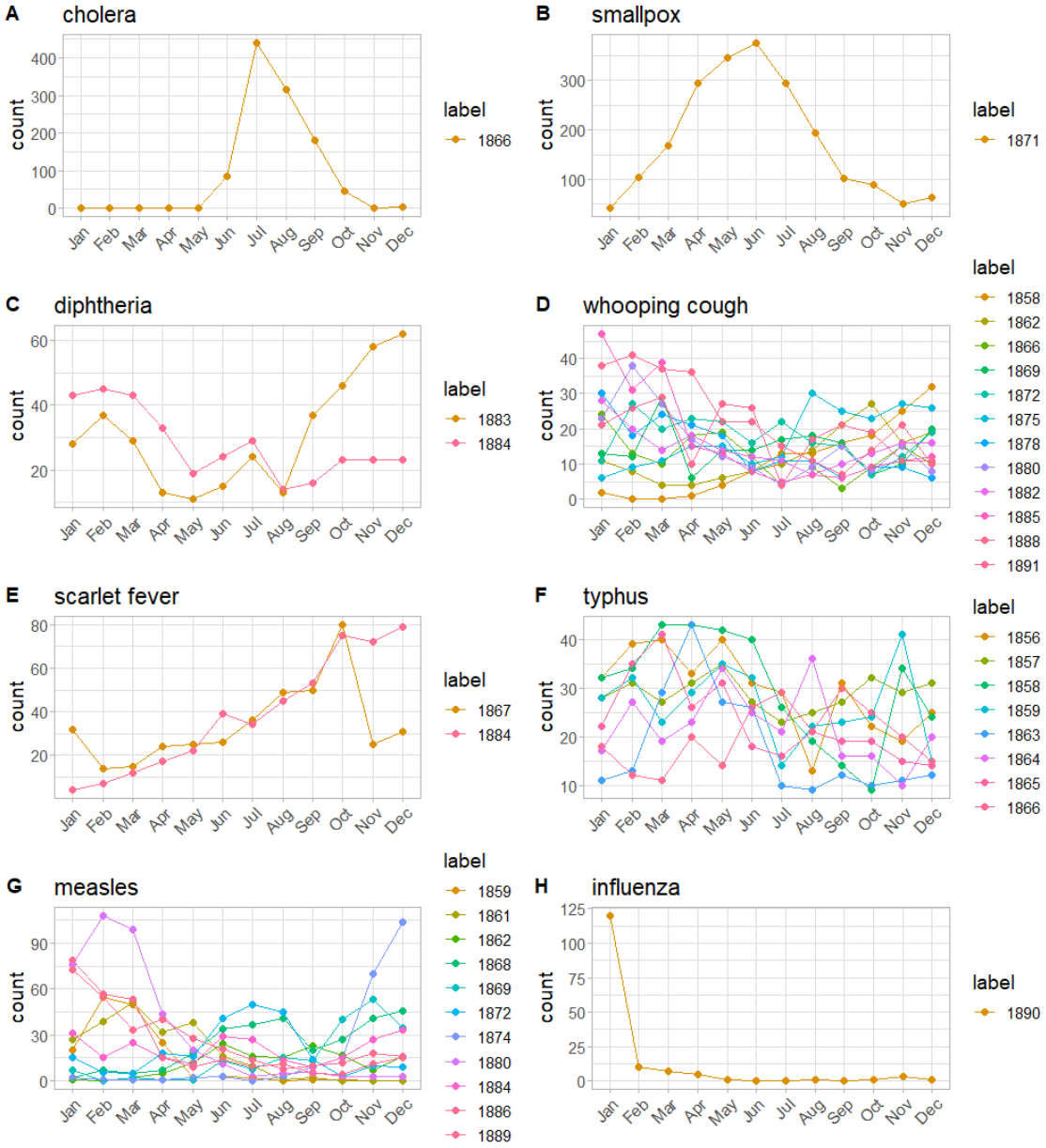
Figure 4 *Wavelet power spectrum for all-cause mortality, excluding several causes-of-death, Amsterdam, 1856–1891*



When diarrheal diseases, a typical summer disease group, are excluded (Panel D), the annual mortality cycle strengthens, as summer mortality declines, making winter peaks more pronounced. The next step, removing endemic diseases (Panel E), such as diphtheria, whooping cough, scarlet fever, typhus fever, measles and influenza, eliminates the longer-than-annual cycles, confirming that these diseases contributed to the multi-year mortality fluctuations observed earlier. The remaining pattern closely resembles that of Panel B, though winter seasonality is even stronger in the 1860s, likely due to the removal of non-winter-seasonal endemic diseases such as typhus, measles, and whooping cough, as well as a major scarlet fever outbreak in October 1867 (see Figure 5 for the seasonality of specific epidemics).

Finally, when all major infectious disease categories are removed (Panel F), the 12-month cycle remains dominant, with only a few interruptions in the late 1860s, early 1870s, and early 1880s. These periods align with disruptions seen in Panel E. These findings are consistent with earlier research on contemporary populations, which similarly identified cardiovascular causes as a major contributor to seasonal mortality and emphasized that temperature alone does not fully explain excess winter deaths. This suggests that, while infectious diseases played a significant role in shaping seasonal mortality, other factors, potentially related to environmental conditions, nutritional deficiencies, or non-epidemic causes, also contributed to the broader rhythm of death in Amsterdam.

Figure 5 *Seasonality of specific causes of death in Amsterdam, 1856–1891*



5 DISCUSSION AND CONCLUSION

This study has shown that mortality in Amsterdam during the 19th and early 20th centuries followed a strong seasonal rhythm, with deaths typically peaking in winter. This pattern was largely driven by airborne infectious diseases. However, winter excess mortality remained visible even after excluding these causes, suggesting that other factors, particularly cardiovascular diseases, also played an important role. Major epidemics disrupted this pattern, introducing additional mortality peaks in the summer months. These findings align with existing research but also point to broader and more complex drivers of excess winter mortality beyond seasonal viruses alone. In turn, a key contribution of this study is the demonstration of how a long-term perspective, combined with cause-specific mortality data, can deepen our understanding of seasonal mortality patterns.

To build on our exploratory work, future research should take a more detailed approach, focusing on specific age groups and what individual-level causes of death can reveal about how, and why, the rhythm of death was shaped. One promising direction is the integration of historical temperature

data, which is available in the HISKLIM database (Brandsma et al., 2000; Ekamper et al., 2009). This would allow for an investigation into the physiological effects of cold exposure on cardiovascular mortality, particularly deaths from heart attacks and strokes. Additionally, because the database includes general weather descriptions, researchers could also explore the impact of reduced sunlight exposure, which has been linked to lower Vitamin D levels and potential health consequences. Most interesting, however, is that the effects of cold and sunlight are unlikely to have been experienced equally across socio-economic groups. The urban poor, often living in poorly insulated housing in shadowly alleyways, would have had fewer resources to protect themselves from the cold or benefit from direct sunlight. This raises the possibility that clear differences in seasonal mortality patterns existed between social classes in a city like 19th-century Amsterdam, which may add to the current debates on the development of inequalities in health during this period.

While taking a closer look at environmental factors, such as temperature and sunlight, is important, they do not act in isolation. Future research should acknowledge that behavioural changes linked to social norms and cultural practices may also have shaped seasonal health outcomes. Winter brings subtle shifts in behaviour, such as reduced physical activity, dietary changes, and increased alcohol consumption, that could have contributed to excess mortality. In addition, these effects could differ by gender, age and social economic status. Although historical studies may not always be able to account for these nuances directly, they remain vital to consider when interpreting past patterns. This highlights the broader value of our historical demographic approach in uncovering how environmental, physiological, and behavioural mechanisms behind seasonal mortality evolved over time. By combining long-term cause-of-death data with interdisciplinary insights, we can continue to refine our understanding of the history of health — an endeavour that remains as relevant today as it was when Angelique Janssens' started her pioneering work.

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