School Closures and Respiratory Infections Transmission and Mortality: Evidence from School Holidays in Poland

Abstract

This study examines the impact of temporary school closures on influenza transmission and respiratory mortality, leveraging a natural experiment from winter break timings in Polish schools. Using causal inference methods on 12 years of ILI (Influenza-Like Illness) data and two decades of respiratory death records, the analysis shows significant reductions in ILI incidence within four weeks post-closures in an average season: 75% among school-aged children, 55% in adults, 26% in pre-school children, and 31% in the elderly, compared to pre-vacation averages. Notably, a 7% decrease in respiratory mortality was observed among the elderly, highlighting school closures as an effective public health intervention for reducing influenza spread and mortality among high-risk groups.

Keywords: Influenza, School Closure, Respiratory Mortality

1 Introduction

WHO [1] estimates that influenza affects approximately a billion individuals globally each year, resulting in between 229,000 and 650,000 fatalities. Children have been identified as key contributors to influenza transmission due to their relatively high infectivity [2]. To halt the spread of infectious diseases, policymakers consider school closures, acknowledging schools as significant hubs for transmission. Yet, most respiratory deaths are concentrated among the elderly [3], which raises questions regarding the efficacy of school closures in preventing such deaths. This paper documents the causal impact of temporary school closures on the incidence of influenza and related respiratory mortality in Poland.

The underlying hypothesis posits that school closures reduce contact rates among students [4], effectively reducing the spread of the virus. Schools serve as a prime environment for viral transmission, with children spending 5-8 hours daily in classrooms that typically house around 30 students. In such settings, a single ill student can spread the infection to numerous peers. Moreover, infected students may carry the virus home, transmitting it to family members across different age groups, including siblings, parents, and grandparents. Interrupting this transmission pathway through school closures could potentially reduce infections among school-aged children and limit the source of spillovers to other age groups.

Empirical evidence demonstrates that school closures indeed diminish the incidence of Influenza-Like Illness (ILI). Adda [5] and Cauchemez et al. [6] use French data[1] to show a significant decline in ILI incidence following schools holidays. This conclusion is further supported by Chu et al. [7] who find a decrease in ILI among children and adults after school closures in Beijing. Similar results are obtained in studies throughout the world [8], in India [9], in Israel [10] , and in the USA [11]. An exception is the study in Hong Kong by Cowling et al. [12], which shows a limited impact of school vacations when influenza activity is low.

This paper leverages the plausibly exogenous variation in the timing of winter breaks across Polish schools as a natural experiment to identify the causal impact of temporary school closures on the incidence of Influenza-Like Illness and respiratory mortality. To achieve this aim, the study utilizes detailed surveillance and administrative data, encompassing 12 years of weekly, county-level reports of ILI cases and two decades of respiratory infection-related mortality data. The disaggregation of the data by age enables me to answer the critical question of whether this policy also affects groups at elevated risk of mortality.

While school closures can have immediate health benefits in terms of fewer infections and deaths, such measures are not without their costs, particularly concerning human capital development and the productivity of caregivers. This paper focuses predominantly on the health implications of school closures. Readers interested in the broader socio-economic consequences of school closures are encouraged to consult existing literature on the topic [13, 5, 14, 15, 16].

2 Methods

This study uses detailed surveillance data on ILI cases and mortality records. It employs an event study framework leveraging the variation in timing of school

[1] Similar to data used in this project

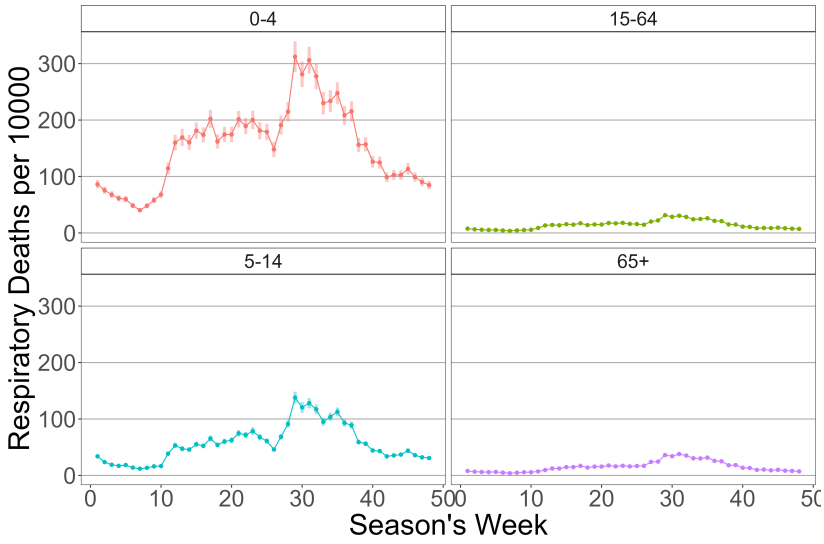
vacations across Polish regions to capture the causal effects of school closures on ILI incidence and respiratory mortality.

2.1 Data

I use two primary datasets: surveillance data on ILI cases and administrative mortality records.

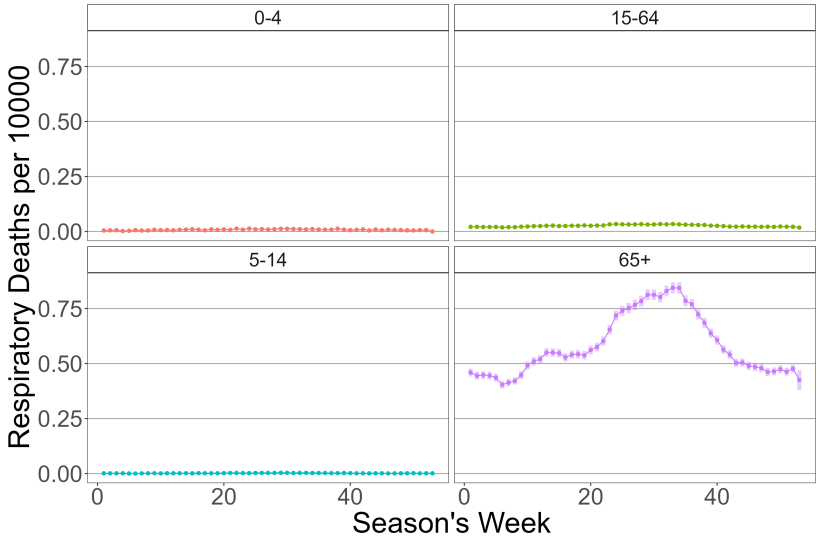
Surveillance Data: ILI case data are derived from weekly reports by primary care physicians as part of the national influenza surveillance initiative. For surveillance purposes, an illness is an ILI when a patient exhibits a fever of 38 °C (100 °F) or higher along with a cough that started within the last 10 days. The reports detail the number of patients presenting influenza symptoms, categorized into four age groups: 0-4, 5-14, 15-64, and 65+ years. The reporting is performed by physicians who voluntarily take part in the program. Nationally, about 1000 health practitioners participate in the program. Data is aggregated at the county and week level, with "county" here referring to the administrative division known as "powiat" in Poland. Access to these reports was facilitated through county and regional epidemiological stations, covering 168 of Poland's 380 counties from 2005 to 2019. Figure A.1 in the appendix shows counties that made the data available. The dataset includes the total reported cases and the contributing doctors per county each week, disaggregated by age group for the majority of counties. The surveillance data received from the epidemiological stations is organized into 48 "epidemiological weeks" per year, following the structure set by Polish authorities. Specifically, each month is divided into 4 "weeks", starting respectively on the 1st, 8th, 16th, and the 23rd day of the month. My main outcome of interest is the weekly incidence rates per age group and county, defined as the number of reported cases in a week per 10,000 persons in a given age group and county. Figure 1a illustrates the weekly averages of these rates. Weeks are expressed in influenza season terms with the 1st week of the season corresponding to the first week of July. A clear seasonal pattern is observed, with incidence peaking typically in February-March, diminishing during the summer, and resurging in September, aligning with the start of the school year. More detailed figures are available in the appendix, including Figure A.15 , which shows the weekly average incidence for each season, and Figure A.16, which illustrates the weekly average incidence in relation to the timing of vacations.

Mortality Data: The mortality dataset encompasses all Polish counties from 2000 to 2018, detailing deaths by cause, age group, county, week, and year. Weeks



(a) Average ILI Incidence by Week and Age Group

Note: Each dot represents the average weekly incidence of ILI cases per 10,000 people, aggregated across seasons and counties. First week of the season corresponds to the first week of July. Data were supplied by the epidemiological stations in 168 Polish counties detailing the ILI reports from the primary care providers spanning years 2005-2019. Bands represent 95% confidence intervals for the means. Pre-school children (0-4 years) report the highest incidence, followed by school-aged children (5-14 years), the elderly (65+ years), and adults (15-64 years).



(b) Average Respiratory Mortality by Week and Age Group

Note: Each dot represents the average weekly mortality from respiratory diseases (ICD-10 category J) per 10,000 people, aggregated across seasons and counties. First week of the season corresponds to the first week of July. The administrative mortality data provided by Polish Statistical Office (GUS) covers all Polish counties (360) across years 2000-2018. Bands represent 95% confidence intervals for the means. Seniors exhibit the highest mortality rates, reflecting a seasonal pattern aligned with ILI incidence.

Figure 1: Average ILI and Mortality by Week and Age Group

in mortality data follow ISO norms. This analysis focuses on deaths attributed

to respiratory system diseases (ICD-10 category J), using annual population data by age to compute mortality rates per 10,000, with both datasets from the Polish Statistical Office (GUS). Similar to ILI incidence, a seasonal mortality pattern arises, particularly pronounced among seniors, with negligible rates in younger age groups (Figure 1b). Figures disaggregated by season (A.17) and vacation timing (A.18) are available in the appendix.

2.2 Empirical Strategy and Statistical Methods

The empirical strategy leverages a unique natural experiment arising from the staggered timing of school breaks across Polish regions. During the winter months from January to March, schools close for a two-week period[2]. However, the specific timing of these breaks within a year varies by region (which encompasses multiple counties)[3]. Regions are divided each year into four groups. The first group begins their vacation in mid-January, while the last group starts in mid-February. Furthermore, whether a region has an earlier or later school break changes every year. The school calendar, set in June before winter vacation, is determined without knowledge of the upcoming flu season's dynamics, making the timing of these breaks exogenous to geographical and time variations in ILI incidence. This suggests that counties in early and late vacation regions are similar before the vacation starts, supporting the assumption that ILI trends would be parallel without school closures. Using late-vacation counties as a control, I can construct a counterfactual for early-vacation counties and estimate the causal effects of school closures on ILI incidence and associated mortality rates.

The empirical strategy is intuitively illustrated in the Figure 2, showing the average incidence of influenza-like illnesses (ILI) per 10,000 children aged 5-14 during the years 2015-2016, segmented by the week they start vacation. The orange line represents the group of counties starting vacation in week 28, while the grey line represents the group starting vacation in week 32.

Both lines overlap during the period before any group starts vacation, reflecting the quasi-random assignment of vacation timing across counties, which should result in similar baseline ILI rates. This overlap supports the use of the later-starting group as a counterfactual for the earlier-starting group.

In week 28, the orange group starts their vacation, effectively becoming the "treated" group. The incidence of ILI in this group remains relatively low during their vacation period. Conversely, the grey group does not start their vacation until

[2]Pre-schools and care centers do not observe winter breaks.

[3]There are 16 regions (wojewodztwa) in Poland

week 32, remaining "untreated" during this interval, and their ILI incidence rises. Their outcomes can be conceived as what would happen in the "treated" group had they not experienced vacation.

The difference in ILI rates between the two groups represents the treatment effect of the vacation. Specifically, it shows the reduction in incidence caused by the vacation.

The graph ends at week 31 because, from week 32 onwards, both groups have had a vacation, removing a pure counterfactual comparison. Although longer-term effects cannot be inferred from the figure alone, they can be estimated under certain assumptions using the framework detailed below.

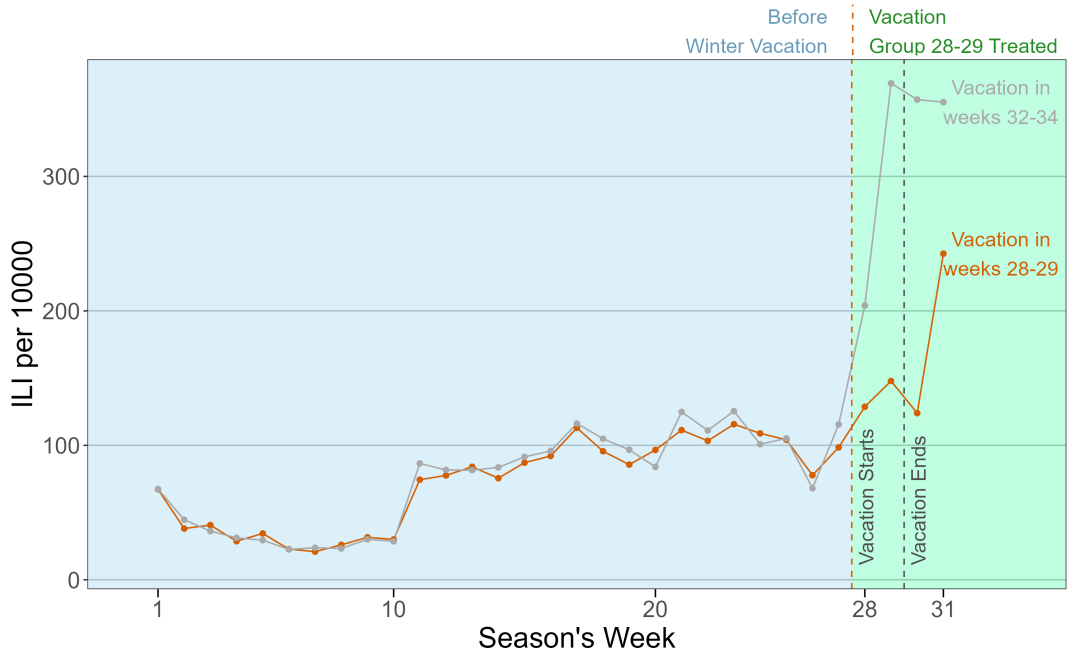


Figure 2: Winter Vacation and ILI Incidence among School-Age Children

Note: Graph shows the average incidence of influenza-like illnesses per 10,000 among children aged 5-14 years in 2015-2016, divided by the week in which they start vacation. The orange line represents the incidence for the group starting vacation in week 28, while the grey line represents the group starting vacation in week 32. The x-axis shows the week of the season. The first dashed line indicates the start of vacation for the group having vacation in weeks 28-29, and the next dashed line indicates the end of vacation for the same group. In the light blue shaded area, none of the groups have experienced vacation. In the light green shaded area, the group with vacation in weeks 28-29 has experienced vacation, but the group with vacation in weeks 32-34 has not. Starting in week 32, both groups would have experienced vacation.

To assess the dynamic, week-by-week effects of school closures, I employ an event study approach following the equation 1:

$$y_{cwya} = \sum_{\substack{T=-6 \\ T\neq -3}}^{12} \delta_{Ta} I\{wy - (wy)_c^V = T\}_{cwy} + \phi X_{cwy} + \gamma_{wya} + \theta_{cwa} + \lambda_{cya} + e_{cwya} \quad (1)$$

This equation models the outcome variables y_{cwya}, representing either the number of reported flu cases or respiratory deaths per 10,000 individuals in county c, during week w, of year y, across different age groups a.

The treatment effect is captured through a series of indicator functions. Let $(wy)_c^V$ be the first week of vacation in year y and county c. Then $I\{wy - (wy)_c^V = T\}_{cwy}$ equals 1 if a county c is T weeks away from the start of the winter vacation. As T ranges from -6 to 12, the coefficients δ_{Ta} capture effects from 6 weeks prior to the vacation to 12 weeks afterward. Therefore, δ_{Ta} are the primary coefficients of interest showing the change in the outcome for each week relative to a counterfactual scenario where no vacation occurs. The key period of intervention is marked by $T = 0$, corresponding to the week when the recess begins. The analysis is normalized to the third week before the vacation ($T = -3$) as the reference point for two reasons. First, it allows for detecting anticipatory effects, as individuals might change their behavior before $T = 0$. Setting $T = -3$ is far enough to ensure behavior is unaffected by the start of the vacation. Second, it addresses the misalignment between the 48-week epidemiological calendar and the school year calendar, where vacations may begin before $T = 0$, causing effects to appear in $T = -1$[4]. Therefore, $T = -3$ is chosen because vacations never overlap with it, avoiding underestimation of effects.

The model also includes a control variable for the number of reporting doctors per 10,000 individuals (X_{cwy}) and fixed effects: week-year-age-group γ_{wya}, week-county-age-group θ_{cwa}, and year-county-age-group λ_{cya}, to account for varying seasonality and reporting practices. The model is estimated separately for each age group to capture age-specific effects. Standard errors are clustered at the regional level.

The reduced-form approach used in this paper estimates the impact of interventions by comparing observed outcomes, such as infection rates post-vacation, to a counterfactual scenario without the intervention. Being model-agnostic, it avoids assumptions about transmission dynamics, incubation periods, or the size of the susceptible population, making it flexible and more robust to mis-specification. Reduced-form estimates can capture both the average effect on incidence and growth rates across different contexts, and they can be disaggregated by specific epidemic phases, provided these phases occur within the sample (results disaggregated by

[4]In case of a discrepancy, the first week of vacation is set to be the first epidemiological week which has more than 3 days of vacation

epidemic context are in the appendix). However, these estimates combine multiple factors (e.g., changes in contact rates, shifts in the susceptible population) without differentiating between them. Additionally, the reduced-form approach does not allow for reconstructing the full epidemic curve.

In contrast, structural models used in other work estimate the underlying parameters of the transmission process, enabling them to simulate how interventions affect different stages of the epidemic. These models can predict whether an intervention will slow growth or accelerate decline and can assess the impact of varying the timing or duration of interventions. Structural models provide richer, more generalizable insights, but they rely on assumptions about the transmission process that may be more vulnerable to mis-specification. They also help distinguish the specific mechanisms driving changes in outcomes and can simulate a wider range of hypothetical scenarios (an excellent example being Cauchemez et al. [6]).

Both approaches offer value to policymakers and work well together. Structural models can guide reduced-form analysis by helping to evaluate whether observed outcomes, such as changes in growth rates, align with model-based predictions. Reduced-form methods, on the other hand, deliver clear, empirical estimates of overall effects without imposing any structure, which can inform and help calibrate structural models—for example, by identifying whether cases rise after schools reopen. Together, these methods provide a fuller, more comprehensive understanding of intervention effects.

Although the estimates are model-independent, Section A.5 illustrates how the event study coefficients relate to parameters of a simplified SIR model if that were the infection-generating process. In Section A.6, I validate the event study method through simulations, showing it accurately estimates the treatment effect in a controlled environment where the true effect is known. The event study exhibits minimal bias, with any positive bias related to the heterogeneity of treatment effects across groups experiencing vacations at different times [17].

This issue arises because the direct counterfactual for a county experiencing a vacation is an "untreated" county that has not yet experienced a vacation. However, since all counties have vacations within a 4-week window, there is no "untreated" county for periods further than 3 weeks after the first group's vacation. Using estimates from the earlier periods, it is still possible to causally infer the longer-term effects, but this requires the assumption that the path of effects is the same for counties with early and late vacations. In Section A.5, I argue why the assumptions behind this method are likely to hold true in this context.

To address this potential issue, I also provide an estimate using a method de-

signed by Callaway and Sant'Anna [18], which is robust to the issue arising due to the heterogeneity of treatment effects. Section A.6 shows this method exhibits minimal bias. However, this approach comes at the cost of not being able to extrapolate longer-term effects. These findings are presented in Appendix Figures A.7 and A.8.

The main results are presented in terms of incidence, similar to some other studies on school closures [10, 7, 19]. This approach offers a straightforward empirical summary of the extent to which the epidemic burden is alleviated due to school vacations by comparing observed incidence with a counterfactual scenario without vacations. However, it does not necessarily provide a complete picture of the underlying dynamics that lead to the causal decline in cases. To address this limitation and gain a more comprehensive understanding, I present additional analyses described below.

In the Appendix (Figure A.10), I assess how school vacations affect the epidemic growth rate, exploring how the average causal difference in growth rates evolves compared to a counterfactual scenario without vacations. During vacations, growth rates may slow or cases may decline more rapidly. After vacations, growth rates may increase relative to the counterfactual, potentially leading to case numbers that remain lower or eventually surpass those without vacations. Combined with the analysis of total incidence, this approach provides insights into the overall effects and some intermediary mechanisms of the intervention. However, these estimates do not fully capture the epidemic's complex dynamics; modeling could complement this empirical analysis by offering more detailed insights into specific transmission pathways and predicting outcomes under different scenarios.

I also analyze the data using the logarithm of incidence plus one, $\log(y + 1)$, as the outcome variable (see Figures A.3 and A.4), or percentage interpretations of the effects. Recognizing the challenges associated with the $\log(y + 1)$ transformation and zero outcomes [20], I employ Poisson regression models on the count data as well, with results presented in Figures A.5 and A.6. This methodology follows approaches used in previous studies [21, 22]. Additionally, the analysis is extended to evaluate the policy's impact on the reproductive number, not merely incidence rates, as detailed in Appendix Section A.3.

3 Results

Temporary school closures significantly reduce the incidence of ILI among all age groups and respiratory mortality among the elderly.

Figure 3 shows the estimated δ_{Ta} coefficients, reflecting the impact of school

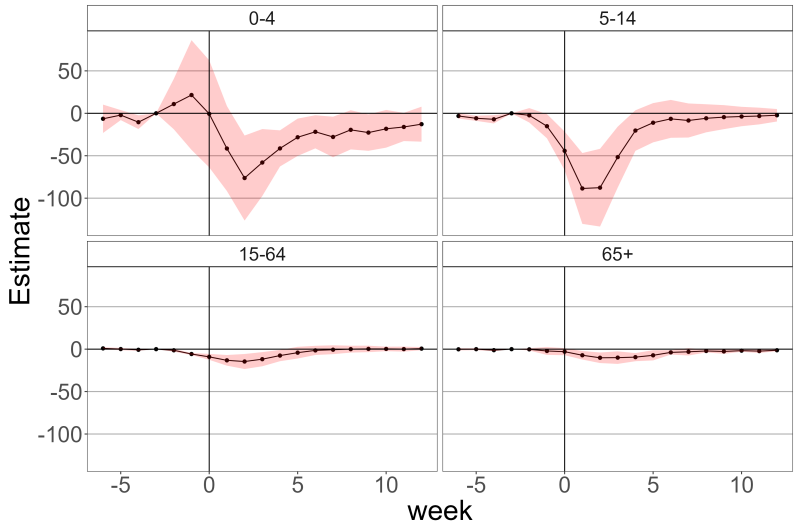


Figure 3: Event study: ILI Incidence

Note: Graph shows the impact of winter vacation on the ILI incidence by age group. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. Line represents estimates of the coefficients δ_T from equation where the outcome is number of reported cases in a given county and week, per 10 000 inhabitants (of a given age-group). These parameters show the change in incidence in week T, compared to the counterfactual the vacation. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

closures on ILI incidence for various age groups over time. These results represent the average effect of school vacations across the seasons in the sample, for the 6 weeks before and 12 weeks after the vacations. Each coefficient for a week T represents the difference in incidence in T weeks after the vacation, compared to what the incidence would have been in that week had the vacation not occurred. These coefficients come from the context where school vacations typically occur around the peak of the influenza season. The effect in a particular season might depend on the timing of the vacation within the epidemic curve; this heterogeneity is analyzed in the appendix, while average results are summarized here. Children aged 5-14 are the most significantly affected. The reduction begins during the vacation week and extends to the fourth week after. In the vacation week, incidence decreases on average by 44 cases per 10,000 (95% confidence interval (CI): -71.6, -16.9), relative to the counterfactual, which is notable given the pre-vacation weekly average of approximately 91 cases per 10,000 in this demographic. The decline continues with, on average, 88 fewer cases per 10,000 in the first and second weeks after the vacation begun (95% CI: Week 1: -138.9, -38.2; Week 2: -142.9 -32.4) and 51 fewer cases per 10,000 on average in the third week (95% CI: -94.9, -8.4), all statistically significant at 5% level. Most coefficients preceding vacation are zero, suggesting parallel ILI trajectories in early and late counties without vacation. The sole deviation at

$T = -1$ may arise from discrepancies between epidemiological and school calendar alignment. Aggregating the effects over the 4 weeks since the start of the recess, the vacation cause a decrease of around 272 cases per 10,000 (95% CI: -414, -130) in an average season, roughly a 75% reduction from the pre-vacation period of 4 weeks (with average of 364.6 cases per 10,000). Cumulative effects are detailed in the Table A1 [5].

The implementation of school closure policies effectively slows the spread of influenza-like illness among school-aged children, lowering their infection rates and potential to spread the virus. As the subsequent results demonstrate, school closures also mitigate the incidence in other age groups, including the most vulnerable populations: preschool children and the elderly.

For preschool children (0-4 years), infection incidence decreases on average by 76 cases per 10,000 compared to the counterfactual (95% CI: -136, -15.88) starting in the second week after the break, and the effects persist until the fifth week. The total decrease over the first 4 weeks after the start of the vacation accumulates to 217 cases fewer per 10,000 (95% CI: -362, -72.2) in an average season, a 26% reduction compared to the 4 weeks pre-vacation average for this age group (of 824.8 cases per 10,000).

Adults also show a decline in ILI cases post-vacation. Although the absolute decrease is smaller, the relative reduction is more pronounced. There is a significant drop of 9 cases per 10,000 on average in the first week (95% CI: -13.2, -4.9), extending until the third week. Over the first four weeks, the decrease totals 48.6 cases per 10,000 (95% CI: -74.9, -22.4) in an average season, a 55% reduction compared to the pre-vacation average of 87.62 per 10,000. This underscores that school-aged children are a significant source of infections for adults.

Finally, the 65+ population also experiences a decline in infections. Beginning with the second week of vacation, there are, on average, 7 fewer cases per 10,000 (95% CI -13.23, -1.1) compared to the counterfactual. The protective effect extends over the following five weeks, though it gradually wanes. Overall, the first 4 weeks of vacation contribute to averting 30.3 cases per 10,000 (95% CI -50.9, -9.75) in this age group in an average season, a 31% decrease from the 4 weeks pre-vacation period (with average of 98.5 per 10,000).

The reduction in ILI cases across various age groups is likely due to decreased intra-household transmission, with school-age children being key carriers. This highlights the broader protective impact of school closures beyond the immediate school environment.

[5] The summary statistics including the fit of the model are presented in table A6

Given the positive spillovers of school closures on at-risk age groups, a decrease in mortality is expected. Figure 4 presents the results of the estimation where the outcome variable is respiratory mortality, indicating that school closures in an average season indeed contribute to a decline in respiratory disease-related deaths among the elderly population aged 65 and above.

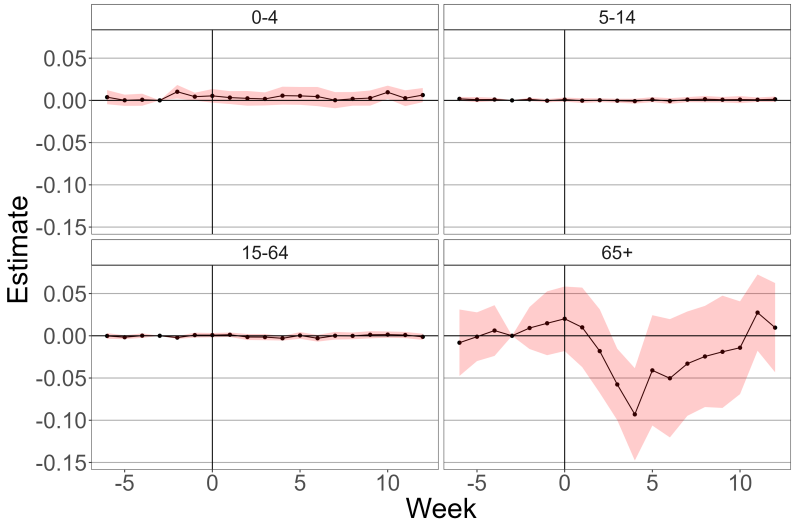


Figure 4: Event study: Respiratory Mortality

Note: Graph shows the impact of winter vacation on the ILI incidence by age group. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. Line represents estimates of the coefficients δ_T from equation where the outcome is number of reported cases in a given county and week, per 10 000 inhabitants (of a given age-group). These parameters show the change in incidence in week T, compared to the counterfactual without the vacation. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

Four weeks before the vacation, baseline single-week average respiratory mortality among the elderly is 0.73 per 10,000. After the vacation starts, significant declines occur in an average season: 0.0576 per 10,000 (95% CI: -0.1, -0.01) in the third week and 0.093 per 10,000 in the fourth week (95% CI: -0.15, -0.03). While the coefficient at later dates are negative, they are not statistically significant at 5%. The timing of the statistically significant reductions aligns with the incubation to death period for influenza, consistent with the hypothesized impact of school closures. The cumulative effect over weeks 2-5 is 0.21 fewer deaths per 10,000 (95% CI: -0.4, -0.01) in an average season, a 7.2% decrease from the cumulative 4 weeks pre-vacation average of 2.92 per 10,000.

These results confirm that student interactions significantly propagate influenza-like illnesses. School closures, when implemented around the peak of the epidemic, lead to a reduction in viral transmission across age groups and contribute to a temporary decrease in seasonal mortality among high-risk groups, such as the elderly.

Overall, these findings offer compelling justification for the implementation of school closures as a public health strategy aiming to temporarily reduce the incidence and mortality.

4 Discussion

This study's causal estimates of the effects of school closures on ILI incidence and respiratory mortality highlight the significant public health benefits. Analyzing data from a natural experiment involving staggered school vacation schedules reveals substantial reductions in ILI incidence across all age groups in an average season, with the most significant decrease of 75% among school-aged children. While children often recover quickly from mild symptoms, very young children and the elderly are more susceptible to severe complications, making their cases more impactful from a healthcare perspective. School closures also reduce incidence in these high-risk groups by breaking the transmission chain, demonstrating the critical role school-aged children play in spreading influenza. Consequently, the implementation of vacations not only decreased the incidence among the elderly, a group particularly vulnerable to severe outcomes from influenza, but also attenuated their respiratory mortality.

This study's impact on incidence is at the higher end compared to other findings. Similar work by Adda [5] reports decreases of 30-40% in children, 20-30% in adults, and 20% in the elderly. Adda's inclusion of preschool children and older teenagers in the children's group may account for the smaller effect size. Additionally, the confidence intervals for the elderly in Adda's study overlap with the estimates reported here.

Comparative studies from China [7], Argentina [21], Israel [10], and the USA [11] typically demonstrate smaller effects ranging from $13 - 42\%$ decline for children. Nonetheless, these studies, often compare incidence ratios during or post-break to the periods before, rather than to a counterfactual scenario in the absence of holidays—potentially underestimating the effects relative to the causal estimates presented here. Chowell et al. [23], found reductions similar to this study in Chile, with a 67% decline in children and 37% in adults. The estimates from Cauchemez et al. [6], which are informed by a robust mathematical model of transmission, also reported smaller reductions.

Regarding mortality, while no direct comparisons exist, the magnitude of significant effects on the elderly in this study is more than double the increase in influenza mortality linked to local events, such as the presence of a home team in the Super

Bowl, associated with a mortality increase of 0.07 per $10,000$ among the elderly [24].

According to my estimates in the Polish context, and considering an average season with recess close to the epidemic peak, winter vacations may prevent approximately $107,400$ infections annually among school-age children, $25,736$ infections in the $0-4$ age group, $125,580$ cases among adults aged $15-64$, and $18,177$ cases among those aged 65 and older in the four weeks following the vacation. Crucially, disrupting transmission during the winter vacation is estimated to prevent, on average, 101 deaths in the elderly population annually in the five weeks after vacation.

It is important to note that the presented estimates reflect the average effects across multiple seasons. Vacation during each individual season may have its own distinct impact, influenced by factors such as the severity of the epidemic or the timing of the vacation relative to the peak. The event study approach aggregates these effects, providing an average summary across the contexts in the sample.

One limitation of this analysis is that most school vacations in Poland occur symmetrically in short time around the epidemic peak, typically within five weeks before or after (see Figure A.19 for the distribution). As a result, the findings are most applicable to interventions implemented near the peak, a period when such measures may be most effective. In contrast, the effects of vacations scheduled further from the peak may be limited, as the potential impact of public health interventions could vary depending on the current influenza activity.

To better understand how the effects vary across different contexts, I perform a heterogeneity analysis. Ideally, one would desegregate the analysis by individual season; however, due to limited statistical power, this approach introduces excessive noise in the estimates. Instead, I group the seasons by factors likely to influence the overall effect, such as epidemic severity and timing of vacation relative to the peak. When desagregated by epidemic severity, the results for incidence and mortality are presented in Figures A.12 and A.13, with corresponding cumulative effect estimates provided in Tables A3 and A4. For timing of vacation relative to the peak, see Figure A.14 and Table A5.

As anticipated, the effect is more pronounced during severe epidemics, with a greater number of infections and deaths being averted during these periods. This finding aligns with existing literature [12], which observed limited effects in milder seasons when epidemic activity was less intense.

Regarding the timing relative to the epidemic peak, splitting the sample leads to noisier estimates. Nevertheless, the results suggest that the effects of school vacations occurring before and during the peak are consistent with the overall average estimate. In contrast, vacations scheduled after the peak show a diminished effect.

It is important to note that in my sample, vacations rarely occur significantly after or before the peak, which should be considered when interpreting these estimates.

Another limitation is that the data from primary care reports may under-detect infections if individuals avoid seeking medical attention. Additionally, not all doctors report cases, leading to missing data, and not all reported cases are confirmed as influenza.

Vacation periods may influence the propensity to visit a doctor, but this is unlikely to account for the observed effect for four reasons. Firstly, changes in reporting during the vacation cannot explain the decline observed after the vacation period. Notably, the continued decrease in the third week, despite students having returned to school, is statistically significant and large compared to the mean. While increased travel and related absences may contribute to decreased reporting during the recess, the subsequent drop after the recess cannot be attributed to these absences, as students must return to school the following week. If vacation only affected reporting and not actual infections, there should be no effect in the third week when reporting normalizes. However, the effects persist, suggesting that the observed drop during vacation corresponds to an initial decrease in the pool of infected individuals due to fewer interactions, which subsequently leads to a lower source of new infections and hence lower incidence after the vacation. This pattern is inconsistent with a temporary decrease in reporting, as this could not produce a persistent drop after the vacation.

Secondly, it is unlikely that elderly people change their reporting patterns due to school vacations, yet the significant effects in this group further indicate a genuine decline in infections.

Thirdly, while the number of doctors submitting reports changes during vacation (see Figure A.33), the regression analysis controls for the number of reporting physicians, adjusting for any fluctuations. The estimated effect on infections is on top of the changes in reporting. Furthermore, the vacation effects remain strong even when using the number of reports per doctor as the outcome (see Figure A.34), which would not be the case if the effects were solely due to fewer reporting doctors. Detailed analysis of the impact on reporting doctors and alternative outcomes is provided in the appendix section A.8.

Fourthly, there is a clear effect on mortality, where reporting issues are irrelevant because mortality data are derived from administrative records, and the timing of death is unlikely to be manipulated.

An additional limitation of this method is that it can only evaluate short-to-medium term effects, not long-term effects. Specifically, it cannot assess whether

having a vacation versus not having a vacation affects the cumulative rate of infections over an entire season, as all counties eventually have vacations. While variation in timing helps to identify short and medium term effects, it is not possible to determine what would happen without vacations altogether. This leaves open the possibility that some of the infections prevented by vacations may occur later in the season. To address this concern, I produce an additional analysis of the effect on the growth rate and incidence in an extended window.

Epidemic models predict that school closures decrease the growth rate of infections compared to the counterfactual, followed by a relatively higher growth rate upon reopening due to a larger susceptible population. This pattern is observed in Figure A.10, where the weekly growth rate of cases is used as the outcome in equation (1) to show the causal effect of vacations on growth rates. Depending on the relative magnitudes of these effects, there may either be a catch-up in incidence after vacations-if the post-reopening growth offsets the initial decline-or no catch-up if it does not.

To empirically investigate potential 'catch-up' later in the season, I extend the analysis window to 16 weeks (Figure A.9). In the event study framework, a catch-up would manifest as an increase in incidence in weeks post-reopening compared to the counterfactual, offsetting the previous decline. The analysis reveals no evidence of a significant rebound in incidence. Although the growth rate increases post-vacation, it does not raise total cases to or above the levels observed without school closures. Most coefficients in Figure A.9 for weeks 0–16 across all age groups are zero or negative, indicating no meaningful increase in cases up to 16 weeks post-closure compared to the no-vacation scenario. Furthermore, the upper bounds of the 95% confidence intervals for the cumulative effect over this period remain negative for all age groups except adults, where it includes zero (Table A2). While a rebound could theoretically occur after the 16-week window, this is unlikely, as infection rates are typically very low by June. Evaluating the effect throughout the totality of the season is technically not feasible, as it would require comparisons with seasons in which some counties do not have vacations. In section A.7, I also test whether having earlier versus later vacations affect cumulative incidence in a season, though the timing difference is at most four weeks. These small differences in timing do not produce statistically significant differences in total incidence.

Limitations also include the imperfect alignment between the epidemic and school calendars due to the predefined week structure in the Polish data, which could result in the misassignment of vacation periods by up to three days. This may also limit generalizability to diseases with different transmission patterns. Another

concern arises from the resulting uneven duration of weeks, which vary between 7 and 8 days. The median week length is 7 days (mean = 7.6, SD = 0.76), with a range from 6 to 9 days. Nonetheless, this variation is unrelated to random vacation timing and any potential bias is addressed through the use of fixed effects, ensuring that comparisons between early and late counties are based on the same week length. Additionally, to enhance interpretability, I include a robustness analysis where all cases are scaled to a 7-day equivalent (Figure A.11), and the results remain nearly identical to the main results.

Despite these limitations, the results appear robust, as substantiated by the various robustness checks, exploring alternative outcome definitions, specifications, and methodological approaches.

While school closures can effectively reduce infections and mortality, this benefit is just one part of the decision. Policymakers need to consider the trade-offs, balancing health gains against educational disruptions and the extra caregiving load on families.

References

[1] WHO. Influenza (Seasonal), 2023.

[2] Terho Heikkinen. Influenza in children. *Acta Paediatrica (Oslo, Norway: 1992)*, 95(7):778–784, July 2006.

[3] CDC. Past Seasons Estimated Influenza Disease Burden | CDC, February 2024.

[4] Charlotte Jackson, Emilia Vynnycky, and Punam Mangtani. The Relationship Between School Holidays and Transmission of Influenza in England and Wales. *American Journal of Epidemiology*, 184(9):644–651, November 2016.

[5] Jérôme Adda. Economic activity and the spread of viral diseases: Evidence from high frequency data. *The Quarterly Journal of Economics*, 131(2):891–941, 2016. Publisher: MIT Press.

[6] Simon Cauchemez, Alain-Jacques Valleron, Pierre-Yves Boëlle, Antoine Flahault, and Neil M. Ferguson. Estimating the impact of school closure on influenza transmission from Sentinel data. *Nature*, 452(7188):750–754, April 2008.

[7] Yanhui Chu, Zhenyu Wu, Jiayi Ji, Jingyi Sun, Xiaoyu Sun, Guoyou Qin, Jingning Qin, Zheng Xiao, Jian Ren, Di Qin, Xueying Zheng, and Xi-Ling Wang.

Effects of school breaks on influenza-like illness incidence in a temperate Chinese region: an ecological study from 2008 to 2015. *BMJ Open*, 7(3):e013159, March 2017.

[8] Charlotte Jackson, Emilia Vynnycky, Jeremy Hawker, Babatunde Olowokure, and Punam Mangtani. School closures and influenza: systematic review of epidemiological studies. *BMJ Open*, 3(2):e002149, 2013.

[9] Sheikh Taslim Ali, A.S. Kadi, and Neil M. Ferguson. Transmission dynamics of the 2009 influenza A (H1N1) pandemic in India: The impact of holiday-related school closure. *Epidemics*, 5(4):157–163, December 2013.

[10] Anthony Heymann, Gabriel Chodick, Brian Reichman, Ehud Kokia, and Joseph Laufer. Influence of School Closure on The Incidence of Viral Respiratory Diseases Among Children and on Health Care Utilization. *The Pediatric Infectious Disease Journal*, 23(7):675–677, July 2004.

[11] Colleen C Wheeler, Laura M Erhart, and Megan L Jehn. Effect of school closure on the incidence of influenza among school-age children in Arizona. *Public health reports*, 125(6):851–859, 2010. Publisher: SAGE Publications Sage CA: Los Angeles, CA.

[12] Benjamin J Cowling, Eric HY Lau, Conrad LH Lam, Calvin KY Cheng, Jana Kovar, Kwok Hung Chan, JS Malik Peiris, and Gabriel M Leung. Effects of school closures, 2008 winter influenza season, Hong Kong. *Emerging infectious diseases*, 14(10):1660, 2008. Publisher: Centers for Disease Control and Prevention.

[13] Howard Lempel, Joshua M Epstein, and Ross A Hammond. Economic cost and health care workforce effects of school closures in the US. *PLoS currents*, 1, 2009. Publisher: Public Library of Science.

[14] Kairon Shayne D. Garcia and Benjamin W. Cowan. The Impact of U.S. School Closures on Labor Market Outcomes during the COVID-19 Pandemic, January 2022.

[15] Rebecca Jack and Emily Oster. COVID-19, School Closures, and Outcomes. *The Journal of Economic Perspectives*, 37(4):51–70, 2023.

[16] Dan Goldhaber, Thomas J. Kane, Andrew McEachin, Emily Morton, Tyler Patterson, and Douglas O. Staiger. The Educational Consequences of Remote

and Hybrid Instruction during the Pandemic. *American Economic Review: Insights*, 5(3):377–392, September 2023.

[17] Kirill Borusyak, Xavier Jaravel, and Jann Spiess. Revisiting Event-Study Designs: Robust and Efficient Estimation. *Review of Economic Studies*, page rdae007, February 2024.

[18] Brantly Callaway and Pedro H. C. Sant'Anna. Difference-in-Differences with multiple time periods. *Journal of Econometrics*, 225(2):200–230, December 2021.

[19] Victor Chernozhukov, Hiroyuki Kasahara, and Paul Schrimpf. The association of opening K–12 schools with the spread of COVID-19 in the United States: County-level panel data analysis. *Proceedings of the National Academy of Sciences*, 118(42):e2103420118, October 2021.

[20] Jiafeng Chen and Jonathan Roth. Logs with zeros? Some problems and solutions, November 2023. arXiv:2212.06080 [econ, stat].

[21] Roberto C. Garza, Ricardo Basurto-Dávila, Ismael R. Ortega-Sanchez, Luis Oreste Carlino, Martin I. Meltzer, Rachel Albalak, Karina Balbuena, Pablo Orellano, Marc-Alain Widdowson, and Francisco Averhoff. Effect of Winter School Breaks on Influenza-like Illness, Argentina, 2005–2008. *Emerging Infectious Diseases*, 19(6):938–944, June 2013.

[22] Ryan B. Simpson, Tania M. Alarcon Falconi, Aishwarya Venkat, Kenneth H. H. Chui, Jose Navidad, Yuri N. Naumov, Jack Gorski, Sanjib Bhattacharyya, and Elena N. Naumova. Incorporating calendar effects to predict influenza seasonality in Milwaukee, Wisconsin. *Epidemiology & Infection*, 147, 2019.

[23] Gerardo Chowell, Sherry Towers, Cécile Viboud, Rodrigo Fuentes, and Viviana Sotomayor. Rates of Influenza-like Illness and Winter School Breaks, Chile, 2004–2010. *Emerging Infectious Diseases*, 20(7):1195, July 2014.

[24] Charles Stoecker, Nicholas J Sanders, and Alan Barreca. Success Is something to sneeze at: Influenza mortality in cities that participate in the Super Bowl. *American Journal of Health Economics*, 2(1):125–143, 2016. Publisher: MIT Press.

[25] Liyang Sun and Sarah Abraham. Estimating dynamic treatment effects in event studies with heterogeneous treatment effects. *Journal of Econometrics*, 225(2):175–199, December 2021.

A Appendix: For Online Publication

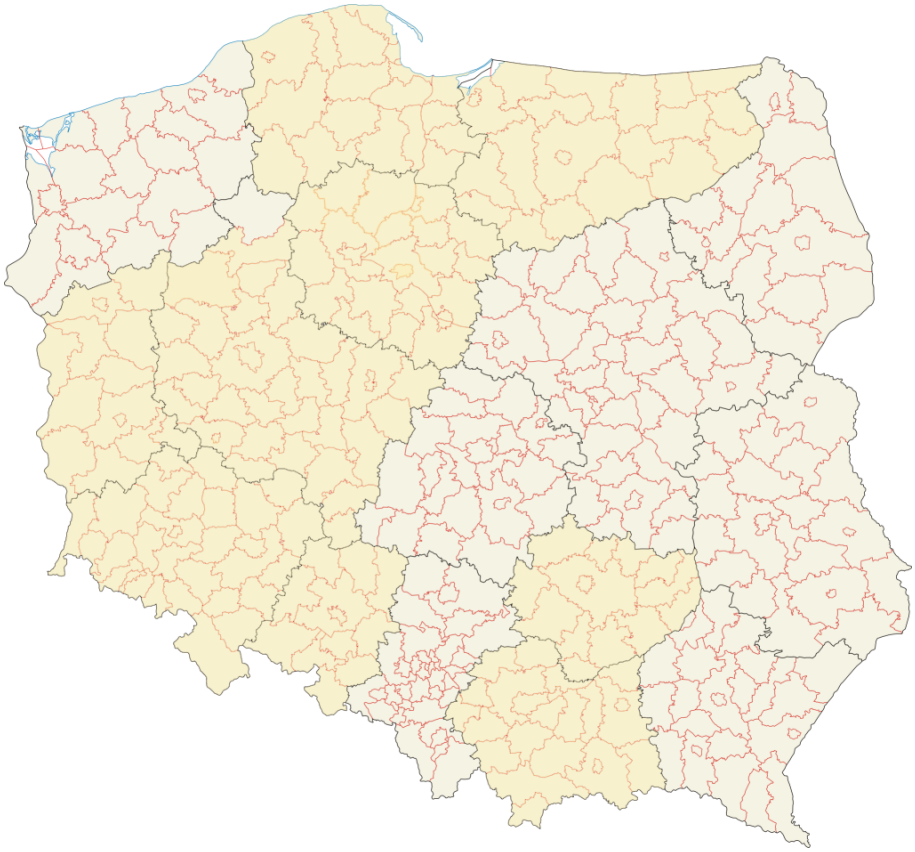


Figure A.1: Counties with data available

Note: Shaded areas represent counties which made their data available

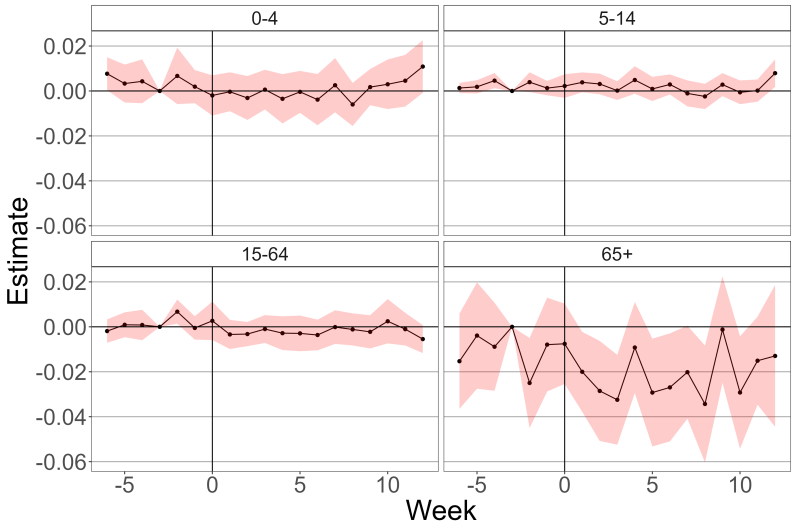


Figure A.2: Event study: Mortality due to external causes by age

Note: Graph shows the impact of winter vacation on the external causes mortality (cat. V in ICD-10) by age. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is the number of deaths due to external causes in the given age category in a county i and week t per 10 000 of inhabitants (of given age). Hence the coefficients show the change in the number of deaths in each week prior and after the vacation, where week 0 is the first week of vacation. Shaded area represents 95% confidence interval for the estimates.

A.1 Robustness Checks

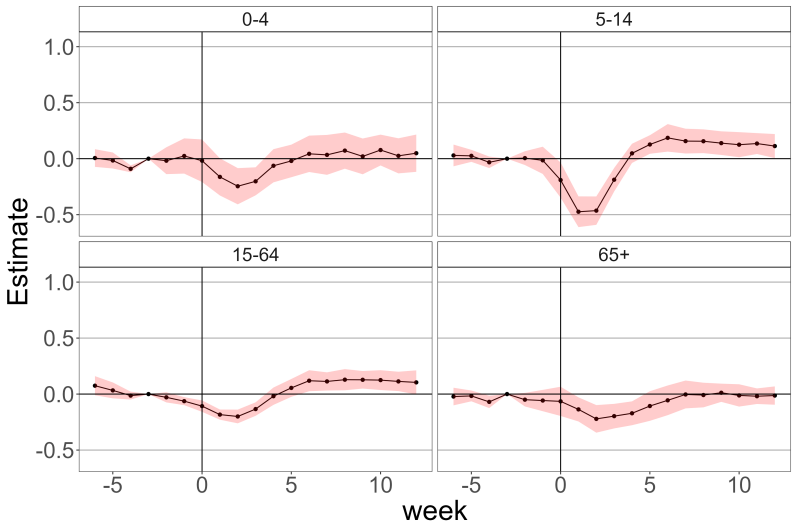


Figure A.3: Event study: log(Incidence+1)

Note: Graph shows the impact of winter vacation on the weekly incidence of infections. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is log of the incidence plus 1. Hence, the coefficients show the percentage change in the incidence in each week prior and after the vacation, where week 0 is the first week of vacation, compared to counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates. Errors are clustered at the region level.

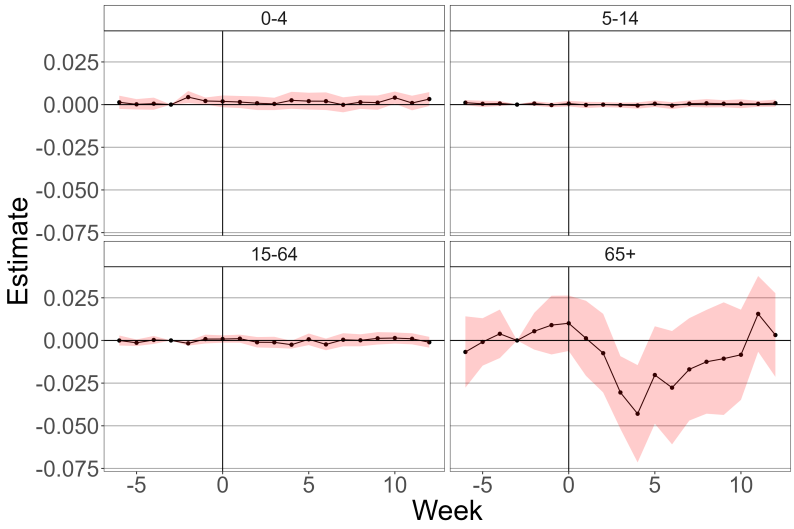


Figure A.4: Event study: log(Mortality+1)

Note: Graph shows the impact of winter vacation on the weekly respiratory mortality. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is log of the mortality plus 1. Hence, the coefficients show the percentage change in the incidence in each week prior and after the vacation, where week 0 is the first week of vacation, compared to counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates. Errors are clustered at the region level.

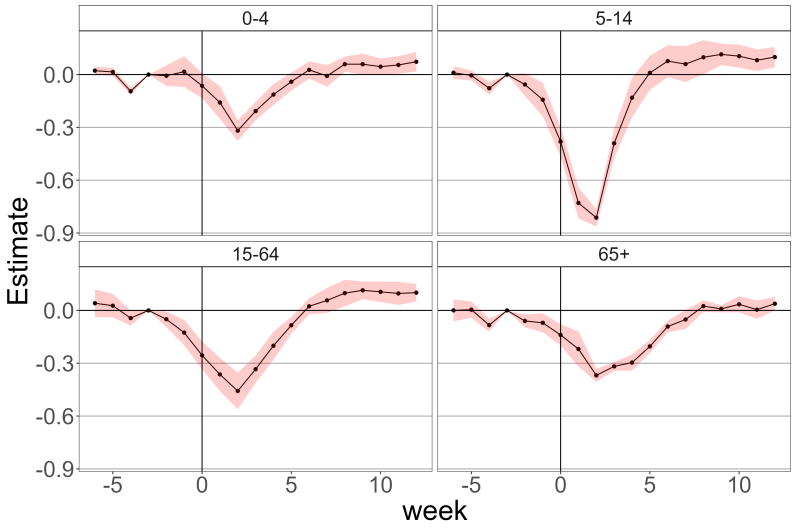


Figure A.5: Poisson Regression: Infections

Note: Graph shows the impact of winter vacation on the external causes mortality (cat. V in ICD-10) by age. Each sub-figure represents results of fitting a Poisson regression following the specification of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is the number of reported infections in a given county and week. Hence, the coefficients show the percentage change in the incidence in each week prior and after the vacation, where week 0 is the first week of vacation, compared to counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates.

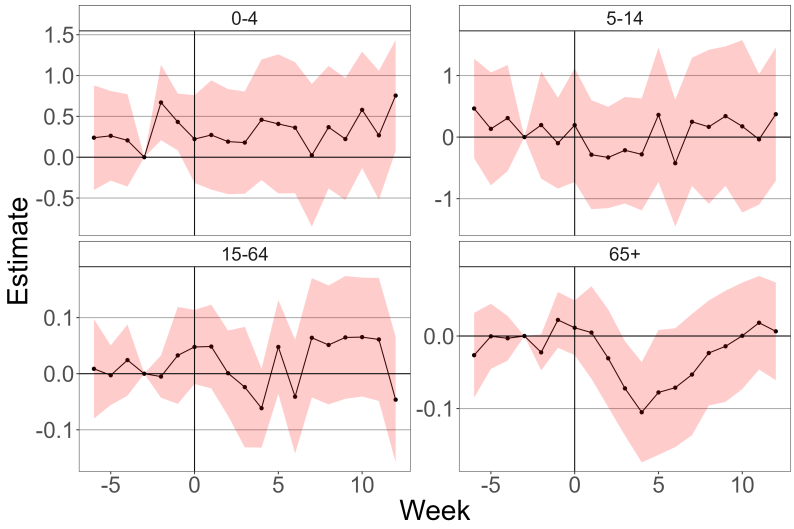


Figure A.6: Poisson Regression: Deaths

Note: Graph shows the impact of winter vacation on the external causes mortality (cat. V in ICD-10) by age. Each sub-figure represents results of fitting a Poisson regression following the specification of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is the number of respiratory deaths in a given county and week. Hence, the coefficients show the percentage change in the incidence in each week prior and after the vacation, where week 0 is the first week of vacation, compared to counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates.

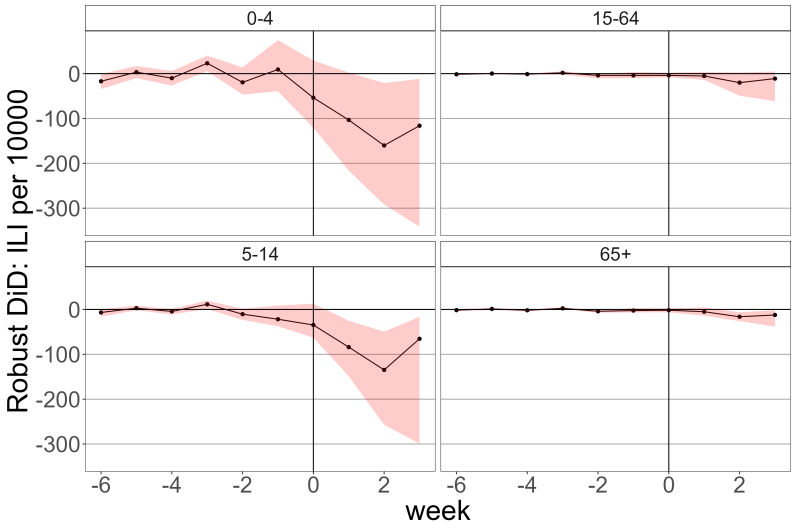


Figure A.7: Robust DiD: Incidence

Note: Graph shows the robust event study estimates based on [18]. Each sub-figure represents results of the estimation on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is the number of reported infections in the given age category in a county c and week w per 10 000 of inhabitants (of given age). Each season is treated as a separate experiment. Only the effect up to week 3 can be estimated, because the latest region starts vacation 4 weeks after the earliest region and hence there is no more untreated units. Standard errors are boostrapped, with randomization at the regional level. Shaded area represents 95% confidence interval for the estimates.

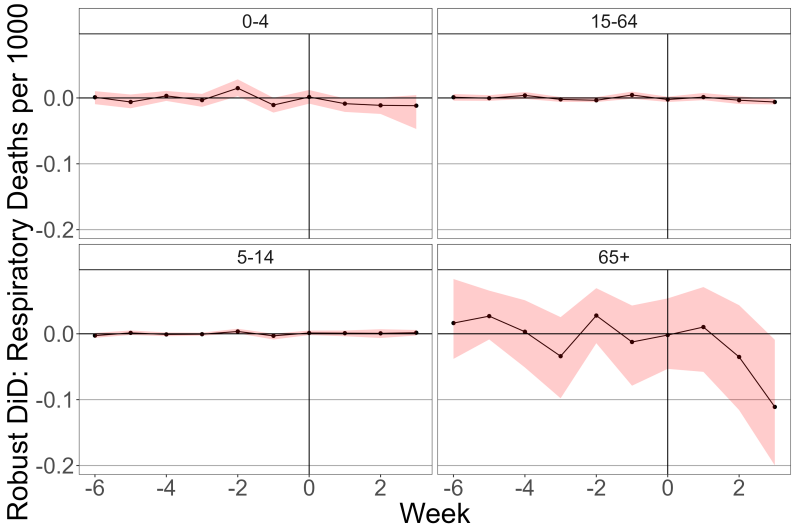


Figure A.8: Robust DiD: Mortality

Note: Graph shows the robust event study estimates based on [18]. Each sub-figure represents results of the estimation on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is the number of deaths in the given age category in a county c and week w per 10 000 of inhabitants (of given age). Each season is treated as a separate experiment. Only the effect up to week 3 can be estimated, because the latest region starts vacation 4 weeks after the earliest region and hence there is no more untreated units. Standard errors are boostrapped, with randomization at the regional level. Shaded area represents 95% confidence interval for the estimates.

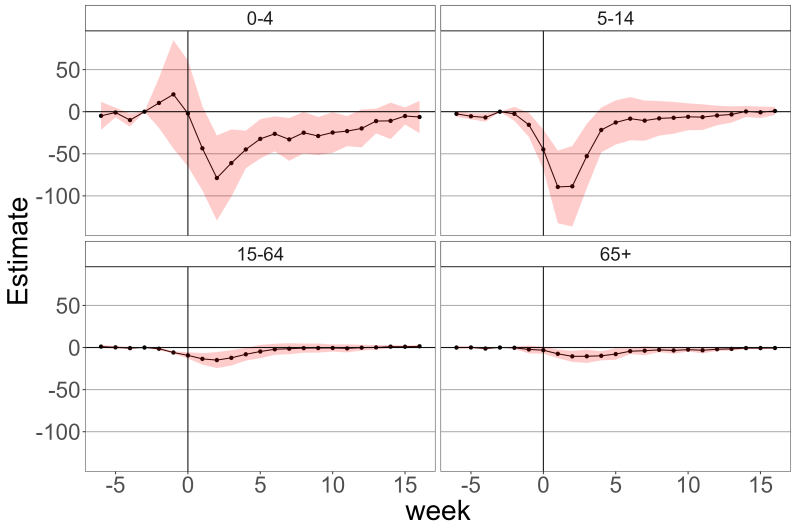


Figure A.9: Incidence Effect up to 16 Weeks

Note: Graph shows the impact of winter vacation on the ILI incidence by age group. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. Line represents estimates of the coefficients δ_T from equation where the outcome is number of reported cases in a given county and week (up to week 16 after vacation), per 10 000 inhabitants (of a given age-group). These parameters show the change in incidence in week T, compared to the counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

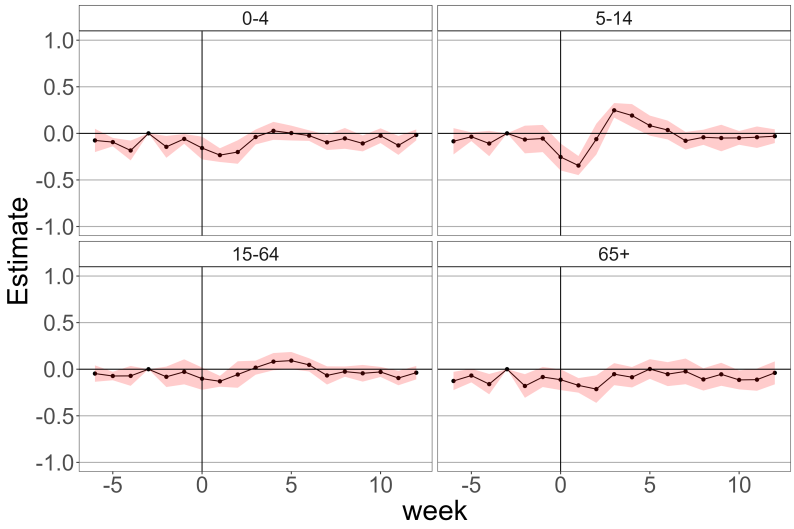


Figure A.10: Effect on Cases Growth Rate

Note: Graph shows the impact of winter vacation on the ILI growth rate by age group. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. Line represents estimates of the coefficients δ_T from equation where the outcome is the growth rate of cases. Growth rate is defined as the difference in log of cases between current and previous week, where -1 is assigned for weeks without cases following [19]. These parameters show the change in growth rate in week T, compared to the counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

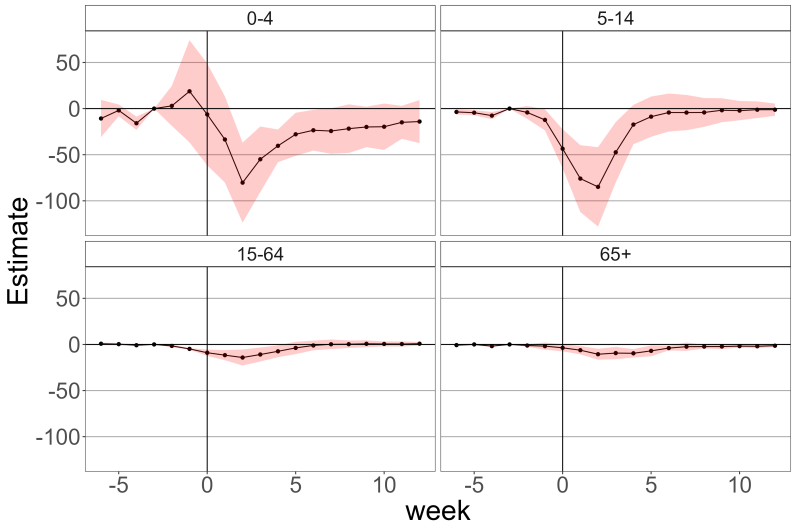


Figure A.11: Effect on Incidence with Standardized Week

Note: Graph shows the impact of winter vacation on the ILI incidence by age group. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. Line represents estimates of the coefficients δ_T from equation where the outcome is number of reported cases per 10 000 inhabitants in a given county and standardized week. The number of cases is adjusted by scaling them to a uniform 7-day period, i.e., multiplying by $\frac{7}{\text{week length}}$. These parameters show the change in incidence in a standardized week T, compared to the counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

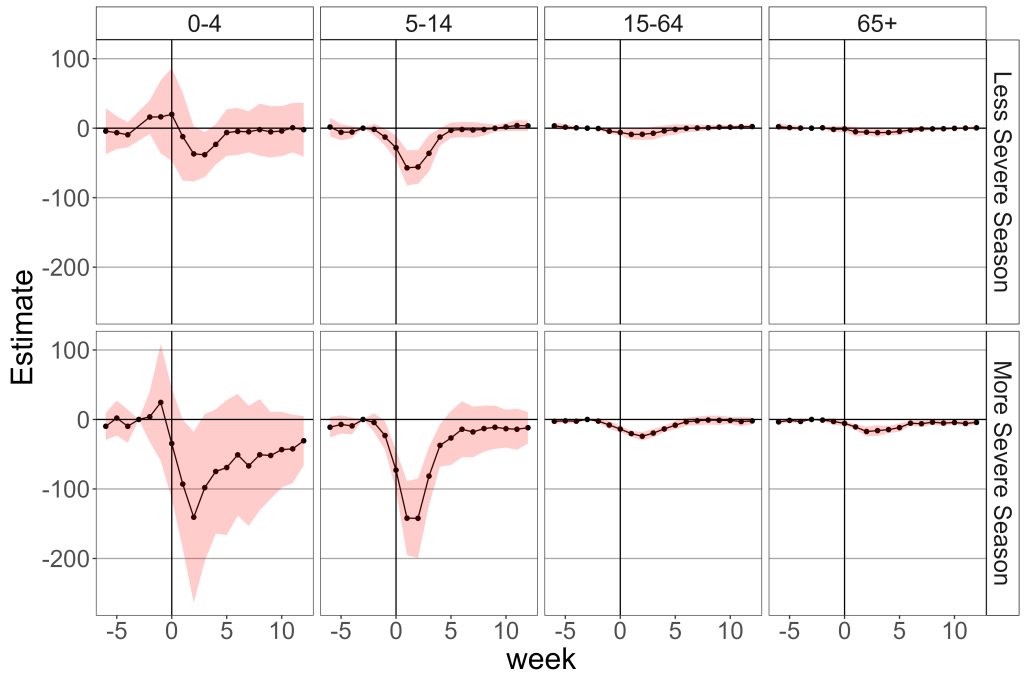


Figure A.12: Effect on Incidence by Season Severity

Note: Graph shows the impact of winter vacation on the ILI incidence by age group and season severity. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+ and to either more or less severe epidemics. More severe epidemics are those epidemics that had total incidence of at least 1000 cases per 10 000. Line represents estimates of the coefficients δ_T from equation where the outcome is number of reported cases per 10 000 inhabitants in a given county and week. These parameters show the change in incidence in a week T, compared to the counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

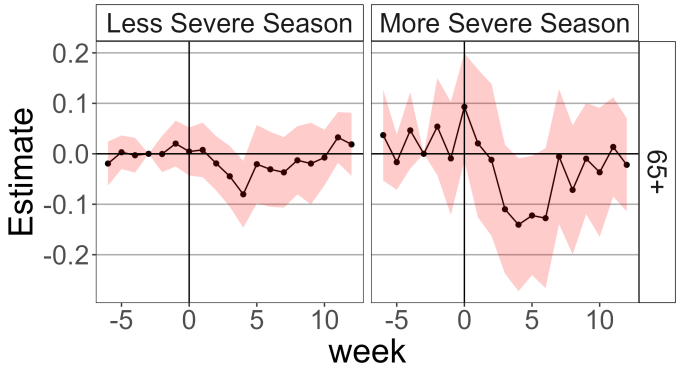


Figure A.13: Effect on Respiratory Mortality by Season Severity among the elderly

Note: Graph shows the impact of winter vacation on the weekly respiratory mortality of individuals 65+ by the season severity. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age group 65+ and to either more or less severe epidemics. More severe epidemics are those seasons that had total incidence of at least 1000 cases per 10 000. Line represents estimates of the coefficients δ_T from equation where the outcome is number of respiratory deaths per 10 000 inhabitants in a given county and week. These parameters show the change in mortality in a week T, compared to the counterfactual without vacation. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

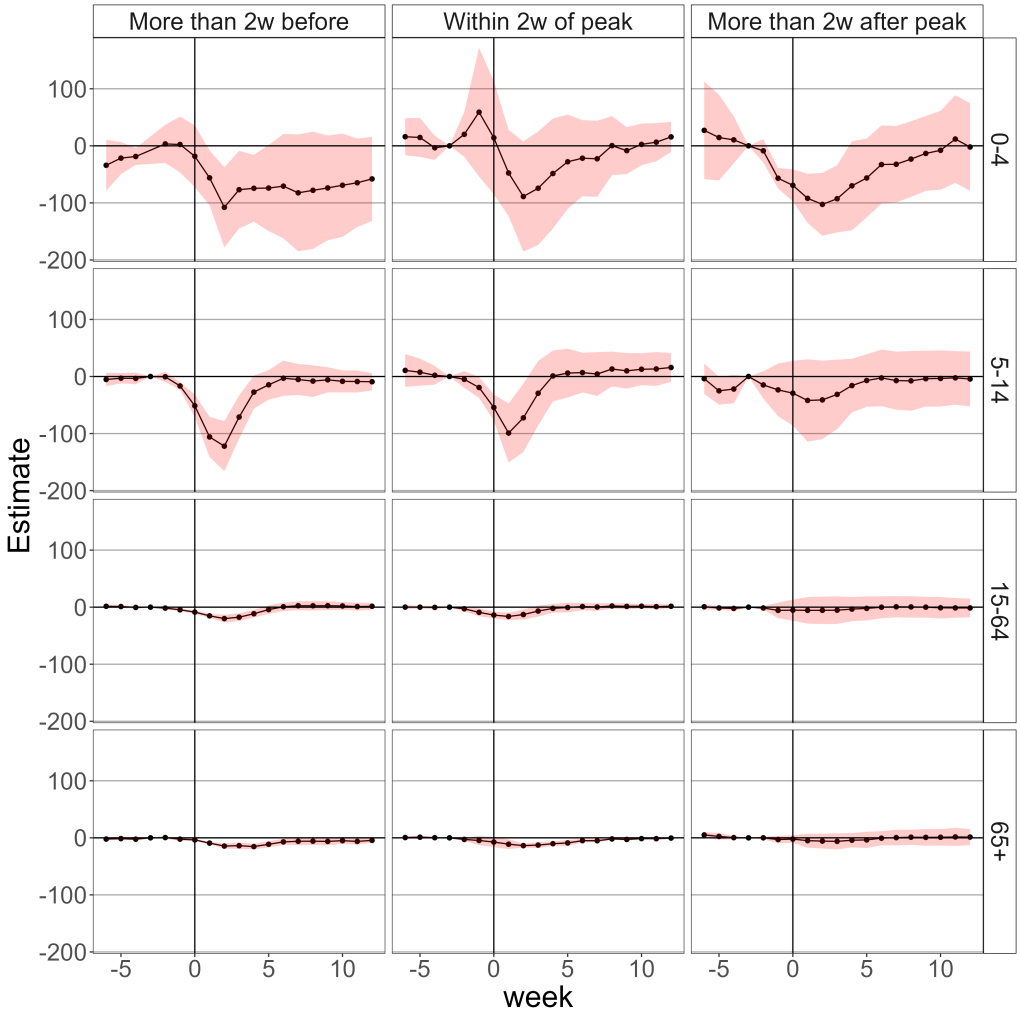


Figure A.14: Effect on Incidence by Peak Timing

Note: Graph shows the impact of winter vacation on the ILI incidence by age group and the timing of the peak. Each sub-figure represents results of the estimation of the event study in equation 1 where the relative week is interacted with an indicator for whether the vacation start occurs two week before, withing or two weeks after the peak. Line represents estimates of the coefficients. These parameters show the change in incidence in a week T, compared to the counterfactual without vacation for three scenarios regarding the peak timing. Shaded area represents 95% confidence interval for the estimates. Standard errors are clustered at the region level.

Table A1: Cumulative 4-Week Decline in Incidence

Age Group	Cumulative 4-Weeks Decline per 10,000	95% Confidence Interval	4-Weeks Average Pre-Vacation per 10,000	Percent Effect
0-4 years	-217	[-362, -72.2]	824.8	-26%
5-14 years	-272	[-414, -130]	364.6	-75%
15-64 years	-48.6	[-74.9, -22.4]	87.62	-55%
65+ years	-30.3	[-50.9, -9.75]	98.5	-31%

Note: Cumulative 4-Weeks Average Pre-Vacation Incidence and cumulative 4-Weeks decline by age group estimated as the linear combination of the event study estimates from equation 1

Table A2: Cumulative Effects of Weeks 0-16 on Incidence

Model	Estimate	95% Confidence Interval
0-4 years	-477	[-671, -282]
5-14 years	-364	[-706, -22.5]
15-64 years	-65.2	[-150, 19.6]
65+ years	-74.4	[-128, -20.4]

Note: Cumulative effect of weeks 0-16 estimated as the linear combination of the event study estimates.

Table A3: Cumulative 4-Week Decline in Incidence by Season's Severity

	Less Severe Season		**More Severe Season**	
Age Group	Estimate	95% CI	Estimate	95% CI
0-4	-67.5	[-265, 130]	-366	[-756, 23.4]
5-14	-177	[-261, -93.7]	-439	[-612, -265]
15-64	-31.2	[-60.1, -2.18]	-78	[-94.1, -61.9]
65+	-18.3	[-38.9, 2.35]	-49.7	[-67.5, -32]

Note: Cumulative effect of weeks 0-4 on incidence estimated as the linear combination of the event study estimates interacted with season's severity dummy. Severe season is defined as the one with total incidence of at least 1000 per 10 000.

Table A4: Cumulative Decline in Mortality in Weeks 2-5 by Season's Severity

	Less Severe Season		**More Severe Season**	
	Estimate	95% CI	Estimate	95% CI
0-4	0.00876	[-0.0192, 0.0367]	0.041	[-0.0295, 0.112]
5-14	-0.003	[-0.0171, 0.0111]	-0.015	[-0.0411, 0.0111]
15-64	0.00185	[-0.00845, 0.0122]	-0.00921	[-0.0295, 0.0111]
65+	-0.165	[-0.408, 0.0782]	-0.385	[-0.873, 0.104]

Note: Cumulative effect of weeks 2-5 after vacation on respiratory mortality estimated as the linear combination of the event study estimates interacted with season's severity dummy. Severe season is defined as the one with total incidence of at least 1000 per 10 000.

Table A5: Cumulative 4-Week Decline in Incidence by Peak Timing

Age	**More than 2w before**		**Within 2 of peak**		**More than 2w after peak**	
Group	Estimate	95% CI	Estimate	95% CI	Estimate	95% CI
0-4	-258.9	[-488.6, -29.3]	-196.7	[-501.4, 107.8]	-356.6	[-533.3, -179.834]
5-14	-350.5	[-483.2, -217.7]	-255.2	[-432.0, -78.4]	-143.5	[-399.9, 112.883]
15-64	-61.4	[-79.9, -42.9]	-49.9	[-73.3, -26.5]	-21.5	[-111.5, 68.3]
65+	-40.9	[-53.5, -28.3]	-44.8	[-64.0, -25.7]	-18.9	[-64.4, 26.5]

Note: Cumulative effect of weeks 0-4 on incidence estimated as the linear combination of the event study estimates interacted with season's severity dummy. Severe season is defined as the one with total incidence of at least 1000 per 10 000. Sample includes only seasons with full number of weeks.

Table A6: Summary Statistics

Incidence				
	0-4	**5-14**	**15-64**	**65+**
Observations	72,231	72,003	72,003	72,239
R^2	0.84	0.86	0.8	0.8
Within R^2	0.05	0.13	0.11	0.09
Mortality				
	0-4	**5-14**	**15-64**	**65+**
Observations	374,899	374,899	374,899	336,107
R^2	0.078	0.095	0.074	0.17
Within R^2	0.00009	0.00008	0.00005	0.00024

Note: These are summary statistics from estimating the regression in equation 1 and represented in figures 3 and 4

A.2 Additional Plots

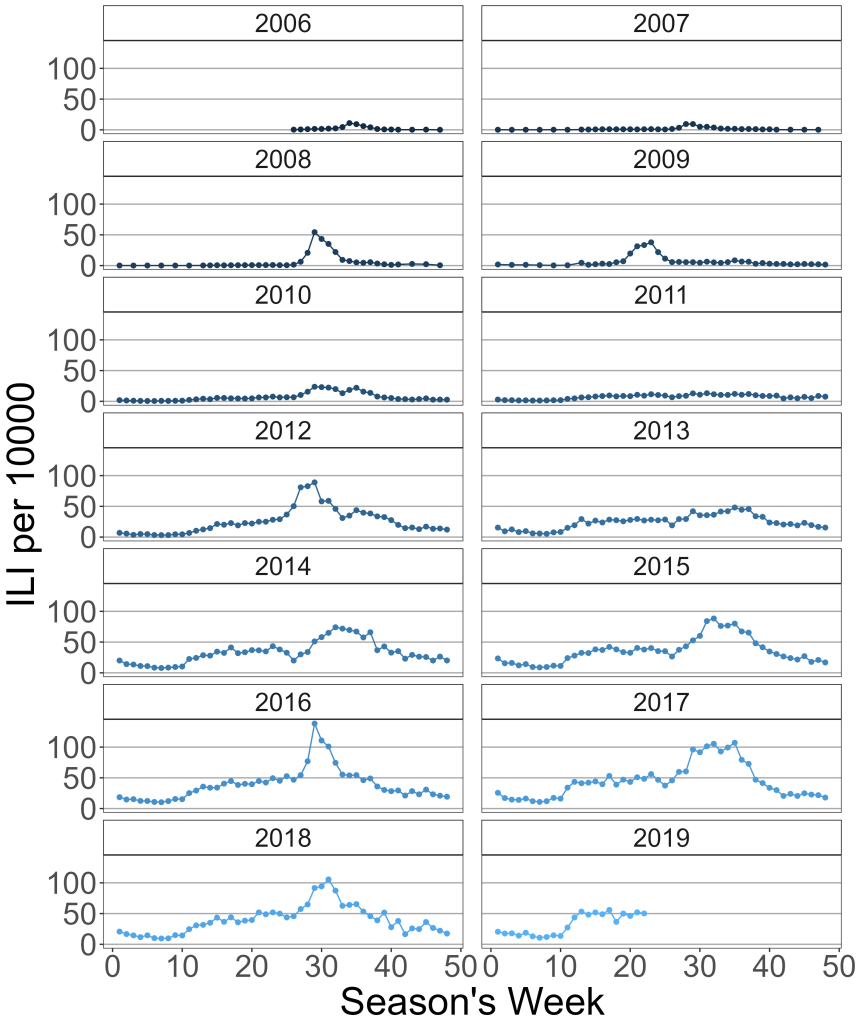


Figure A.15: Incidence by Season

Note: Graph shows the average incidence in each week of each season. Note that in earlier years the reporting was relatively low which resulted in low incidence. Week 1 corresponds to the first week of July (of year t-1) and year is divided into 48 weeks.

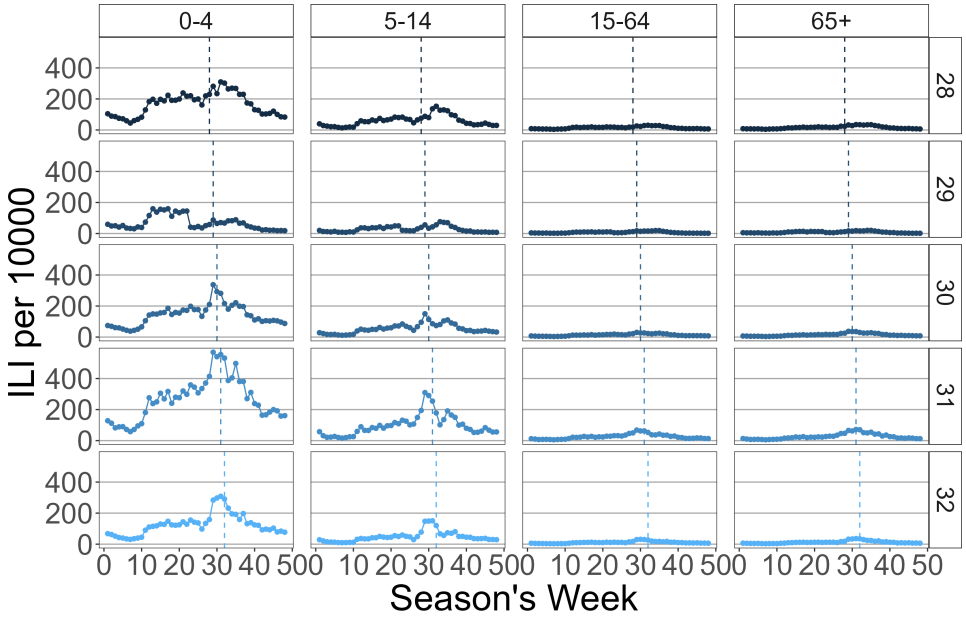


Figure A.16: Incidence by Age and Vacation Week

Note: Graph shows the average incidence in each week divided by groups of the vacation timing (first week of vacation on the left) and age (on the top). Week 1 corresponds to the first week of July and year is divided into 48 weeks.

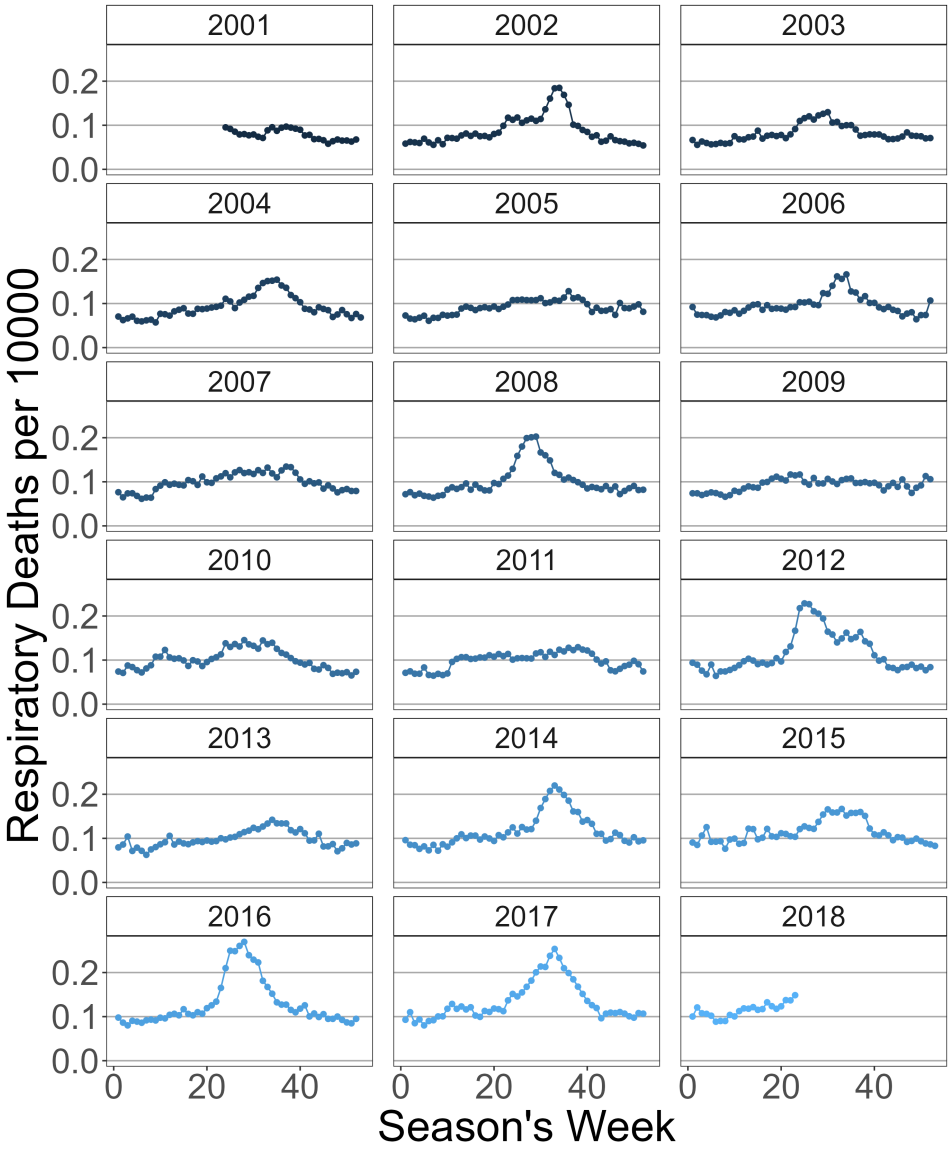


Figure A.17: Mortality by Season

Note: Graph shows the average respiratory mortality in each week of each season. Week 1 corresponds to the first week of July (of year t-1) and year in the mortality data is divided into 52/3 weeks.

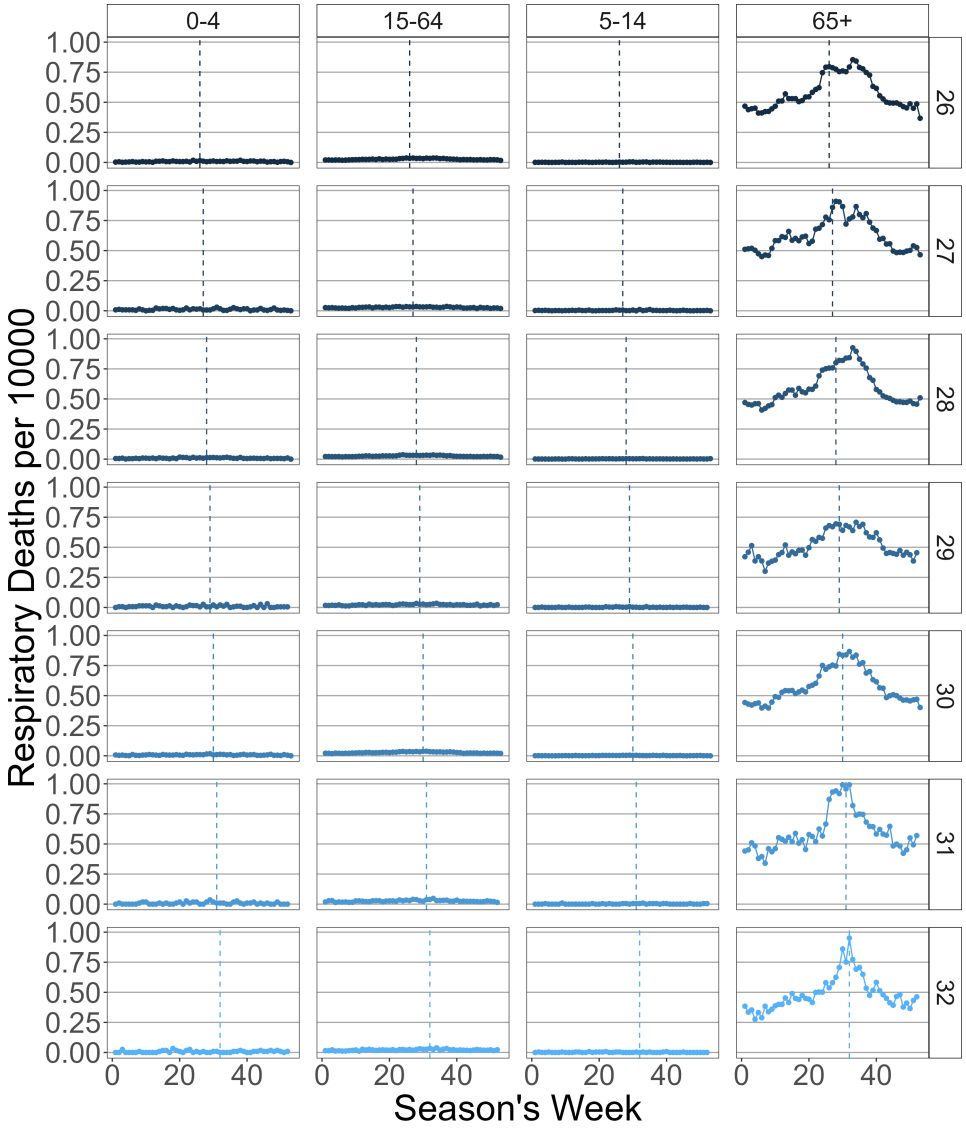


Figure A.18: Mortality by Age and Vacation Week

Note: Graph shows the average respiratory mortality in each week divided by groups of the vacation timing (first week of vacation on the left) and age (on the top). Week 1 corresponds to the first week of July and year in the mortality data is divided into 52/3 weeks.

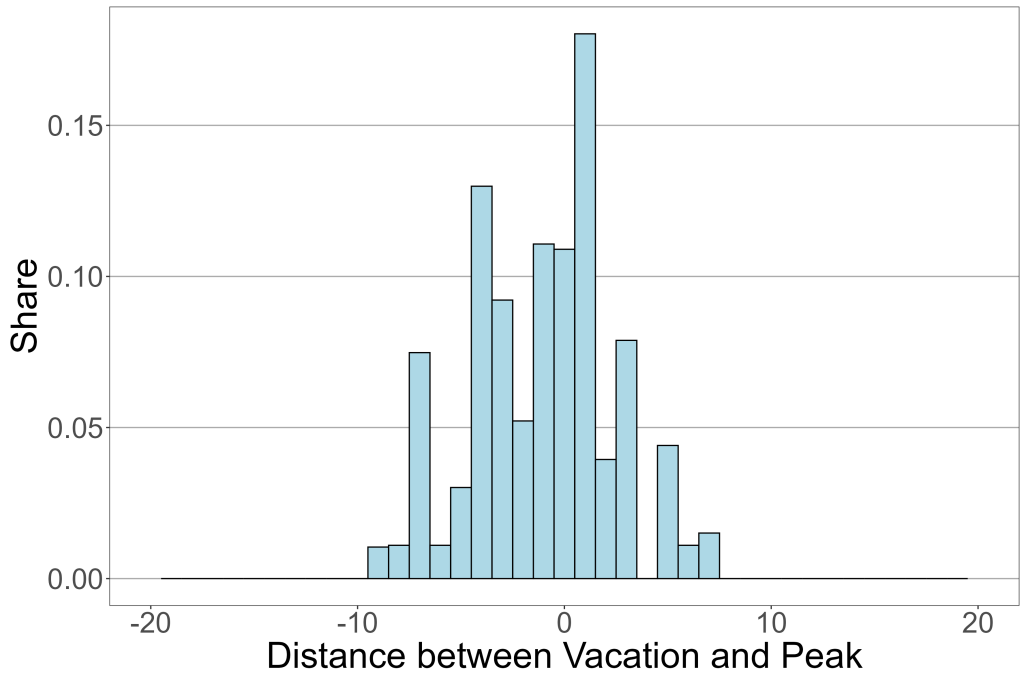


Figure A.19: Distribution of Vacation Weeks Relative to Seasonal Peaks
Note: The graph displays the distribution of the number of weeks between the vacation week and the peak week in the corresponding season. The X-axis represents the number of weeks, with negative values indicating that the vacation occurs before the peak and positive values indicating that it occurs after.

A.3 Event Study: Impact on Transmission

This event study looks at the impact of the winter vacation on the transmission of the virus. It measures the impact of the break on the interactions which fuel the spread of the virus. School recess disrupts interactions in weeks 0 and 1 (recess lasts two weeks). Hence the virus will have a lower reproduction rate from week 0 to 1 and from week 1 to 2. The event study follows the equation:

$$\frac{I_{cwy}}{L_{cwy}} = \sum_{T=-5}^{10} \alpha_T I\{wy-(wy)_c^V = T\}_{cwy} * \widetilde{S_{cwy}} \frac{\widetilde{I_{cwy}}}{L_{cwy}} + \alpha_0 \widetilde{S_{cwy}} \frac{\widetilde{I_{cwy}}}{L_{cwy}} + \delta X_{cwy\cdot} + e_{cwy} \quad (2)$$

Where the L_{cwy} represents the number of reporting doctors in county c, week w and year y. The number of infected individuals (I_{cwy}) is the number of newly reported cases. The susceptible share $\widetilde{S_{cwy}}$ is the total population minus the cumulative number of reported infections since the start of the epidemic season (epidemiological week 24).

Figure 15 shows the results. Vacation clearly disrupts the transmission of the flu, as evidenced by the high decline in the reproduction of the virus in weeks 1 and

2. In other words, the same amount of infections results in fewer secondary cases in weeks 1 because of fewer interactions among children in respective previous weeks. As expected, the parameter returns to the pre-vacation value three weeks after the vacation began as children start interacting again at a usual rate in week 2.

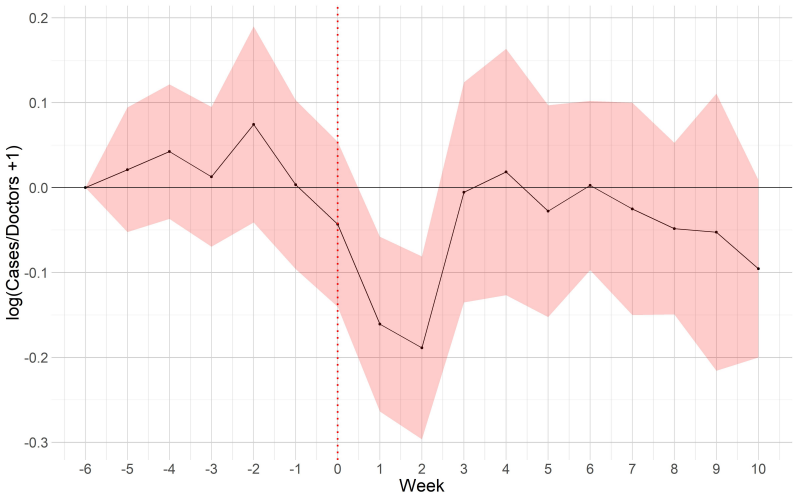


Figure A.20: Impact of winter vacation on transmission

Note: Graph shows the impact of winter vacation on the reported influenza cases. Lines represents estimates of the coefficients α_T from the event study in equation 2 where the outcome has been changed to $log(\frac{I_{i,t}}{L_{i,t}} + 1)$. Shaded area represents 95% confidence interval for the estimates.

A.4 Motivating Model

This section interprets the impact of school closures through the parameters of a simple SIR model. Specifically, school closures directly affect infection rates by temporarily reducing the contact rate and indirectly by decreasing the stock of infections. This is not presented as an accurate depiction of the contagion process but rather as a simple illustration of how the policy operates. In the following subsection, I relate the reduced-form estimates from the event study to this model.

I use the "Susceptible-Infected-Recovered" (SIR) epidemiological model (similar to the one used in [4]). It is a framework wherein an individual's state transitions through being susceptible, infected, and then recovered. The evolution of the infected population within this model is guided by both the inflow of new infections and the outflow of recoveries, as detailed in the following equation:

$$I_{t,a} = \beta_{aa} \frac{S_{t-1,a}}{P_{t-1,a}} I_{t-1,a} + \sum_{b \neq a} \beta_{ab} \frac{S_{t-1,a}}{P_{t-1,a}} I_{t-1,b} \tag{3}$$

The susceptible population evolves according to:

$$S_{t,a} = S_{t-1,a} - I_{t,a} \tag{4}$$

In this equation, $I_{t,a}$ represents the number of infected individuals in age group a at time t, with the infection dynamics determined by three principal factors:

1. **Intra-Age-Group Infections**: New infections within age group a are proportional to the transmission rate (β_{aa}) within this age group between susceptible and previously infected individuals in that age group ($I_{t-1,a}$), adjusted for the the proportion of the population still susceptible ($S_{t-1,a}/P_{t-1,a}$). Transmission rate is a function of contact rate and per-contact transmission probability.

2. **Inter-Age-Group Infections**: New infections stemming from contacts with individuals from age group b (β_{ab}), summed over all age groups.

3. **Recoveries**: Individuals who were sick in the previous time period recovers and add to the susceptible population.

Equation 4 can be used to demonstrate how the policy of school closures impacts infection levels by temporarily reducing contact rates among school-aged children, leading to a decrease in the parameter $\beta_{5-14,5-14}$. This reduction in contact rates results in lower transmission and, consequently, an immediate decline in infections following the start of school vacations. Importantly, the effects of this intervention may extend beyond the vacation period, even after the contact rate has returned to its prior level. As the number of infected individuals decreases, the likelihood of new infections correspondingly falls, potentially maintaining reduced infection rates for an additional 2 to 3 weeks after the recess period. Furthermore, the decline in infections in this group, can fuel a further decline in other age groups.

A.5 Event Study Coefficients

The event study coefficients represent reduced-form estimates of the policy's treatment effects on infections and deaths as the difference between actual outcome in a given week and counterfactual outcome that would have occured in that week without vacation. They are independent of the underlying transmission model specification. While this approach provides robust estimates that are less sensitive to model misspecification, it may be valuable to illustrate how these coefficients relate to parameters in a potential transmission model. This section aims to establish this relationship, focusing on a simplified SIR (Susceptible-Infectious-Recovered) model

for intuitive understanding, while acknowledging that more complex models could be employed for a more comprehensive analysis.

Event study estimates can be conceptualized as a series of difference-in-differences estimates. They approximate the change in outcomes between the time of vacation implementation and an excluded period within a county and age group (first difference), compared to the same change in a control county that has not yet experienced vacation (second difference). Formally, following Sun and Abraham [25], under the assumption of parallel trends, no anticipation, treatment effect heterogeneity across treatment waves, we can express this as:

$$\delta_{ta} = \sum_{c,a} \omega_c E\left[(I_{c,t,a}(1) - I_{c,t,a}(0))\right] \tag{5}$$

Where:

- δ_{ta} is the event study coefficient for time t and age group a
- e is the excluded period
- c is a treated county
- ω_c are weights
- $I_{c,t,a}(1)$ is the outcome in county c, at relative time t, and age group a if treated
- $I_{c,t,a}(0)$ is the counterfactual outcome in county c, at relative time t, and age group a had it been non-treated. Typically it's also a function of outcomes in a county which has not been treated yet.

Under these assumptions, the coefficients represent the average treatment effect at relative period t. In other words, they indicate the difference between what occurs t periods after the vacation versus what would happen in that week without the vacation.

It is important to note that this interpretation is only valid under the stated assumptions. Nonetheless, in the context of this study, these assumptions are likely satisfied.

Firstly, the treatment timing is randomly assigned as counties are quasi-randomly assigned to the vacation group. Therefore, the untreated county k and its outcome $I_{k,t,a}$ serves as a valid counterfactual for what would have occurred in county c without the vacation, that is: $E(I_{c,t,a}(0)) = E(I_{k,t,a})$. The estimator requires a weaker assumption: that the trends in treated and untreated counties would be parallel (in expectation) in the absence of treatment, that is: $E(I_{c,t,a}(0) - I_{c,-3,a}(0)) =$

$E(I_{k,t,a} - I_{k,-3,a})$. Given random assignment, there is no reason to expect a difference between the groups of counties, and consequently, their trends should evolve in parallel.

Secondly, the no anticipation assumption requires that there are no effects during or before the reference period. Since the reference period is set to three weeks before the vacation, it is very unlikely to be affected. This assumption would be violated if people behaved differently knowing that a vacation will happen three weeks ahead, which is improbable.

The final assumption states that the treatment effect in counties treated early and counties treated late would be the same. Random assignment ensures that these counties should, on average, be similar, which limits concerns regarding these assumptions. However, differences can still arise if the counties having vacations at different times have, for instance, different susceptible populations at the time of treatment. While it is likely that counties with later vacations might have smaller susceptible populations, all counties are treated within four weeks, making significant differences unlikely. To address this concern, I also provide estimates from a method robust to this issue designed by Callaway and Sant'Anna [18].

Given the parallel trends assumption, the difference should be zero before the policy implementation, as the treatment only affects outcomes post-intervention. Assuming the policy starts at time $t = 0$, we expect:

$$\delta_{ta} = 0 \quad \text{for} \quad t < 0 \tag{6}$$

A.5.1 Relating Event Study Coefficients to SIR Model Parameters

To illustrate the relationship between event study coefficients and transmission model parameters, we will use a simplified age-structured SIR model. Let $S_{c,t,a}$, $I_{c,t,a}$, and $R_{c,t,a}$ represent the number of susceptible, infectious, and recovered individuals, respectively, in county c, at time t, for age group a. The total population in each group is denoted by $P_{c,t,a}$.

We define the following transmission rates:

- $\beta^s_{a,b}$: transmission rate from age group b to age group a during the school term
- $\beta^v_{a,b}$: transmission rate from age group b to age group a during vacation

For simplicity, we assume that only school-aged children (5-14 years old) are directly affected by school vacation, and other transmission rates stay constant. This assumption can be relaxed in more complex models, to allow other groups to

be affected directly as well. I will also assume, for the clarity of the illustration, that the effects is homogenous and hence the ω_k can be ommited.

1. At the start of the intervention ($t = 0$) for school-aged children:

$$\delta_{0,5-14} = E\left[I_{c,0,5-14}(1) - I_{c,0,5-14}(0)\right] \tag{7}$$

$$= E\left[\beta^v_{5-14,5-14}\frac{S_{c,-1,5-14}I_{c,-1,5-14}}{P_{c,-1,5-14}} + \sum_{b\neq 5-14}\beta_{5-14,b}\frac{S_{c,-1,b}I_{c,-1,b}}{P_{c,-1,5-14}}\right] \tag{8}$$

$$- E\left[\beta^s_{5-14,5-14}\frac{S_{c,-1,5-14}I_{c,-1,5-14}}{P_{c,-1,5-14}} + \sum_{b\neq 5-14}\beta_{5-14,b}\frac{S_{c,-1,5-14}I_{c,-1,b}}{P_{c,-1,5-14}}\right] \tag{9}$$

$$= E\left[\left[\beta^v_{5-14,5-14} - \beta^s_{5-14,5-14}\right]\frac{S_{c,-1,5-14}I_{c,-1,5-14}}{P_{c,-1,5-14}}\right] \tag{10}$$

This coefficient represents the immediate effect of changing the transmission rate due to vacation. Note that other age groups are not affected in this first period.

2. For the subsequent period during vacation for school-aged children:

$$\delta_{1,5-14} = E\left[I_{c,1,5-14}(1) - I_{c,1,5-14}(0\right] \tag{11}$$

$$+ E\left[\beta^v_{5-14,5-14}\frac{S_{c,0,5-14}(1)I_{c,0,5-14}(1)}{P_{c,0,5-14}} + \sum_{b\neq 5-14}\beta_{5-14,b}\frac{S_{c,0,b}I_{c,0,b}}{P_{c,0,5-14}}\right] \tag{12}$$

$$- E\left[\beta^s_{5-14,5-14}\frac{S_{c,0,5-14}(0)I_{c,0,5-14}(0)}{P_{c,0,5-14}} + \sum_{b\neq 5-14}\beta_{5-14,b}\frac{S_{c,0,5-14}I_{c,0,b}}{P_{c,0,5-14}}\right] \tag{13}$$

$$= E\left[\beta^s_{5-14,5-14}\frac{S_{c,0,5-14}(1)}{P_{c,0,5-14}}I_{c,0,5-14}(1) - \beta^v_{5-14,5-14}\frac{S_{c,0,5-14}(0)}{P_{c,0,5-14}}I_{c,0,5-14}(0)\right] \tag{14}$$

This coefficient captures both the change in transmission rates and the cumulative effects on the stocks of susceptible and infected individuals in the previous period.

3. For other age groups ($b \neq 5-14$), in the first week after vacation start:

$$\delta_{1,b} = E\left[I_{c,1,b}(1) - I_{c,1,b}(0)\right] \tag{15}$$

$$= E\left[\sum_{a\neq 5-14} \beta_{b,a} I_{c,0,a} \frac{S_{c,0,b}}{P_{c,0,b}} + \beta_{b,5-14} I_{c,0,5-14}(1) \frac{S_{c,0,b}}{P_{c,0,b}}\right] \tag{16}$$

$$- E\left[\sum_{a\neq 5-14} \beta_{b,a} I_{c,0,a} \frac{S_{c,0,b}}{P_{c,0,b}} + \beta_{b,5-14} I_{c,0,5-14}(0) \frac{S_{c,0,b}}{P_{c,0,b}}\right] \tag{17}$$

$$= E\left[\beta_{b,5-14} \frac{S_{c,0,b}}{P_{c,0,b}} \left[I_{c,0,5-14}(1) - I_{c,0,5-14}(0)\right]\right] \tag{18}$$

This coefficient reflects the indirect effects of the vacation on other age groups through changes in the infection patterns of school-aged children.

4. After the vacation ends, for all age groups, the coefficient become complex non-linear function of the cumulative effect on stocks of infected and susceptible individuals in the previous periods.

$$\delta_{t,a} = E\left[I_{c,t,a}(1) - I_{c,t,a}(0)\right] \tag{19}$$

$$= E\left[\sum_b \beta_{a,b} \frac{S_{c,t-1,a}(1)}{P_{c,t-1,a}} I_{c,t-1,b}(1)\right] \tag{20}$$

$$- E\left[\sum_b \beta_{a,b} \frac{S_{c,t-1,a}(0)}{P_{c,t-1,a}} I_{c,t-1,b}(0)\right] \tag{21}$$

$$= E\left[\sum_b \beta_{a,b} \left[\frac{S_{c,t-1,a}(1)}{P_{c,t-1,a}} I_{c,t-1,b}(1) - \frac{S_{c,t-1,a}(0)}{P_{c,t-1,a}} I_{c,t-1,b}(0)\right]\right] \tag{22}$$

This coefficient captures the lingering effects of the vacation on infection patterns, even after transmission rates return to pre-vacation levels.

A.5.2 Discussion and Limitations

While this framework provides insights into the relationship between event study coefficients and SIR model parameters, it has several limitations in it interpretation:

1. Simplification: The model assumes a simple SIR structure. More complex models (e.g., SEIR, SIAR) might provide more nuanced insights.

2. Parameter identification: The event study coefficients alone do not allow for

direct identification of specific SIR model parameters. They provide aggregate effects that combine multiple underlying parameters..

3. Spatial heterogeneity: The current framework does not explicitly model spatial interactions between counties.

Despite these limitations, this approach offers valuable insights for policymakers by providing causal treatment effects that are robust to model misspecification. This simplified derivation serves to bridges the gap between reduced-form empirical analysis and structural modeling, providing a more comprehensive toolkit for epidemiological policy evaluation.

A.6 Simulations

A.6.1 Methodology

The goal of these simulations is to verify the performance of the estimation methods in a controlled setting where the true treatment effects are known. By comparing the estimated treatment effects to the true treatment effects, we can assess the accuracy of the methods. The treatment effects are defined as the differences between outcomes with and without vacations.

A.6.2 Setting

1. **Setting**: Infection outcomes were generated for 381 Polish counties over 5 seasons for each of the age groups: `0-4`, `5-14`, `15-64`, and `65+`. The simulation will produce outcomes aggregated at a weekly level for 48 weeks(as in the original data) in each season.

A.6.3 Data Generation

2. **Simulation Process without Vacation**: The simulation iteratively calculated the number of new infections (I) and susceptible individuals (S) over time according to the simple process below. This simplified process was chosen for illustrative purposes, but similar results could be shown for other data generating processes. Since the focus is on estimating the effects of a policy on outcomes rather than particular parameters of the model, the estimation is independent of the underlying data generating process.

$$I_t = \min\left(\beta_s I_{t-1} S_{t-1}, 1\right)$$

$$S_t = \max(S_{t-1} - I_t, 0)$$

Where I_{t-1} and S_{t-1} are 4×1 vectors of the share of infected and susceptible populations in each age group. The time period corresponds to half a week to match the generation time of influenza. Once the infections are generated at each step, the number of infections is aggregated at a weekly level to assimilate the data used in the paper.

3. **Initialization**: Initial susceptible (S_0) and infected (I_0) fractions were set at 0.85 and 5×10^{-4}, respectively, across all age groups.

4. **Transmission Rates**: The following transmission matrix was used in the absence of school vacation (row and column entries are in order: `0-4`, texttt5-14, `15-64`, and `65+`).

   - **School Year Transmission Matrix (β_s)**:

$$\begin{pmatrix} 0.18 & 0.12 & 0.06 & 0.06 \\ 0.12 & 0.6 & 0.36 & 0.24 \\ 0.06 & 0.36 & 0.6 & 0.36 \\ 0.06 & 0.24 & 0.36 & 0.6 \end{pmatrix}$$

This results in the following infection patterns across the age groups:

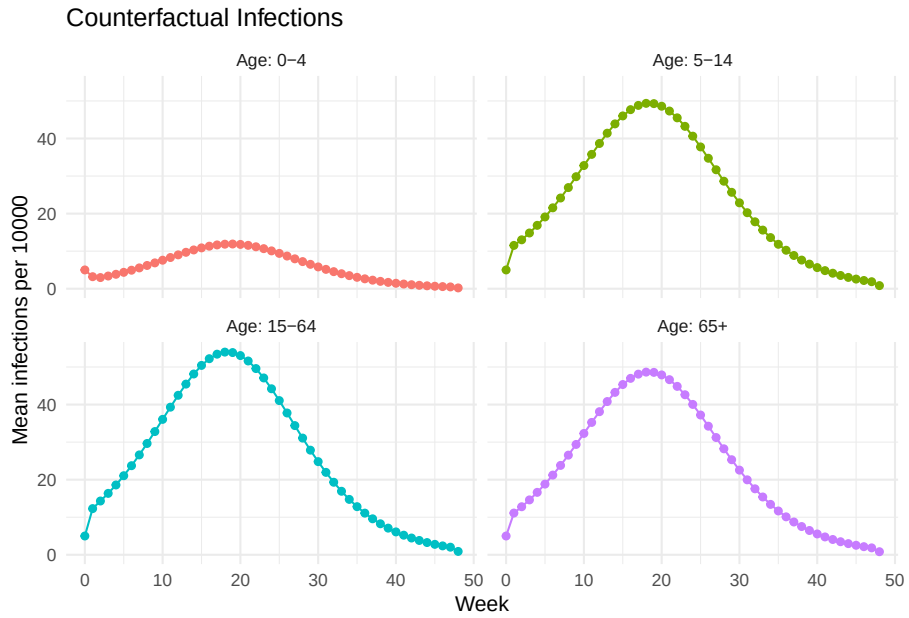


Figure A.21: Counterfactual Simulated Incidence without Vacation

5. **Process with Vacation**: The process is identical to the one described above until the vacation periods. Each county was randomly assigned to one of the 4 vacation periods. Vacation can start in weeks 18, 19, 20, 21. During the vacation periods, the transmission matrix β_s switches to β_v to account for the lower contact rate among school-aged children (2nd group):

   - **Vacation Transmission Matrix (β_v)**:

$$\begin{pmatrix} 0.18 & 0.12 & 0.06 & 0.06 \\ 0.12 & 0.06 & 0.36 & 0.24 \\ 0.06 & 0.36 & 0.6 & 0.36 \\ 0.06 & 0.24 & 0.36 & 0.6 \end{pmatrix}$$

After the vacation, the transmission matrix returns to β_s. This procedure produces the following infection patterns across the age groups:

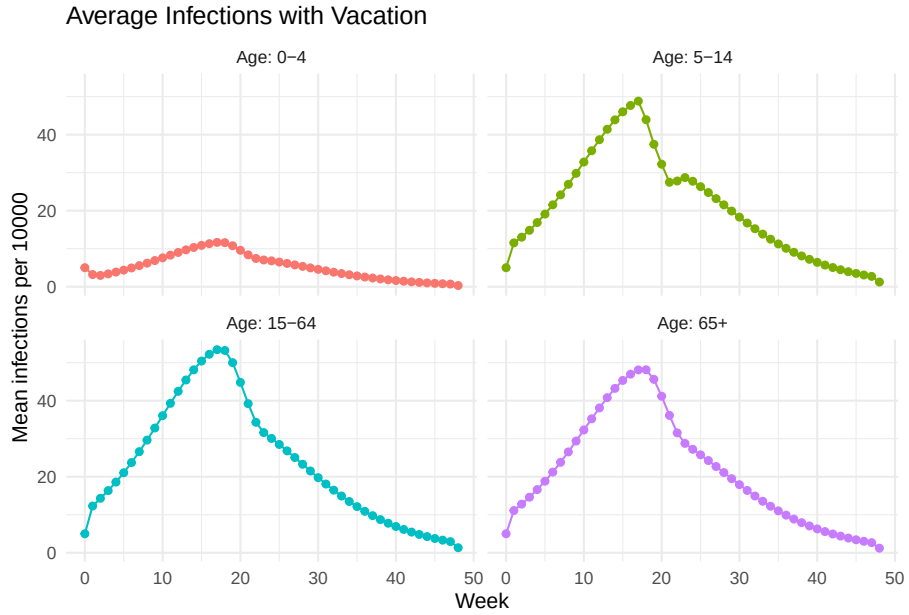


Figure A.22: Actual Simulated Incidence with Vacation

Similarly, below are the infection patterns split by when the vacation occurs, with a dotted line showing the first week of vacation. The outcomes before the vacation are identical to the counterfactual scenario without vacations.

They change after the vacation. The difference between the counterfactual outcomes and outcomes under the scenario of vacation is the treatment effect. It is represented below for each vacation period and age group:

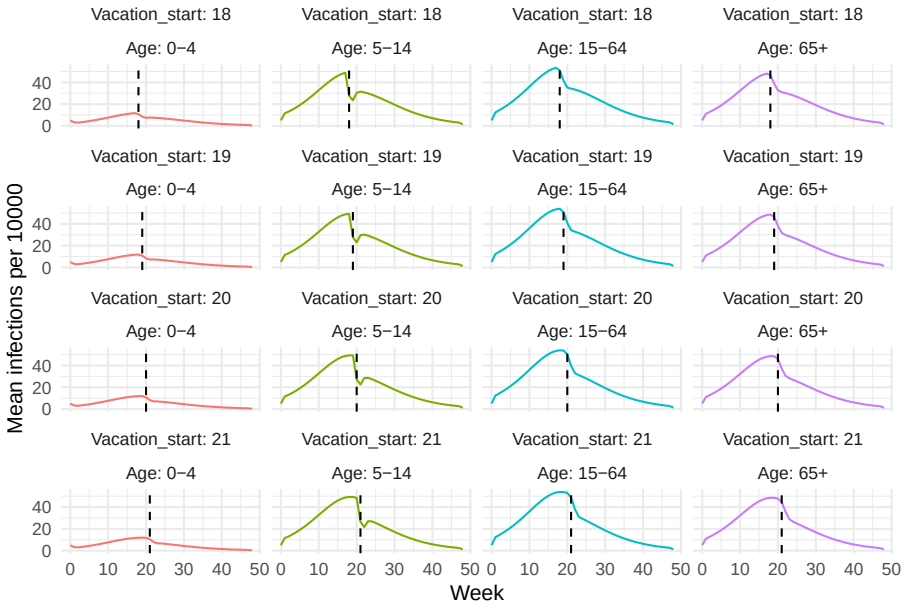


Figure A.23: Actual Simulated Incidence with Vacation by Vacation Week

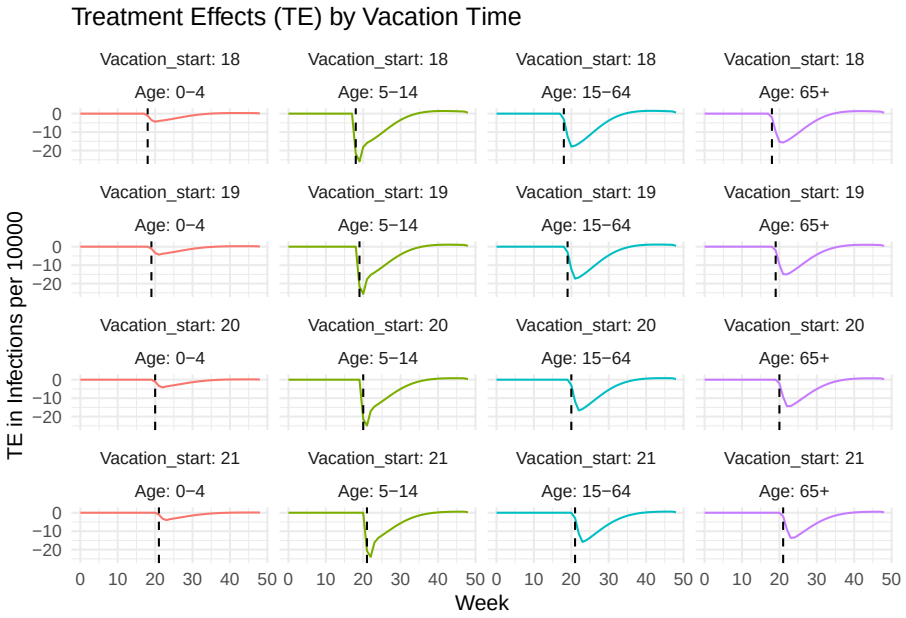


Figure A.24: Treatment Effect by Vacation Week

Similarly, we can focus on the average treatment effect across all vacation periods for the 6 weeks before and 12 weeks after vacation (which corresponds to the estimation target).

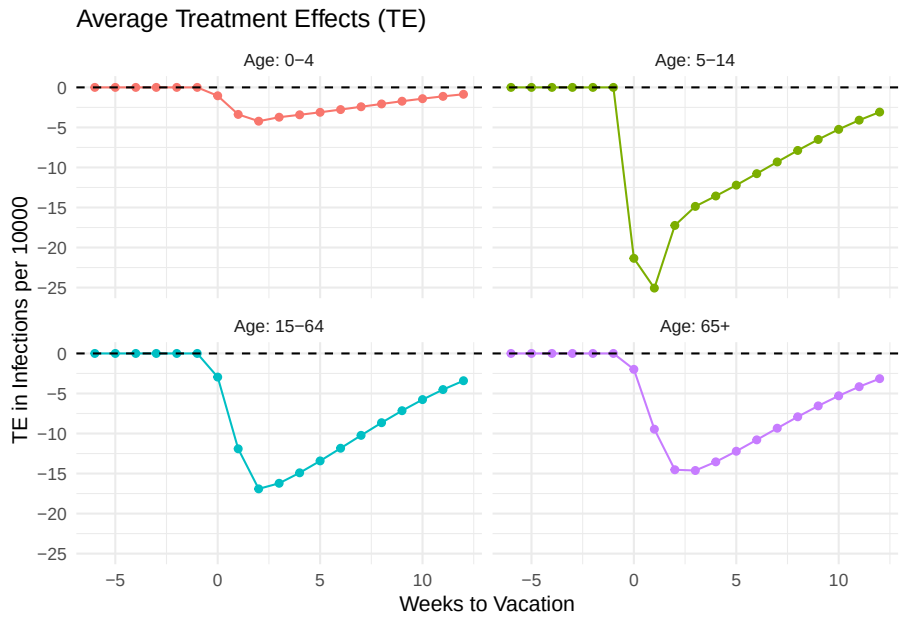


Figure A.25: Average Treatment Effect

A.6.4 Estimation

6. **Event Study Design**: I analyze the impact of school vacations on infection rates using an event study framework. The week of vacation start was treated as the event week, with weeks relative to the event week being considered as `event` time. I estimate the following regression:

$$y_{cwya} = \sum_{\substack{T=-6 \\ T\neq -3}}^{12} \delta_{Ta} I\{wy - (wy)_c^V = T\}_{cwy} + \gamma_{wya} + \theta_{cwa} + \lambda_{cya} + e_{cwya}$$

Please refer to the paper for the exact interpretation of the coefficients and variables. The coefficients δ_{Ta} represent the estimates of the treatment effects for each age group a and week T relative to vacation. I fit models with multiple outcomes as detailed below:

7. **Outcomes**:

   - **Levels**: Direct levels of infection rates per 10000 (I_{cwya}).
   - **Log Transformation**: Log-transformed infection rates plus one ($\log(I_{cwya}+1)$). The one aims to avoid the issue of the log of zero. The coefficient in this case represents the treatment effect in percentage terms.

- **Inverse Hyperbolic Sine Transformation**: A transformation similar to the log but defined for all real numbers. The coefficient in this case represents the treatment effect in percentage terms.
- **Poisson Regression**: Suitable for count data. The coefficient in this case represents the treatment effect in percentage terms.
- **Robust Difference-in-Differences**: An estimator from Callaway & Sant'Anna (2021). Used to address the potential bias stemming from the heterogeneity in the treatment effects across cohorts. It can be used only for periods for which untreated units exist. Hence, extrapolation further than week 2 after the start of vacation is not possible because by that time all counties are treated, meaning they experience vacation.

Below are the plots of estimation results plotted against the true target treatment effect.

First is the estimation in levels vs the true treatment effect:

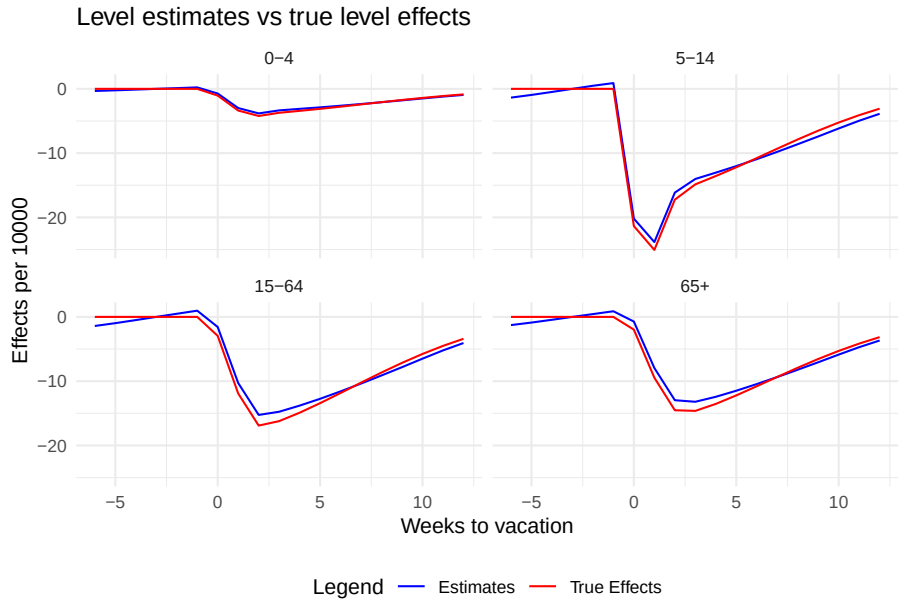


Figure A.26: Estimates and True Effects: Levels

Second is the estimation with outcome in log vs the true percentage treatment effect:

Third is the estimation with outcome in inverse hyperbolic sine transformation vs the true percentage treatment effect:

Fourth is the estimation of the Poisson model vs the true percentage treatment effect (relative to the mean in the untreated group):

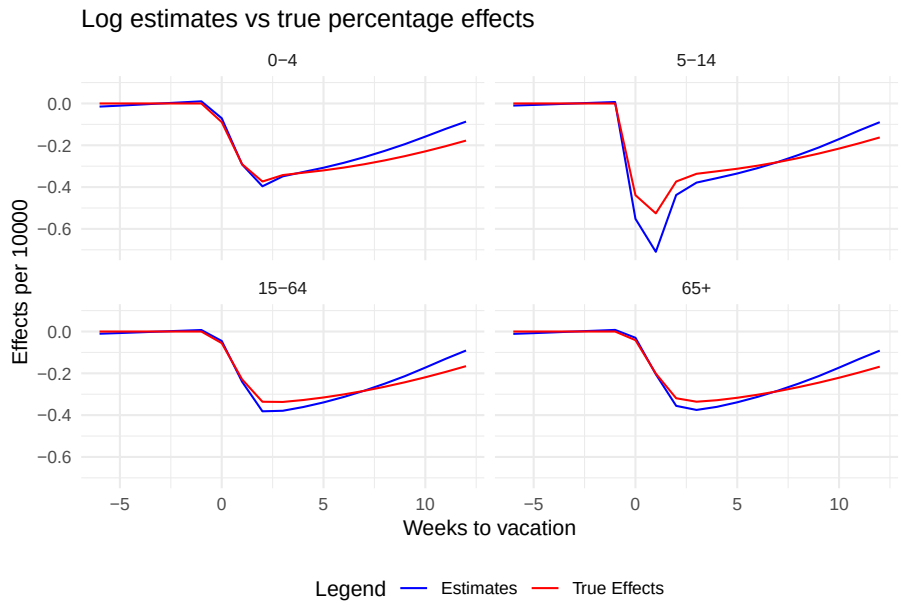


Figure A.27: Estimates and True Effects: Log+1

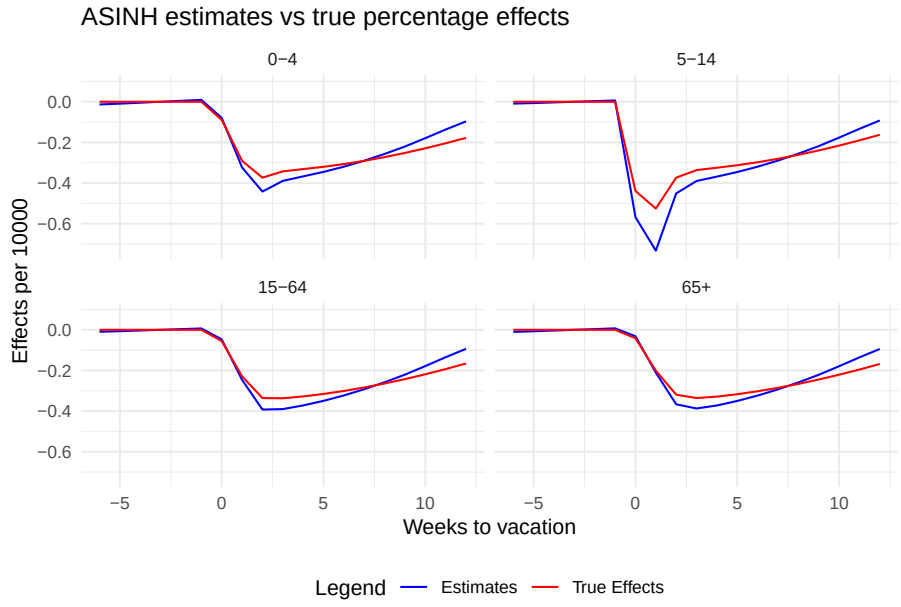


Figure A.28: Estimates and True Effects: Inverse Hyporbolic Sine

Last is the estimation of the robust difference-in-differences model vs the true level treatment effects for units with available untreated comparison. It is only available for the number of periods corresponding to weeks with at least one untreated county.

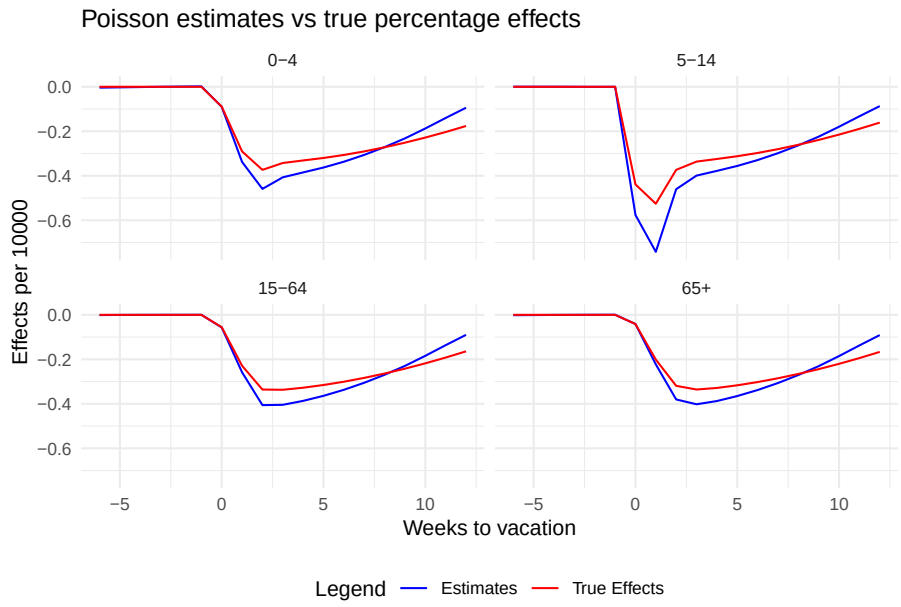


Figure A.29: Estimates and True Effects: Poisson

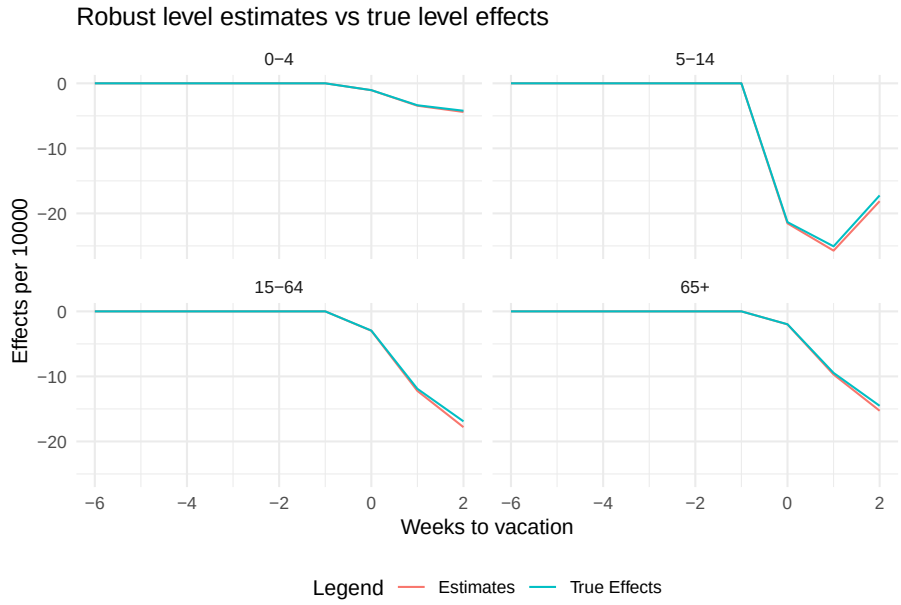


Figure A.30: Estimates and True Effects: Robust DiD

A.6.5 Bias Calculation

8. **Bias Estimation**: The bias of each estimation method was calculated by comparing the estimated treatment effects to the true treatment effects from the simulations.

I sum the absolute differences between the estimated treatment effects and the true treatment effects and divide it by the sum of true treatment effects. Thus, it represents the total deviation from the true treatment effect in percentage terms.

$$Bias_a = \frac{\sum_{t=-6}^{12} |\delta_{Ta} - TE_{Ta}|}{\sum_{t=-6}^{12} |TE_{Ta}|}$$

Age	Levels	Log(I+1)	Inverse Hyperbolic Sine	Poisson	Robust DID
0-4	0.11	0.15	0.15	0.16	0.00
5-14	0.09	0.18	0.20	0.21	0.00
15-64	0.12	0.13	0.14	0.16	0.00
65+	0.13	0.13	0.14	0.16	0.00

Table A7: Biases for Different Methods and Age Groups

A.6.6 Discussion

1. Levels

- Description: This method estimates the effects on direct levels of infection rates per 10,000 individuals in an event study framework.

- Results: The plots show that the level estimates closely track the true treatment effects across all age groups. The deviations are relatively small, indicating good accuracy.

- Bias: The total bias for this method is relatively small and ranges from 0.09 to 0.13 across different age groups, with the lowest bias in the 5-14 age group and the highest in the 65+ age group. Most of the estimates are conservative, slightly underestimating the true effect initially and overestimating it in future periods. This difficulty in extrapolating results for future periods is linked to potential heterogeneity of the treatment effect, as discussed in [17]. This issue is addressed by the robust difference-in-differences method.

2. Log Transformation (Log(I+1))

- Description: This method is an event study which uses a log transformation of the infection rates (plus one) to avoid the issue of taking the log of zero. The coefficients aim to represent the treatment effect in percentage terms.

- Results: The log estimates are slightly less accurate than the level estimates, particularly for the younger age groups (0-4 and 5-14). However, they still follow the true treatment effects reasonably well.

- Bias: The bias ranges from 0.13 to 0.18, with the highest bias observed in the 5-14 age group. This indicates a higher tendency to overestimate or underestimate percentage changes compared to the level estimates. This method tends to overestimate the impact at the beginning of the treatment and underestimate the long-term impact.

3. Inverse Hyperbolic Sine Transformation

- Description: This transformation is similar to the log but defined for all real numbers. It is useful for handling data with zeros or negative values.

- Results: The results are comparable to the log transformation, with slightly higher deviations in the younger age groups. The method captures the overall trend of the true treatment effects but with more variability.

- Bias: The bias is similar to the log transformation, ranging from 0.14 to 0.20. The highest bias is again in the 5-14 age group, suggesting similar strengths and weaknesses as the log transformation.

4. Poisson Regression

- Description: This method is suitable for count data and represents the treatment effect in percentage terms relative to the mean in the untreated group.

- Results: The Poisson estimates perform relatively worse compared to other methods.

- Bias: The bias ranges from 0.16 to 0.21, with the highest bias in the 5-14 age group. While the Poisson method effectively handles periods with zero infections, it comes at the cost of slightly higher bias.

5. Robust Difference-in-Differences (DID)

- Description: This method from Callaway and Sant'Anna [18] addresses bias from treatment effect heterogeneity and is only available for periods with untreated counties. It minimizes bias by comparing treated and untreated units over time, thus avoiding any extrapolation.

- Results: The Robust DID method shows the closest match to the true treatment effects for counties which have an untreated comparison available. It performs consistently well across all age groups.

- Bias: This method is unbiased. This highlights its strength in providing accurate estimates, especially when heterogeneity is a concern. However, the long-term effects cannot be extrapolated because, after three weeks, all counties have experienced vacations, and there is no pure control group.

Comparison of Method

Across all methods, those expressed in levels perform the best and give a relatively clear picture of the policy's effects. The event study in levels performs better than event studies with other outcome formulations and has relatively little bias. The bias can be further reduced by using the robust method, but it comes at the cost of long-term extrapolation.

In particular, all counties experience vacation within four weeks. Hence, for the group of counties having vacation first, there exists a pure control for the next three weeks (counties which experience vacation last). After that time, an extrapolation using previously estimated treatment effects is necessary, hinging on the assumption that treatment effects are the same across all groups. This may not be true for various reasons [25].

The robust difference-in-differences (DID) method outperforms traditional DID methods in handling the differential treatment timing and the treatment effect heterogeneity. The robust DID method, developed by Callaway and Sant'Anna [18], effectively compares treated and untreated units over time without using extrapolation, hence it has a lower bias. It provides the best estimate for the short-term impact of the vacations. However, the robust DID method has its limitations. It can only interpolate results within a short window when untreated units are available, making it less effective for longer-term extrapolation.

A.7 Vacation Timing and Cumulative Burden

In this section, I describe the methodology used to estimate the effect of vacation timing on the cumulative incidence and mortality in a season. Estimating the impact of having versus not having a vacation is not feasible because all counties eventually have vacations. Instead, I focus on the timing of vacations.

First, I aggregate all incidence (or mortality) across the season for each county and age group, resulting in one observation per county, age group, and season. This aggregated measure is then regressed on indicators of vacation timing, controlling for county and season fixed effects.

I use two types of indicators for vacation timing. The first set of indicators consists of dummies representing the absolute week of vacation, defined in terms of

weeks since the start of July. I take the first possible vacation week (week 28) as the reference point. The coefficients δ_w from this regression represent differences in cumulative incidence for vacations taken in week w compared to week 28, as shown in Equation 23.

$$y_{ita} = \sum_{w=29}^{32} \delta_w I\{vac_{it} = w\} + \alpha_i + \gamma_t + e_{ita} \tag{23}$$

Here, y_{ita} denotes the cumulative incidence (or mortality) for county i, age group a, and season t. The term $I\{vac_{it} = w\}$ is an indicator variable that equals 1 if the vacation for county i in season t occurs in week w. The terms α_i and γ_t represent county and season fixed effects, respectively, and e_{ita} is the error term.

The second set of indicators measures the timing of vacations relative to the peak incidence week. I first identify the week with the maximum incidence in each county and season. I then calculate the difference between this peak week and the vacation week, categorizing the differences into five bins: more than 4 weeks before the peak, 4-2 weeks before the peak, 1 week before to 1 week after the peak, 2-4 weeks after the peak, and more than 4 weeks after the peak. The bin "more than 4 weeks before the peak" serves as the reference point. This specification is represented in Equation 24.

$$y_{ita} = \sum_{w=\text{bins}} \delta_w I\{rel_{it} = w\} + \alpha_i + \gamma_t + u_{ita} \tag{24}$$

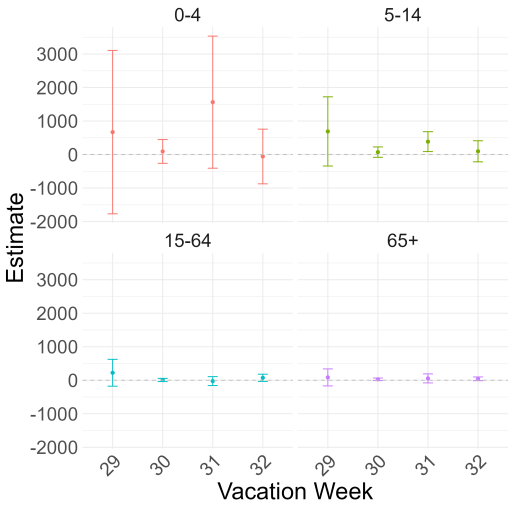
In this equation, $I\{rel_{it} = w\}$ is an indicator variable that equals 1 if the vacation for county i in season t falls into the wth relative bin. The other terms are defined as in Equation 23.

The results are presented in the figures below. Points represent point estimates of coefficients δ_w with w on the x axis, and bands represent 95% confidence intervals for the coefficients.

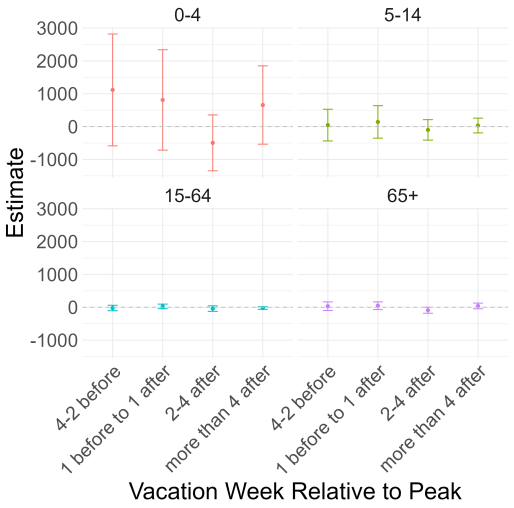
As shown on the figures, virtually none of the coefficients are statistically significant from zero at 5% level, suggesting that the timing of the vacation does not significantly affect the incidence or mortality rates. This lack of significance may be due to the relatively small differences in vacation timing, with the earliest timing being only 4 weeks before the latest timing.

A.8 Effects on Reporting

This subsection assesses how vacation periods impact the number of physicians who submit reports. The original data from epidemiological stations specify how



(a) Absolute Timing

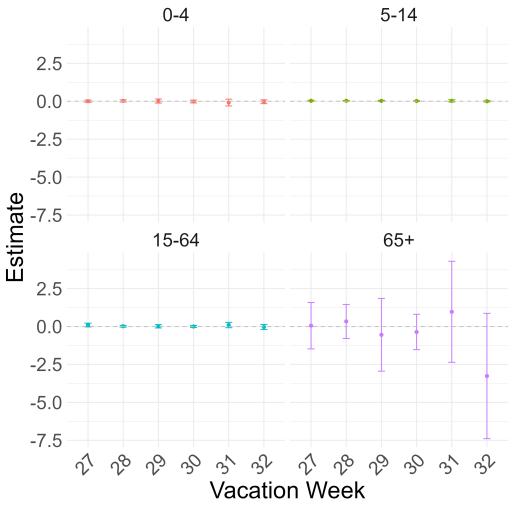


(b) Relative to Peak Timing

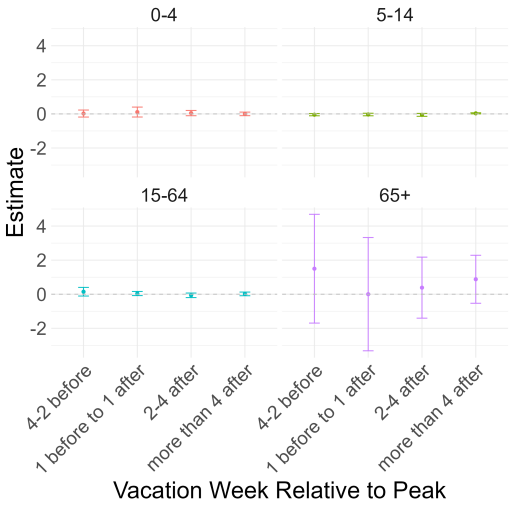
The graph shows the results of the regression specified in Equation 23 with the outcome of cumulative ILI incidence per 10,000 in a season. Points represent point estimates, and bands represent 95% confidence intervals.

The graph shows the results of the regression specified in Equation 24 with the outcome of cumulative ILI incidence per 10,000 in a season. Points represent point estimates, and bands represent 95% confidence intervals.

Figure A.31: Effects of Vacation Timing on Cumulative Incidence of Infections



(a) Absolute Timing



(b) Relative to Peak Timing

The graph shows the results of the regression specified in Equation 23 with the outcome of cumulative respiratory mortality per 10,000 in a season. Points represent point estimates, and bands represent 95% confidence intervals.

The graph shows the results of the regression specified in Equation 24 with the outcome of cumulative respiratory mortality per 10,000 in a season. Points represent point estimates, and bands represent 95% confidence intervals.

Figure A.32: Effects of Vacation Timing on Cumulative Respiratory Mortality

many physicians submitted the reports. However, it is important to highlight that physicians will only appear as submitting reports if they reported a positive number of cases. If a physician was in the office but had zero cases to report, they will not

be represented in the data. This distinction is crucial, as any changes in the number of reporting physicians may reflect either their presence or absence in the office, or fluctuations in the number of patients presenting with infections.

First, I estimate the regression in equation 1 using the number of physicians who submitted reports per 10,000 population as the outcome. The estimates are shown in Figure A.33.

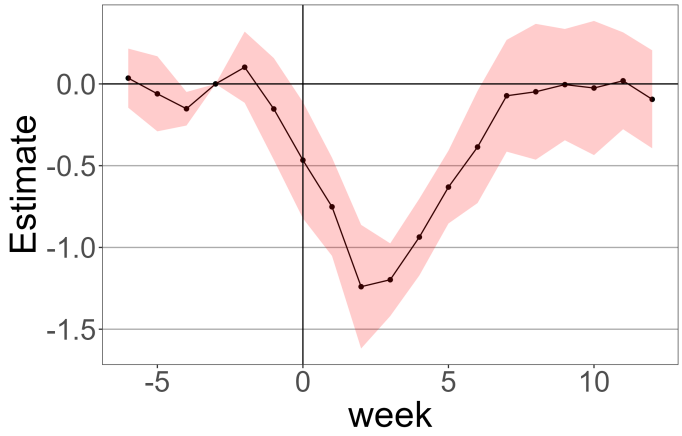


Figure A.33: Effect on Physicians who Submitted Reports

Note: Graph shows the impact of winter vacation on the weekly number of doctors submitting the reports. The outcome is number of doctors who submitted the report per 10 000 population. Hence the coefficients (as in equation 1) show the change in number of reporting physicians in each week prior and after the vacation, where week 0 is the first week of vacation. Shaded area represents 95% confidence interval for the estimates. Errors are clustered at the region level.

There is a clear decline in the number of physicians submitting reports both during vacation and in the five weeks following. This decline mirrors the decrease in incidence. There are two potential explanations for this decline. The first possibility is that physicians are absent and do not submit reports during the vacation and up to five weeks afterward. The second possibility is that there are fewer cases both during and after the vacation, leading physicians to submit fewer reports because there are fewer cases to report. Unfortunately, with the available data, it is impossible to accurately distinguish between these two explanations. Nonetheless, the observed pattern closely follows the decline in infections. While it is plausible that doctors might be more likely to leave the office during vacation if they have school-aged children, it is more difficult to argue why doctors would be absent specifically in the weeks following the vacation.

To address the concern that a lower number of reports is due to fewer reporting physicians, I also ran the regression in equation 1 replacing the outcome with the number of reports divided by the number of physicians. If the decline in incidence is

entirely due to fewer physicians reporting, the ratio of reports to physicians should remain unaffected. Figure A.34 demonstrates the results. While the results are noisier, there is a clear decline in the number of reports per physician, indicating that even physicians who do report see fewer patients with influenza-like illnesses.

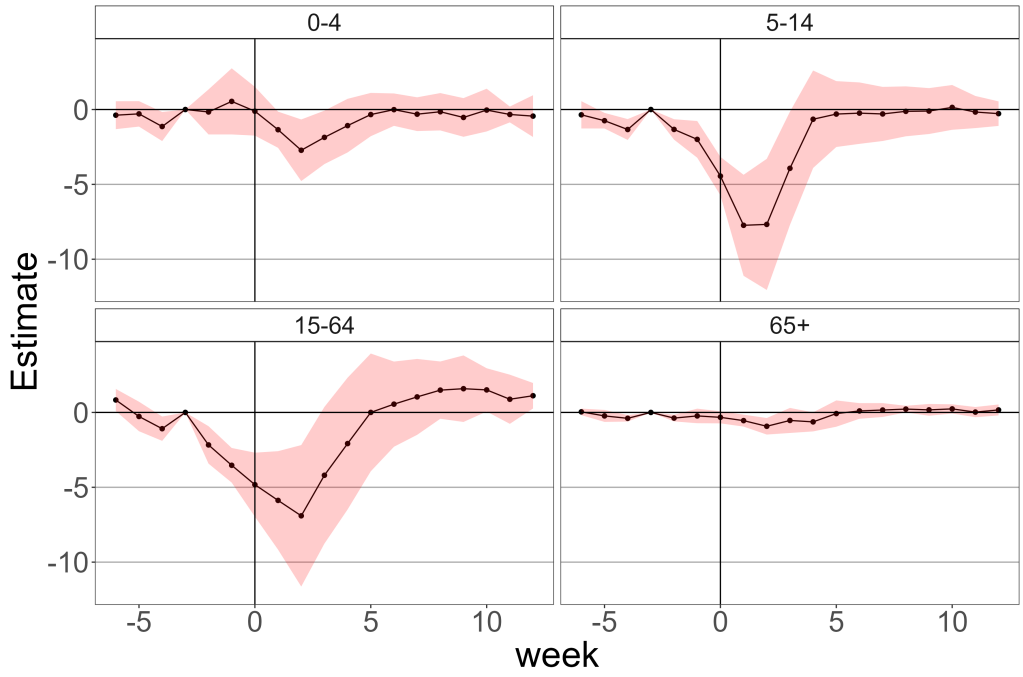


Figure A.34: Effect on Cases per Reporting Physician

Note: Graph shows the impact of winter vacation on the number of ILI reports per reporting physician. Each sub-figure represents results of the estimation of the event study in equation 1 on a sub sample restricted to age groups 0-4,5-14,15-64, 65+. The outcome is the number of reports per reporting physician in a given week. Hence the coefficients show the change in the above ratio in each week prior and after the vacation, where week 0 is the first week of vacation. Shaded area represents 95% confidence interval for the estimates. Errors are clustered at the region level.