The Effects of Employment on Influenza Rates*

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Abstract

The seasonal influenza virus afflicts millions of people in the U.S. population each year, imposing significant costs on those who fall ill, their families, employers, and the health care system. The flu is transmitted via droplet spread or close contact, and certain environments, such as schools or offices, promote transmission. In this paper, we examine whether increases in employment are associated with increased incidence of the flu. We use state-level data on the prevalence of the flu from the Centers for Disease Control and Prevention. In our preferred specification, we find that a one percentage point increase in the employment rate increases the number of influenza related outpatient health care visits by 19 percent, and these effects are highly pronounced in the retail sector and healthcare sector, the sectors with the highest levels of interpersonal contact.

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1 Introduction

The seasonal influenza epidemic imparts surprisingly large costs on the United States economy. According to the Centers for Disease Control and Prevention (CDC), the flu infects between 9 and 35 million people in the United States each year, resulting in between 140,000 and 710,000 hospitalizations, and between 12,000 and 56,000 deaths (Centers for Disease Control and Prevention, 2018a). Estimates of the economic burden of the flu range from 34.7 billion dollars to nearly 90 billion dollars per year (Molinari et al., 2007; Rothman, 2017).

The flu is transmitted from sick to well individuals when a healthy person either touches a surface containing the flu virus or inhales the virus after an infected person coughs or sneezes. As we discuss in detail below, many of the characteristics or environments associated with modern daily work life may encourage the transmission of the flu. These characteristics include commuting via public transportation, working in climate controlled indoor offices, sharing workspaces, and placing young children in daycare. If the features of the working environment promote the spread of the flu, then being out of the labor force and away from these environments could help reduce the spread of the flu.

This paper explores the proposition that employment rates affect the spread of the flu. We use data from the CDC on the incidence of the influenza virus for the 2010/2011 to 2016/2017 flu seasons in conjunction with an employment rate constructed from data from the Bureau of Labor Statistics (BLS). Using dynamic panel data models to account for the infectious nature of the flu, we find evidence that increased employment indeed aids the spread of the flu. In our preferred model, we find that a one percentage point increase in the employment rate will lead to about a 19 percent increase in flu prevalence. Since the average average absolute change in the employment rate from one month to the next in our sample is 0.31 percentage points, an average monthly increase in the employment rate increases the flu incidence by about six percent.

We also examine the relationship between employment and flu prevalence at the sector level, hypothesizing that sectors with more personal contact should be more related to the flu. We construct a measure of interpersonal contact levels for each industry sector using data from O*Net and construct employment measures at the industry level using data from the BLS Current Employment Statistics (CES) data set. We find that employment changes in the retail and healthcare

sectors, the two sectors with the highest levels of interpersonal contact, drive the aggregate results. Moreover, we find that employment changes in the construction and manufacturing sectors, the two sectors with the lowest levels of interpersonal contact, are not associated with statistically significant changes in flu prevalence.

2 Background

2.1 Influenza Virus and Transmission

There are four types of influenza viruses, A, B, C, and D, although only types A and B are responsible for the seasonal epidemics in humans (Centers for Disease Control and Prevention, 2018e). Within types A and B there are different strains of the virus.[1] The virus mutates at a very rapid rate, resulting in an ever changing combination of active strains. The constant and rapid evolution of new strains means that people cannot become indefinitely immune to the virus from adaptive immunity or through vaccination (Centers for Disease Control and Prevention, 2018c). Having recovered from a past flu infection or having been vaccinated against known strains of the flu does not protect against infection from new strains of the virus. Occasionally an abrupt change in a type A virus can occur, leaving the majority of the population with little to no immunity against the novel strain. This can lead to flu pandemics, during which widespread infection, hospitalization, and death are common.

Influenza epidemics are seasonal, occurring mainly in the winter months. The CDC actively monitors flu activity during the flu season, defined by the CDC as between October and May (Centers for Disease Control and Prevention, 2018c). There is no consensus among the scientific community as to why flu outbreaks occur mainly in the winter months (Cox and Subbarao, 2000). Since the 1982-1983 flu season, the peak month of flu activity occurred in February nearly half of the time. However, the peak month of activity can occur as early as November, and regularly occurs in March.

The mechanism for flu spread is sick-to-well transmission. That is, healthy people become

[1]There are multiple subtypes of influenza A, of which two are commonly found in humans. A(H1N1) was responsible for the "swine flu" pandemic in the 2009/2010 flu season, and A(H2N3) was largely responsible for the particularly severe 2017/2018 flu season. Each subtype of influenza A has multiple strains. Influenza B has no subtypes, but multiple strains.

infected with influenza by coming into contact with infected individuals. According to the CDC, the transfer of the virus occurs through droplet spread. The influenza virus is present in the saliva and mucus of infected individuals, and when an infected person coughs or sneezes, the virus is introduced into the air in aerosol form. The virus can either be inhaled when airborne, called aerosol transmission, or introduced into the body after touching a surface containing the droplets (and then touching the eyes, nose or mouth), called close contact transmission. Experimental evidence suggests that, under laboratory conditions, aerosol transmission is the most successful mechanism of infection (Tellier, 2006). However, the virus can only survive for 16 hours in aerosol form. On the other hand, the virus can survive for up to 48 hours on a non-porous, dry surface. This evidence, coupled with experimental evidence showing up to 60 percent of the objects surrounding an infected person have an infectious concentration of the virus, leads some researchers to believe close contact transmission is the primary mechanism of flu spread (Brankston et al., 2007; Tellier, 2006).

Schools and workplaces both offer the close contact between individuals and commonly touched surfaces conducive to the spread of influenza. Cox and Subbarao (2000) report that the highest attack rates of influenza occur among school-age children, and that school absenteeism is typically followed by work absenteeism. Individuals in families with school-age children (or children in daycare) are more than twice as likely to become infected with influenza, which suggests schools are a primary environment for the spread of the flu (Cox and Subbarao, 2000). But because schooling is compulsory, the large variability in flu incidence between regions and both within and across flu seasons is unlikely to be explained by school enrollment.

Employment provides an analogous environment for adults, and employment rates vary significantly across regions and time. A person who is employed could be much more likely to come into close contact with the influenza virus than individuals out of work or out of the labor force. Since employed people typically commute to and from the workplace, they are more likely to use public transportation or car pools, which are favorable environments for flu transmission. According to the 2016 American Community Survey, over 7.6 million Americans commute using public transportation and another 13.6 million commute by carpool. This puts roughly 14 percent of working Americans at risk of catching the flu in these manners. Moreover, the majority of Americans work indoors. Working environments like offices are ideal for both aerosol and close contact transmission

of the influenza virus. In addition to the close proximity of workers, Tellier (2006) reports that the influenza virus in aerosol form survives better in low humidity environments. Thus, a climate-controlled office building would be a conducive environment for aerosol transmission. Commonly touched surfaces, such as keyboards, water coolers, and communal restrooms, also promote close contact flu transmission. Finally, employed individuals may be more likely to put young children in daycare, and as noted above, school and daycare are fertile environments for the spread of the flu.

It is worth noting that there are alternative (though not generally accepted) views concerning influenza transmission. This literature suggests that influenza does not behave like a typical infectious disease, and sick-to-well transmission may not adequately explain the epidemiology of the flu. One theory suggests epidemics of the flu are due to vitamin D deficiency and its importance to innate immunity (Cannell et al., 2008).[2] Although this theory of flu transmission is very different from the generally accepted view, influenza transmission could still be highly influenced by the work environment. Since the majority of Americans work during the daylight hours, which limits sun exposure, it follows that vitamin D deficiency may increase in prevalence during times of increased indoor employment. Additionally, person-to-person contact is still necessary for the flu spread, even if it is not responsible for the outbreak of an epidemic. Thus, in the event that this theory of flu transmission has more substance than is currently attributed to it, we should still see an effect of employment on flu rates.

2.2 Influenza and Employment

The above discussion points to a possible strong positive relationship between periods of high employment levels and high rates of the flu, and this is the primary hypothesis that we test in the paper. As discussed below in the methodology section, we estimate a reduced form model of the relationship between employment and flu rates, with the caveat that this approach does not distinguish among the mechanisms through which the relationship might occur. The above discussion states that the human interaction that goes along with employment-based activities directly contribute to the spread of the flu. Other indirect or intermediary mechanisms may be responsible as well, and some of these may have opposing effects. For example, Cornwell (2012)

[2]See White (2008) for a description of vitamin D's importance for innate immunity.

hypothesizes that higher levels of unemployment that occur during recessions are associated with increases in flu activity through the mechanisms of financial strain, lack of access to health care and low vaccine rates, and stress, which can increase susceptibility to viral infections.

There is certainly validity to the argument that high levels of unemployment can have detrimental effects on overall health, and it is reasonable that one might expect to see higher levels of flu cases as a result. However, the mechanisms here are more distal than that of the opposing hypothesis where higher levels of human interaction that go along with employment promote the transmission of the flu. In the case of stress, it is also not clear whether unemployment or employment promotes more stress given that some jobs may be highly stress-inducing. Ultimately, it is an empirical question and our results show the net effects that come about from changes in these possible mechanisms.

In regards to the existing literature, the paper by Cornwell (2012) is the most closely related to ours. To test his hypothesis, Cornwell regresses state-level data on the probability of observing "widespread" or "widespread/regional" levels of flu activity on the state unemployment rate. Using a lagged dependent variable panel data model, he finds that an increase in the unemployment rate is associated with a higher likelihood of widespread flu cases. However, the estimates are likely biased since he ignores the endogeneity of the lagged dependent variable.

We also argue that the unemployment rate is not an ideal measure to use in this context. First, it is unclear whether unemployed workers are regularly engaged in the employment search activities that would contribute to the spread of the flu. The employment to population ratio does not suffer from this ambiguity. Second, the unemployment rate can change because of workers switching status or by entering or exiting the labor force. These transitions have different implications for the stress and financial strain stories. For example, the unemployment rate can rise as people enter the labor force during an economic recovery and this may be accompanied by reduced stress and increased hopefulness among previously discouraged workers. Again, the employment to population ratio is a much cleaner measure and easier to interpret.

Our paper adds to the literature concerning the effect of macroeconomic conditions on health. The bulk of this literature relates local unemployment rates to a variety of health outcomes. For example, Ruhm (2000) finds that higher state-level unemployment rates are associated with decreased mortality rates, including deaths from influenza and pneumonia (in direct contrast to the

results by Cornwell (2012)). Interestingly, Ruhm (2015) revisits the question and finds that over time, mortality shifted to being weakly or even unrelated to unemployment, but he does not examine influenza in this newer article. Also, Gerdtham and Ruhm (2006) find that a one percent increase in national unemployment rates among OECD countries are associated with a 1.1 percent rise in deaths from influenza and pneumonia. There is some evidence to suggest that these types of analyses may be sensitive to the business cycle measure used (Gerdtham and Johannesson, 1999).[3] In work related to this paper, Adda (2016) estimates the effect of various types of economic activity in France on the spread of viral diseases, including influenza. He finds that influenza spread is positively related to inter-regional trade, but finds mixed effects for unemployment.[4] Our paper helps sort out the divergent results from the existing papers that have included a measure of influenza as an outcome. We also focus on the employment to population ratio as opposed to the unemployment rate, which as previously discussed, is not an ideal measure and is hard to interpret in the context of a communicable disease. However, we show results using the unemployment rate in a supplemental analysis.

While our paper is not the first to link business cycles to health, our focus on a common communicable disease in the United States adds a new dimension to the discussion. Most of the previous literature relies on changes in individual health-related behaviors as the mechanisms behind the observed relationships between either mortality or health status and unemployment. These can vary dramatically among individuals and among circumstances, and as previously stated, the relationship appears to be changing over time (Ruhm, 2000, 2015). Communicable diseases are different. The human-to-human transmission mechanism of the flu is well established and likely not to vary over time. Also, while it is interesting from an academic perspective to know whether we can expect lower mortality or improved health status during a recession, from a policy perspective, little can be done to alter these outcomes via changes in the business cycle. The results of our study are more concrete and can help predict when a flu season will be "bad." This is particularly important since, as Dr. Anne Schuchat of the CDC states, "...flu is incredible difficult to predict."[5] Barring

[3] A number of other studies examine how various health behaviors and health outcomes vary with unemployment rates including a series of papers by Ruhm (Ruhm and Black (2002), Ruhm (2003) Ruhm (2005), and Ruhm (2007)). We refer the reader to papers by Currie et al. (2015) and Ruhm (2016) for comprehensive reviews of this literature.

[4] Another related paper is Stoecker et al. (2016), who show that traveling to a Super Bowl event dramatically increases incidence of the flu in the travelers' home counties. Our study differs by focusing on economic activity as the conduit for increased interpersonal contact and flu spread, rather than travel.

[5] Her interview is here: `https://www.cdc.gov/media/releases/2018/t0209-flu-update-activity.html`, last ac-

major economic shocks, there tends to be strong month-to-month persistence in employment trends so when we find ourselves in expansions, our results predict higher rates of flu cases. This knowledge can help public health practitioners prepare. For example, the 2017-2018 flu season was particularly severe. Hospitals found themselves unprepared and struggling to keep up with the influx of patients. The ability to predict the potential severity of an upcoming flu season would be vital information for hospitals and public health officials.

3 Data

3.1 Employment Data

We construct state-level monthly employment rates using data from the Bureau of Labor Statistics. Each month, Census workers interview people in 60,000 households about their labor market activities during the week that includes the 12th day of the month. These values are adjusted for sample demographics and extrapolated to state-level or region-level employment figures.[6] From these surveys, we construct an employment rate measuring the percent of the civilian population currently working. The employment rate is the employed population divided by the non-institutional civilian population multiplied by 100. We use this employment rate rather than the more well-known unemployment rate because the unemployment rate denominator is the labor force and not the working-age population. As mentioned above, since the unemployment rate denominator fluctuates as people enter or exit the labor force, the unemployment rate will not fully capture the changes in the percentage of the population working.

We use unadjusted employment data rather than seasonally adjusted numbers. In our context, seasonally unadjusted employment data is preferable to the seasonally adjusted series. The seasonally adjusted series, by design, diminishes month-to-month variation, making it hard to identify any effect of the seasonally adjusted employment rates on outcomes that vary monthly. Also, the seasonally adjusted series removes the seasonal component of employment. Since our interest is in the spread of the flu related to movements in and out of employment for any reason, the unadjusted number is preferable.

cessed April 2, 2019.

[6]For more information on the construction of the BLS data, see `https://www.bls.gov/cps/documentation.htm`, last accessed April 2, 2019.

3.2 Flu Data

The measure of flu prevalence comes from the CDC. The CDC heads an influenza surveillance system comprised of, among other surveillance measures, an Outpatient Influenza-like Illness Network (ILINet). The ILINet consists of some 3,500 health care providers across all fifty states and Washington D.C. Each week during the flu season, providers voluntarily submit the number of patient visits for all causes and the number of patients with an influenza-like illness (ILI). The symptoms necessary for an ILI diagnosis include a fever greater than 100 degrees Fahrenheit, a cough and/or sore throat, and a lack of a known cause besides influenza. From this we generate state-by-week flu prevalence rate defined as the number of ILI cases per number of outpatient medical care visits. Starting with the 2010-2011 flu season, the CDC reports the ILINet data at the state level (prior years were at a regional level only), so we begin our sample with this flu season and continue it through the 2016-2017 flu season. We drop the state of Florida because they do not report ILINet data, and we additionally exclude Alaska and Hawaii because the ILINet data includes only the contiguous 48 states plus Washington D.C.

Since the flu data are reported on a weekly basis and our other variables are reported on a monthly basis, we aggregate the flu variable to a monthly level by taking the average of all the weekly observations during that month. According to the CDC, an infected person may not show symptoms for up to four days after infectious contact and is capable of infecting others for up to a week after symptoms start. There is also some lag between when symptoms start and when that person visits a health care provider regarding his or her illness. Given these timing issues, it seems likely that a few weeks or more would pass before the effect of a change in employment could alter flu prevalence as recorded by ILINet. Thus, we lag our employment variables by one month. Since the employment variables are measured over the week containing the 12th day of the month and the flu is measured during the next month, the measurement of the flu starts two to three weeks later on average.

There are some potential pitfalls with our flu measure. First, the location and coverage of the health providers are unknown. There may be a bias towards urban locations and larger, well-staffed medical providers. Second, the reporting may not accurately reflect the severity of the flu. It may be that patients with flu-like symptoms are more from populations at risk from serious complications

arising from the flu, such as children, the elderly, and people with concurrent illnesses. Since these populations are less likely to work, it may bias our estimates. Health care providers' reactions may also change based on the severity of the flu. It may be that they diagnose and report patients with the flu more often during the height of the flu season. If the measurement error from reporting is not systematically related to employment, our estimates will be unbiased, but the standard errors will rise. However, if the measurement error is correlated with employment, identifying the direction and magnitude of the measurement error bias will be more difficult (Bound et al., 2001).

Another potential problem with our dependent variable is that labor market conditions may affect the rate at which people seek health care providers. If people go to the doctor less when they are not employed, the number of flu-related outpatient health care visits could likewise be affected. We examine this issue in Section 5.1. A final potential problem is that the spread of influenza may affect employment status. For example, someone may be fired for being sick too often, or someone may leave their job to take care of a sick parent, spouse or child. Our use of lagged employment should mitigate this problem.

Table 1 shows summary statistics from our sample. Our main analysis sample contains 2,688 state-month cells (48 states x 7 flu seasons x 8 months in each flu season). Our measure of the flu from the CDC indicates that during the flu season, about two percent of outpatient visits are flu-related. The employment to population ratio is just under 60 percent. Unsurprisingly, temperatures tend to be in the lower ranges during the flu season, and almost 85 percent of our state-month cells have average temperatures less than 60 degrees. Average precipitation levels are generally lower than seven inches for most state-year cells.

4 Methodology

We exploit variations within states across time in both influenza incidence and labor market activity to test the theory that increases in employment levels increase individuals' risk of exposure to the influenza virus. We examine the spread of the flu during the flu season, which we define as running from October through May of the next year. We only focus on the flu season because there is minimal flu activity during the summer. Since influenza is a communicable disease, the number of individuals that will be infected depends on the number that were previously infected. Arellano-

Bond tests for autocorrelation suggests that the rate of current influenza infection is significantly correlated with the past three months' infection rates. Therefore, for state s, flu season y, and month m, we first estimate the following panel data model with a lagged dependent variable:

$$ln(F_{sym}) = \alpha + \sum_{i=1}^{3} \rho_i ln(F_{sym-i}) + \beta E_{sym-1} + \gamma X_{sym} + \sigma_{sy} + \mu_{sm} + \epsilon_{sym}, \quad (1)$$

In Equation 1, the dependent variable is the natural log of a measure of flu prevalence, $ln(F_{sym})$.[7] The right-hand side variable of interest is the employment to population ratio, lagged by one month, E_{sym-1}. The coefficient β identifies the reduced form effect of marginal changes in labor market activity on influenza rates. Since not everyone in a given area will experience a change in flu exposure when local employment conditions change—and those that do will experience varying degrees of change to their exposure—this estimate is best viewed as an intent-to-treat (ITT) effect.

We also include three lags of the flu prevalence on the right hand side, F_{sym-i}, given the results of the autocorrelation tests.[8] The vector X_{sym} accounts for other factors likely related to the spread of the flu. Since research suggests the spread of the flu is related to temperature and humidity, X_{sym} also includes temperature and precipitation data for each state from the ηClimDiv dataset from the National Oceanic and Atmospheric Administration.[9] Finally, we control for state by flu season and state by month fixed effects, given by σ_{sy} and μ_{sm}.

Other variables of potential relevance are only available annually and likely do not vary much within a flu season. Variables such as the average education level, the percent of the population with health insurance, the percent immunized against the flu, the percent of the population living in rural areas, and the age distribution, may all contribute to the spread of the flu and will be captured by the state by flu season fixed effects. The omission of two of these excluded variables, the percent of the population with health insurance and the percent of the population immunized against the flu, may be of some concern. Excluding monthly health insurance data from the analysis could bias results due to the correlations with both employment and physician visits. However,

[7] There are four observations which have a flu measure of zero (two in Delaware and two in Montana, all four in the month of May), so we add 0.01 to every flu measure before taking the natural log.

[8] Versions of these tests can be seen in Table 2 and show that once three lags of the flu variable are included, only lags one and two are significantly correlated with the dependent variable, which is what we would expect if our errors were uncorrelated after the inclusion of the lags.

[9] See `https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-divisions.php` and `ftp://ftp.ncdc.noaa.gov/pub/data/cirs/climdiv/` for more information on the ηClimDiv data (last accessed April 2, 2019).

employment and health insurance are positively correlated, while the correlation between health insurance and influenza rates is negative or zero. While this suggests that any bias may drive our results toward zero, signing the direction of any bias is again difficult, and this remains a possible limitation of our analysis.

Regarding the influenza vaccine, a vaccination is designed to protect an individual for an entire flu season. That is, it is likely the case that the benefits from the vaccination are not seen in the same month (or even adjacent months) in which the vaccine is received, assuming that the vaccine is a good match. Estimates of the seasonal effectiveness of the vaccine range from 10 to 60 percent (Centers for Disease Control and Prevention, 2018d). In the most recent U.S. flu season, the vaccine effectiveness was 36 percent in 2017-18 (Centers for Disease Control and Prevention, 2018b). It is therefore unlikely that monthly vaccination data would add to the analysis. The state by flu season fixed effects absorb the impact of different vaccination rates each season in each state, which is likely a more informative control than any monthly data that may exist.

4.1 Endogeneity in Dynamic Panel Data Model

As Nickell (1981) points out, when variables are effectively transformed to be deviations from their mean in a fixed-effects model, the lagged-dependent variable is correlated with the error. Thus, Equation 1 may generate biased and inconsistent coefficients, and this inconsistency will translate to the coefficients of other regressors that are correlated with the lagged-dependent variable. This problem becomes more severe in instances where the number of cross-sectional observations is small (Beggs and Nerlove, 1988). In order to deal with the problem, we first use the Anderson and Hsiao (1981) first difference two stage least squares estimator. Anderson and Hsiao (1981) first difference all terms to purge the fixed effects, then instrument for the lagged dependent variable with a further lagged *level* of the dependent variable. In our situation, this model is

$$\Delta ln(F_{sym}) = \sum_{i=1}^{3} \rho_i \Delta ln(\widehat{F_{sym-i}}) + \beta \Delta E_{sym-1} + \gamma \Delta ln(X_{sym}) + \Delta \mu_{sm} + \Delta \epsilon_{sym}, \tag{2}$$

where $\Delta ln(\widehat{F_{sym-1}})$, $\Delta ln(\widehat{F_{sym-2}})$, and $\Delta ln(\widehat{F_{sym-3}})$ are instrumented for with $ln(F_{sym-4})$, $ln(F_{sym-5})$, and $ln(F_{sym-6})$. Equation 2, while consistent, is possibly not efficient. Arellano and Bond (1991)'s dynamic panel data model uses further lags of the dependent variable in a "difference GMM" frame-

work. However, one issue with the first-difference transformation is that it increases the number of missing observations. Arellano and Bover (1995) suggest using "forward orthogonal deviations," where each observation is subtracted from the average of all available values of that variable.[10] Thus, our final model implements an orthogonal forward deviations model as described in Arellano and Bover (1995). We show that this model is robust to a number of specification tests in Section 5.1.

It is worth noting that this estimation technique is only meant to correct for the endogeneity of the lagged dependent variables and does not directly affect employment, which remains the source of identifying variation in our models (as discussed in Section 2). An alternative strategy would be to omit the lagged dependent variables and simply use a two-way fixed effects model. However, this method would introduce bias to the coefficient estimates of any variables that are correlated with lagged flu rates. This is concerning because the lagged flu rates are themselves determined by further lags of the explanatory variables. Thus, endogeneity would be almost guaranteed for any explanatory variables that are serially correlated, which is highly likely for all of the variables included in our model, including employment. Nonetheless, we use the two-way fixed effects model (with no lagged dependent variable) as a point of comparison and find qualitatively similar results.

We weight all our regressions using state population, although our results are robust to not using weights. When estimating the fixed effects model, we cluster our standard errors at the state by flu-season level to account for the possible intraseasonal correlation within states in the error term, and when estimating dynamic panel data models, we calculate robust standard errors.

We are concerned with a number of other threats to validity and potential sources of bias. These include issues surrounding logs versus levels, weighted and unweighted models, changing the lag structure, unemployment rates versus the employment-to-population ratio, and spurious and concurrent trends. These are all address below in Section 5.1. Lastly, we examine the results by industry sector to examine whether the results are stronger in industries with higher levels of interpersonal contact, and we discuss these results in Section 5.2.

[10] See Arellano and Bover (1995) and Roodman (2009a) for more information.

5 Results

Table 2 displays our main regression results. Starting from left to right, we first examine a fixed-effects model without incorporating lags of the flu prevalence, then estimate a lagged-dependent variable model, then an Anderson and Hsiao (1981) style first-difference 2SLS model, and finally an Arellano and Bover (1995) style orthogonal forward deviations GMM model. In the first column, a one percentage point increase in the employment rate will lead to about a 28 percent increase in flu prevalence. However, accounting for the communicable nature of the flu through lagged dependent variables reduces the size of the employment coefficient. In our preferred specification in Column (4), a one percentage point increase in the employment rate increases the flu prevalence by 19 percent.[11] The coefficient for the first-difference 2SLS model is consistent with that of the Arrelano-Bover model, but is imprecisely estimated.[12]

To put the 19 percent effect into context, the average ILI measure is just over two percent. Thus, a one percentage point increase in the employment rate translates to an average marginal increase in flu prevalence of about 0.42 percentage points, or 4.2 additional flu-related visits per 1000 outpatient visits for all causes. Since the average employment rate is about 59 percent, the elasticity of flu incidence with respect to employment is about 11 (0.19×59.11). However, a one percentage point change in employment over the course of a month is quite large. The average absolute change in the employment rate from one month to the next in our sample is 0.31 percentage points, so an average monthly increase in the employment rate increases the flu incidence by 0.13 percentage points (0.42×0.31), or about six percent. We advise some caution in interpreting the magnitude of this effect, however, as the data available are limited and may not be a representative sample of flu seasons.

[11] The autocorrelation tests in Column (4) show that only lags one and two of the dependent variable are correlated with the contemporaneous dependent variable, which suggests that the errors of our lagged model are not serially correlated. We examined the robustness of changing number of lags which are included in the Arrelano-Bover model. Including two lags of the dependent variable (instead of three) did not change the size or statistical significance of the coefficient on employment, but autocorrelation tests from this model suggested that more lags of the dependent variable were needed to account for autocorrelation. We additionally examined including four lags of the dependent variable. The size of the coefficient on employment became somewhat smaller (0.13) but is still statistically significant at the five percent level.

[12] This is likely due to the inefficiency of the first-difference 2SLS estimator, which we discuss in Section 4.1.

5.1 Robustness Checks

Next, we demonstrate that our results are robust to estimation with different dynamic panel data model specifications. Table 3 reestimates our results from Column (4) in Table 2. A common concern with models derived from Arellano and Bond (1991) is overidentification. In the main specifications, all possible lags of the dependent variable are used as instruments which generates 42 instruments. In the first four columns, we undertake various strategies to reduce the number of instruments, highlighted in Roodman (2009a). First, we limit the number of lags to one, instead of using all available lags. This reduces the instruments to 24 (one for each lag for each of the eight months we estimate our regressions). Next, we use principal components to reduce the instruments, following Roodman (2009a), Bai and Ng (2010), and Kapetanios and Marcellino (2010). Finally, we collapse the instrument matrix (Roodman, 2009b). In the final column, we show that our results are robust to using the more standard first differences transformation, instead of the orthogonal deviations in our main results. Across all the columns, we still see a positive and statistically significant effect of employment on the flu.

Next, we demonstrate that our results are robust to other possible identification concerns. First, as mentioned in Section 3.2, we use the employment-to-population ratio, instead of the unemployment rate, because the denominator of the unemployment rate is the labor force. We examine this issue in more depth in the first three rows of Appendix Table A1. First, we run our results with the unemployment rate and find smaller coefficients which are not statistically significant. However, as we mention above, this is likely due to the construction of the unemployment rate, where the denominator is the labor force, not the population. When we separately regress flu prevalence on the labor force-to-population ratio and the unemployment-to-population ratio, we find positive and statistically significant coefficients for the labor force and small, not statistically significant, coefficients for the unemployment-to-population ratio regressions. Since the labor force is made up of the employed and unemployed individuals, this provides further evidence that it is the employed individuals affecting the transmission of the flu.

Additionally, as mentioned in Section 3.2, our flu measure may be inaccurate if the denominator, total outpatient visits, is affected by employment. To check, we run a regression of the natural log of the number of visits on the employment rate and other controls, using the same specifications

as in Table 2. The coefficients on employment are positive but very small and not statistically significant. As a second check, we change our dependent variable to be the natural log of the number of flu-related visits, adding in the natural log of the total number of outpatient visits as a right-hand side control variable. The coefficients on the employment rate remain positive and statistically significant and very similar to our main results. We display these results in rows four and five of Appendix Table A1.

Finally, we also test the robustness of our results to adding one to the flu rate (instead of 0.01), using the unlogged flu rate, logging the employment rate, adding in a control for quarterly real state per-capita income from the Bureau of Economic Analysis, lagging the other controls (temperature/precipitation), using contemporaneous (rather than lagged) employment, not using sample weights, clustering our standard errors at the state (rather than state x flu season) level, and running our regressions on all months instead of the flu season. These results, all shown in the bottom columns of Appendix Table A1 continue to show a positive, statistically significant relationship between employment and the flu.

Despite controlling for trends in flu incidence through state by month fixed effects, one might worry that the positive relationship we find between employment and the flu reflects a spurious relationship reflecting concurrent trends in the series. For example, a positive but spurious correlation might occur if the flu incidence increases in the early winter months at the same time employment increases during the Christmas shopping season. An inspection of the data, however, reveals a different story. Employment figures typically peak in the summer and fall throughout the autumn and winter months. There is usually a slight rise in employment around December due to the Christmas season, but the downward trend continues thereafter, typically until February. On the other hand, flu cases rise during the fall and winter months, and nearly always peak around February, which is usually the trough of the employment trend. In fact, the raw correlation between the two time series is negative (r=-0.12). Thus, a positive coincidental seasonality of employment and influenza is not of particular concern.

5.2 Results by Industry Sector

The degree to which increased employment contributes to the spread of the flu is likely influenced by heterogeneity in person-to-person exposure in different types of employment. That is, we expect

that industries with frequent interpersonal contact, like retail sales, should promote flu spread. Likewise, we may expect jobs that limit the employees' exposure to people, like manufacturing, to have an insignificant or negative effect on flu prevalence. The results of total employment will reflect a combination of the effects in the different sectors. To see these differential effects, we run models focusing on specific industries that are especially likely or unlikely to contribute to flu prevalence, based on their levels of interpersonal contact. As the employment data from the Current Population Survey based statistics is not easily broken down by industry, for these results we use monthly BLS employment numbers based on the CES surveys. The CES is a survey of nearly 150,000 businesses and government agencies, and its monthly employment numbers are available at the state and industry level.

We use data from O*Net, which classifies occupations and occupational characteristics, and measure the level of activities relating to interpersonal contact for each occupation.[13] We calculate the mean levels of how often each occupation engages in interpersonal contact activities (measured on a scale of 1 to 7). Next, we match occupations to industries using the share of each occupation in each industry according to the Bureau of Labor Statistics Industry-Occupation Matrix.[14] Table 4 shows the results of this analysis. The retail trade and healthcare sectors have the two highest average measures of interpersonal contact while the manufacturing and construction sectors have the two lowest average measures of interpersonal contact.

We re-estimate our regressions from Table 2, Column (4) (the Arellano and Bover (1995) specification), replacing the aggregate employment rate with the employment rates from the sectors with the highest and lowest levels of interpersonal contact. We expect that sectors with more personal contact will be more positively related to the spread of the flu. Results from these regressions are found in Table 5. We find that the retail sector and health sectors are positively and statistically significantly related to the flu, with larger coefficients than in our results in Table 2. The coefficients

[13]Specifically, we use 13 employment activities: "Communicating with Supervisors, Peers, or Subordinates," "Communicating with Persons Outside Organization," "Establishing and Maintaining Interpersonal Relationships," "Assisting and Caring for Others," "Selling or Influencing Others," "Resolving Conflicts and Negotiating with Others," "Performing for or Working Directly with the Public," "Training and Teaching Others," "Guiding, Directing, and Motivating Subordinates," "Coaching and Developing Others," "Provide Consultation and Advice to Others," "Performing Administrative Activities," and "Staffing Organizational Units."

[14]We first map the occupations in O*Net to occupations in the Bureau of Labor Statistics using a crosswalk located here: `https://www.onetcenter.org/taxonomy/2010/soc2018.html`, last accessed April 2, 2019. The Bureau of Labor Statistics Industry-Occupation Matrix is located here `https://www.bls.gov/emp/tables/industry-occupation-matrix-industry.htm`, last accessed April 2, 2019.

for employment in the manufacturing and construction sectors are much smaller in magnitude (and negative for the construction sector), and are not statistically significant.

6 Conclusion

This paper examines whether labor market conditions affect the spread of the flu. Labor market based activities, such as using public transportation and carpools, working in offices, putting children in daycare, and having frequent contact with the public, are likely conducive to the spread of the flu. We hypothesize that increases in the rates of people working increase the spread of the flu. We test our theory using state-level data on flu prevalence from the CDC and the employment rate calculated from the BLS. We find evidence that the spread of the influenza virus is linked with higher employment, particularly in industry sectors with the highest levels of interpersonal contact. Specifically, we find that a one percentage point increase in the percent of the population that is employed will increase the prevalence of the flu-related outpatient visits by about 19 percentage points or about six percent. Our results are robust to changing both the empirical strategy and the specification of the model.

Our results are generally consistent with Ruhm (2000) and Adda (2016) and are in contrast to Gerdtham and Ruhm (2006). However, differences in methodology, time periods, countries studied, outcomes, and measures of economic activity prevent direct comparison. The use of unemployment rates rather than the employment-to-population ratio is another important difference, and as we argue previously, unemployment rates are harder to interpret in the context of the spread of a communicable disease. One limitation to our paper is that we are constrained to a relatively short period of time, and Ruhm (2015) finds that when studying deaths and recessions, short time panels can produced unstable results that vary with the time period examined.

These results are relevant for several reasons. First, employment conditions can be forecast, to a fairly accurate degree, several months in advance. This information could be used by the public health community to plan for the severity of an upcoming flu season. For example, if the economy is on an upswing, the public health community should plan for an above normal increase in flu incidence. Furthermore, our results imply that employment in service industries—particularly retail and health care—is a particularly strong mechanism for flu spread. If our economy continues

to shift to more service-oriented employment, the results presented here suggest there is greater potential for flu spread in the future. Therefore, we suggest that monitoring shifts in employment from goods producing jobs to service jobs could help officials plan for the severity of flu incidence.

Our results are also of interest to firms concerning productivity during the flu season. Employers should consider differences in the lost productivity from many employees becoming infected with influenza versus the lost productivity from a few infected individuals taking sick leave. Workers concerned about missing pay or losing their jobs as the result of staying home from work due to illness will be less likely to heed early signs of influenza infection and stay home. Since a person may be infectious while experiencing mild symptoms, this greatly increases the probability that the virus will spread to other workers in the firm. This implies that firms should consider more generous "sick day" policies, particularly during the flu season.

Future research should concentrate on more micro-level data or testing this relationship internationally. Individual level data will allow a more direct estimation of the impact of employment status on the incidence of flu. Additionally, this will allow the determination of what types of jobs are most likely to aid in the spread of the flu virus.

References

Adda, J. (2016). Economic activity and the spread of viral diseases: Evidence from high frequency data. The Quarterly Journal of Economics 131(2), 891–941.

Anderson, T. W. and C. Hsiao (1981). Estimation of dynamic models with error components. Journal of the American Statistical Association 76(375), 598–606.

Arellano, M. and S. Bond (1991). Some tests of specification for panel data: Monte carlo evidence and an application to employment equations. The Review of Economic Studies 58(2), 277–297.

Arellano, M. and O. Bover (1995). Another look at the instrumental variable estimation of error-components models. Journal of Econometrics 68(1), 29–51.

Bai, J. and S. Ng (2010). Instrumental variable estimation in a data rich environment. Econometric Theory 26(6), 1577–1606.

Beggs, J. J. and M. Nerlove (1988). Biases in dynamic models with fixed effects. Economics Letters 26(1), 29 – 31.

Bound, J., C. Brown, and N. Mathiowetz (2001). Chapter 59 - measurement error in survey data. Volume 5 of Handbook of Econometrics, pp. 3705 – 3843. Elsevier.

Brankston, G., L. Gitterman, Z. Hirji, C. Lemieux, and M. Gardam (2007). Transmission of influenza a in human beings. The Lancet Infectious Diseases 7(4), 257 – 265.

Cannell, J. J., M. Zasloff, C. F. Garland, R. Scragg, and E. Giovannucci (2008). On the epidemiology of influenza. Virology Journal 5, 29.

Centers for Disease Control and Prevention (2018a). Disease burden of influenza.

Centers for Disease Control and Prevention (2018b). Interim estimates of 2017-18 seasonal influenza vaccine effectiveness Ů United States, February 2018.

Centers for Disease Control and Prevention (2018c). Seasonal influenza (flu).

Centers for Disease Control and Prevention (2018d). Seasonal influenza vaccine effectiveness, 2005-2018.

Centers for Disease Control and Prevention (2018e). Types of influenza viruses.

Cornwell, B. (2012). Unemployment and widespread influenza in America, 1999–2010. Influenza and other respiratory viruses 6(1), 63–70.

Cox, N. J. and K. Subbarao (2000). Global epidemiology of influenza: past and present. Annual Review Of Medicine 51, 407 – 421.

Currie, J., V. Duque, and I. Garfinkel (2015). The great recession and mothers' health. The Economic Journal 125(588), F311–F346.

Gerdtham, U. and M. Johannesson (1999). New estimates of the demand for health: results based on a categorical health measure and Swedish micro data. Social Science & Medicine (1982) 49(10), 1325 – 1332.

Gerdtham, U. and C. J. Ruhm (2006). Deaths rise in good economic times: evidence from the OECD. Economics And Human Biology 4(3), 298 – 316.

Kapetanios, G. and M. Marcellino (2010). Factor-GMM estimation with large sets of possibly weak instruments. Computational Statistics & Data Analysis 54(11), 2655–2675.

Molinari, N.-A. M., I. R. Ortega-Sanchez, M. L. Messonnier, W. W. Thompson, P. M. Wortley, E. Weintraub, and C. B. Bridges (2007). The annual impact of seasonal influenza in the U.S.: Measuring disease burden and costs. Vaccine 25(27), 5086 – 5096.

Nickell, S. J. (1981). Biases in dynamic models with fixed effects. Econometrica 49(6), 1417 – 1426.

Roodman, D. (2009a). How to do xtabond2: An introduction to difference and system GMM in Stata. Stata Journal 9, 86–136.

Roodman, D. (2009b). A note on the theme of too many instruments. Oxford Bulletin of Economics and statistics 71(1), 135–158.

Rothman, T. (2017). The cost of influenza disease burden in the U.S. population. International Journal of Economics & Management Science 6(4), 1–3.

Ruhm, C. J. (2000). Are recessions good for your health? Quarterly Journal of Economics 115(2), 617 – 650.

Ruhm, C. J. (2003). Good times make you sick. Journal Of Health Economics 22(4), 637 – 658.

Ruhm, C. J. (2005). Healthy living in hard times. Journal of Health Economics 24(2), 341 – 363.

Ruhm, C. J. (2007). A healthy economy can break your heart. Demography 44(4), 829 – 848.

Ruhm, C. J. (2015). Recessions, healthy no more? Journal of Health Economics 42, 17–28.

Ruhm, C. J. (2016). Health effects of economic crises. Health Economics 25, 6–24.

Ruhm, C. J. and W. E. Black (2002). Does drinking really decrease in bad times?. Journal of Health Economics 21(4), 659 – 678.

Stoecker, C., N. J. Sanders, and A. Barreca (2016). 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.

Tellier, R. (2006). Review of aerosol transmission of influenza a virus. Emerging Infectious Diseases 12(11), 1657 – 1662.

White, J. H. (2008). Vitamin D signaling, infectious diseases, and regulation of innate immunity. Infection And Immunity 76(9), 3837 – 3843.

Table 1: Summary Statistics

	Mean	Std. Dev	Min	Max
% Flu-Related Dr. Visits	2.21	1.60	0.00	14.02
Empl. Rate	59.11	3.27	48.32	71.37
% Retail Empl.	6.16	0.51	3.53	9.25
% Health Empl.	7.39	1.26	4.45	12.45
% Manufacturing Empl.	5.09	1.77	0.18	10.28
% Const. Empl.	2.41	0.53	1.36	6.97
% Avg. Temp < 30	12.65	33.24	0.00	100.00
% Avg. Temp 30-39	16.64	37.25	0.00	100.00
% Avg. Temp 40-49	26.25	44.01	0.00	100.00
% Avg. Temp 50-59	26.96	44.38	0.00	100.00
% Avg. Temp ≥ 60	17.51	38.01	0.00	100.00
% Avg. Precip < 1	13.43	34.10	0.00	100.00
% Avg. Precip 1-2.9	39.69	48.93	0.00	100.00
% Avg. Precip 3-4.9	32.12	46.70	0.00	100.00
% Avg. Precip 5-6.9	10.86	31.12	0.00	100.00
% Avg. Precip ≥ 7	3.90	19.37	0.00	100.00
N	2,688			

Notes: Data from the CDC ILI, the Bureau of Labor Statistics, and the National Oceanic and Atmospheric Administration. Statistics are for the 2010/2011 to 2016/2017 flu seasons, which span from October to May. Statistics are weighted by state non-institutionalized population.

Table 2: The Effect of Employment on Natural Log of Flu Prevalence

	Fixed Effects	Lagged Dependent Variable	First Difference 2SLS	Arellano Bover
Lagged Employment	0.283***	0.259***	0.178	0.190***
	(0.060)	(0.048)	(0.213)	(0.050)
Avg. Temp < 30	-0.238***	-0.072	-0.252	-0.102
	(0.089)	(0.081)	(0.396)	(0.093)
Avg. Temp 30-39	-0.232***	-0.036	-0.137	-0.074
	(0.077)	(0.073)	(0.380)	(0.085)
Avg. Temp 40-49	-0.165**	-0.025	-0.072	-0.059
	(0.075)	(0.067)	(0.302)	(0.083)
Avg. Temp 50-59	-0.107*	-0.028	0.007	-0.062
	(0.061)	(0.053)	(0.156)	(0.070)
Avg. Precip 1-2.9	0.067**	0.039	0.025	0.027
	(0.032)	(0.030)	(0.068)	(0.032)
Avg. Precip 3-4.9	0.092**	0.037	-0.011	0.029
	(0.037)	(0.035)	(0.049)	(0.038)
Avg. Precip 5-6.9	0.002	-0.039	-0.090*	-0.050
	(0.046)	(0.042)	(0.047)	(0.048)
Avg. Precip ≥ 7	-0.056	-0.062	-0.064	-0.091
	(0.062)	(0.057)	(0.081)	(0.061)
Lagged Flu (1 Month)		0.535***	0.235	0.603***
		(0.032)	(1.030)	(0.040)
Lagged Flu (2 Months)		-0.269***	0.005	-0.263***
		(0.027)	(0.488)	(0.030)
Lagged Flu (3 Months)		-0.063**	-0.059	0.018
		(0.027)	(0.165)	(0.027)
N	2688	2544	1536	2544
Adj. R^2	0.797	0.853	0.385	
AR Test 1				0.000
AR Test 2				0.000
AR Test 3				0.137
AR Test 4				0.912

Notes: Data from the CDC ILI, the Bureau of Labor Statistics, and the National Oceanic and Atmospheric Administration. All models are run for the 2010/2011 to 2016/2017 flu seasons, which span from October to May. The dependent variable is the logged rate of flu diagnoses from the CDC. In addition the coefficients shown, all models include state by flu season fixed effects and state by month fixed effects. Standard errors are in parentheses, and are clustered at the state by flu season level for the Fixed Effects and Lagged Dependent Variable specifications and are robust in the First Difference 2SLS and Arellano Bover specifications. All regressions are weighted by state non-institutionalized population. Stars denote statistical significance levels: *: 10%, **: 5%, and ***: 1%.

Table 3: The Effect of Employment on Natural Log of Flu Prevalence
(Dynamic Panel Data Robustness Checks)

	Limit Number of Instruments	Use PCA to Reduce Instruments	Collapse Instruments	First Difference (Arellano & Bond)
Lagged Employment	0.197***	0.202***	0.132***	0.223***
	(0.046)	(0.047)	(0.048)	(0.069)
% Avg. Temp < 30	-0.001	-0.000	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)
% Avg. Temp 30-39	-0.000	-0.000	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)
% Avg. Temp 40-49	-0.000	-0.000	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)
% Avg. Temp 50-59	-0.000	-0.000	-0.000	-0.000
	(0.001)	(0.001)	(0.000)	(0.001)
% Avg. Precip 1-2.9	0.000	0.000	0.001**	0.001***
	(0.000)	(0.000)	(0.000)	(0.000)
% Avg. Precip 3-4.9	0.000	0.000	0.001**	0.001**
	(0.000)	(0.000)	(0.000)	(0.000)
% Avg. Precip 5-6.9	-0.000	-0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)	(0.000)
% Avg. Precip ≥ 7	-0.001	-0.001	0.000	0.000
	(0.001)	(0.001)	(0.001)	(0.001)
Lagged Flu (1 Month)	0.591***	0.624***	0.591***	0.582***
	(0.038)	(0.095)	(0.047)	(0.044)
Lagged Flu (2 Months)	-0.246***	-0.350***	-0.251***	-0.249***
	(0.026)	(0.082)	(0.027)	(0.028)
Lagged Flu (3 Months)	0.001	0.080	0.040	0.014
	(0.024)	(0.058)	(0.030)	(0.032)
N	2544	2544	2208	2208
AR Test 1	0.000	0.000	0.000	0.000
AR Test 2	0.000	0.598	0.000	0.000
AR Test 3	0.611	0.824	0.385	0.112
AR Test 4	0.597	0.170	0.871	0.699

Notes: Data from the CDC ILI, the Bureau of Labor Statistics, and the National Oceanic and Atmospheric Administration. All models are run for the 2010/2011 to 2016/2017 flu seasons, which span from October to May. The dependent variable is the logged rate of flu diagnoses from the CDC. In addition the coefficients shown, all models include state by flu season fixed effects and state by month fixed effects. Robust standard errors are in parentheses. All regressions are weighted by state non-institutionalized population. Stars denote statistical significance levels: *: 10%, **: 5%, and ***: 1%.

Table 4: Industry Personal Contact Summary

Industry	Total Employment (thousands)	Interpersonal Contact Level		
		Mean	Std. Error	Rank
Retail Trade	7,954.6	3.296	0.022	1
Healthcare	8,019.1	3.286	0.029	2
Management	1,094.5	3.203	0.031	3
Information	967.3	3.146	0.036	4
Finance	3,369.7	3.143	0.030	5
Utilities	234.1	3.135	0.039	6
Government	3,552.7	3.118	0.029	7
Other Services	3,053.2	3.111	0.034	8
Education	2,755.2	3.101	0.029	9
Wholesale Trade	3,210.1	3.100	0.030	10
Professional	3,985.0	3.089	0.029	11
Hospitality And Food	6,683.5	3.015	0.032	12
Arts	924.1	3.000	0.039	13
Real Estate	1,170.5	2.992	0.043	14
Transportation	1,488.9	2.983	0.030	15
Administrative Support	3,899.9	2.939	0.030	16
Mining	247.2	2.896	0.046	17
Construction	3,664.3	2.892	0.033	18
Manufacturing	5,231.2	2.849	0.035	19

Notes: Data from the O*Net Database (version 22).

Table 5: The Effect of Employment on Natural Log of Flu Prevalence by Industry (Arellano Bover Models)

	High Personal Contact Sectors		Low Personal Contact Sectors	
	Retail Trade	Healthcare	Manufacturing	Construction
Lagged Employment	0.982**	0.741*	0.549	-0.104
	(0.426)	(0.397)	(0.442)	(0.240)
Avg. Temp < 30	-0.088	-0.114	-0.107	-0.103
	(0.094)	(0.094)	(0.094)	(0.094)
Avg. Temp 30-39	-0.066	-0.085	-0.080	-0.078
	(0.086)	(0.085)	(0.085)	(0.085)
Avg. Temp 40-49	-0.054	-0.070	-0.067	-0.066
	(0.083)	(0.083)	(0.083)	(0.084)
Avg. Temp 50-59	-0.057	-0.069	-0.066	-0.060
	(0.069)	(0.069)	(0.069)	(0.070)
Avg. Precip 1-2.9	0.017	0.019	0.020	0.013
	(0.032)	(0.032)	(0.032)	(0.032)
Avg. Precip 3-4.9	0.023	0.022	0.022	0.005
	(0.038)	(0.038)	(0.038)	(0.037)
Avg. Precip 5-6.9	-0.051	-0.055	-0.054	-0.080*
	(0.048)	(0.048)	(0.048)	(0.047)
Avg. Precip ≥ 7	-0.100	-0.103	-0.103	-0.121*
	(0.063)	(0.063)	(0.062)	(0.063)
Lagged Flu (1 Month)	0.603***	0.601***	0.605***	0.590***
	(0.040)	(0.040)	(0.040)	(0.041)
Lagged Flu (2 Months)	-0.254***	-0.260***	-0.261***	-0.267***
	(0.030)	(0.030)	(0.030)	(0.032)
Lagged Flu (3 Months)	0.020	0.019	0.020	0.009
	(0.027)	(0.027)	(0.027)	(0.029)
N	2544	2544	2544	2438
AR Test 1	0.000	0.000	0.000	0.000
AR Test 2	0.000	0.000	0.000	0.000
AR Test 3	0.170	0.157	0.127	0.026
AR Test 4	0.974	0.940	0.934	0.605

Notes: Data from the CDC ILI, the O*Net Database (version 22), the Bureau of Labor Statistics, and the National Oceanic and Atmospheric Administration. All models are run for the 2010/2011 to 2016/2017 flu seasons, which span from October to May. The dependent variable is the logged rate of flu diagnoses from the CDC. Each model is estimated with Arellano-Bover dynamic panel data models including two lags of the dependent variable. In addition the coefficients shown, all models include state by flu season fixed effects and state by month fixed effects. Robust standard errors are in parentheses. The AR Test statistics are p-values corresponding to Arrelano-Bond tests for first, second, third and fourth degree autocorrelation. All regressions are weighted by state non-institutionalized population. Stars denote statistical significance levels: *: 10%, **: 5%, and ***: 1%.

Appendix Table

Table A1: Robustness Checks: The Effect of Employment on Flu Prevalence

	Fixed Effects	Lagged Dependent Variable	First Difference 2SLS	Arellano Bover
Replace Emp. Rate w/ Unemp. Rate	-0.009	-0.008	-0.177	0.013
	(0.058)	(0.044)	(0.176)	(0.044)
Replace Emp. Rate w/ LFP Rate	0.237***	0.216***	-0.011	0.170***
	(0.049)	(0.039)	(0.071)	(0.041)
Replace Emp. Rate w/ Unemp. to Pop.	0.022	0.025	-0.262	0.049
	(0.087)	(0.066)	(0.251)	(0.066)
Ln Healthcare Visits as Dep. Var.	0.016	0.029	0.045	0.014
	(0.029)	(0.027)	(0.039)	(0.036)
Ln Flu Visits as Dep. Var.	0.330***	0.297***	1.019	0.236***
	(0.075)	(0.064)	(4.697)	(0.068)
Flu Rate (+1)	0.144***	0.128***	0.618	0.067***
	(0.030)	(0.021)	(1.142)	(0.022)
Flu Rate (Not Logged)	0.478***	0.447***	-0.556	0.220***
	(0.096)	(0.076)	(1.092)	(0.076)
Use Ln Emp Rate	16.415***	14.623***	10.648	10.398***
	(3.450)	(2.758)	(14.188)	(2.878)
Include Ln PC Income as Control	0.273***	0.242***	0.159	0.168***
	(0.059)	(0.047)	(0.239)	(0.049)
Lag Temp/Precip.	0.285***	0.256***	0.222	0.191***
	(0.060)	(0.048)	(0.250)	(0.050)
Contemporaneous Employment	0.185***	0.172***	0.221	0.139***
	(0.059)	(0.049)	(0.198)	(0.049)
Models w/o Weights	0.285***	0.258***	0.192	0.197***
	(0.062)	(0.050)	(0.226)	(0.050)
Cluster Standard Errors at State Level	0.283***	0.259***	0.178	0.190***
	(0.050)	(0.039)	(0.213)	(0.050)
Run on All Months	0.290***	0.213***	0.231	0.200***
	(0.058)	(0.050)	(0.286)	(0.049)

Notes: Data from the CDC ILI, the Bureau of Labor Statistics, the Bureau of Economic Analysis, and the National Oceanic and Atmospheric Administration. All models are run for the 2010/2011 to 2016/2017 flu seasons, which span from October to May. In addition the coefficients shown, all models include state by flu season fixed effects and state by month fixed effects. Standard errors are in parentheses, and are clustered at the state by flu season level for the Fixed Effects and Lagged Dependent Variable specifications and are robust in the First Difference 2SLS and Arellano Bover specifications. All regressions are weighted by state non-institutionalized population unless noted. Stars denote statistical significance levels: *: 10%, **: 5%, and ***: 1%.