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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 doctor visits by about 16 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 economy. According to the Centers for Disease Control and Prevention (CDC), the flu infects between 9 and 35 million people 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 levels affects 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 16 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 five 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. 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

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 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

¹There 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.

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.

The above discussion points to a possible strong positive relationship between periods of high employment levels and high rates of the flu. However, an alternative relationship should be considered where high unemployment and recessions more generally are associated with high rates of flu cases. This idea is elucidated by Cornwell (2012). He hypothesizes that higher levels of unemployment 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. To test this hypothesis, he 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 support for his hypothesis, although the estimates are likely biased since he ignores the endogeneity of the lagged dependent variable.

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

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

the transmission of the flu. Ultimately, it is an empirical question. However, we argue that the unemployment rate is not an ideal measure to use to answer this question. 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.

This paper also 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

³A 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.

⁴Another 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.

in the context of a communicable disease.

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 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

⁵Her interview is transcribed here: <https://www.cdc.gov/media/releases/2018/t0209-flu-update-activity.html>.

⁶For more information on the construction of the BLS data, see http://www.bls.gov/cps/cps_htgm.htm.

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 employment for any reason, the unadjusted number is preferable.

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 2,800 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. 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.

There are some potential pitfalls with this flu measure. First, the location and coverage of the health providers is 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 people visiting the doctor 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. Doctors' reactions may also change based on the severity of the flu. It may be that doctors diagnose and report patients with the flu more often during the height of the flu season. As long as the measurement error from reporting is not systematically related to employment, our estimates will be unbiased, but the standard errors will rise.

Since the flu data are reported on a weekly basis and our other variables are reported on a monthly or quarterly 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 physician 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.

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 physician 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} . 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. First, X_{sym} includes the natural log of state per-capita income reported quarterly from the Bureau of Economic Analysis, which clarifies the interpretation of our parameter of interest. We want to capture how changing employment conditions affect the spread of influenza, not how changes in income, which may be related to health behaviors and health resources, affect the spread of influenza. Additionally, 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 η ClimDiv 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} . We cluster the standard errors at the state by flu-season level to account for the possible intraseasonal correlation within states in the error term.

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

⁷There 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.

⁸Versions 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.

⁹See <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 η ClimDiv data.

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, employment and health insurance are positively correlated, while the correlation between health insurance and influenza rates is negative or zero. Thus if bias is introduced to the estimates by excluding a health insurance variable, the bias is likely driving our results toward zero.

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

Equation 1 may generate biased and inconsistent coefficients. 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. 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

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

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” framework. 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.

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 27 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 16 percent.¹¹ The coefficient for the first-difference 2SLS model is consistent with that of the Arellano-Bover model, but is imprecisely estimated.¹²

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

¹¹The 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 Arellano-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.

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

To put the 16 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 0.35 percentage points, or 3.5 additional flu-related doctor visits per 1000 doctor visits for all causes. Moreover, 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 one case per 1000 doctor visits. On average, approximately 5,000 out of every 100,000 people visit a general practitioner every month (Centers for Disease Control and Prevention, 2015). Thus, of the newly employed, we expect 18 additional flu-related doctor visits above the mean, or a new total of 557 cases for the 100,000 individuals.

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.¹³

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 cor-

¹³We also tested the robustness of our results to not using the logged flu rate, logging the employment rate, and lagging the other controls (per-capita income and temperature/precipitation). These results, available upon request, continue to show a positive, statistically significant relationship between employment and the flu.

relation 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. If this phenomenon is biasing our results, it is driving them toward zero.

Another potential problem with our dependent variable is that labor market conditions may affect the rate at which people go to the doctor.¹⁴ If people go to the doctor less when they are unemployed, the number of flu-related physician visits could be likewise affected. To check, we ran a regression of the log of the number of physician visits on the employment rate and other controls. We found that as employment activity increases, the number of physician visits increases by a very small, not statistically significant, amount. Our base models account for this potential bias by using as the denominator for the dependent variable the number of physician visits rather than the population. Thus, both the numerator (number of flu-related physician visits) and the denominator (total number of physician visits) are affected by a change in insurance prevalence in the population. We investigated the potential bias by regressing the log of the number of flu-related physician visits on the variables in our preferred model, but adding in the log of the total number of physician visits as an explanatory variable. The coefficients on the employment rate remain positive and statistically significant and nearly identical to our main results.

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.

¹⁴Another 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. In the aggregate, these sources of bias are likely to be small.

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.

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.¹⁵ 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.¹⁶ 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 for employment in the manufacturing and construction sectors are much smaller in magnitude (and negative for the construction sector), and are not statistically significant.

¹⁵Specifically, 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."

¹⁶We 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>. The Bureau of Labor Statistics Industry-Occupation Matrix is located here <https://www.bls.gov/emp/tables/industry-occupation-matrix-industry.htm>, accessed August 7th, 2018.

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 numbers 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 going to work will increase the prevalence of the flu-related physician visits by about 3.5 flu-related physician visits per 1000 physician visits for all reasons. Our results are robust to changing both the empirical strategy and the specification of the model.

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 the 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.

These 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.

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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
Personal Income (thousands)	48.94	7.50	34.72	78.74
% 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 \geq 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 \geq 7	3.90	19.37	0.00	100.00
N	2,688			

Notes: Data from the CDC ILI, the Bureau of Labor Statistics, the Bureau of Economic Analysis, 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 Flu Prevalence

	Fixed Effects	Lagged Dependent Variable	First Difference 2SLS	Arellano Bover
Lagged Employment	0.273*** (0.059)	0.242*** (0.047)	0.159 (0.239)	0.168*** (0.049)
Log Personal Income	1.624 (1.131)	1.769* (0.974)	2.980 (4.044)	2.791** (1.109)
Avg. Temp < 30	-0.223** (0.090)	-0.056 (0.082)	-0.218 (0.447)	-0.075 (0.093)
Avg. Temp 30-39	-0.220*** (0.077)	-0.023 (0.073)	-0.106 (0.426)	-0.052 (0.085)
Avg. Temp 40-49	-0.158** (0.075)	-0.017 (0.067)	-0.047 (0.338)	-0.046 (0.082)
Avg. Temp 50-59	-0.103* (0.061)	-0.024 (0.053)	0.019 (0.174)	-0.055 (0.070)
Avg. Precip 1-2.9	0.067** (0.032)	0.040 (0.030)	0.020 (0.075)	0.029 (0.032)
Avg. Precip 3-4.9	0.093** (0.037)	0.039 (0.035)	-0.016 (0.053)	0.034 (0.038)
Avg. Precip 5-6.9	0.002 (0.046)	-0.036 (0.042)	-0.094** (0.047)	-0.046 (0.048)
Avg. Precip ≥ 7	-0.056 (0.062)	-0.061 (0.057)	-0.063 (0.083)	-0.088 (0.060)
Lagged Flu (1 Month)		0.538*** (0.032)	0.309 (1.145)	0.600*** (0.039)
Lagged Flu (2 Months)		-0.267*** (0.027)	-0.016 (0.524)	-0.261*** (0.029)
Lagged Flu (3 Months)		-0.061** (0.027)	-0.057 (0.171)	0.016 (0.026)
N	2688	2544	1536	2544
Adj. R ²	0.797	0.854	0.375	
AR Test 1				0.000
AR Test 2				0.000
AR Test 3				0.178
AR Test 4				0.882

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. 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 in parentheses are clustered at the state by flu season level. Stars denote statistical significance levels: *: 10%, **: 5%, and ***: 1%.

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

	Limit Number of Instru- ments	Use PCA to Reduce Instru- ments	Collapse Instru- ments	First Difference (Arellano & Bond)
Lagged Employment	0.175*** (0.046)	0.181*** (0.047)	0.121** (0.048)	0.201*** (0.068)
Log Personal Income	2.769*** (1.072)	2.700** (1.092)	4.671*** (1.419)	5.630*** (1.530)
% Avg. Temp < 30	-0.000 (0.001)	-0.000 (0.001)	-0.001 (0.001)	-0.001 (0.001)
% Avg. Temp 30-39	-0.000 (0.001)	-0.000 (0.001)	-0.001 (0.001)	-0.001 (0.001)
% Avg. Temp 40-49	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	-0.001 (0.001)
% Avg. Temp 50-59	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.000)	-0.000 (0.001)
% Avg. Precip 1-2.9	0.000 (0.000)	0.000 (0.000)	0.001** (0.000)	0.001*** (0.000)
% Avg. Precip 3-4.9	0.000 (0.000)	0.000 (0.000)	0.001** (0.000)	0.001** (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)	-0.001 (0.001)	0.000 (0.001)	0.000 (0.001)
Lagged Flu (1 Month)	0.589*** (0.038)	0.617*** (0.093)	0.584*** (0.046)	0.577*** (0.044)
Lagged Flu (2 Months)	-0.244*** (0.026)	-0.341*** (0.081)	-0.246*** (0.026)	-0.243*** (0.028)
Lagged Flu (3 Months)	-0.000 (0.024)	0.072 (0.057)	0.039 (0.030)	0.012 (0.031)
N	2544	2544	2208	2208
AR Test 1	0.000	0.000	0.000	0.000
AR Test 2	0.000	0.527	0.000	0.000
AR Test 3	0.697	0.787	0.545	0.183
AR Test 4	0.617	0.197	0.851	0.671

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. 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 in parentheses are clustered at the state by flu season level. 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) and the Bureau of Labor Statistics.

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

	High Personal Contact Sectors		Low Personal Contact Sectors	
	Retail Trade	Healthcare	Manufacturing	Construction
Lagged Employment	0.825* (0.429)	0.788** (0.397)	0.158 (0.454)	-0.182 (0.252)
Log Personal Income	3.222*** (1.147)	3.553*** (1.141)	3.405*** (1.180)	3.214*** (1.194)
Avg. Temp < 30	-0.059 (0.095)	-0.080 (0.094)	-0.074 (0.094)	-0.069 (0.094)
Avg. Temp 30-39	-0.042 (0.086)	-0.056 (0.085)	-0.052 (0.085)	-0.050 (0.085)
Avg. Temp 40-49	-0.040 (0.082)	-0.052 (0.082)	-0.048 (0.082)	-0.049 (0.083)
Avg. Temp 50-59	-0.050 (0.069)	-0.060 (0.069)	-0.056 (0.069)	-0.051 (0.069)
Avg. Precip 1-2.9	0.020 (0.032)	0.022 (0.032)	0.022 (0.032)	0.015 (0.031)
Avg. Precip 3-4.9	0.029 (0.038)	0.029 (0.037)	0.027 (0.038)	0.012 (0.037)
Avg. Precip 5-6.9	-0.047 (0.048)	-0.048 (0.048)	-0.050 (0.048)	-0.075 (0.047)
Avg. Precip ≥ 7	-0.095 (0.061)	-0.097 (0.061)	-0.098 (0.061)	-0.115* (0.061)
Lagged Flu (1 Month)	0.600*** (0.039)	0.597*** (0.040)	0.602*** (0.039)	0.585*** (0.040)
Lagged Flu (2 Months)	-0.253*** (0.029)	-0.258*** (0.029)	-0.258*** (0.029)	-0.265*** (0.032)
Lagged Flu (3 Months)	0.018 (0.026)	0.017 (0.027)	0.018 (0.026)	0.006 (0.027)
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	0.223	0.223	0.188	0.038
AR Test 4	0.922	0.998	0.973	0.642

Notes: Data from the CDC ILI, the O*Net Database (version 22), 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. The dependent variable is the logged rate of flu diagnoses from the CDC. Each model is estimated with Arellano-Bond dynamic panel data models including two lags of the dependent variable. In addition to 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 Arellano-Bond tests for first, second, third and fourth degree autocorrelation. Stars denote statistical significance levels: *: 10%, **: 5%, and ***: 1%.