Influenza, the *in utero* environment, and later developmental outcomes in children

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Abstract

In a well-known demonstration of the "fetal origins hypothesis," Almond (2006) showed that exposure to the 1918 influenza epidemic in the prenatal period had substantial and long lasting effects on income and educational attainment in adulthood. However, while the historical link for the prominent 1918 epidemic is clear, much less is known about the modern day implications of prenatal exposure to seasonal influenza epidemics. Moreover, these implications may be much broader in scope, possibly contributing to well-known seasonal differences in outcomes over birth quarter, and pointing to inequality in health and ability at the earliest stage of life. Using comprehensive data on influenza epidemics over the last two decades, this paper estimates the effect of prenatal exposure on birth outcomes, and tracks the impact on health and skill accumulation throughout early childhood.

JEL: I12, I18, J13

In a well-known demonstration of the "fetal origins hypothesis," Almond (2006) showed that exposure to the 1918 influenza epidemic in the prenatal period had substantial and long lasting effects on income and educational attainment in adulthood. However, while the historical link for the prominent 1918 epidemic is clear, much less is known about the modern day implications of prenatal exposure to seasonal influenza epidemics. Moreover, these implications may be much broader in scope, possibly contributing to well-known seasonal differences in outcomes over birth quarter, and pointing to inequality in health and ability at the earliest stage of life. Using comprehensive data on influenza epidemics over the last two decades, this paper estimates the effect of prenatal exposure on birth outcomes, and tracks the impact on health and skill accumulation throughout early childhood. Evidence on this relationship is timely, particularly in view of recent literature suggesting that gaps in early developmental status can widen over time, and that the optimum time for investment in child well-being could be at the earliest, even before birth (Almond and Currie 2011; Cunha and Heckman 2010; Cunha, Heckman and Schennach 2010; and Case and Paxson 2010).

In order to provide evidence on the relationship between prenatal exposure and childhood outcomes, we will combine recent data on influenza prevalence in Canadian provinces with the information on early childhood development collected in the National Longitudinal Survey of Children and Youth, Early Childhood Development File (NLSCY -- ECD). Given that the NLSCY documents child development from birth into early childhood, it is uniquely suited to provide evidence on these relationships.

Since our aim is to disentangle the causal mechanism between prenatal exposure and childhood outcomes, we address the possibility that differences in prenatal influenza exposure may be confounded by other factors related to childhood outcomes. For instance, previous literature

shows that seasonal factors affecting infant health (such as weather and family characteristics) vary according to birth timing (Buckles and Hungerman 2008; Deschenes, Greenstone, and Guryan 2009). At the same time influenza risk has a seasonal component. We deal with the endogeneity of influenza exposure by including direct controls for family background characteristics, accounting for province specific differences, and allowing for flexible functions of week and year of conception to capture alternative seasonal factors related to the health endowment at birth. This strategy will identify the effect of influenza exposure by capturing the effect of unseasonably high exposure rates within province.[1]

We find that influenza exposure *in utero* has a deleterious effect on cognition and health measured at age 4 to 5. This is particularly when high rates of influenza are circulating in the *first* trimester of pregnancy. For instance, an increase of 1 standard deviation from the average first trimester surveillance rate is associated with a negative and statistically significant decrease of 1.1 points on the PPVT test, and an increase of 2.8 percentage points in likelihood that the child has a chronic condition.

This paper is organized as follows. First, we describe the data used in the analysis and discuss the method for matching influenza exposure rates to child gestational periods. Second, we describe our methodology. The third sections presents results, and the last sections offers conclusions and describes avenues for future research.

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[1] In the future, we plan to perform further analysis to support this methodology: First, we will instrument influenza exposure with unanticipated mismatches in the influenza vaccine, The idea here is use variation in the type of circulating influenza strains as it relates to the yearly vaccine. Since mismatches occur from random mutations in the influenza virus, the increase in exposure risk is largely unpredictable, particularly in the first months of occurrence. See Ward (2010) for further details on influenza strains, vaccination, and influenza exposure. In an alternative approach, we will focus on within family differences in prenatal influenza exposure by include maternal fixed effects in our model for the sample of NLSCY observations that includes sibling pairs.

Data

Our empirical strategy requires data on childhood developmental outcomes, detailed information on the conception-birth period, and comprehensive data on influenza exposure. Data on child outcomes and birth information comes from the National Longitudinal Study of Children and Youth (NLSCY) and the primary source of influenza exposure is the rate of laboratory confirmed influenza. This section describes and summarizes these data, detailing measures of influenza exposure and then describing how these data are matched to the NLSCY.

Influenza

We compile several sources of information on weekly influenza exposure. Our primary measure is influenza laboratory surveillance rates from the Respiratory Virus Detection/Isolation Surveillance System (RVDI) reported by the Public Health Agency of Canada (PHAC).[2] These data comprise weekly reports of tests performed and tests positive for influenza in each province, and have the advantage of measuring only *laboratory confirmed* assessments of influenza. A further advantage of the RVDI data is that it is available in the early 1990s, which allows us to capture earlier cohorts in the NLSCY (specifically, the data span the period between 1992-week 37 to 2011-week 34).

Since our aim is to match these data to the exact timing of conception-birth periods, our methodology depends on timely reporting of influenza activity relative to actual exposure rates. In particular, since previous evidence on *in utero* exposure to the 1918 influenza epidemic indicates that the impact on long term outcomes is largest for exposure earlier in pregnancy

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[2] These data are available from PHAC at this link: http://www.phac-aspc.gc.ca/bid-bmi/dsd-dsm/rvdi-divr/index-eng.php

(Almond 2006; Mazumdar et al. 2009), it is important to accurately match exposure timing to the timing of each gestational window. For instance, miss assignment in early gestation would occur if influenza reporting happens with a significant lag. We account for this concern in two ways. First, rather than back dating each birth date by average human gestation, we use individual specific information on gestational length to define the conception date. Second, we directly assess the timeliness of influenza reporting by supplementing the influenza surveillance data with data from two other sources and comparing the timing of each of these measures.

The first supplementary source of data is hospital counts from the Hospital Morbidity Database (HMDB) maintained by the Canadian Institute for Health Information. The HMDB includes complete records of inpatient discharges for hospitals in Canada. Each discharge abstract consists of information on patient age, sex and home postal code as well as detailed medical information including: date of hospital admittance and discharge, discharge disposition (i.e. living or deceased), and detailed diagnosis information. Each abstract reports one diagnosis labelled the most responsible diagnosis (MRD) and up to 15 co-diagnoses. We use the HMDB data to construct weekly hospitalization counts for influenza and pneumonia admissions and date these hospitalizations using the date of admission.[3] These data span the time period of 1996-week 14 to 2006-week 13.[4]

The second supplementary source is Google Flu Trends data from google.org.[5] These data use aggregated influenza associated search term activity to " estimate current flu activity around the

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[3] We include all admissions listing influenza or pneumonia as a diagnosis (either as most responsible or contributory). All hospital counts are adjusted for changes in diagnosis coding from International Classification of Disease edition nine to edition ten.

[4] Hospitals in Quebec and non-Winnipeg Manitoba did not submit to the HMDB prior to 2001.

[5] Data for Canadian provinces is available here: http://www.google.org/flutrends/ca/#CA

world in near real-time" (Ginsberg et al. 2009; Google 2012)[6]. The aggregated search queries have been validated through comparison with official historic influenza data on the number of physician visits for influenza-like-illness (ILI) and the search data have been normalized to represent ILI cases per 100,000 physician visits for each week in each province.[7] These data span the period 2003-week 39 to 2011-week 34.

Figure 1 shows a comparison of average weekly influenza rates for all three measures for all available data points. All three measures display the same seasonal aspect of influenza exposure with epidemic peaks occurring in similar weeks in each year. For instance, over province and season, the average range between peak weeks for all influenza measures is 3 weeks, and in 35 percent of all province-seasons combinations, peak weeks for these measures fall within one week of each other.[8] In terms of the timing of each seasonal influenza epidemic, the average seasonal peak occurs in week 6 of each year (i.e. averaging the peak week for all three influenza measures over each province and season).

To further understand the relative timing of the influenza measures, Figure 2 shows estimates of lag and lead effects for our primary measure of influenza (the rate of positive influenza surveillance tests) and the other measures of influenza circulation. The first panel shows the correlation of the surveillance rate with up to four week lags and leads of the log of influenza/pneumonia hospitalizations (controlling for province and season specific factors). These results show that changes in hospital admissions in a particular week are associated with contemporaneous changes in the surveillance rate. The second panel shows results for up to four

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[6] Further information on Google Flu Trends is available here: http://www.google.org/flutrends/about/how.html

[7] Flu trends data are not available for Prince Edward Island due to small population size and Google confidentially regulations.

[8] Each influenza season is defined as the year running from week 35 to week 34 of the following year.

lags and leads of Google influenza search volume (normalized to ILI per 100,000 physician visits). Given the nature of this influenza measure, changes in influenza exposure probabilities likely translate into changes in influenza associated search activity with little delay. The second panel shows that Google search volume appears to lead the surveillance rate relative to hospital admissions. However, the timing of influenza associated Google search terms still corresponds well with the timing of the influenza surveillance rate. Given this evidence, we interpret the surveillance rate as closely tracking the timing of actual influenza exposure probabilities.

Summary statistics for influenza are given in Table 1. The means for weekly rates/counts by province are given in the first rows. For instance, per week per province, an average 5 percent of surveillance tests are positive for influenza, an average of 232 cases of influenza/pneumonia are admitted to hospital, and, based on Google search activity, there are about 1,911 cases of ILI per 100,000 physicians visits.

The statistics also show substantial variation in surveillance rates. The last two columns of the Table break this variation down into "between" province and season variation and "within" province and season variation. Here, the between standard deviation is calculated using variable means for each province and season, and the within standard deviation is calculated using residual variation for each variable after differencing out variable means for province-by-season. The results show that most of the variation in influenza surveillance rates occurs within province and season.

Since we will account for province and season fixed effects in the analysis, identification relies on ample variation in *in utero* influenza exposure within province and season. The remainder of Table 1 shows mean rates of influenza exposure by pregnancy trimester (i.e. 13 week duration)

and by gestational period (i.e. 41 week duration). These variables calculate, for each possible conception week, the sum of influenza counts over the next 13 (or 41) weeks. Since influenza epidemics each year occur rather sharply (yielding substantially elevated rates for a period of about 10 weeks), there is still room for variation in *in utero* exposure within province and season. Since previous evidence has identified early pregnancy as a vulnerable period for exposure, we will focus on estimating effects by trimester. The within-variation for the full 41 week gestational period is lower, but variation in influenza surveillance rates still remains high with a within-standard deviation at 55 percent of the mean.

NLSCY

The National Longitudinal Survey Children and Youth, Early Child Development File (NLSCY - ECD) documents child development from birth to early childhood, and hence, it is uniquely suited to provide evidence on the relationship between *in utero* exposure to influenza and later developmental outcomes. Data are collected from the 'person most knowledgeable' about the child (typically the mother) during in-home interviews with a Statistics Canada employee at two-year intervals.

We pool 6 cohorts of children for whom we know both place of residence at 0/1 and pre-natal/birth history as well as later pre-school developmental outcomes at age 4/5. Cohorts include children with conception dates between 1992 and 2004 and pre-school developmental indicators between 1996 and 2008. At this stage in the project, we have focussed on just two outcomes.[9] The first is a marker of cognitive development – the Peabody Picture Vocabulary Test score (PPVT-R). The PPVT is a measure of receptive vocabulary often used as a measure

[9] In later work, we plan to expand to include additional indicators of cognitive development (e.g., indicators of general knowledge and number knowledge) as well as indicators of behavioural development (e.g., anxiety, hyerpactivity).

of school readiness in young children (xx). Given parental permission, the test is administered by the interviewer in the child's home. The child is shown pictures on an easel and asked to point to the one that matches the word the interviewer says (the test can be taken in French or English). We use the age-standardized PPVT-R score. Our second outcome of interest is measure of health – specifically, a pmk-reported indicator that the child has a chronic condition diagnosed by a physician and having lasted (or being expected to last) at least six months.

Table 2 gives summary statistics for the NLSCY sample, and indicates an average standardized PPVT score of 101 with a standard deviation of 15 points. An average of 19 percent of 4 to 5 year olds have been diagnosed with a chronic condition. The remainder of the table shows our base control variable means and standard deviations along with the our controls for health at birth.

There are differences in birth and developmental outcomes, including birth weight and PPVT scores, across conception dates (not shown here). If differences in fertility by date of conception reflect differences in other health and socioeconomic characteristics, then associations between influenza exposure and childhood outcomes may be confounded. We address this concern by using year-by-week-by-province level variation in influenza exposure and controlling directly for province and conception-year differences as well as a conception-month fixed effects to capture province invariant seasonal aspects related to date of conception.

Econometric Strategy

Our analysis is based on the following model relating child outcomes to *in utero* influenza exposure:

$$y_i = \delta_1 flu_{ywp} + \delta_2 flu_{ywp}^2 + X_i\beta + \alpha_y + \gamma_m + \theta_p + \varepsilon_i$$

Where y_i is an outcome for child i, conceived in year y and month m, in province p.[10] The independent variables X are controls for child, PMK and household characteristics at the time of survey. The error term is ε_i, and we report standard errors clustered at the province level to account for serial correlation across time. All regressions are weighted by the survey sample weight.

The variables of interest are flu_{ywp}, and $flu^2_{ywp.}$, which denote influenza exposure over the gestational period for each child conceived in year y, week w, and province p. Since previous evidence has identified the first trimester as the most vulnerable to harmful effects of influenza, we also modify this definition to capture the first trimester of exposure only. The quadratic term is included to capture any nonlinear effect in the effect of influenza exposure. To further explore any nonlinearity, we divide gestation exposure rates into five groups: gestational periods that have surveillance rates of zero, and quartile rankings of the remaining surveillance rates.

The conception date is defined as the number of gestational weeks before each child's birth date where gestational length is based on the date of the mother's last menstrual period. This is preferable to using a 41 week back-date of the birth date since gestational length may itself be a function of influenza exposure. Given that the surveillance rate appears to closely track the timing of influenza exposure, we count the number of surveillance tests from the first week of gestation. For the first trimester, we calculate the rate of positive influenza surveillance during the first 13 week period.

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10 We define the birth province as the province listed during survey administration, and we use the earliest listed province of residence when there has been an indication of a move between surveys. Mobility between date of conception and date of first survey is unlikely a concern since household mobility (particularly across provincial lines) is low during a child's gestational period and early life.

The year and province fixed effects, α_y and θ_s, capture time invariant differences in child outcomes across province, and differences across conception year that are common to all provinces. We define the conception year as the "flu season year" running from week 35 to week 34 of the following year.

We deal with the endogeneity of influenza exposure by including direct controls for seasonal factors and individual background characteristics. Individual level controls measured at the time of survey are represented by X_i and include for the child: sex, age in months, number of siblings; for the PMK: age, education, immigrant status, race and self perceived health status; and for the household: family structure and household income. In order to capture the effect of influenza exposure that is independent of observable measures of health at birth, we also include birth weight and gestational length in some specifications. Since the summary statistics in Figure 1 and in Table 1 demonstrate the strong seasonal pattern in influenza exposure, seasonality in the health endowment is another concern. For instance, previous literature shows that other factors influencing infant health (such as weather and family characteristics) vary according to birth timing (Buckles and Hungerman 2008; Deschenes, Greenstone, and Guryan 2009). In addition to the individual level controls we include, we also include month fixed effects, γ_m, to capture the effect of any secular differences in child outcomes within the year that are independent of influenza exposure.

Results

Table 3 shows the result for the effect of *in utero* influenza exposure on later cognitive and health outcomes of 4 to five year olds. Panel A shows results for a quadratic in the surveillance rate, where exposure is measured over the full 41 week gestational period following conception.

The results are small and imprecisely measured. For instance, for the PPVT test, an increase of 1 standard deviation form the mean surveillance rate is associated with a increases of 0.17 points on the PPVT test (about 1 percent of a standard deviation). There is a 0.6 percentage point increase in likelihood that the child has a chronic condition (a 3 percent decrease relative to the mean).

To assess the extent that this effect is apparent in observable measures of health at birth, we also include gestational length and birth weight in the specification.The results are similar when controlling for health at birth, which indicates that the impact of influenza exposure *in utero* has a separate effect on outcomes measured at ages 4 to 5 that is not captured by measures of health at birth. This could mean that standard measures of health at birth and measures of cognition and health later on are capturing different aspect of child development, or that any impact on *in utero* exposure is latent until later in life.

Previous evidence has shown that the effects of influenza exposure *in utero* are most apparent for exposure earlier in pregnancy. To asses this in our context, we isolate variation in influenza exposure in the first trimester. These results (Panel B) show much stronger effects of cognition and health. For instance, for the PPVT test, an increase of 1 standard deviation form the mean surveillance rate is associated with a negative and statistically significant decreases of 1.1 points on the PPVT test (about 7 percent of a standard deviation). In terms of health, there is an increase a 2.8 percentage point increase in likelihood that the child has a chronic condition (a 14 percent decrease relative to the mean). For further comparison, we can compare these results to the impact of maternal health (i.e. PMK health). The average change in the PPVT score for a mother in poor health versus a mother in excellent health is 3 PPVT points and the average change in the likelihood of a diagnosed chronic condition is 14 percentage points.

Figure 3 offers a more flexible specification by comparing different influenza surveillance quartile-bins to conception periods where the surveillance rate for influenza is zero. In all cases, there is a deleterious effect of positive influenza exposure relative to zero exposure. For PPVT, this effect increases (in absolute terms) over the first 3 quartiles. For the 41 week measure of exposure, this effect drops to zero, whereas the effect in the first 13 weeks remains negative. This could be due to differences in maternal response to influenza epidemics over the gestational period whereby high levels of influenza in later pregnancy induce compensatory avoidance behaviours.[11] This possibility will be addressed in future analysis.

Future Directions

Future work will extend analysis to address any remaining concerns with endogeneity of maternal behaviours an influenza exposure. First, we will account for unobservable seasonal changes that are correlated with impending flu seasons, by instrumenting influenza exposure with unanticipated mismatches in the influenza vaccine. We can show that seasonal mismatches in the influenza vaccine lead to an increase in influenza exposure risk. This increase in risk occurs either because: (1) the vaccine provides less protection to the overall population or (2) mismatched strains are more virile or infectious. In either case, since mismatches occur from random mutations in the influenza virus, the increase in exposure risk is largely unpredictable, particularly in the first months of occurrence. By collecting data on seasonal influenza matches, we will explore the implications of this in the empirical work. In a supplementary approach, we will include maternal fixed effects in our model. Since seasonal variation in unobserved maternal characteristics may explain seasonal differences in infant health outcomes, we will provide

[11] This may also occur because the 13 and 41 week quartile cut offs differ.

supplementary analysis focusing on within-mother differences in prenatal influenza exposure. Using the more limited sample of observations that includes sibling pairs, this analysis will allow us to compare the main specification described above to results controlling for unobserved maternal characteristics.

Conclusions

Using weekly data on influenza surveillance over two decades, we show that exposure to influenza *in utero* is associated with lower scores on the PPVT test and higher likelihood of a chronic condition at age 4 to 5. This effect is primarily apparent when there are high rates of influenza circulating in the first trimester of pregnancy. In this case, an increase of 1 standard deviation from the mean surveillance rate is associated with a negative and statistically significant decreases of 1.1 points on the PPVT test, and an increase of 2.8 percentage points in likelihood that the child has a chronic condition.

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Table 1
Summary Statistics - Influenza

	Mean	S.D.	"Between" Province-Season S.D.	"Within" Province-Season S.D.
Weekly				
Surviellance rate	0.054	0.122	0.029	0.119
Influenza/Pneumonia hospitalizations	231.9	299.2	277.6	111.6
Google search volume	1911.2	1294.8	326.6	1252.9
Over 13 week trimester				
Surviellance rate	0.077	0.123	0.063	0.106
Sureveillance exposure bin				
Zero	0.268			
Quartile 1: (0.00, 0.02]	0.183			
Quartile 2: (0.02, 0.07]	0.183			
Quartile 3: (0.07, 0.15]	0.182			
Quartile 4: (0.15, 1]	0.183			
Influenza/Pneumonia hospitalizations	3217.9	4024.0	3871.2	1098.3
Google search volume	26795.0	13145.0	4249.9	12439.0
Over 41 week gestation				
Surviellance rate	0.122	0.132	0.114	0.066
Sureveillance exposure dummy				
Zero	0.038			
Quartile 1: (0.00, 0.05]	0.240			
Quartile 2: (0.05, 0.1]	0.241			
Quartile 3: (0.1, 0.16]	0.241			
Quartile 4: (0.16, 1]	0.241			
Influenza/Pneumonia hospitalizations	9673.0	11708.4	11665.9	996.2
Google search volume	79966.3	19314.5	15152.3	11977.3

Note: Weekly data is per week, per province. The surveillance rate gives the rate of positive influenza tests out of total tests performed. Hospitalizations denote the number of hospital admissions for influenza or pneumonia. Google search volume denotes weekly search counts for influenza-associated terms and is normalized to represent visits for ILI (influenza like illness) per 100,000 physician visits. Statistics over a 13 (or 41) week duration sum influenza counts over the next 13 (or 41) weeks for each of the 52 possible conception weeks during the year. The “between” standard deviation is calculated using variable means for each province and season (where season is defined as the year running from week 35 to next year week 34). The “within” standard deviation is calculated using residual variation for each variable after differencing out variable means for province and season.

Table 2
Summary Statistics - NLSCY

	Mean	S.D.
Outcomes		
PPVT score	100.90	15.20
Chronic condition	0.191	0.393
Base controls		
Child		
Number of Siblings	1.275	0.940
Age in months	58.58	6.50
Male		
PMK		
Age (PMK)	34.453	5.413
University Education (PMK)	0.275	0.446
Immigrant (PMK)	0.168	0.373
Non White (PMK)	0.152	0.359
Self percieved Health (PMK)	3.030	0.914
Household		
Family Stucture		
Biological Family	0.838	0.368
Step Family	0.026	0.160
Lone Parent	0.135	0.342
Income	40523.0	29275.8
Controls for Health at birth		
Birth weight (kgs)	3.419	0.581
Gestational length (days)	272.53	13.32
Number of Observations	11,888	

Table 3
Regression results - Cognitive and Health outcomes

	Cognitive - PPVT test		Health - Chronic condition	
Panel A - Exposure over 41 week gestation				
Flu rate	1.07	1.37	0.06	0.06
	(2.45)	(2.59)	(0.10)	(0.10)
Flu rate squared	0.78	0.42	-0.05	-0.05
	(2.21)	(2.36)	(0.11)	(0.11)
Effect of 1 S.D. change at mean flu rate	0.166	0.194	0.006	0.006
Point of inflection	-0.688	-1.628	0.554	0.559
Select controls				
Male	-1.344**	-1.558**	0.0262**	0.0259**
	(0.532)	(0.557)	(0.009)	(0.009)
Gestational length (days)		-0.0238***		-0.001
		(0.004)		(0.001)
Birth weight (kgs)		1.616***		0.002
		(0.186)		(0.016)
Panel B - Exposure over 13 week trimester				
Flu rate	-11.41**	-11.41**	0.266*	0.279*
	(4.02)	(4.07)	(0.13)	(0.13)
Flu rate squared	15.82*	15.62*	-0.27	-0.28
	(7.03)	(6.96)	(0.17)	(0.16)
Effect of 1 S.D. change at mean flu rate	-1.104	-1.108	0.028	0.029
Point of inflection	0.361	0.365	0.498	0.507
Select controls				
Male	-1.450**	-1.654**	0.0288**	0.0285***
	(0.573)	(0.591)	(0.009)	(0.009)
Gestational length		-0.0230***		-0.001
		(0.003)		(0.001)
Birth weight		1.552***		0.002
		(0.161)		(0.015)
Base Controls (X)	Y	Y	Y	Y
Conception year, week, province FE	Y	Y	Y	Y

Note: Standard errors in parentheses Source: NLSCY * p < 0.1, ** p < 0.05, *** p < 0.01.

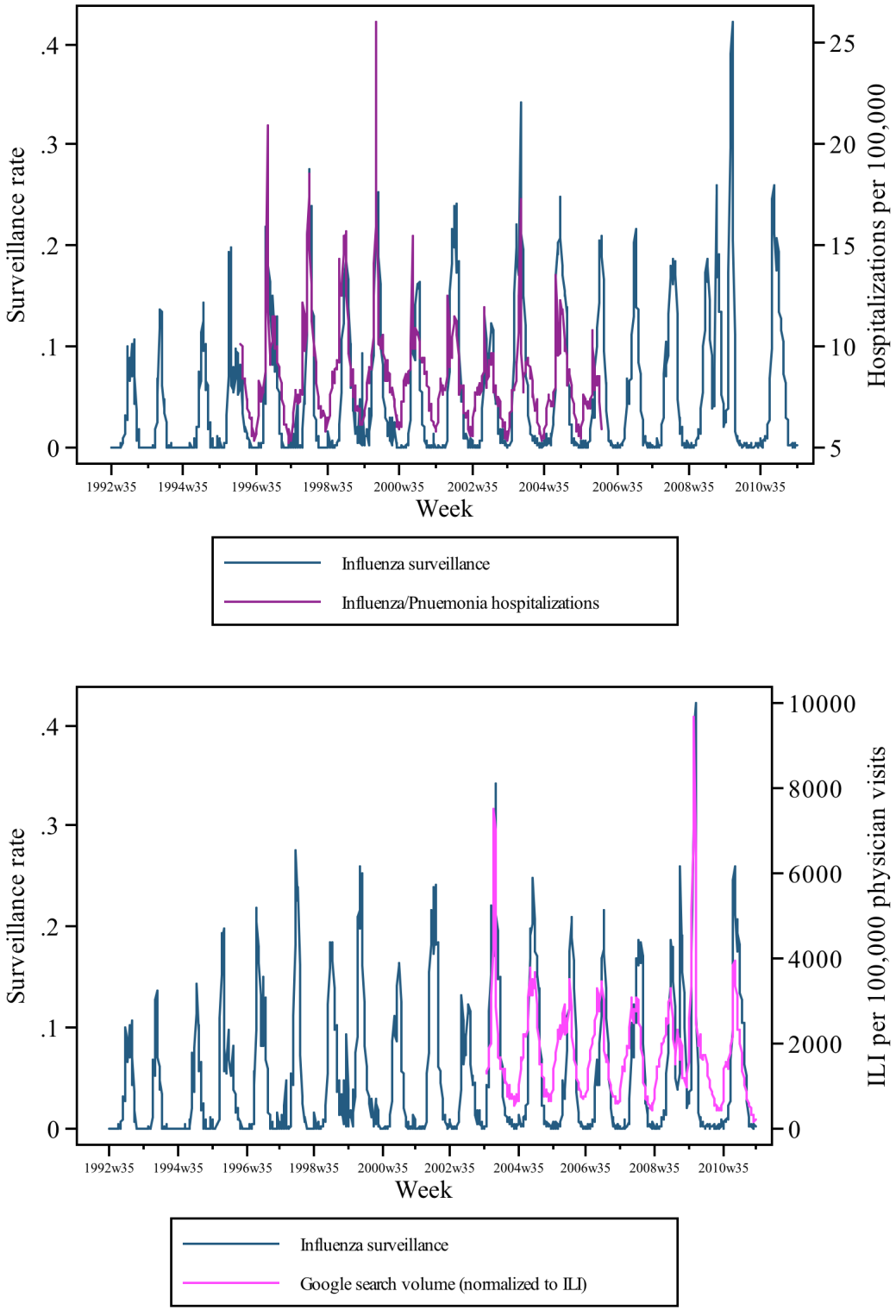
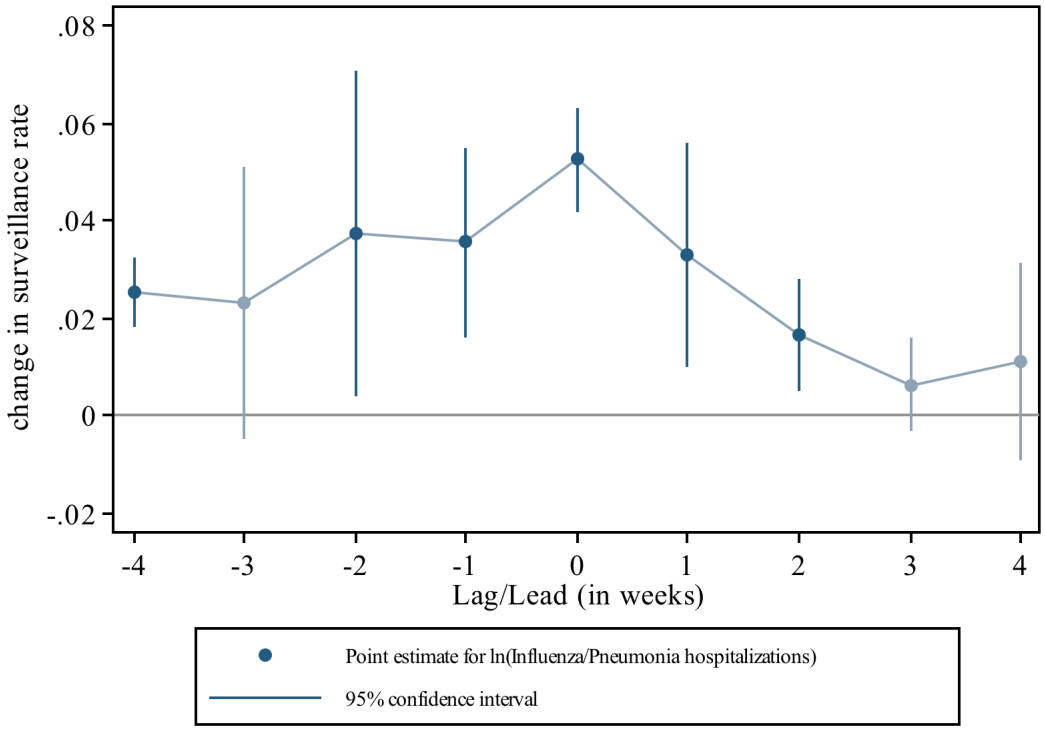


Figure 1: Average weekly influenza rates



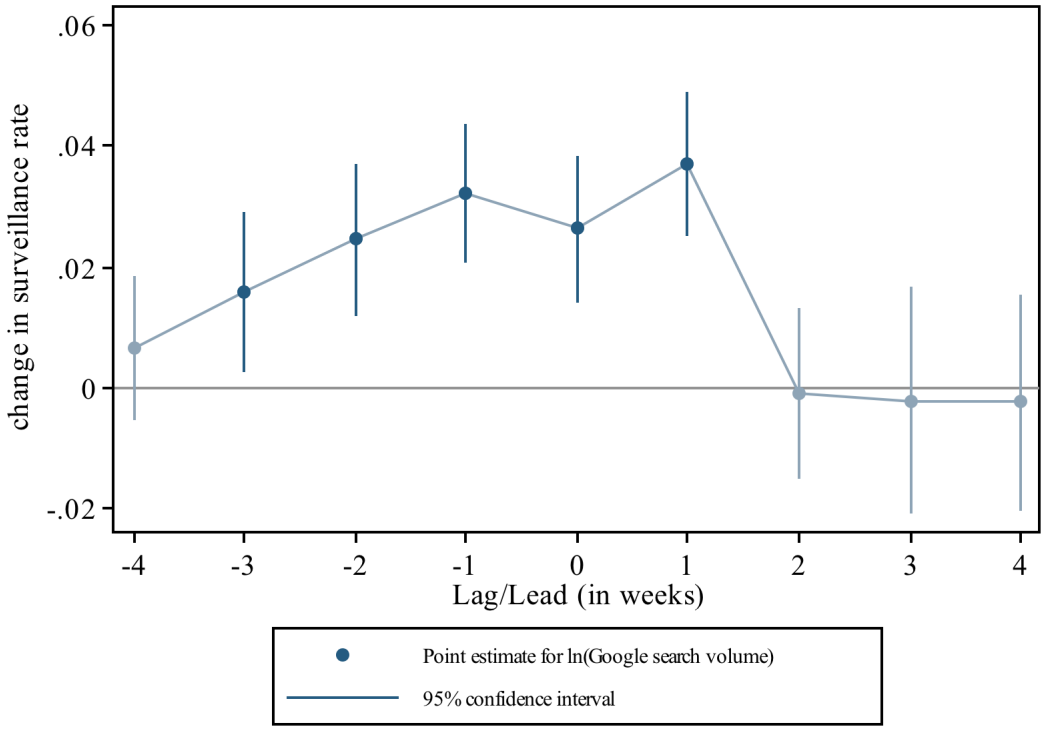


Figure 2: Estimated lag and lead coefficients for influenza measures

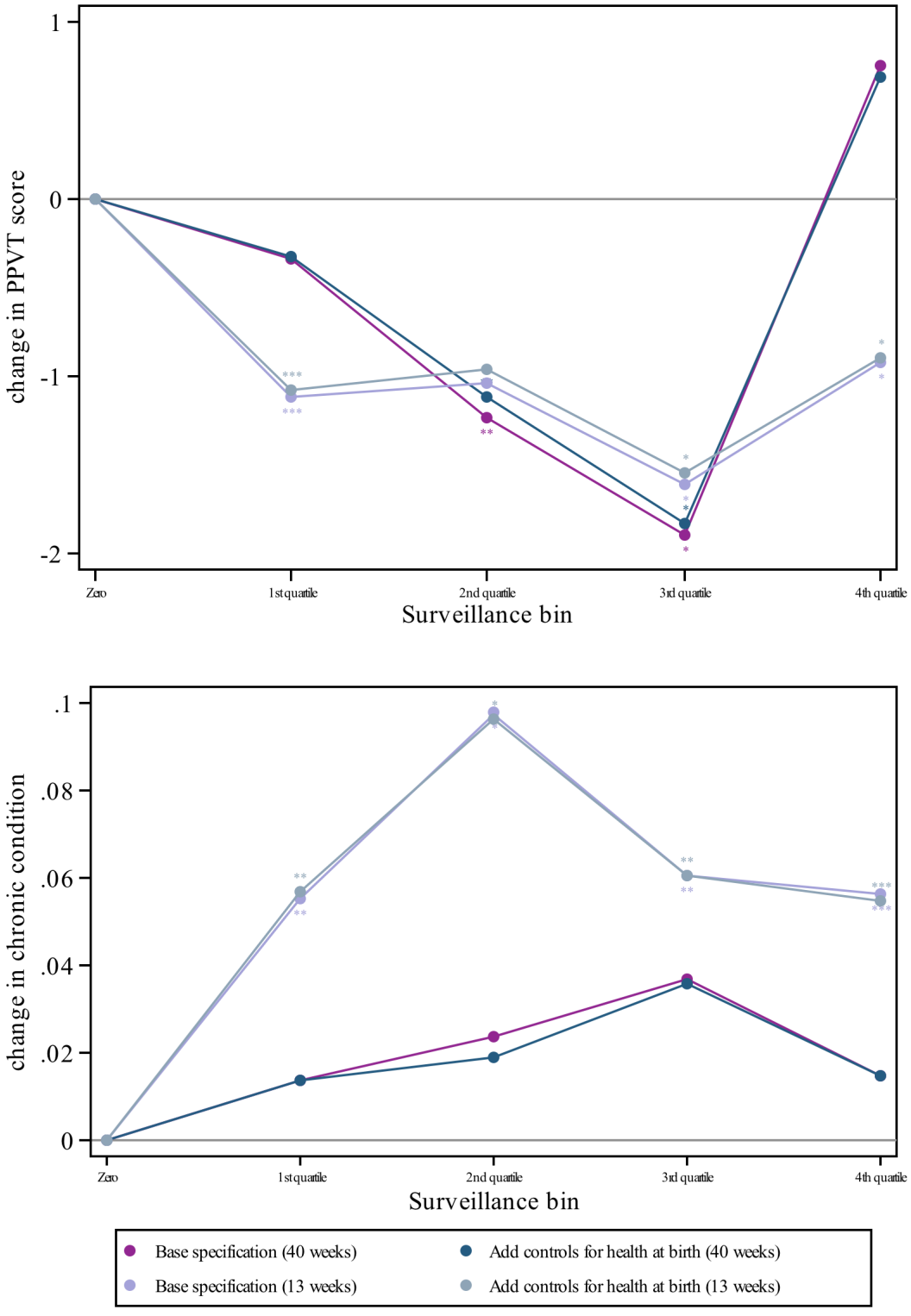


Figure 3: Effect of influenza on cognitive and health outcomes by surveillance bin