The Economic Impacts of Ecosystem Disruptions: Private and Social Costs From Substituting Biological Pest Control*

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Abstract

This study examines how ecosystem degradation and substitution of natural inputs result in private and social costs. I leverage variation from the sudden emergence of a wildlife disease in the United States that caused large mortality shocks to bats, a natural predator of insects. Exploiting the staggered expansion of the wildlife disease, I find that farmers increased their insecticide use, experienced a drop in crop revenues, and that the counties that experienced the bat die-offs saw increases in human infant mortality. The findings demonstrate that disruptions to ecological systems can have substantial impacts on economic activities and human health.

Keywords: substitution, natural inputs, ecosystems, biodiversity, willingness-to-pay, bats, white nose syndrome, pesticides, pollution, agricultural productivity, infant health.
JEL Codes: I10, Q53, Q57.

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1 Introduction

Economists and ecologists theorize that the well-documented reduction in the abundance and diversity of animals and plants (Cardinale et al. 2012; Dirzo et al. 2014; Ceballos et al. 2015; Díaz et al. 2019; Cardoso et al. 2020; Paul et al. 2020) will result in large social costs (Weitzman 1992; Nehring and Puppe 2002; Brock and Xepapadeas 2003; Díaz et al. 2019; Dasgupta 2021; Paul et al. 2020). Despite growing interest in assessing the economic impacts of ecosystem degradation it remains an understudied empirical area (Heal 2000; Fenichel and Abbott 2014; Ferraro et al. 2019), with current knowledge largely not informed by quasi-experimental evidence (Costanza et al. 1997; Polasky et al. 2019). Improving our understanding on this topic is important because substituting environmental inputs as they decline is central to the concept of sustainability in economics (Arrow and Fisher 1974; Dasgupta and Heal 1974; Stiglitz 1974; Solow 1993). The knowledge gap about how changes in *biological* features of the environment affect human well-being is even more striking when contrasted with the large body of work that studies the willingness-to-pay (WTP) for *non-biological* features of the environment such as lower pollution levels (Chay and Greenstone 2003; Currie and Walker 2011; Ebenstein 2012; Zivin and Neidell 2012; Schlenker and Walker 2015; Currie et al. 2015; Ebenstein et al. 2017; Deryugina et al. 2019; Keiser and Shapiro 2019; Marcus 2020), or stable weather conditions (Schlenker et al. 2006; Deschênes and Greenstone 2007; Deschênes et al. 2009; Schlenker and Roberts 2009; Dell et al. 2014; Costinot et al. 2016; Fujiwara et al. 2016; Hsiang et al. 2017; Proctor et al. 2018; Corno et al. 2020).

Valuations of ecosystems have mostly relied on survey data to elicit monetary values for the preservation of species, costs of off-the-shelf substitutes in back of the envelope calculations, or using revealed preferences in observational, non-experimental settings (Daily et al. 2000; Heal 2000). See Hanemann (1994) and Carson (2012) for excellent reviews on the use of those methods and their findings. The lack of identifying variation makes valuation studies hard to interpret, even in terms of orders of magnitude. In one highly controversial example, Costanza et al. (1997) valued global ecosystems and natural capital at nearly twice the output of the global economy, criticized as an "Audacious bid to value the planet" (1998). The value people place on stable ecosystems is an open, and important, empirical question.

The primary goal of this paper is to test whether disruptions to the functioning of ecosystems have an effect on economic activity, and human well-being. I provide large-scale evidence on the costs of declining wildlife populations in the context of agricultural pests and their natural enemies. Specifically, I study the role that insectivorous bats play in the pro-

vision of biological pest control.[1] In the absence of pest control by bats, farmers might face crop losses unless they use insecticides as a substitute (Boyles et al. 2011; Kunz et al. 2011). While these defensive expenditures might be privately optimal, they might not be socially optimal because agrochemicals, of which insecticides are a subset, have been linked to negative health effects (Brainerd and Menon 2014; Lai 2017; Taylor 2021; Dias et al. 2020).

I use plausibly exogenous variation in biological pest control in the form of mortality shocks to bats caused by an invasive fungus species, which has spread to 36 states in the U.S. Exploiting the staggered expansion of the wildlife disease from 2006, I use a difference-in-differences (DD) strategy and find that farmers compensate and increase their insecticide use by 34.5%. While farmers are able to maintain production quantities close to baseline levels, I estimate that crop revenues drop by over 26.7%, potentially due to insect damages that lower crop quality. Following the substitution towards insecticides, which are toxic compounds by design and can be transported by air and water off the target field, I document an increase in human infant mortality rate of 5%.

These findings provide causally interpretable estimates regarding defensive expenditures following the degradation of a key ecosystem function, namely biological pest control. The key contribution I make in this paper is to document the substitution of a declining natural input: compensating with insecticides for the reduction in bat populations that provide biological pest control. While the existence of this substitution has long been theorized, well-identified empirical validation had not thus far been established, to the best of my knowledge (Frank and Schlenker 2016; Hsiang et al. 2019). Fungal diseases as a direct shock to crop productivity have been studied in the past (O'Rourke 1994; Schlarbaum et al. 1997; Banerjee et al. 2010), but I demonstrate here that even when the invasive fungus does not directly target crops, it can have large effects on environmental conditions through an ecological interaction channel.

The quasi-experimental shock to biological pest control and insecticide use allows me to make a second contribution to the literature on the effects of chemical pollution on human health. Existing research documents negative health impacts of air and water pollution from *industrial* activity (Chay and Greenstone 2003; Currie and Walker 2011; Ebenstein 2012; Schlenker and Walker 2015; Ebenstein et al. 2017; Deryugina et al. 2019; Marcus 2020). However, there is less work that leverages quasi-experimental variation to estimate the health effects of pollution from *agricultural* production. Notable exceptions are several papers that find negative health effects from agrochemicals using variation in growing seasons and fertilizer use in India (Brainerd and Menon 2014), changes to agricultural policy in China

[1] The term "biological control" is mostly used to describe interactions between natural enemies where one organism preys, parasites, or causes disease in, another organism (Sawyer 1990).

(Lai 2017), being downstream from an increase in herbicide-tolerant crop adoption in Brazil (Dias et al. 2020), the spread of an invasive fruit pest in the U.S. (Jones 2020), or increased insecticide use that follows cicada cycles in the U.S. (Taylor 2021). The line of work that examines the causal impacts of agrochemicals on health builds on a broad literature that associates pesticide exposure with negative health outcomes (Garry et al. 2002; Regidor et al. 2004; Winchester et al. 2009; Larsen et al. 2017).

I use the empirical estimates along with data on chemical expenditures, and the value of mortality risk reduction to arrive at a total welfare loss of $31.85 billion in the affected counties from 2006 to 2017 ($1,545 per-capita), and $24.3 billion when excluding the infant health damages ($1,1176 per-capita). Combined, these findings reflect both private costs to farmers, as well as social costs to the neighboring populations through the channel of short-term infant health.

In what follows, I review the detection and expansion of White Nose Syndrome in the U.S.; describe the data and I how use it in the DD research design; present the main findings; followed by a set of tests for potential threats to identification along with robustness checks.

2 Emergence of White Nose Syndrome

White Nose Syndrome (WNS) is an infectious wildlife disease that develops in certain bat species as a result of exposure to an invasive cold-loving fungus species. The disease receives its name because the fungus grows around the nose of the bat and creates a cluster of white flakes (Turner et al. 2011). The infection of bats and growth of the fungus benefit from the environmental conditions found in the caves that bats use during the day and throughout winter. Specifically, temperatures below 16 degrees Celsius as the fungus has an upper temperature limit of 20 degrees Celsius (Verant et al. 2012), and is extremely sensitive to ultraviolet light (Palmer et al. 2018).

The earliest evidence of WNS in the U.S. dates back to February 2006 from photos taken at Howes Cave outside of Albany, New York (Blehert et al. 2009). The effects of WNS were first detected during a routine bat survey in March 2007 in the same cave outside of Albany. Instead of finding a healthy bat colony the surveyors found a large number of dead bats on the floor of the cave (Kolbert 2014). Using DNA sequencing of the fungus, researchers have determined the fungus originated from the European continent, and the current scientific consensus regarding its introduction to the U.S. is that it was brought over as spores on the shoes or backpacks of hikers (Frick et al. 2010; Drees et al. 2017).

Several bat species that get infected with the fungus experience high mortality rates. The fungus spreads throughout the skin of the bat, essentially consuming the top layers of skin

tissue. The most common symptoms are premature awakening during hibernation (Reeder et al. 2012; Fenton 2012), damages to the wings' membrane (Reichard and Kunz 2009; Cryan et al. 2010), and evaporative water loss (Willis et al. 2011). Bats that wake up during the winter, when they are supposed to still be hibernating, face an almost non-existent food supply, and an increased caloric use rate due to the low temperatures. They generally do not survive the winter (Thomas et al. 1990; Blehert et al. 2009). By 2010, mortality rates were between 30% and 99%, with a mean of 73% (Frick et al. 2010), and by 2012 the mortality estimate was at least 5.7 million bats (Froschauer and Coleman 2012). However, not all bat species are adversely affected by the fungus, meaning that they do not develop WNS even if they serve as hosts for the fungus. Consequently, there are counties where the fungus is present, but the biological pest control by bats is not disrupted by WNS.

In Figure 1, I plot the gradual expansion of WNS across the U.S., and highlight the states and counties that are included in the main estimation sample. The pattern of county contagion seems to follow the migration path of bats as well as hiking trails along the Appalachian. As seen in Figure 1, there are counties that are further south and west that appear to have been exposed to WNS around the same time as counties closer to the northeast. This suggests that the spores of the fungus were transported to those counties by people, not bats, and it appears that a radial dispersion pattern emerged around these new epicenters. The expansion of WNS contagion remains a complex function of environmental conditions, host genetics, and behavioral responses of both bats and people (Maher et al. 2012; Wilder et al. 2015), making it hard for ecologists to predict which counties will contract the fungus. Because the fungus can survive as spores even without an available bat host, an exposed county remains in exposed status.

3 Data

In the main analysis, I use county-level data from 1997 to 2017 on pesticide use, crop sales, expenditures on agrochemicals, infant health outcomes, and the expansion of WNS. I report summary statistics for the main variables in Table A1, and summarize the secular trends of the main outcomes in Figure A1.

In order to classify the counties that experienced a negative shock to the provision of biological pest control, I use WNS detection data from the Fish and Wildlife Service (FWS) across U.S. counties. Each county is classified as either confirmed, suspected, or not-detected. Because previous research has documented delays in detecting WNS in counties (Verant et al. 2018), and as I demonstrate in the Appendix, those delays average at two years, I uniformly assign WNS treatment onset two years before the formal FWS classification.

The main prediction regarding the defensive expenditures by farmers is that insecticides will be used to substitute for biological pest control. I use the data from the U.S. Geological Survey (USGS) on estimated agricultural pesticide use, by chemical compound, at the county-year level (Baker and Stone 2013; 2015). Using data from the Environmental Protection Agency (EPA), and several other sources, I classify and aggregate the 528 different chemical compounds to three aggregated pesticide use classes: insecticides, fungicides, and herbicides. In order to compare the used amounts between counties of different sizes and different scales of agricultural activity, I normalize each variable by the total county area.

To examine the net effects on agricultural productivity, I use data on the total crop sales value and chemical expenditures, which are reported in the agricultural census, conducted in 5 year intervals. I use the data for the census years of 1997, 2002, 2007, 2012, and 2017. Unfortunately, a balanced panel of crop yield data for the majority of crops are only available for a subset of counties. This limits my ability to estimate the direct damages to agricultural productivity (see the Appendix for additional details).

In order to test for the potential negative health impacts due to insecticide applications, I use restricted-access data on linked birth and death certificates from the National Center for Health Statistics. Following previous work that uses this source of data, I construct infant mortality rates (IMR) for all causes of death, as well as for internal and external causes (Chay and Greenstone 2003; Sanders and Stoecker 2015). The main focus in the analysis is on the internal IMR, which excludes deaths due to accidents and homicides. In addition, I construct variables for the number of live births, mean birth weight, mean gestation length, and share of births that classified as low birth weight or as premature births.

4 White Nose Syndrome as a Natural Experiment

The sudden and unexpected emergence of WNS provides a natural experiment that approximates random assignment of counties to high or low bat population levels. As the fungus expands its range each year, more counties phase into the treatment group. This allows me to estimate the effect of declining bat populations on the outcomes of interest as a function of time from exposure to WNS. Using a staggered difference-in-differences (DD) strategy, I define the counties that are classified as WNS confirmed as the treatment group, and non-WNS counties as the control group. WNS confirmed counties are those where both the fungus and bats suffering from WNS have been detected. Counties where only the fungus has been detected, but no bat species have developed WNS are classified as WNS suspected. The treatment status of WNS suspected counties is ambiguous. Consequently, I exclude the WNS suspected counties from the main analysis in order to compare only the WNS

confirmed to non-WNS counties.

The classification of WNS exposure most likely lags by two years relative to the first signs of decline in bat populations. Recent research has documented this lag (Verant et al. 2018), and I provide additional evidence for it in the Appendix (see Figures A3a and A3b). As a result, I uniformly shift the exposure timing by two years earlier relative to the official FWS year, which simply re-centers the event time around exposure. To allow for enough post-treatment years for each WNS confirmed county, I focus on counties that were classified by the FWS as WNS confirmed counties up to, and including, 2014, while omitting counties exposed post-2015. Finally, I compare the WNS confirmed counties to other non-WNS counties residing in states with WNS confirmed counties.

The main identifying assumption is that counties would have had their outcomes develop similarly in the absence of WNS exposure. In addition to estimating pre-treatment trends, I also verify that lagged values of the outcomes are not strong predictors of WNS exposure. In the context of changing environmental conditions, another important assumption is that neighboring counties are not affected by the exposure of a nearby county. However, because bats can forage for insects in neighboring counties, and migrate between seasons, a sharp decline in a bat population in one county can also affect its adjacent counties.[2] This potential violation of the stable unit treatment value assumption (SUTVA) will negatively bias and attenuate my estimates if those counties also increase their insecticide use. To address this concern, I provide results that estimate the scope of spatial spillovers.

A key threat to the interpretation of the results as identifying the effects of a decline in biological pest control, is that the fungus responsible for WNS might have other effects not through the channel of bat mortality. This violation of the exclusion restriction is made less plausible by two facts. First, the fungus requires cold and humid conditions, with temperatures well below 20 degrees Celsius, as well as little exposure to ultraviolet light to thrive (Palmer et al. 2018; Verant et al. 2012). That makes the caves that serve as bat hibernacula an ideal setting for it, but also reduces the likelihood of it surviving outside of the caves, and having direct impacts on agriculture and health. In addition, the fungus has been prevalent throughout the European continent for years, if not centuries (Leopardi et al. 2015), where no direct link between the fungus, agriculture, and infant health has been suggested. In the analysis, I am able to provide empirical evidence that helps support the exclusion restriction. Specifically, I exploit the fact that some counties have bat species

[2] Most bats fly between 20 to 30 km a night and are considered to have a maximum nightly flying radius of up to 50 km (Williams et al. 1973; Troxell et al. 2019). In the Appendix, I demonstrate that the centroid distance between only 6.7% of the second degree neighboring counties (adjacent to the adjacent counties) have a distance below 50 km. This makes it unlikely that bats fly more than one county away in a given night.

that act as hosts of the fungus, but those species do not develop the symptoms that lead to massive die-offs.

4.1 Main Estimation Specifications

For the outcome of interest, y_{cst}, in county c, state s, in period t, I estimate the post-treatment effect of WNS exposure using the following DD specification:

$$y_{cst} = \beta_1 WNS_{ct} + \boldsymbol{X_{ct}\theta} + \lambda_c + \delta_{st} + \varepsilon_{cst} \tag{1}$$

Where y_{cst} is either insecticide use, crop revenue or chemical expenditures, normalized by county-area; or the infant mortality rate due to internal causes of death.[3] The treatment variable, WNS_{ct}, is a dummy variable that is equal to 1 for county c for all periods t following WNS detection. The parameter of interest is β_1, which captures the average effect of WNS exposure.

I weight observations in order to avoid distortions to the estimated effects that could arise from counties with either low agricultural activity, or low population sizes. Specifically, I use three main sets of sample weights. First, population size, which I use across all outcomes to allow the results to be easily compared to one another. Second, the amount of cropland area prior to the emergence of WNS, as measured in the 2002 agricultural census. Weighting by baseline cropland prevents counties with unstable input use rates to distort the magnitude of the estimates. Third, I use the number of live births in order to reduce the sensitivity of the infant health estimates to unstable rates.

I include county, λ_c, and state-by-year, δ_{st}, fixed effects, controlling for any time-invariant unobserved county characteristics, and for flexible time-trends at the state level, respectively. A key source of local variation over time is weather, which agricultural input use is highly responsive to. To control for local weather variation, I add to the parsimonious specification a set of county-year control variables, $\boldsymbol{X_{ct}}$, which include weather variables for the April through September growing period in the form of degree days in 5 degree bins, as well as linear and squared terms for precipitation. When estimating infant mortality, I also report results where I include population shares by age groups. Any remaining unobserved heterogeneity is part of the error term, ε_{ct}, which I cluster at the county level.

I examine whether there are differential time trends between treatment and control counties before exposure by modifying the specification in Equation (1) to include leads and lags

[3] I focus on estimating a level effects for insecticide use because farmers' decisions regarding insecticide applications rely on whether the number of insects captured in a pheromone trap exceeds a certain subjective threshold, which results in insecticide applications in discrete batches (Metcalf 1980; Bateman 2003)

for WNS exposure:

$$y_{cst} = \sum_{r \leq -7}^{-2} \beta_r \mu_c^r + \sum_{r=0}^{\geq 7} \beta_r \mu_c^r + \boldsymbol{X_{ct}\theta} + \lambda_c + \delta_{st} + \varepsilon_{cst} \tag{2}$$

Where μ_c^r is a dummy variable receiving the value of 1 when the county is r years away from exposure. The effects are all estimated relative to the omitted category of the year just prior to exposure. I bottom code 7 years and above prior to exposure, and top code 8 years and above from exposure.[4] If the identifying assumption holds, we should not expect to observe changes in the outcomes of interest prior to WNS exposure. All remaining variables are the same as in Equation (1).

5 The Effects of Declining Biological Pest Control

In this section, I report the main findings, in Table 1, of an average increase in insecticide use of 34.5%, a decline in crop revenue of 26.7%, and an increase in infant mortality, due to internal causes of death, of 5%. After I establish the main findings of the paper, I review additional results for the validity of the exclusion restriction, the extent of spatial spillovers, the quasi-randomness of WNS expansion, as well as robustness checks for potential outliers and misspecification issues. In the Appendix, I report a set of diagnostic results and alternative estimators addressing concerns regarding two-way-fixed-effects estimators and differential timing of treatment.

5.1 Average Treatment Effects

The main results corroborate the theoretical prediction regarding the substitution pattern following a decline in a natural input. On average, insecticide use increases by 2.59 kg per-squared-km, reflecting an increase of 34.5% relative to the population-weighted mean (Table 1, Panel A, column 2). Insecticides should be the most responsive to a decline in biological pest control as it offers the most direct substitution. However, other pesticide inputs can respond differently, depending on how related they are to insecticide use. Agronomic literature has documented that insect damage opens more pathways for fungi to spread, and weakens the defenses of the plant, in addition to insects dispersing fungal spores as they move between plants (Fennell et al. 1975; Kluth et al. 2002). In Tables A3-A4, I show

[4] When reporting the estimation results, I omit the bottom and top coded coefficients because they are not directly interpretable.

that on average, fungicide use increases, albeit imprecisely, and that herbicide use declines post-WNS exposure. The decline in herbicide use suggests substitution between pesticide classes, either due to binding budget constraints or regulatory limits on pesticide use.[5]

Damages from crop pests could lower the quality of the agricultural output even when overall production does not decrease substantially, resulting in lower prices paid to the farmer and lower revenue. The revenue from the sales of all crop commodities falls, on average, by $7,420 per-squared-km (column 4), and is only slightly offset by an average reduction in chemical expenditure of $410 per-squared-km (column 6).[6] Documenting the effect of environmental conditions on crop quality is challenging because we often lack detailed price data on crop quality and the prices paid to the farmers. However, Dalhaus et al. (2020) established that when apple quality declines due to extreme weather conditions, those quality effects have a larger negative impact on farm revenue than the reduction in yield.

Even if farmers are responding optimally to any observed elevated levels of crop pests, and are correctly targeting their fields, that does not guarantee that sprayed insecticides remain on the field. In Figure A2, I demonstrate that insecticide detections in water samples during the agricultural growing season increase by at least 100%. This sharp increase is in agreement with previous work that documented water and wind erosion carrying pesticide off the target field (Burkhard and Guth 1981; Rüdel 1997; Arias-Estévez et al. 2008; Winchester et al. 2009).

The erosion of insecticides from the field can adversely affect health through off-farm exposure. I test for this potential health channel by using the infant mortality rate, due to internal causes of death, which increases by 0.33 deaths per-1,000 live births, on average, following WNS exposure (column 8). This reflects an increase of 5% relative to the mean, population-weighted level of internal IMR. These findings are robust to different weights, inclusion of weather and population share controls, and are not observed when estimating the effect on infant deaths due to external causes of death such as accidents and homicides (see Tables A5-A7). I verify that the characteristics of mothers who gave birth before and after WNS exposure do not change in WNS counties relative to non-WNS counties (summarized in Figure A6).

The findings on infant mortality are well bounded by previous estimates on environmental pollution and infant health. Combining the average increase of 34.5% in insecticide use, and 5% increase in internal IMR, suggests an elasticity of 0.15. Using the same source of data

[5] In the U.S., the number of pesticide applications, the amounts used, and the allowed pesticide residue on food crops is subject to federal regulations by the Environmental Protection Agency, Department of Agriculture, and Food and Drug Administration.

[6] Unfortunately, to the best of my knowledge, there are no data on chemical expenditures by pesticide class.

for infant health, Chay and Greenstone (2003) estimate that a 1% decline in ambient air pollution levels leads to a 0.3% decline in internal infant mortality. Focusing on agricultural water pollution in India, Brainerd and Menon (2014) find that a 1% increase in agrochemicals in the water leads to a 0.46% increase in infant mortality. In Table A8, I report small effects on birth weight and gestation length, similar to previous papers that find these outcomes do not necessarily deteriorate even when infant mortality increases (Chay and Greenstone 2003; Currie and Neidell 2005).

5.2 Heterogeneous Treatment Effects

The average increase in insecticide use is masking considerable heterogeneity between counties, specifically: their baseline use. The rationale for these heterogeneous treatment effects is that farmers in counties that saw the largest declines in biological pest control are likely to be those that will have a higher compensatory response. In addition, counties with low baseline use levels also might have more slack with respect to regulatory constraints regarding insecticide applications.

Because there is a data gap regarding the counties that directly benefit from the pest control that bats provide, I use the amount of insecticide use prior to the emergence of WNS as a proxy. In counties with high provision levels of biological pest control, we would expect to see low levels of baseline use, and vice-versa. I calculate baseline use as the mean insecticide use from 1997 to 2005, and define counties as high or low baseline insecticide use as above or below the national median level. I estimate a single regression where I interact the WNS exposure dummy variable with dummy variables for being either above or below the median baseline level.

Counties that were below the median baseline insecticide use exhibit a stronger response following the emergence of WNS. In Table 1, Panel B, I report the heterogeneous treatment effects post-WNS exposure. Counties that were previously using low levels of insecticides increase their use by 3.62 kg per-squared-km following WNS exposure, relative to 1.61 kg per-squared-km in already high insecticide using counties (column 2). This pattern repeats across all the main outcomes where the effect in the low baseline insecticide use counties is larger by a factor of 2 to 4 than in the high baseline counties (columns 4, 6, and 8).

5.3 Dynamic Treatment Effects

To examine whether the treatment effects of WNS exposure are driven by existing pre-trends in the outcomes of interest, I plot the event study results in Figure 2. I report three sets of results. First, in the left-most column of the figure, I report the results with census region-by-

year fixed effects, and weight the outcomes by either baseline cropland area in 2002 (except for internal IMR), or the number of live births (for internal IMR). For the outcomes that I can observe annually, insecticide use and internal IMR, there does not appear to be a systematic difference between counties that become WNS confirmed to non-WNS counties, however, following WNS exposure, the treated counties diverge from the untreated counties, and the magnitude of the effects increases over time. For the results I can observe during agricultural census years, I interact the census year with the WNS exposure dummy. Crop revenue is not declining in WNS confirmed counties before 2007, but falls sharply in 2012, and remains significantly lower in 2017. Chemical expenditure declines in 2012 and 2017, but by an order of magnitude less than crop revenue.

These dynamic patterns persist when I more flexibly control for time trends by using state-by-year fixed effects (the second column of figures), and as I use population weights across all results (third column of figures). The main exception is that the dynamic pattern for internal IMR is dampened as more of the treatment effect is absorbed by the state-by-year fixed effects. This reflects similar difficulties with estimating dynamic responses in infant health as documented in previous work studying the effects of air pollution exposure (Chay and Greenstone 2003; Currie and Neidell 2005).

The dynamic response for internal IMR is larger, and more precisely estimated, when I exclude the nearest neighbors to the WNS confirmed counties (see Figure A7). This suggests that the internal IMR results are attenuated due to spatial spillovers, which I examine in greater detail in the following section.

5.4 Evaluating Threats to Identification

An important concern is that the fungus could affect agriculture and health through channels not related to bat mortality. I leverage a feature of the empirical setting where not all bat species develop the conditions that lead to WNS, even if the fungus is detected in the county they hibernate in. I do not find evidence for a fungus-presence effect that does not also involve bat mortality from WNS. In Table 2, I re-estimate the post-WNS results when including the WNS suspected counties in the sample (columns 1, 3, 5, and 7). I then estimate a separate effect for the presence of the fungus in the absence of confirmed bats suffering from WNS (columns 2, 4, 6, and 8), and fail to detect an effect in the WNS suspected counties. The somewhat lower point estimates for the average post-WNS effects suggest that some of the WNS suspected counties are also experiencing declines in biological pest control.

Counties that are adjacent to WNS confirmed counties potentially also experience some degree of treatment. If these spillovers result in increased insecticide use then they will

negatively bias, and attenuate the results. I perform two tests to evaluate the impacts of these spillovers. First, I seek to estimate the magnitude of the spillovers themselves. I repeat the main post-WNS estimation, only now I exclude the set of counties that ever become a WNS confirmed county, and define the counties that are adjacent to at least one WNS confirmed county as the set of treated counties. I also estimate the effect of having an increasing share of adjacent counties becoming WNS counties. Specifically, I include two dummies, for the share of surrounding WNS adjacent counties in the interval of 33-66%, and 66-100%, estimated relative to the omitted category of 0-33%. The second test involves removing the potential source of bias by excluding the counties that are potentially experiencing spillover effects, causing a SUTVA violation. Specifically, I exclude the counties that are adjacent to WNS confirmed counties, but never become classified as a WNS confirmed county themselves.

I estimate small, and imprecise spatial spillover effects. The signs of the effects in Table 3, Panel A (columns 1, 3, 5, and 7) is in agreement with the signs of the effects in Table 1, but the magnitudes are smaller. However, in Table 3, Panel B, when estimating the treatment effects for the WNS confirmed counties but when excluding their adjacent counties, the magnitude and precision of the estimates increase (column 1, 3, 5, and 7). The estimates increase in size when the share of neighboring WNS confirmed counties increase (columns 2, 4, 6, and 8), but the effects are not statistically significant from one another. Finally, I verify that clustering at the county level does not systematically underestimate the standard errors by accounting for their potential spatial correlation (see Table A9).

5.5 Quasi-Randomness, Sensitivity to Sample Composition &Misspecification

I provide supporting evidence for the interpretation of the spread of WNS as being quasi-random with respect to the outcomes of interest. In Table A10, I report precisely estimated zeros for the effect of lagged values of the main outcomes on the probability of WNS presence, while spatial proximity appears as the only strong predictor for WNS. To test if the results are driven by outliers, I report jackknifing distributions in Figure A11, which are narrowly centered around the estimates from the main sample. I use permutation inference to test if the DD specification fails to account for important cross-sectional or temporal trends. I re-estimate the post-WNS specification in Equation (1) for each new bootstrap sample, for each outcome, and summarize the results in Figure A12. The estimated effects from the non-bootstrap samples are all in the tails of the bootstrap distributions, resulting in exact p-values well below 5%.

6 Conclusions

In this paper, I demonstrate that farmers compensate for the reduction in biological pest control, provided by bats, by using more insecticides. This validates theoretical predictions in the economic literature regarding substitution patterns when natural inputs decline. The reduction in biological pest control lowers crop revenue, as predicted by ecological theory and by the agronomic literature. I estimate higher infant mortality rates following the increase in pesticide use, consistent with associations made in the epidemiological literature.

Using the main estimates, I evaluate the magnitude of the welfare losses due to the decline in bat populations in the following way. I start with the results on overall chemical expenditure, that show a total decline in expenditures despite the increased use of insecticides. I multiply the mean land area of a county, times the number of treated county-year pairs, times the mean reduction in chemical expenditure of \$410 per-squared-km, which results in a \$1.47 billion lower total chemical expenditure. However, this reduced expenditure, likely a result of shifting resources from herbicide to insecticide applications, also has an effect on total revenue. I repeat the above calculation using the mean loss of \$7,420 per-squared-km in crop revenue, which results in total losses of \$26.6 billion. To anchor these magnitudes, consider that chemical expenditure and crop revenue across all of the U.S. in 2017 alone were around \$17 billion and \$190 billion (2017 dollars), respectively. Finally, for the health impacts, I multiply the total number of live births in the treated counties, after WNS exposure, by the mean effect of 0.33 additional deaths per-1,000 live births, resulting in an increase of 821 infant deaths. Using the EPA's recommended central mortality risk reduction value of \$9.24 million (2017 dollars), this reflects a welfare loss of \$7.6 billion. Combining these estimates results in a total net loss of \$31.85 billion, or \$2.7 billion per-year, during 2006-2017, due to agricultural losses and damages to infant health.

The findings in this paper highlight two policy topics: conservation of bats, and monitoring agrochemical pollution levels. If the costs of conserving bat populations are low and the damages from the pesticides used to substitute for their pest control are high, then it is potentially efficient and welfare-improving to preserve bat populations. Increasing the capacity to monitor chemical pollutants would allow for more research on the health effects of pesticide exposure. Currently, pesticides are regulated individually, meaning that the effects of various permutations are not tested. As a result, we remain with observational data to alert us regarding the effects of chemical mixtures.

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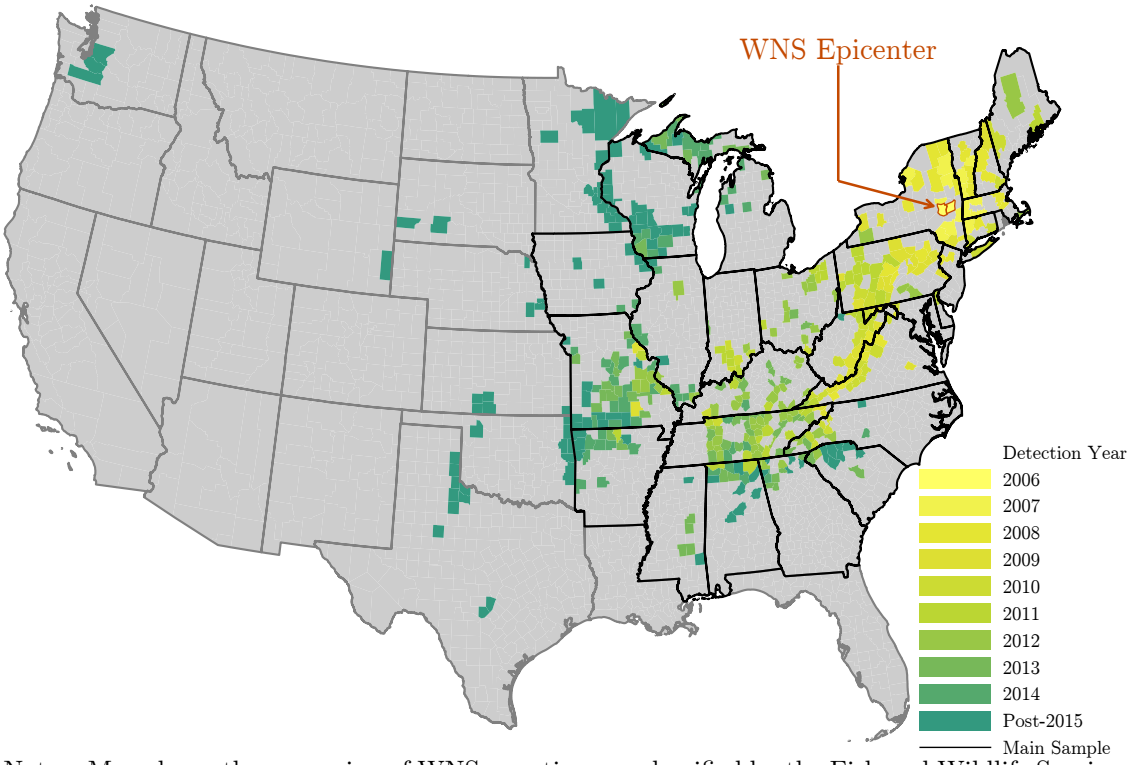
Figure 1: Expansion of White Nose Syndrome Across the United States



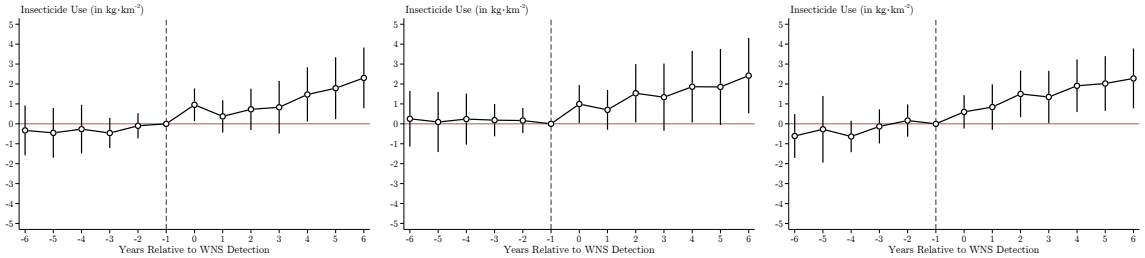
Notes: Map shows the expansion of WNS over time, as classified by the Fish and Wildlife Service, with the two first detections highlighted as the WNS epicenter. The solid black lines highlight the states in the main estimation sample, based on having a county with a WNS status by the end of 2014.

Figure 2: Dynamic Effects of WNS Exposure

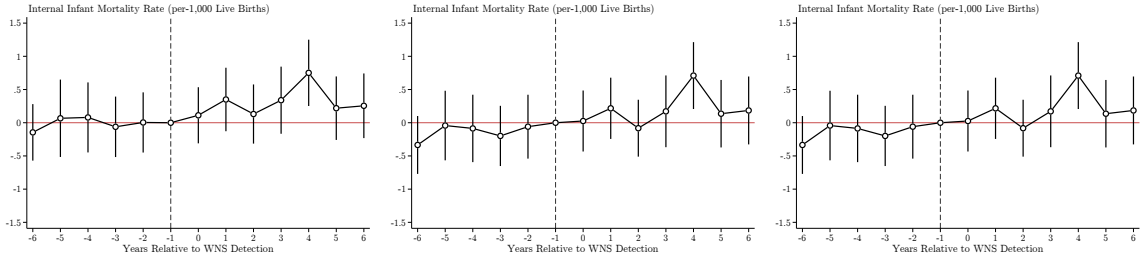
Census Region-by-Year FEs	State-by-Year FEs	
Weighted by either Baseline Cropland or Live Births		Population-Weighted

Outcomes Observed Annually

(a) Insecticide Use (b) Insecticide Use (c) Insecticide Use

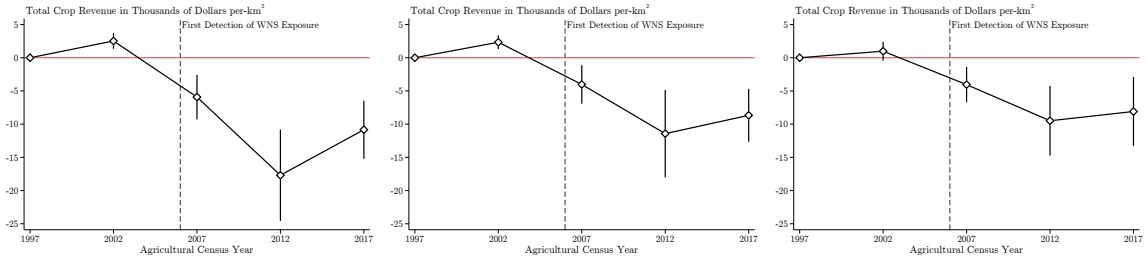


(d) Internal IMR (e) Internal IMR (f) Internal IMR

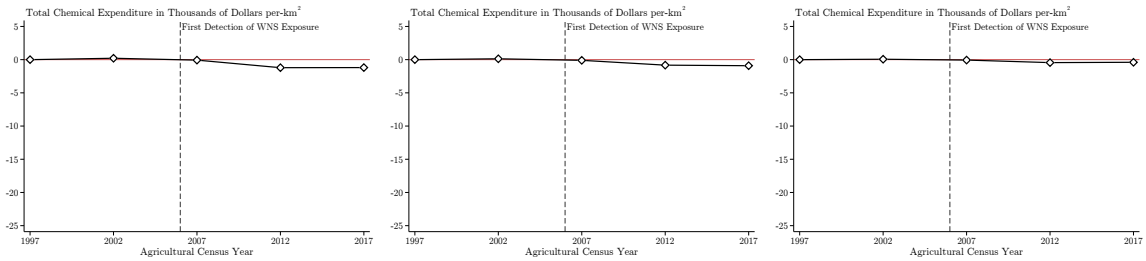


Outcomes Observed In Five-Year Intervals

(g) Crop Revenue (h) Crop Revenue (i) Crop Revenue



(j) Chemical Expenditure (k) Chemical Expenditure (l) Chemical Expenditure



Notes: (a-f) Estimated coefficients and 95% CIs from the specification in Equation (2). Top and bottom coded event-time coefficients are not reported. (g-l) Estimated coefficients and 95% CIs from interacting a year dummy with the WNS exposure dummy in Equation (1). In the first column of results, each regression includes county and census region-by-year fixed effects, and observations are weighted by cropland acres in 2002, except for internal IMR, which is weighted by live births. Results in the second column use state-by-year fixed effects, with the same weighting scheme as in the first column. In the third column, all regressions are population-weighted. Standard errors are clustered at the county level.

Table 1
Average & Heterogeneous Treatment Effects Post-WNS Exposure

	Insecticide Use		Crop Revenue		Chemical Expenditure		Internal IMR	
Panel A. Average Treatment Effects								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS	2.24	2.59	-8.25	-7.42	-0.72	-0.41	0.48	0.33
	(0.49)	(0.66)	(1.08)	(2.15)	(0.09)	(0.13)	(0.20)	(0.11)
R^2	0.53	0.58	0.93	0.91	0.92	0.90	0.15	0.33
Panel B. Heterogeneous Treatment Effects								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS×Low Baseline	3.80	3.62	-12.02	-13.11	-1.03	-0.77	0.50	0.42
	(0.46)	(0.53)	(1.03)	(2.45)	(0.09)	(0.15)	(0.28)	(0.14)
WNS×High Baseline	0.03	1.61	-3.32	-2.15	-0.34	-0.08	0.44	0.24
	(0.68)	(0.97)	(1.64)	(2.38)	(0.12)	(0.14)	(0.21)	(0.12)
R^2	0.53	0.58	0.93	0.92	0.92	0.91	0.15	0.33
County FEs	X	X	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X	X	X
Weighted	E	P	E	P	E	P	E	P
Dep. Var. Mean	9.28	7.50	28.73	27.53	2.45	1.81	6.77	6.56
N	33,474	33,474	7,350	7,350	7,590	7,590	33,474	33,474
Clusters	1,594	1,594	1,470	1,470	1,518	1,518	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are weighted equally (E), or by the total population in the county-year observation (P), using estimated population sizes as reported by the Census Bureau. The reported mean for the dependent variable is the weighted mean. Insecticide use, crop revenue, and chemical expenditure are normalized by county area, and are measured in either kg·km^{-2} (insecticide use), or thousands of dollars per-km^{-2} (revenue and expenditure). Internal infant mortality rate (IMR) is meausred in deaths per-1,000 live births. Panel A reports average post-WNS exposure effects. Panel B interacts the WNS exposure dummy with a dummy for being below or above the median insecticide use in the pre-exposure period of 1997-2005, which acts as a proxy for higher baseline levels of biological pest control. Standard errors are clustered at the county level.

Table 2
Falsification Test Using WNS Confirmed & Suspected Counties DD Estimates

	Insecticide Use		Crop Revenue		Chemical Expenditure		Internal IMR	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS Confirmed	1.69	2.20	-5.54	-6.37	-0.33	-0.38	0.23	0.24
	(0.58)	(0.58)	(1.68)	(1.84)	(0.10)	(0.11)	(0.09)	(0.10)
WNS Suspected		-2.41		3.43		0.22		-0.06
		(0.65)		(1.58)		(0.16)		(0.14)
R^2	0.58	0.58	0.92	0.92	0.90	0.90	0.33	0.33
County FEs	X	X	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X	X	X
Weighted	P	P	P	P	P	P	P	P
Dep. Var. Mean	7.46	7.46	27.04	27.04	1.77	1.77	6.46	6.46
N	36,666	36,666	8,050	8,050	8,290	8,290	36,666	36,666
Clusters	1,746	1,746	1,610	1,610	1,658	1,658	1,746	1,746

Notes: Estimation results for the specification similar to that in Equation (1). The sample now also includes counties that are only WNS suspected, where the fungus has been detected but there have not been sufficient detections of bats developing symptoms to classify the county as WNS confirmed. Insecticide use, crop revenue, and chemical expenditure are normalized by county area, and are measured in either $kg \cdot km^{-2}$ (insecticide use), or thousands of dollars per-km^{-2} (revenue and expenditure). Internal infant mortality rate (IMR) is meausred in deaths per-1,000 live births. Observations are population-weighted. The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table 3
Evaluating the Scope of Attenutation From Spatial Spillovers

	Insecticide Use		Crop Revenue		Chemical Expenditure		Internal IMR	
Panel A. Effects on Adjacent Counties When Excluding WNS Counties								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS^{N1}	0.29		-1.24		-0.09		0.27	
	(1.35)		(1.89)		(0.14)		(0.11)	
Surr. WNS [33%,66%)		0.40		-3.29		-0.05		0.11
		(1.11)		(2.23)		(0.15)		(0.17)
Surr. WNS [66%,100%]		1.01		-13.62		-0.06		0.09
		(2.49)		(5.96)		(0.44)		(0.32)
R^2	0.59	0.59	0.92	0.92	0.90	0.90	0.34	0.34
Dep. Var. Mean	8.16	8.16	29.51	29.51	1.98	1.98	6.64	6.64
N	28,329	28,329	6,205	6,205	6,440	6,440	28,329	28,329
Clusters	1,349	1,349	1,241	1,241	1,288	1,288	1,349	1,349
Panel B. Excluding Counties Adjacent to WNS Counties								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS	3.52		-8.53		-0.50		0.63	
	(1.18)		(2.37)		(0.16)		(0.14)	
Surr. WNS [33%,66%)		2.00		-8.67		-0.56		0.41
		(0.76)		(2.77)		(0.17)		(0.14)
Surr. WNS [66%,100%]		2.04		-10.13		-0.62		0.57
		(0.94)		(2.98)		(0.19)		(0.26)
R^2	0.57	0.57	0.91	0.91	0.91	0.91	0.34	0.34
Dep. Var. Mean	7.88	7.88	27.39	27.39	1.88	1.88	6.76	6.76
N	24,885	24,885	5,430	5,430	5,640	5,640	24,885	24,885
Clusters	1,185	1,185	1,086	1,086	1,128	1,128	1,185	1,185
County FEs	X	X	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X	X	X
Weighted	P	P	P	P	P	P	P	P

Notes: Estimation results for the specification similar to that in Equation (1). In Panel A, I exclude the WNS confirmed counties, and define the non-WNS counties that share a border with the WNS counties, as the treated group. In Panel B, I include the WNS-confirmed counties excluded in Panel A, but exclude the non-WNS counties that share a border with the WNS confirmed counties. I estimate both an average effect for a county being adjacent to a WNS county (Panel A), or the county being classified as a WNS county (Panel B), as well as treatment intensity effects where larger shares of neigboring counties get classified as WNS counties. All observations are population-weighted. Standard errors are clustered at the county level.

Online Appendix

A Additional Results

A.1 Secular Trends in Main Outcomes

In addition to the summary statistics reported in Table A1, I provide visual summaries for the variation over time in the four main outcome variables in Figure A1. In Figure A1a, I plot the mean pesticide amount used across all counties each year, by pesticide class, scaled to 1997. With the greater availability and adoption of genetically modified herbicide-resistant crop seeds, the use of herbicides has gone up. Conversely, new genetically modified seed varieties include insecticide and fungicide properties, lowering the use of those pesticide classes. Figure A1b shows growth in overall crop revenues in real terms since 1997, albeit a lower amount in 2017 relative to 2012. Similarly, chemical expenditure (Figure A1c) has also gone up, and was much higher in 2017 and 2012, relative to previous years. Infant mortality rates have been declining from a level of 7.7 deaths per-1,000 live births in 1997, to 6.6 deaths per-1,000 live births in 2017. However, as Figure A1d shows, there is considerable variation between states. Table A1 reports summary statistics for the main outcomes and additional variables used in the analysis.

A.2 Evidence on Potential Exposure Channels

There is limited data on the concentration of pesticides in water and air, even though research has shown that both wind and surface runoff erode pesticide from target fields, potentially generating off-farm exposure. Here I use data from the USGS, for the period of 1997 to 2017, on detections of different compounds in water samples. I focus on the insecticide compounds that reflect at least 2% and above of overall detections.[7]

Because stations enter and exit the sample frequently, I cannot construct a representative balacned set of monitoring stations to test if concentrations increase following WNS exposure. However, as the simple descriptive summary in Figure A2 shows, the number of insecticide detections in water samples starts to increase in April, remains high throughout May to August, and starts to decline in September. This is consistent with the timing of the main agricultural growing season of April through September, and demonstrates that insecticide applications make their way into waterways. The descriptive results in Figure A2 are only suggestive of there being an exposure pathway through drinking water, yet they

[7] Results are fairly insensitive to the choice of this cutoff.

serve as important evidence of there being elevated levels of insecticides during the pesticide application season.

A.3 Evaluating Potential Earlier WNS Exposure

In order to correctly estimate the impacts of declining biological pest control both the status and timing of its change need to be precisely measured. Because the U.S. Fish and Wildlife Service coordinates survey efforts between state and federal agencies under the White Nose Syndrome Response Team, there is a lower risk of failing to detect a county with WNS. Efforts to detect include surveys in caves and mines, where bats hibernate, as well as randomly testing subsets of bats that are collected for rabies testing (Griggs et al. 2012). Another potential concern is with the measurement accuracy of treatment onset. In order for a county to be classified as WNS confirmed, several bat samples need to be tested positive for the fungus in order to not mistakenly classify a county due to an infected bat flying over from another county.

As recent research has documented, classification of WNS status lags by at least one year following the appearance of symptoms (Verant et al. 2018). In the case of counties that transitioned from a WNS suspected to confirmed status, the average lag is two years (see Figure A3a). In fact, most counties report cases of dead bats due to WNS a year before their formal classification (see Figure A3b). This means that WNS could be having an effect well before the formal WNS determination by the FWS.

To account for the potential lag in WNS classification, I uniformly assign each county's detection date as two years prior to the formal classification. This change only affects the re-centering around WNS exposure, but does not change the assignment of treatment and control counties. This is consistent with documented delays in the classification of counties as WNS confirmed, as in Verant et al. (2018), as well as state wildlife agencies that in some cases consider true exposure to have started even 4 years prior to the official determination.[8]

Here, I use the data that is publicly available for 25 counties that are now considered as WNS confirmed, but were previously classified as WNS suspected. That is, there was a delay between the time of the official detection of the fungus, and a sufficient number of bat detections with WNS. In Figure A3a, I summarize the difference between the fungus and WNS detection years for those 25 counties. On average, these counties potentially experienced a delay of 1.8 years, with a standard deviation of 1.1 years. I also verify that these delays appear in the USGS Wildlife Health Information Sharing Partnership Event Reporting System (WHISPers). In Figure A3b, I plot the difference in the earliest bat

[8] Unfortunately, I cannot share the data or summary results on the timing of WNS exposure according to state agencies until the Fish and Wildlife Service makes it publicly available.

mortality event attributed to WNS that appears in WHISPers, to the year of confirmed WNS status. Around 90% of the 205 counties have a WNS event report in WHISPers 1 year before they are classified with a confirmed WNS status.

A.4 Detailed Results for Insecticides, Fungicides & Herbicides

With a decline in biological pest control, farmers are most likely to increase their use of Insecticides. However, fungicides are highly correlated with insecticide use because higher pest pressure results in higher transmission rates of fungi. Agronomic literature has documented that with higher insect pest pressures, plants and crops are more susceptible to damages from fungi. There are two main mechanisms that explain the connection between insects and fungi spread in crops. First, as insects damage the plant, they open more pathways for fungi to spread, and weaken the defenses of the plant. Second, as insects move across plants they carry and disperse fungal spores (Fennell et al. 1975; Kluth et al. 2002). These mechanisms are consistent with a high correlation between insecticide and fungicide use in the data of 0.87. Conversely, herbicide use is not directly linked to either insecticide or fungicide use, consistent with a lower correlation between insecticide and herbicide use of 0.31.

In Table A3, I report the results for fungicide use, that confirm that insecticide use and fungicide use covary. In contrast, results for herbicide use, Table A4, do not show similar increases. In fact, herbicide use declines in larger quantities, but also has a baseline use level that is an order of magnitude larger relative to insecticides and fungicides.

A.5 Including All WNS Confirmed Counties

In the main analysis, I exclude the counties that were exposed after 2014 in order to maintain a balanced composition in each event-time bin. In Figure A4, I use all the counties that were exposed up to 2017. This uses all the data, on all the WNS confirmed and non-WNS counties, in states with WNS exposure up to 2014. However, each event-time dummy has a somewhat different composition now. This is not a threat to the identification of the effect, but it complicates its interpretation. Results are similar in their magnitude and precision to those reported in the main text.

A.6 Limited Data Availability on Crop Yields

Data availability issues present a challenge for the analysis of agricultural productivity. Repeated measurements of crop yields across a balanced set of multiple counties are largely available in intervals of 5 years as part of the agricultural census. While there are annual

survey records on crop yields, they are available for only a few of the crops, and are only reported every year for a subset of the counties.

Average crop yields after WNS exposure summarize the net effect of different responses by farmers and changes to biological pest control conditions. If farmers are able to substitute most of the lost biological pest control, then any negative effect on yields might only be temporary. Unfortunately, even for the two most surveyed crops, corn and soybeans, less than a third of the 1,594 counties included in the main sample have data on yields reported every year.[9] These data gaps make it hard to draw strong conclusions regarding how yields change following WNS exposure.

Agricultural productivity, as measured in crop yields, declines for some crops, but not others, following exposure to WNS. The results are often imprecise and are sensitive to the set of sample weights, and inclusion or exclusion of weather controls. Consequently, I report the distribution of 12 different estimated coefficients for each crop in Figure A5. Specifically, I estimate effects for a set of crops with sufficiently large sample sizes and representation throughout the states in the sample. On average, 4 of the 7 crop experience a decline in yields, but are small in magnitude. The most negatively hit crop is tobacco, with an average 6.8% decline in yield.

A.7 Detailed Results for All-Cause, Internal-Causes & External Causes of Infant Mortality

In Table A5, I summarize the average effects post-WNS exposure. On average, internal IMR increases by 0.33 deaths per-1,000 live births, reflecting an increase of 5% relative to the mean (Panel A, column 6). These results are robust to the inclusion of both weather controls, and time varying population shares.[10] The increase in infant mortality is larger and more precisely estimated in the counties that were below the median insecticide use at baseline (Table A5, Panel B.).

In Tables A6 and A7, I report the estimates for infant mortality from both internal and external causes (all-cause mortality), and separately for external causes, respectively. The results confirm that the effect on infant mortality rates is observed even when lumping together the full data on infant deaths. This means that the observed increase in interanl IMR is not an artifact of a systematic measurement error – correlated with the spread of WNS – regarding the cause of death. More importantly, there is no average effect on infant mortality due to external causes of death, which should not respond to an increased

[9] Specifically, there are only 499 and 514 counties with balanced data for corn and soybeans, respectively.
[10] In intervals of 5 years, starting from age group 0 to 4, up to 80 to 84, with 85 and above as the omitted category.

insecticide use following WNS exposure.

A.8 Estimates for Additional Health Outcomes

In Table A8, I report the estimated effects for mean birth weight, mean gestation length, mean APGAR5 score, the share of births classified as low birth weight, and the share of births classified as premature births. None of these outcomes exhibit a meaningful change. In fact, given the magnitude of the coefficients, standard errors, and the means of each outcome, I can reject that there are any large effects on one of these additional outcomes. As previous work on the effects of pollution exposure on birth outcomes has documented, these outcomes do not necessarily deteriorate even when infant mortality increases (Chay and Greenstone 2003; Currie and Neidell 2005).

In addition for evaluating the effects on weight and gestation, I also estimate whether live births were classified as going below established thresholds. Results for an increase in the share of low weight births are positive, yet imprecise, and are an order of magnitude lower than previous estimates using the opening of industrial plants (Currie et al. 2015). Results on the share of premature births are positive and imprecise.

A.9 Estimating Changes in Mothers' Characteristics

I use the data in the birth certificates on the age category, educational attainment category, share of mothers who are white, married, the mean number of prenatal care visits, and the share of mothers that were smoking for at least some part of the pregnancy. The results in Figure A6 show extremely small, often very close to zero, differences. I also report the mean of each outcome to make it easy to verify that the reported differences can be interpreted as precise zeros.

A.10 Examining the Impacts of Spatial Spillovers on Internal IMR Attenuation

In the main text, the results for the effect of WNS on internal IMR almost double in size, from 0.33 to 0.63 deaths per-1,000 live births (Table 1, column 8 and Table 3, column 7, respectively). The key difference between the two results is the exclusion of the first degree neighbors (nearest neighbors) to the WNS confirmed counties – those that are most likely to experience spatial spillovers through wind and water erosion channels.

This suggests that, to some degree, the dampening in the effect in the event-study results for internal IMR in Figure 2 is due to spatial spillover attenuating the effect of the increased

insecticide use following WNS exposure. To further establish that this attenuation is masking the dynamic effect of WNS exposure, I repeat the estimation of the event-study while excluding the first degree neighbors to the WNS confirmed counties and report the results in Figure A7.

The results show internal IMR increasing from about 0.2 to 0.4 deaths per-1,000 live births after exposure to WNS, while there is no systematic difference between exposed and non-exposed counties prior to WNS exposure. This result supports the finding that internal IMR increases in counties that experience bat die-offs.

A.11 Evaluating Spatial Correlation of the Standard Errors

I evaluate the degree of spatial clustering on the precision of the estimates in Table A9. In the first row, I report the coefficients for the four main outcomes: insecticide use, crop revenue, chemical expenditure, and internal IMR. For ease of comparison, I report the standard errors from clustering at the county level, as I do throughout the analysis. I then present the standard errors when clustering at levels above the level of the county. I use the agricultural statistics districts, which divides the state into agriculturally homogeneous areas, or I divide the state by its ecological regions (level III), which creates ecologically homogeneous areas. For more details on these sub-units, see the subsection below on Leave-One-Out Estimation Results. For insecticide use and crop revenue, standard errors increase when clustering at levels above the county level, yet they remain nearly identical for the outcome of internal IMR. Results remain precisely estimated at these two higher levels of clustering.

To allow spatial clustering to not end abruptly at the border of the state, I also use a spatial HAC as suggested by Conley (1999) and Conley and Taber (2011). I use the centroid coordinates for each county to define the distance bandwidths, and allow the standard errors to be flexibly correlated up to, but not above, a certain distance threshold. I report results for 50, 100, 250, 500, 750, 1000, and 2000 km. The standard errors for insecticide use and crop revenue are mostly below or close to their estimated standard error when clustering at the county level. However, for short distance threshold, the standard error for internal IMR nearly doubles, lowering the precision of the estimate to the 10% level. Overall, these results demonstrate that the results remain fairly robust to different forms of spatial clustering, and the clustering at the county level does not systematically underestimate them.

A.12 Supporting Evidence for the Quasi-Randomness of WNS Expansion

In Table A10, I estimate a linear probability model for the binary outcome of WNS exposure. I use lagged values of either the outcomes, pesticide use or internal infant mortality, or the lagged WNS status of neighboring counties. In column 1, I include the value of each pesticide type, lagged by 1 period. The coefficients and standard errors are small enough to reject any meaningful impacts due to previous pesticide use. The same holds true for the lagged value of internal IMR in column 2. I do not include crop revenue and chemical expenditure, as those are not available at an annual resolution.

While lagged outcomes fail to predict WNS status, spatial proximity to other WNS counties acts as a strong predictor. In column 3, I regress the WNS status of county c in period t, on a dummy variable that is equal to 1 if any of the adjacent, first degree, neighbors of the county, N1, were WNS confirmed in the previous period, $t-1$. Having a first degree neighbor with WNS in the previous period, precisely increases the probability of being classified with WNS by 0.24 percentage points. In column 4, I expand the lag structure to include the WNS status of the adjacent county in the last three periods. Having an adjacent county that has been previously classified as a WNS confirmed county remains a strong predictor. The effect is mostly driven by the adjacent counties, as seen in column 5 where I include up to three neighboring degrees. Having a first degree neighbor with WNS is twice as likely to result in WNS detection than having a second degree county with WNS. A third degree neighbor has an imprecise and negative effect on the probability, which is an order of magnitude lower than the effect of a first degree neighbor. This patterns persist even when running a regression with lagged pesticide use, internal IMR, and the lagged WNS status of neighboring counties.

A.13 Evaluating Potential Estimation Issues in Staggered DD

Recent work on the estimation of staggered difference-in-differences (SDDs) designs has revealed potential issues that can arise when estimating a post-treatment average effect using a two-way-fixed-effects (TWFE) estimator. However, the potential problems are solely due to the implementation of the estimation, and not a flaw in the research design itself (Borusyak et al. 2021; Callaway and Sant'Anna 2020; Chaisemartin and D'Haultfoeuille 2020; Goodman-Bacon 2021; Sun and Abraham 2020).[11] See Baker et al. (2021) for a recent review of the estimation issues, and a comparison of the suggested alternative estimation

[11] These papers focus on settings where there are a set of never-treated units, as in the empirical setting I study in this paper.

techniques.

The main intuition around the estimation of an SDD using TWFE is that units that switch treatment status are compared both to non-treated units, but also to previously treated units. In the presence of heterogeneous treatment effects across units, or dynamic treatment effects, comparison of treatment switchers and previously treated units violates the parallel trends assumption. As the TWFE recovers a weighted average of the possible DD comparisons, those weights have undesired properties, and can even be negative.

I begin with using the diagnostic DD-decomposition developed in Goodman-Bacon (2021). In Figure A8, I present the different 2X2 DD estimates from the possible comparisons of treated to never-treated units, and timing-groups that compare early to late, or late to early, treated units. Because the majority of the potential issues are related to having multiple treatment cohorts, and multiple timing-groups comparisons, I focus on the two outcomes that I observe annually: insecticide use and internal IMR. I plot decompositions that use different weights, and include in each plot lines that highlight the ATE recovered by the TWFE estimator, as well as the weighted average of the treated to never-treated comparisons.[12]

The DD-decomposition reveals two key insights. First, most of the weight is assigned to the DD comparisons of treated to never-treated units (the "good" comparisons), and very little is assigned to the timing-groups (the potentially "bad" comparisons). This is due to the fact that in this setting, there are many never-treated units (Borusyak et al. 2021). The second important observation is that as a result of the first one, there is very little difference between the weighted TWFE estimate and the re-weighted ATE that uses only the "good" comparisons. Overall, the results from the DD-decomposition greatly reduce the concerns regarding the use of the TWFE estimator in order to obtain average post-WNS effect estimates.

Following Bertrand et al. (2004), who suggest collapsing the data into pre- and post-period and run a two period DD regression, I implement a similar approach using long differences, as in Burke and Emerick (2016). I use the same composition of counties as in the main estimation reported in the paper, and compare them in two time periods, 2002 to 2004 and 2015 to 2017, before and after they were exposed to WNS.[13] I choose those specific years because they are represent the time right before any indication of WNS in the U.S. and right after the final county in my treatment group phases into treatment. Comparing the WNS confirmed counties to non-WNS counties recovers a weighted average of the dynamic treatment effects, as some counties are treated for over a decade and some for only a few

[12] I use the original weights as assigned by the TWFE estimator, and re-scale them such that they sum up to 1.

[13] For crop revenue, as data are only available in 5 year intervals, this effectively collapses to comparing 2017 to 2002.

years by 2017. The important feature is that in this long-differences post-WNS estimation, I avoid having comparisons between early and late, or late and early, treated units affect the estimate for WNS exposure.

In Table A11, I report the results from this pre- and post-collapse procedure. Overall, coefficients have similar magnitudes, if not larger than their comparable coefficients in Table A2. Because the goal of this test is to provide a simple evaluation to whether the issues of negative weights arise in the full sample DD estimation, the emphasis here is on the coefficients, and less so on their precision, especially when using only two time periods.

Finally, to resolve any potential standing issues with the differential timing of treatment, I also re-estimate the results for insecticide use and internal IMR, using one of the newly developed estimators. Specifically, I use the cohort-weighted estimator developed by Sun and Abraham (2020). The estimator runs a regression where the sets of leads and lags are interacted with a cohort dummy. This effectively estimates a separate dynamic treatment effect path for each cohort. The different leads and lags are then weighted, according to the share of each cohort in the panel, to estimate the average dynamic treatment effect path across cohorts. In Figures A9 and A10, I report the results from the TWFE estimator in the left panel, and the results from the Sun and Abraham (2020) estimator in the right panel. For both outcomes, the results are nearly identical.

To conclude, different diagnostic tools and alternative estimation techniques to the standard TWFE estimator recover very similar estimates for the treatment effects. It appears that in this setting, due to the large number of never-treated units, the staggered timing of treatment does not present a major issue for the estimation of the effects.

A.14 Leave-One-Out Estimation Results

I also test the robustness of the results by excluding one unit from the analysis. I either exclude one county, or a set of counties by excluding one agricultural statistics district, or one ecological region at a time. To obtain the above county classification, I match each county to two sets of groups. First, the Agricultural Statistics Districts (ASDs), as classified by the USDA (U.S. Department of Agriculture 2012). Second, the ecological region the county resides in, as classified by the EPA (U.S. Environmental Protection Agency 2013).

The USDA groups counties into ASDs within each state based on crop composition, environmental conditions, and other factors that help construct a set of counties that are more agriculturally homogeneous. This subdivision of states creates between 1 to 9 ASDs, depending on the size and variation in each state. The EPA defines ecological regions as areas with similar biotic and abiotic components, and how self-contained and connected they

are to nearby regions. I use the third level, out of four, which allows to classify counties by their prevalent ecological region.[14] The main sample of 27 states has 164 ASDs, and 37 ecological regions.

For each one of the excluded unit levels, I re-estimate the specification in Equation (1). I summarize the results in Figures A11a, A11b, and A11d. The distribution of estimates for each outcome is centered around the estimate reported in the main text, with a small dispersion around that center. This helps to rule out that the results are mostly driven by the presence of outliers in the sample.

A.15 Permutation Inference

To further rule out any spurious effects I use Permutation Inference methods (Fisher 1966; Bertrand et al. 2004; Young 2019). I randomly assign treatment status as it is recorded in main sample either by: (i) fully randomizing treatment across counties and year (full randomization), (ii) randomizing the treatment across counties but maintaining the time series component of the treatment (block randomization), and (iii) randomizing only the time series component of the treatment within each treated county (within randomization).[15] In Figure A12, I report the results for the three types of permutation tests for the main outcomes I analyze in the main text. For each outcome, the effect estimated in the observed sample is in the right-tail of all the distributions generated from the permutation procedures, and all distributions are centered around zero.

A.16 Results Using a Strictly Balanced Panel

In the main estimation results, Table 1, I report results for samples that are fully balanced and have the same number of observations and clusters for pesticide use, as well as internal IMR. However, when examining crop sales or chemical expenditures the number of clusters declines because fewer counties are fully balanced across the 5 agricultural censuses. I verify that the changes in composition do no have a meaningful effect on the results by constraining all outcomes to the same strictly balanced sample containing the same counties, and report those results in Table A12.

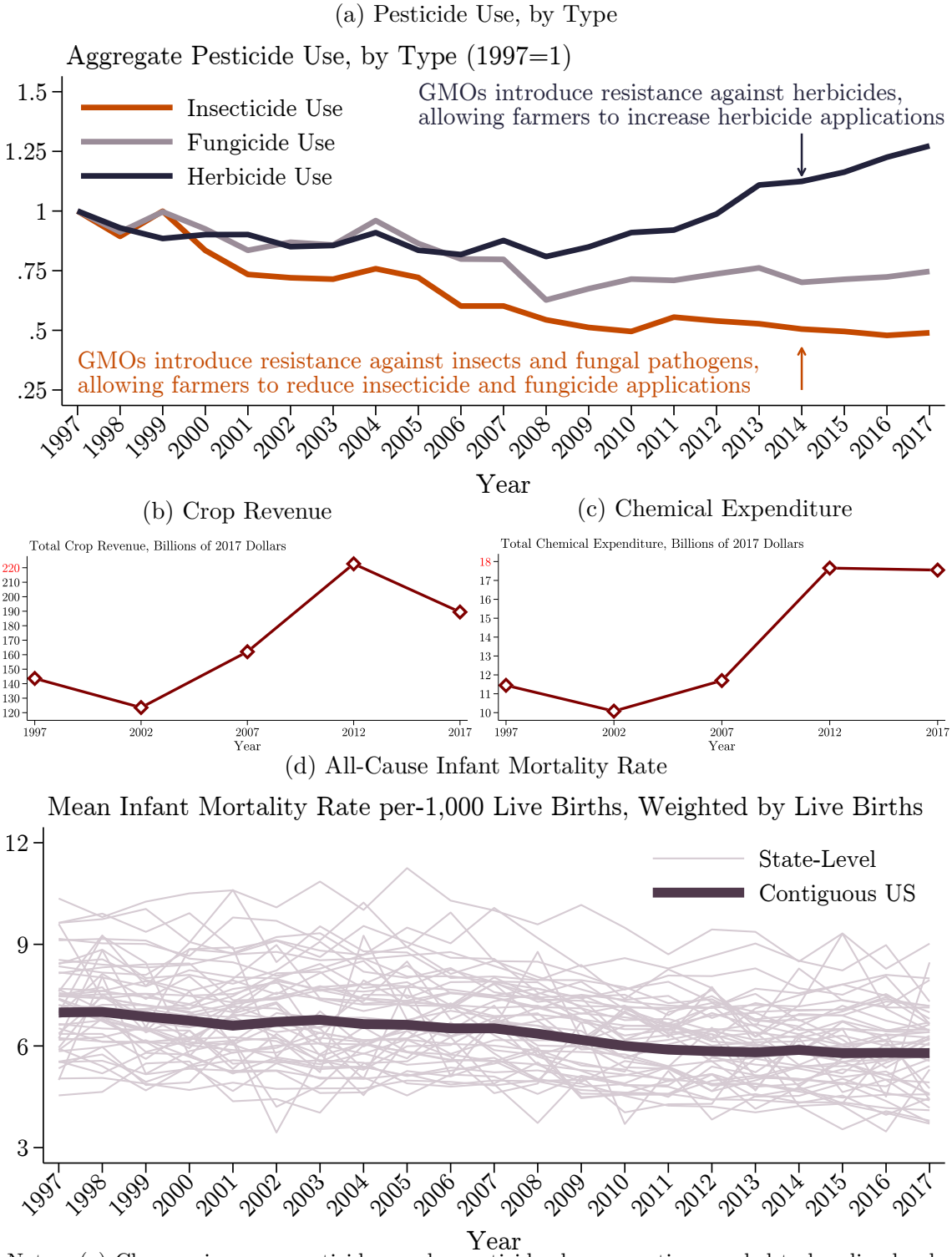
[14] Detailed map is available on: ftp://newftp.epa.gov/EPADataCommons/ORD/Ecoregions/us/Eco_Level_III_US.pdf

[15] Block randomization tests for any time trends that are not properly accounted for, and within randomization tests for any cross sectional differences that are not controlled for.

A.17 Centroid Distance Between Counties

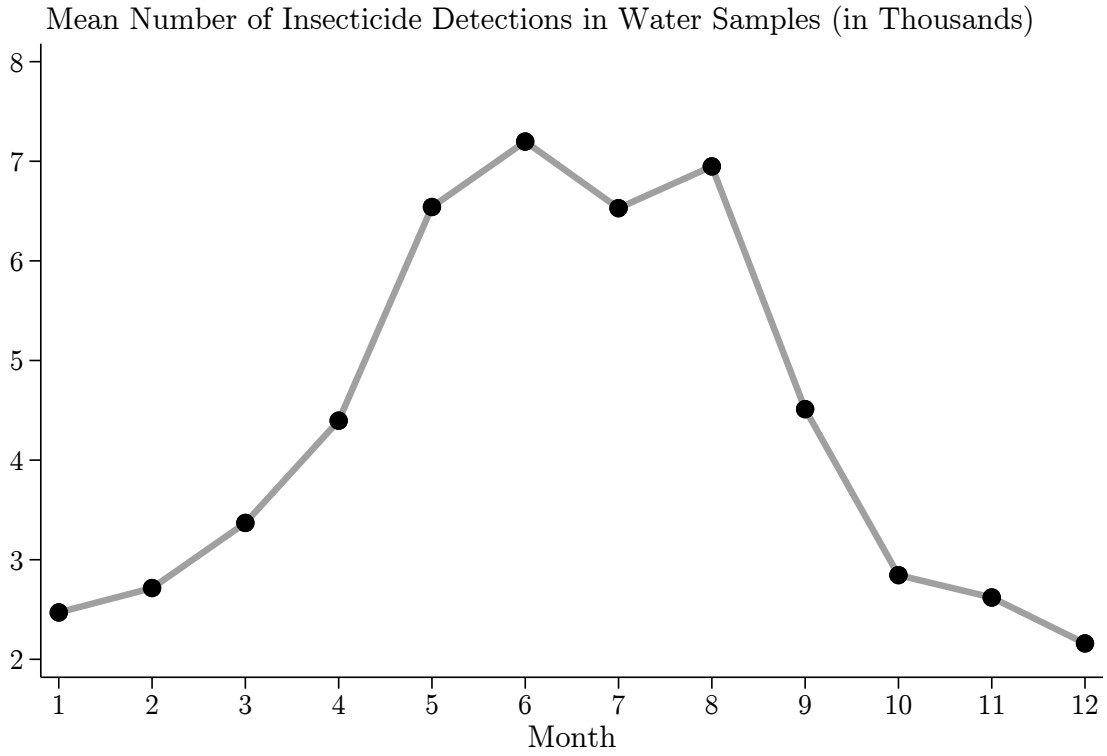
In the analysis, I use the share of adjacent counties that are also WNS confirmed counties. I focus on the adjacent counties because bats are considered to have a maximum nightly flight radius of 50 km, which is close to the highest distance between the centroids of adjacent counties. I summarize the centroid distance between first (adjacent), and second degree neighboring counties in Figure A13.

Figure A1: Secular Trends For Main Outcome Variables



Notes: (a) Changes in mean pesticide use, by pesticide class, over time, scaled to baseline levels in 1997. (b) Change in total crop revenue as measured in demi-decadal agricultural census. (c) Change in total chemical expenditure as measured in demi-decadal agricultural census. (d) Infant mortality rates, from all causes of death, by state or for the contiguous U.S., weighted by live births.

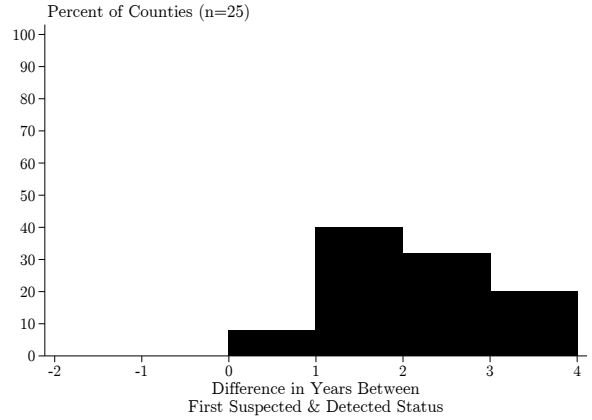
Figure A2: Detections of Insecticides in Water Samples, by Month



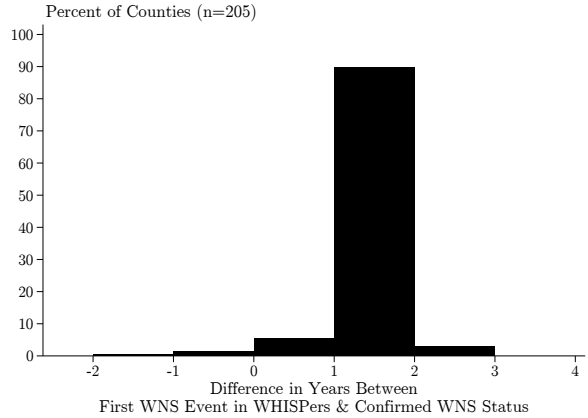
Notes: Data on the number of water samples that had detectable levels of one of the following insecticide compounds: Azinphos-methyl, Carbaryl, Carbofuran, Chlorpyrifos, Diazinon, Disulfoton, Fipronil, Fonofos, Lindane, Malathion, Methyl parathion, Parathion, Phorate, Propargite, Terbufos.
Source: Water quality data from USGS.

Figure A3: Evidence of Delays in WNS Detections

(a) Distribution of Differences Between WNS Confirmed and Suspected Determinations



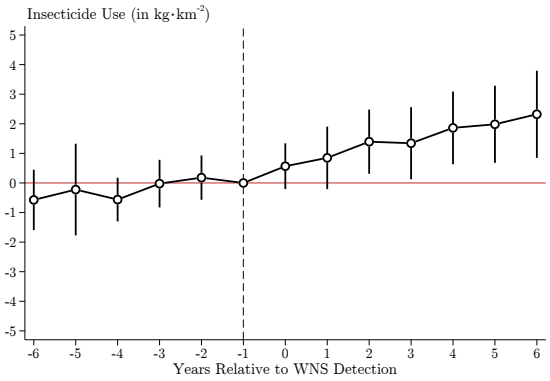
(b) Distribution of Differences Between WNS Cases in WHISPers and Confirmed Detections



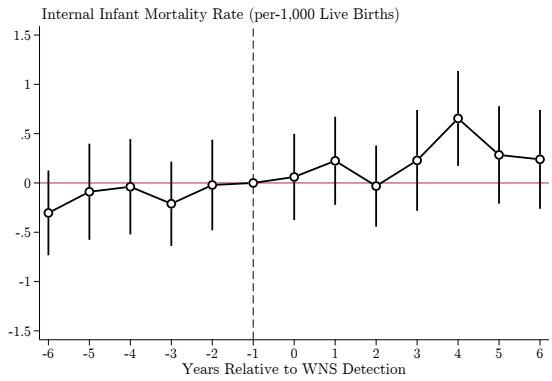
Notes: Distribution of delayed WNS classification, in years, for the (a) 25 counties that have public data on WNS suspected and confirmed determinations, and (b) the 205 counties that have reported mortality of bats due to WNS in the USGS WHISPER system.

Figure A4: Effects on Insecticide Use and Internal IMR Following WNS Exposure, All WNS Confirmed Counties

(a) Insecticide Use

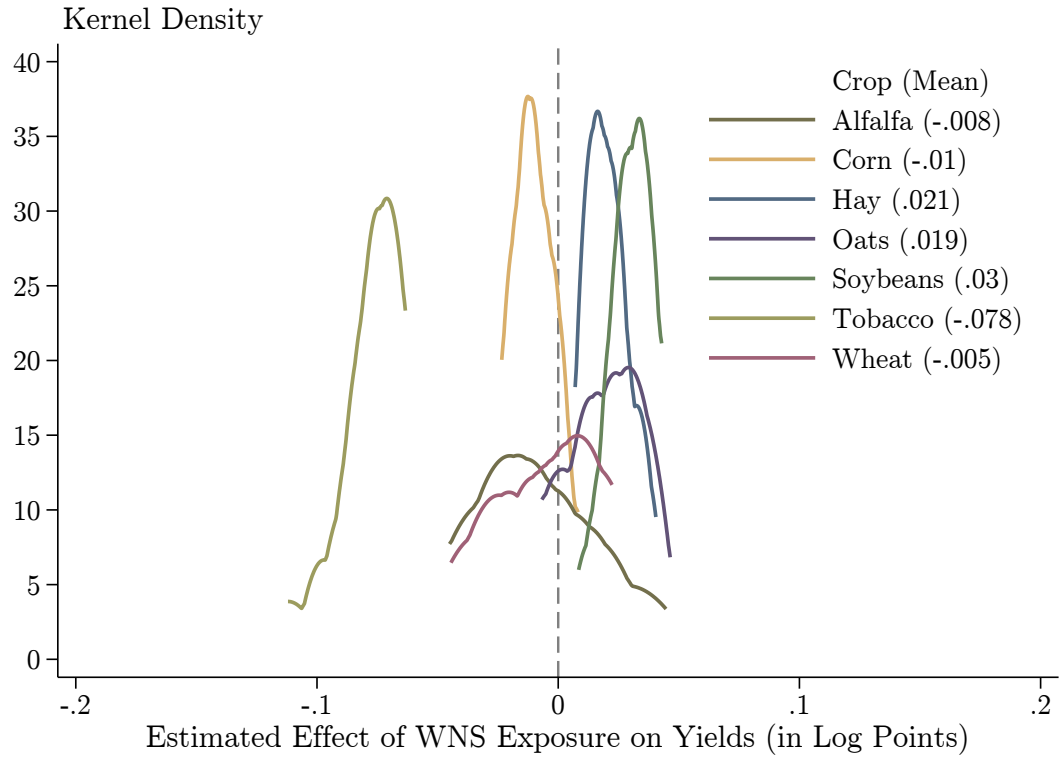


(b) Internal IMR



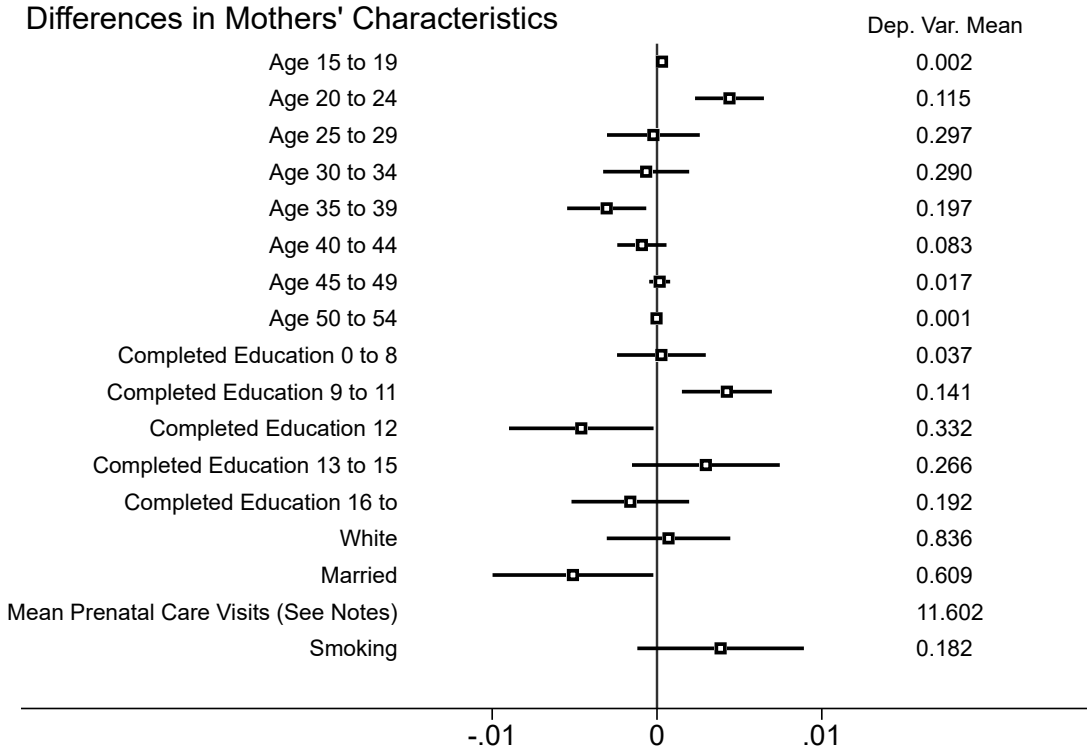
Notes: Estimated coefficients and 95% CIs from the specification in Equation (2). I include all WNS confirmed counties up to 2017, instead of excluding those exposed after 2014, and the composition in each event-time bin is no longer balanced as a result. Top and bottom coded event-time coefficients are not reported. The regression includes county and state-by-year fixed effects. Observations are population-weighted. Standard errors are clustered at the county level.

Figure A5: Distribution of Effects on Yields



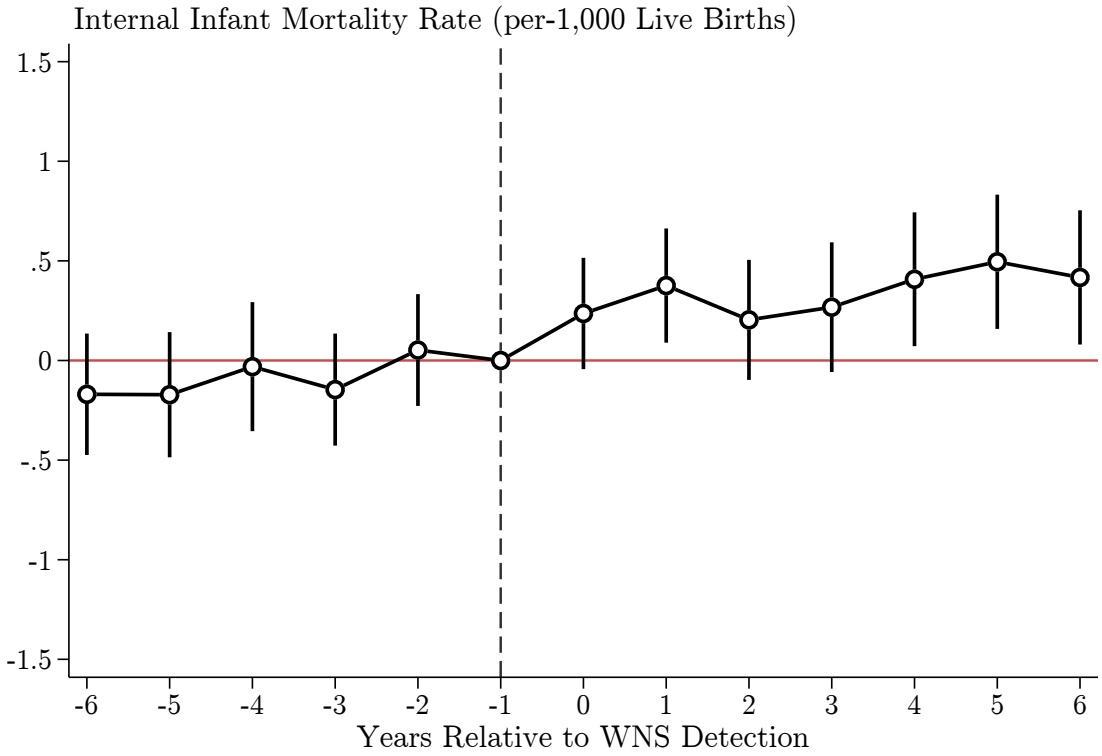
Notes: Kernel densities summarizing the distribution of estimated coefficients from the specification in Equation (1). Each density function summarizes 12 different estimates from a balanced or unbalanced sample, that either include or exclude weather controls, and are either unweighted, weighted by baseline cropland area in 2002, or weighted by specific crop acres in 2002. All regressions include county fixed effects.

Figure A6: Verifying There Are No Differences in Mothers' Characteristics



Notes: Estimated coefficients and 95% CIs from the specification in Equation (1). Each coefficient is estimated in a separate regression. To allow for easier visual inspection of the CIs, I omit the point estimate (-.057) and 95% CI (ranging from -.15 to .037) for the mean prenatal care visits as it distorts the scale. The regressions includes county and state-year fixed effects. Observations are weighted by the number of live births. Standard errors are clustered at the county level.

Figure A7: Dynamic Effects on Internal IMR When Excluding Nearest Neighbors

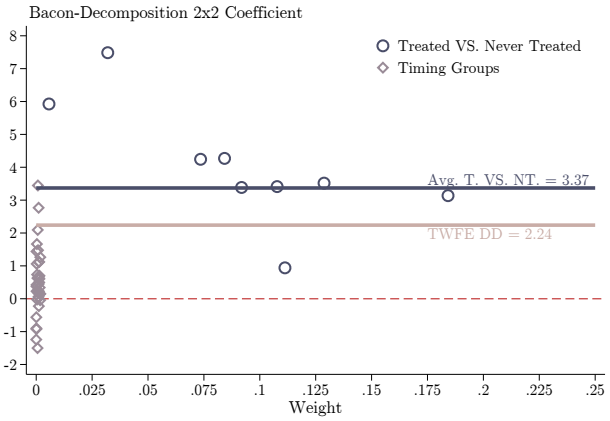


Notes: Estimated coefficients and 95% CIs from the specification in Equation (2). The sample is equivalent to the one used in the results in Figure 2f, with the exclusion of the first degree neighbors of the WNS confirmed counties, in order to reduce the effect of spatial spillovers that might be attenuating the results. The regression include county and state-by-year fixed effects. Observations are population weighted. Standard errors are clustered at the county level.

Figure A8: Bacon DD-Decomposition

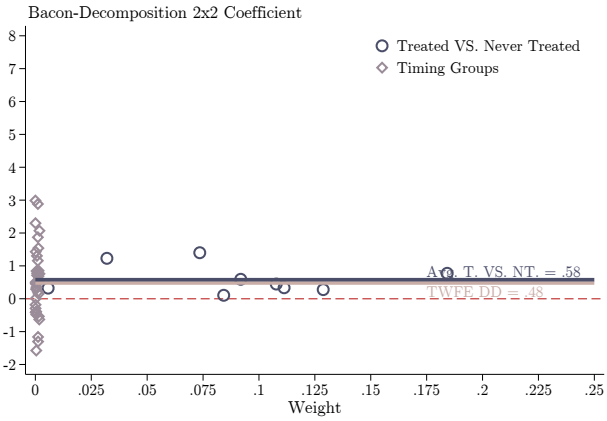
Insecticide Use

(a) Equally-Weighted

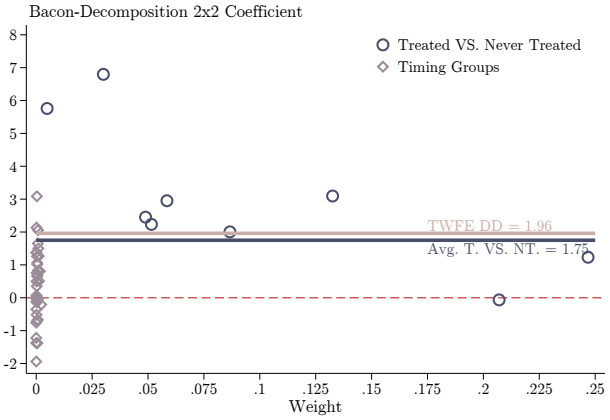


Internal IMR

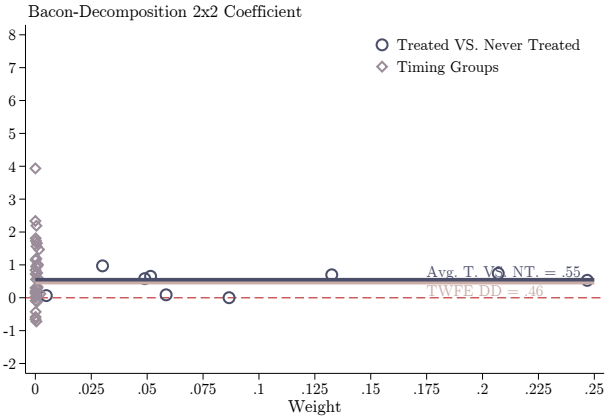
(b) Equally-Weighted



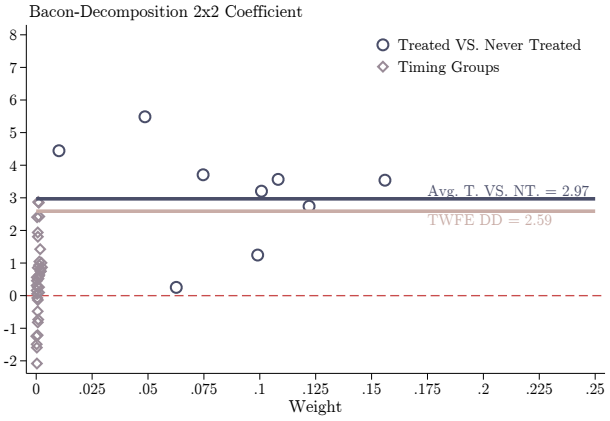
(c) Cropland (2002) Weights



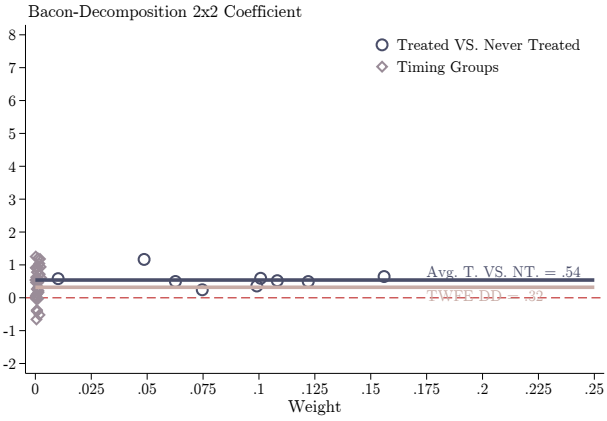
(d) Cropland (2002) Weights



(e) Population (1997) Weights



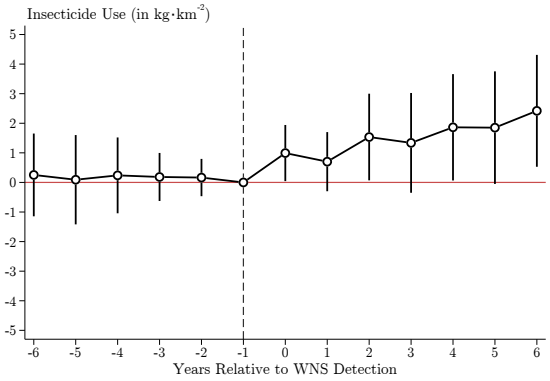
(f) Population (1997) Weights



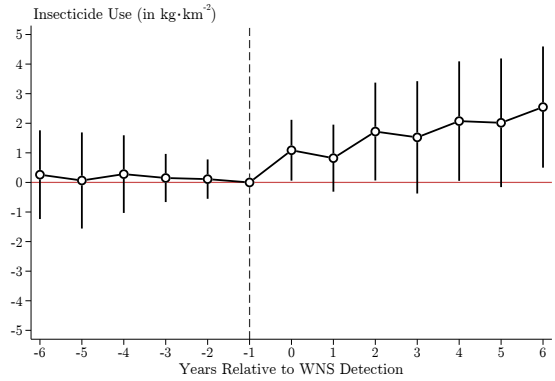
Notes: Results from performing the Bacon Difference-In-Differences Decomposition for insecticide use (in kg per-sq km), and for internal IMR (in deaths per-1,000 live births). Results are either equally-weighted, weighted by baseline cropland area in 2002, or by baseline population in 1997. Each figure shows the separate DD estimates obtained from comparing each treatment switching cohort, either to never-treated control units (circles), or using timing group that compare late to early switchers, or early to late switchers (diamonds).

Figure A9: Insecticide Use Following WNS Exposure

(a) Two Way Fixed Effects Estimator



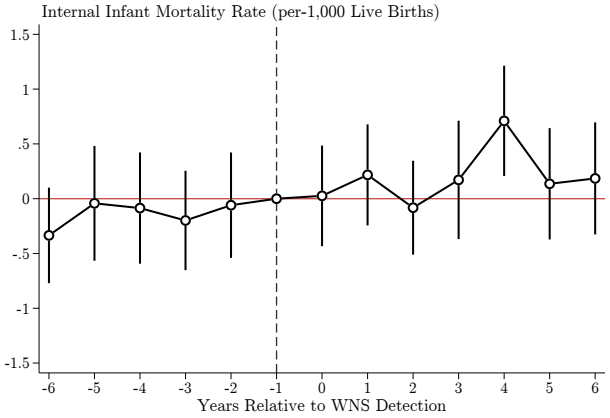
(b) Alternative Differential Timing Estimator



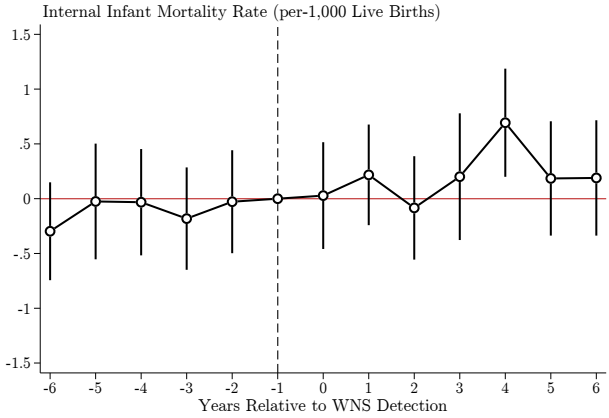
Notes: Estimated coefficients and 95% CIs from the specification in Equation (2). Top and bottom coded event-time coefficients are not reported.The regression includes county and state-by-year fixed effects. Left panel reports estimates from a standard TWFE estimator, while the right panel report estimates from using the estimator developed in Sun and Abraham (2020). Observations are weighted by baseline cropland area in 2002. Standard errors are clustered at the county level.

Figure A10: Internal Infant Mortality Rate Following WNS Exposure

(a) Two Way Fixed Effects Estimator



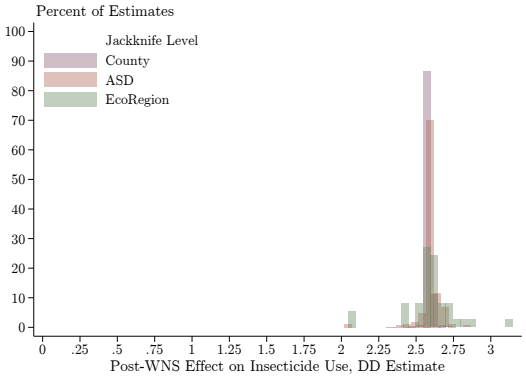
(b) Alternative Differential Timing Estimator



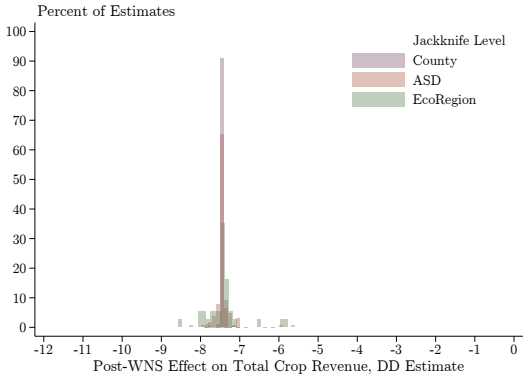
Notes: Estimated coefficients and 95% CIs from the specification in Equation (2). Top and bottom coded event-time coefficients are not reported.The regression includes county and state-by-year fixed effects. Left panel reports estimates from a standard TWFE estimator, while the right panel report estimates from using the estimator developed in Sun and Abraham (2020). Observations are weighted by the number of live births. Standard errors are clustered at the county level.

Figure A11: Leave-One-Out Estimation Results

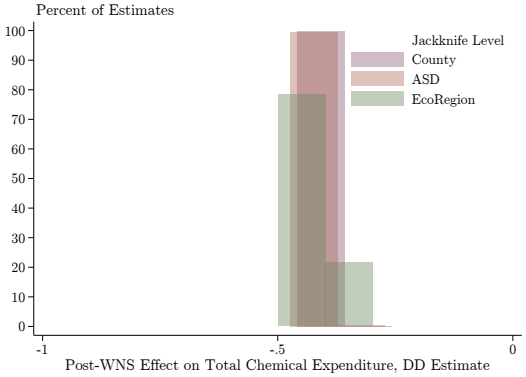
(a) Insecticide Use



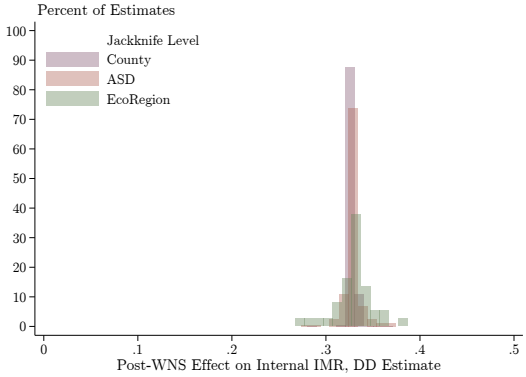
(b) Crop Revenue



(c) Chemical Expenditure



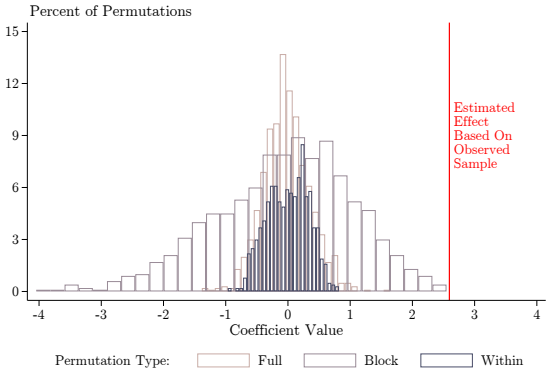
(d) Internal IMR



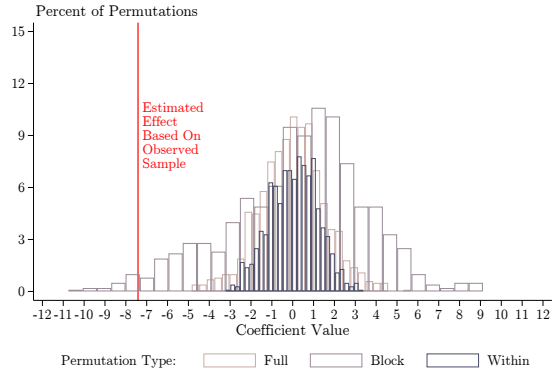
Notes: Distribution of estimated coefficients from the specification in Equation (1), when excluding one unit at a time. All regressions include county and state-year fixed effects. Observations are population-weighted.

Figure A12: Permutation Inference Results

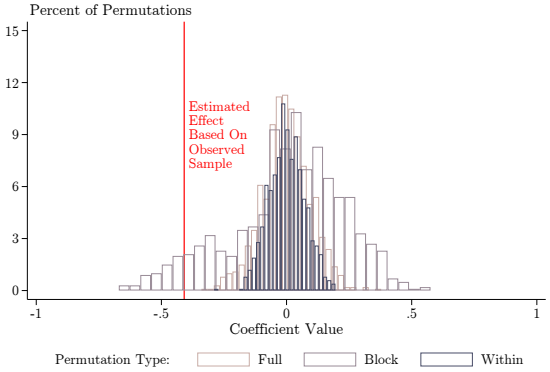
(a) Insecticide Use



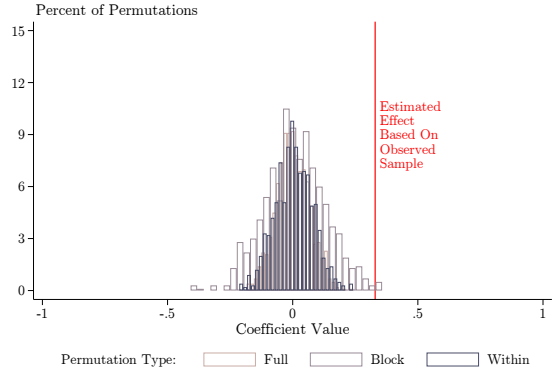
(b) Crop Revenue



(c) Chemical Expenditure

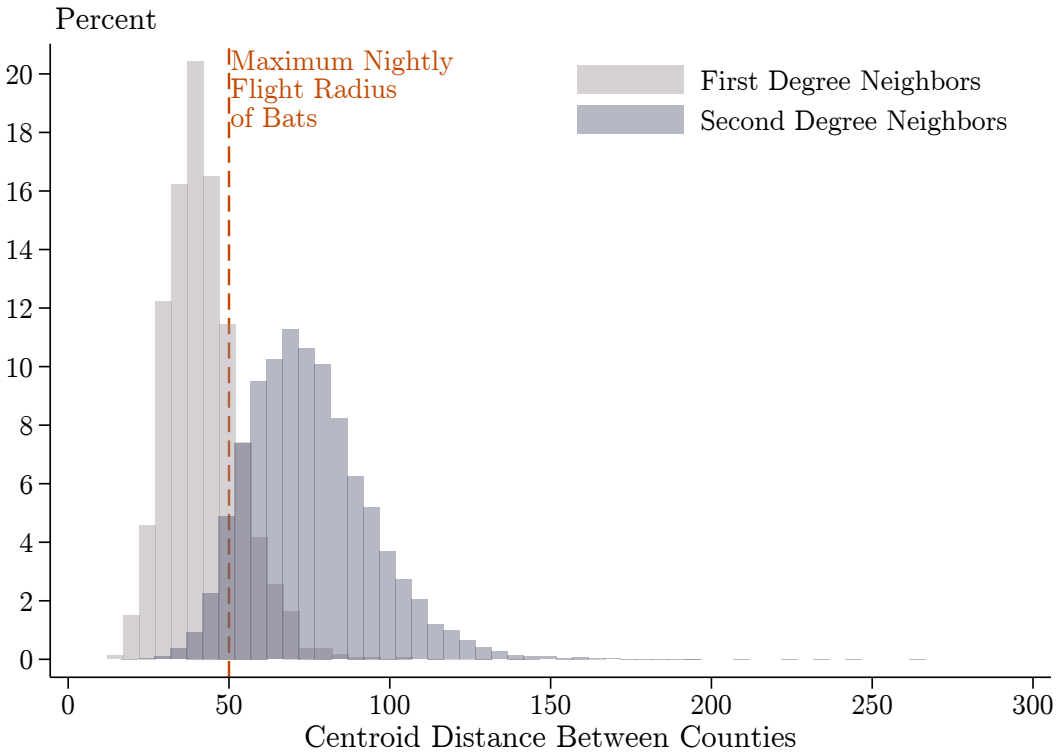


(d) Internal IMR



Notes: Results from randomly reassigning treatment status and re-estimating the treatment effects. I estimate Equation (1) 1,000 times for each permutation type. Each regression include county and state-year fixed effects. Observations are population-weighted. Each histogram shows the distribution of the coefficients on exposure to WNS. The vertical line represents the value of the estimation from the observed sample. Permutation was conducted either on the entire sample (full, in tan), across counties such that the temporal order of the treatment was preserved (block, in purple), or within treated counties such that only the temporal order was randomized (within, in blue).

Figure A13: Distance Between Neighboring Counties



Notes: Histograms showing the centroid distance distribution between adjacent counties (first degree neighbors), and the second degree neighboring counties. Most second degree neighboring counties have a centroid distance well above 50 km, which is approximately the maximum nightly flight radius of bats.

Table A1
Summary Statistics for Main Estimation Sample

Variable	Mean	SD	Min	Max	N
WNS Presence Dummy	0.07	.25	0	1	33,474
Insecticide Use, kg·km^{-2}	9.28	27	0	2,145	33,474
Fungicide Use, kg·km^{-2}	10.96	37	0	2,475	33,474
Herbicide Use, kg·km^{-2}	61.50	73	0	2,184	33,474
Cropland in 2002, km^2	339	379	0.24	2,605	33,474
County Area, km^2	1,434	850	222.74	17,721	33,474
Crop Sales, Thousands of Dollars·km^{-2}	27.93	34	0	289.20	7,769
Chemical Expenditure, Thousands of Dollars·km^{-2}	2.4	2.9	0	21	7,867
Live Births	1,074	2,833	7	85928.00	33,474
All-Cause IMR (per-1,000 Live Births)	7.29	6.2	0	107.14	33,474
Internal IMR (per-1,000 Live Births)	6.77	6	0	107.14	33,474
External IMR (per-1,000 Live Births)	0.43	1.4	0	45	33,474
Population	84,140	202,106	1,326	5,373,418	33,474

Notes: Summary statistics for the main estimation sample 1997 to 2017, for the counties in the states that had at least one WNS confirmed county by 2014. See text for more deatils.
Source: WNS expansion data, and Pesticide use data from the USGS. Crop sales data from the USDA NASS. Infant health outcomes from the NCHS. Population data from the Census Bureau.

Table A2
Insecticide Use DD Estimates
Annual Aggregate Insecticide Use in kg·km^{-2}

Panel A. Average Treatment Effects						
	(1)	(2)	(3)	(4)	(5)	(6)
WNS	2.24	2.19	1.96	2.56	1.98	2.52
	(0.49)	(0.49)	(0.65)	(0.67)	(0.65)	(0.67)
R^2	0.53	0.53	0.58	0.58	0.58	0.58
Panel B. Heterogeneous Treatment Effects						
	(1)	(2)	(3)	(4)	(5)	(6)
WNS×Low Baseline	3.80	3.75	4.48	3.58	4.46	3.60
	(0.46)	(0.47)	(0.75)	(0.54)	(0.76)	(0.52)
WNS×High Baseline	0.03	-0.01	0.63	1.58	0.67	1.49
	(0.68)	(0.68)	(0.70)	(1.00)	(0.70)	(0.99)
R^2	0.53	0.53	0.58	0.58	0.58	0.58
County FEs	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X
Weather Controls		X			X	X
Weighted	E	E	A	B	A	P
Dep. Var. Mean	9.28	9.28	10.94	7.71	10.94	7.50
N	33,474	33,474	33,474	33,474	33,474	33,474
Clusters	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are either equally weighted (E), weighted by cropland area in 2002 (A), prior to the emregence of WNS, weighted by the number of live births (B), or population-weighted (P). The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table A3
Fungicide Use DD Estimates
Annual Aggregate Fungicide Use in kg·km^{-2}

Panel A. Average Treatment Effects						
	(1)	(2)	(3)	(4)	(5)	(6)
WNS	1.14	1.05	0.42	1.91	0.44	1.84
	(0.47)	(0.47)	(0.62)	(0.62)	(0.61)	(0.61)
R^2	0.64	0.64	0.67	0.68	0.67	0.69
Panel B. Heterogeneous Treatment Effects						
	(1)	(2)	(3)	(4)	(5)	(6)
WNS×Low Baseline	2.47	2.38	2.20	2.74	2.07	2.75
	(0.44)	(0.44)	(0.56)	(0.62)	(0.57)	(0.61)
WNS×High Baseline	-0.73	-0.82	-0.52	1.12	-0.42	0.97
	(0.72)	(0.72)	(0.79)	(0.86)	(0.78)	(0.85)
R^2	0.64	0.64	0.67	0.68	0.67	0.69
County FEs	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X
Weather Controls		X			X	X
Weighted	E	E	A	B	A	P
Dep. Var. Mean	10.96	10.96	10.53	9.35	10.53	9.31
N	33,474	33,474	33,474	33,474	33,474	33,474
Clusters	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are either equally weighted (E), weighted by cropland area in 2002 (A), prior to the emregence of WNS, weighted by the number of live births (B), or population-weighted (P). The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table A4
Herbicide Use DD Estimates
Annual Aggregate Herbicide Use in kg·km^{-2}

Panel A. Average Treatment Effects						
	(1)	(2)	(3)	(4)	(5)	(6)
WNS	-6.90	-6.92	-10.76	-0.85	-10.61	-0.85
	(1.50)	(1.50)	(2.92)	(1.70)	(2.90)	(1.55)
R^2	0.89	0.89	0.88	0.90	0.88	0.90
Panel B. Heterogeneous Treatment Effects						
	(1)	(2)	(3)	(4)	(5)	(6)
WNS×Low Baseline	-10.05	-10.15	-20.41	-4.06	-20.71	-3.76
	(1.63)	(1.63)	(3.54)	(1.95)	(3.57)	(1.84)
WNS×High Baseline	-2.47	-2.38	-5.66	2.21	-5.29	1.94
	(2.10)	(2.08)	(3.64)	(2.31)	(3.59)	(2.08)
R^2	0.89	0.89	0.88	0.90	0.88	0.90
County FEs	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X
Weather Controls		X			X	X
Weighted	E	E	A	B	A	P
Dep. Var. Mean	61.50	61.50	123.25	41.52	123.25	41.49
N	33,474	33,474	33,474	33,474	33,474	33,474
Clusters	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are either equally weighted (E), weighted by cropland area in 2002 (A), prior to the emregence of WNS, weighted by the number of live births (B), or population-weighted (P). The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table A5
Infant Mortality DD Estimates
Annual Internal Infant Mortality Rate, per-1,000 Live Births

Panel A. Average Treatment Effects							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
WNS	0.48	0.46	0.49	0.46	0.33	0.33	0.33
	(0.20)	(0.20)	(0.19)	(0.17)	(0.11)	(0.11)	(0.10)
R^2	0.15	0.15	0.15	0.16	0.35	0.35	0.33
Panel B. Heterogeneous Treatment Effects							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
WNS×Low Baseline	0.50	0.49	0.53	0.55	0.44	0.45	0.43
	(0.28)	(0.28)	(0.27)	(0.20)	(0.15)	(0.15)	(0.14)
WNS×High Baseline	0.44	0.42	0.44	0.41	0.22	0.22	0.24
	(0.21)	(0.21)	(0.21)	(0.22)	(0.13)	(0.12)	(0.12)
R^2	0.15	0.15	0.15	0.16	0.35	0.35	0.33
County FEs	X	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X	X
Weather Controls		X				X	X
Population Shares			X			X	X
Weighted	E	E	E	A	B	B	P
Dep. Var. Mean	6.77	6.77	6.77	6.35	6.69	6.69	6.56
N	33,474	33,474	33,474	33,474	33,474	33,474	33,474
Clusters	1,594	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are either equally weighted (E), weighted by cropland area in 2002 (A), prior to the emregence of WNS, weighted by the number of live births (B), or population-weighted (P). The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table A6
Infant Mortality DD Estimates
Annual All-Cause Infant Mortality Rate, per-1,000 Live Births

Panel A. Average Treatment Effects							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
WNS	0.47	0.46	0.49	0.49	0.33	0.33	0.33
	(0.20)	(0.20)	(0.19)	(0.17)	(0.11)	(0.11)	(0.10)
R^2	0.15	0.15	0.15	0.17	0.36	0.36	0.34
Panel B. Heterogeneous Treatment Effects							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
WNS×Low Baseline	0.48	0.47	0.50	0.66	0.44	0.44	0.42
	(0.28)	(0.28)	(0.27)	(0.22)	(0.14)	(0.13)	(0.13)
WNS×High Baseline	0.46	0.45	0.47	0.40	0.23	0.22	0.25
	(0.23)	(0.23)	(0.23)	(0.22)	(0.14)	(0.13)	(0.13)
R^2	0.15	0.15	0.15	0.17	0.36	0.36	0.34
County FEs	X	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X	X
Weather Controls		X				X	X
Population Shares			X			X	X
Weighted	E	E	E	A	B	B	P
Dep. Var. Mean	7.29	7.29	7.29	6.87	7.15	7.15	7.02
N	33,474	33,474	33,474	33,474	33,474	33,474	33,474
Clusters	1,594	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are either equally weighted (E), weighted by cropland area in 2002 (A), prior to the emregence of WNS, weighted by the number of live births (B), or population-weighted (P). The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table A7
Infant Mortality DD Estimates
Annual External Infant Mortality Rate, per-1,000 Live Births

Panel A. Average Treatment Effects	(1)	(2)	(3)	(4)	(5)	(6)	(7)
WNS	-0.018	-0.020	-0.020	0.024	0.011	0.009	0.012
	(0.048)	(0.048)	(0.048)	(0.065)	(0.026)	(0.026)	(0.025)
R^2	0.09	0.09	0.09	0.10	0.18	0.19	0.18
Panel B. Heterogeneous Treatment Effects	(1)	(2)	(3)	(4)	(5)	(6)	(7)
WNS×Low Baseline	-0.010	-0.013	-0.017	0.102	0.013	0.010	0.009
	(0.067)	(0.067)	(0.068)	(0.105)	(0.039)	(0.039)	(0.037)
WNS×High Baseline	-0.030	-0.031	-0.025	-0.017	0.009	0.008	0.015
	(0.058)	(0.058)	(0.057)	(0.077)	(0.029)	(0.027)	(0.028)
R^2	0.09	0.09	0.09	0.10	0.18	0.19	0.18
County FEs	X	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X	X
Weather Controls		X				X	X
Population Shares			X			X	X
Weighted	E	E	E	A	B	B	P
Dep. Var. Mean	0.43	0.43	0.43	0.47	0.38	0.38	0.38
N	33,474	33,474	33,474	33,474	33,474	33,474	33,474
Clusters	1,594	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are either equally weighted (E), weighted by cropland area in 2002 (A), prior to the emregence of WNS, weighted by the number of live births (B), or population-weighted (P). The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table A8
Infant Health Outcomes DD Estimates

	Birth Outcome Mean				Birth Outcome Share	
	Weight	Log(Weight)	Gestation	APGAR5	Low Weight	Premature
	(1)	(2)	(3)	(4)	(5)	(6)
WNS	-5.0934	-0.0015	-0.0132	-0.0084	0.0009	0.0014
	(2.6592)	(0.0008)	(0.0079)	(0.0112)	(0.0006)	(0.0009)
R^2	0.87	0.88	0.81	0.75	0.73	0.73
County FEs	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X
Weighted	B	B	B	B	B	B
Dep. Var. Mean	3,281.69	8.10	38.63	8.84	0.08	0.12
N	33,474	33,474	33,474	33,474	33,474	33,474
Clusters	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for the specification in Equation (1). Observations are weighted by the number of live births (B). Birth weight is measured in grams, gestation in weeks, and APGAR5 is a score between 0 and 10, determined five minutes after delivery. Births where the infant is below 2,500 grams are classified as low birth weight. Births that happen before the 37th week are classified as premature births. The reported mean for the dependent variable is the weighted mean. Standard errors are clustered at the county level.

Table A9
Spatial Clustering at Different Spatial Scales

Coefficients	Insecticide Use (1)	Crop Revenue (2)	Chemical Expenditure (3)	Internal IMR (4)
WNS	2.59	-7.42	-0.41	0.33
Clustering by Administrative Units				
County	(0.66)	(2.15)	(0.13)	(0.11)
Agricultural Statistics District	(0.96)	(2.39)	(0.15)	(0.10)
State-Ecological-Region	(0.95)	(2.49)	(0.15)	(0.11)
Clustering by Distance Thresholds				
Distance: 50 km	(0.40)	(0.88)	(0.07)	(0.18)
Distance: 100 km	(0.42)	(1.13)	(0.09)	(0.17)
Distance: 250 km	(0.46)	(1.34)	(0.11)	(0.17)
Distance: 500 km	(0.52)	(1.03)	(0.08)	(0.16)
Distance: 750 km	(0.55)	(1.14)	(0.11)	(0.13)
Distance: 1000 km	(0.55)	(1.38)	(0.13)	(0.14)
Distance: 2000 km	(0.49)	(1.43)	(0.13)	(0.13)

Notes: Estimation results for the specification in Equation (1), for insecticide use, crop revenue, chemical expenditure, and internal infant mortality rate (IIMR). Observations are population-weighted. There are 1,594 county clusters, 164 agricultural statistics district clusters, and 119 state-ecological-region clusters.

Table A10
WNS Expansion Prediction Estimates
Outcome: Binary WNS Exposure Status

	(1)	(2)	(3)	(4)	(5)	(6)
Insecticide $\text{Use}_{\text{t-1}}$	0.00038					0.00021
	(0.00014)					(0.00009)
Fungicide $\text{Use}_{\text{t-1}}$	0.00012					0.00006
	(0.00013)					(0.00010)
Herbicide $\text{Use}_{\text{t-1}}$	-0.00049					-0.00022
	(0.00011)					(0.00008)
Internal $\text{IMR}_{\text{t-1}}$		0.00037				0.00029
		(0.00017)				(0.00015)
$\text{WNS}^{\text{N1}}_{\text{t-1}}$			0.24085	0.11271	0.22212	0.22103
			(0.01936)	(0.01591)	(0.01922)	(0.01917)
$\text{WNS}^{\text{N1}}_{\text{t-2}}$				0.10671		
				(0.01296)		
$\text{WNS}^{\text{N1}}_{\text{t-3}}$				0.07711		
				(0.01327)		
$\text{WNS}^{\text{N2}}_{\text{t-1}}$					0.07035	0.06990
					(0.01271)	(0.01270)
$\text{WNS}^{\text{N3}}_{\text{t-1}}$					-0.01250	-0.01248
					(0.00841)	(0.00839)
County FEs	X	X	X	X	X	X
EcoRegion-Year FEs	X	X	X	X	X	X
Weighted	E	E	E	E	E	E
R^2	0.58	0.58	0.59	0.63	0.59	0.59
N	31,880	31,880	28,692	25,504	28,692	28,692
Clusters	1,594	1,594	1,594	1,594	1,594	1,594

Notes: Estimation results for a linear probability model with WNS status as the binary outcome regressed on lagged values of pesticide use, or WNS status of neighboring counties (first, second, and third degree neighbors). Observations are equally weighted. Standard errors are clustered at the county level.
Source: Data on White Nose Syndrome status and pesticide use are from the USGS. Eco-region classification is from the EPA.

Table A11
Long Differences DD Estimates

	Insecticide Use		Crop Revenue		Chemical Expenditure		Internal IMR	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS	2.32	2.47	-9.89	-9.09	-0.88	-0.46	0.35	0.41
	(0.67)	(0.83)	(1.26)	(2.75)	(0.11)	(0.16)	(0.40)	(0.19)
R^2	0.76	0.79	0.93	0.91	0.92	0.90	0.58	0.74
County FEs	X	X	X	X	X	X	X	X
Pre/Post by State FEs	X	X	X	X	X	X	X	X
Weighted	E	P	E	P	E	P	E	P
Dep. Var. Mean	8.16	6.70	27.52	26.51	2.56	1.83	6.69	6.40
N	3,188	3,188	2,940	2,940	3,036	3,036	3,188	3,188
Clusters	1,594	1,594	1,470	1,470	1,518	1,518	1,594	1,594

Notes: Estimation results for the specification in Equation (1) for a sample containing the years 2002 to 2004 and 2015 to 2017, collpased as pre- and post-treatment. One exception to that is for the crop revnue results that only use 2002 and 2017 as data are reported every 5 years. The regressions comapre the counties that were classified as WNS confirmed between 2006 and 2014, to counties that were never classified as WNS confirmed or suspected. Observations are either equally weighted (E), or population-weighted (P). The reported mean for the dependent variable is the weighted mean. Insecticide use is measured in $kg \cdot km^{-2}$. Crop revenue and chemical expenditure are meausred in thousands of dollars per-km^{-2}. Internal infant mortality rate (IMR) is meausred in deaths per-1,000 live births. Standard errors are clustered at the county level.

Table A12
Strictly Balanced DD Estimates

	Insecticide Use		Crop Revenue		Chemical Expenditure		Internal IMR	
Panel A. Average Treatment Effects								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS	2.04	2.00	-8.10	-7.15	-0.70	-0.41	0.43	0.25
	(0.52)	(0.61)	(1.12)	(2.20)	(0.09)	(0.13)	(0.17)	(0.10)
R^2	0.56	0.59	0.93	0.91	0.92	0.91	0.16	0.34
Panel B. Heterogeneous Treatment Effects								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
WNS×Low Baseline	3.85	3.42	-12.07	-12.97	-1.01	-0.81	0.54	0.36
	(0.53)	(0.55)	(1.07)	(2.52)	(0.09)	(0.16)	(0.25)	(0.13)
WNS×High Baseline	-0.21	0.72	-3.27	-1.93	-0.32	-0.05	0.28	0.14
	(0.67)	(0.81)	(1.68)	(2.41)	(0.13)	(0.14)	(0.20)	(0.12)
R^2	0.56	0.59	0.93	0.91	0.92	0.91	0.16	0.34
County FEs	X	X	X	X	X	X	X	X
State-Year FEs	X	X	X	X	X	X	X	X
Weighted	E	P	E	P	E	P	E	P
Dep. Var. Mean	9.80	7.88	29.67	27.99	2.52	1.85	6.69	6.50
N	29,715	29,715	7,075	7,075	7,075	7,075	29,715	29,715
Clusters	1,415	1,415	1,415	1,415	1,415	1,415	1,415	1,415

Notes: Estimation results for the specification in Equation (1). The sample if strictly balanced for all outcomes such that they have the same sample compoisition. Observations are weighted equally (E), or by the total population in the county-year observation (P), using estimated population sizes as reported by the Census Bureau. The reported mean for the dependent variable is the weighted mean. Insecticide use is measured in kg·km^{-2}. Crop revenue is meausred in thousands of dollars per-km^{-2}. Chemcial expenditure is measured in thousands of dollars per-km^{-2}. Internal infant mortality rate (IMR) is meausred in deaths per-1,000 live births. Standard errors are clustered at the county level.

B Additional Descriptive Data

Here I include additional summary figures regarding the data, or classifications used in the data.

B.1 Additional Controls

As additional controls, for the results reported in the Appendix, I use the Finescaled Weather Data Set for the U.S. as described in Schlenker and Roberts (2009). I use the number of degree days, in Celsius, in 5 degree bins, as well as data on precipitation, all at a county-year level. I construct weather variables for the agricultural growth period of April through September. Finally, I use population data from the Census Bureau that is interpolated between decadal census years to construct population shares by age group, and calculate the crude birth rate in each year.

B.2 Number of WNS Detections by Year

Figure B1 shows the number of newly classified WNS counties, for either both confirmed and suspected statuses, or just confirmed. The number of confirmed WNS counties by-state, by-year, are summarized in Table B1. The tabulation by-state-year demonstrates that in some cases state-by-year fixed effects might absorb a considerable amount of the treatment effect.

B.3 Confirmed Versus Suspected WNS Counties

In the main analysis, I compare the counties that are classified as confirmed WNS counties to counties never confirmed or suspected as a WNS county. A WNS confirmed county has had multiple samples of bats test positive for the fungus, and multiple bats demonstrating syndromes of WNS. A WNS suspected county is a county that has had bats test positive for the fungus, but have not had bats demonstrate syndromes yet. In the main map, Figure 1, I report the year of WNS classification for both WNS confirmed and suspected counties. In Figure B2, I focus on the group of states that had at least one WNS county by 2014, as used in the main analysis. I highlight the counties that are classified as WNS confirmed counties.

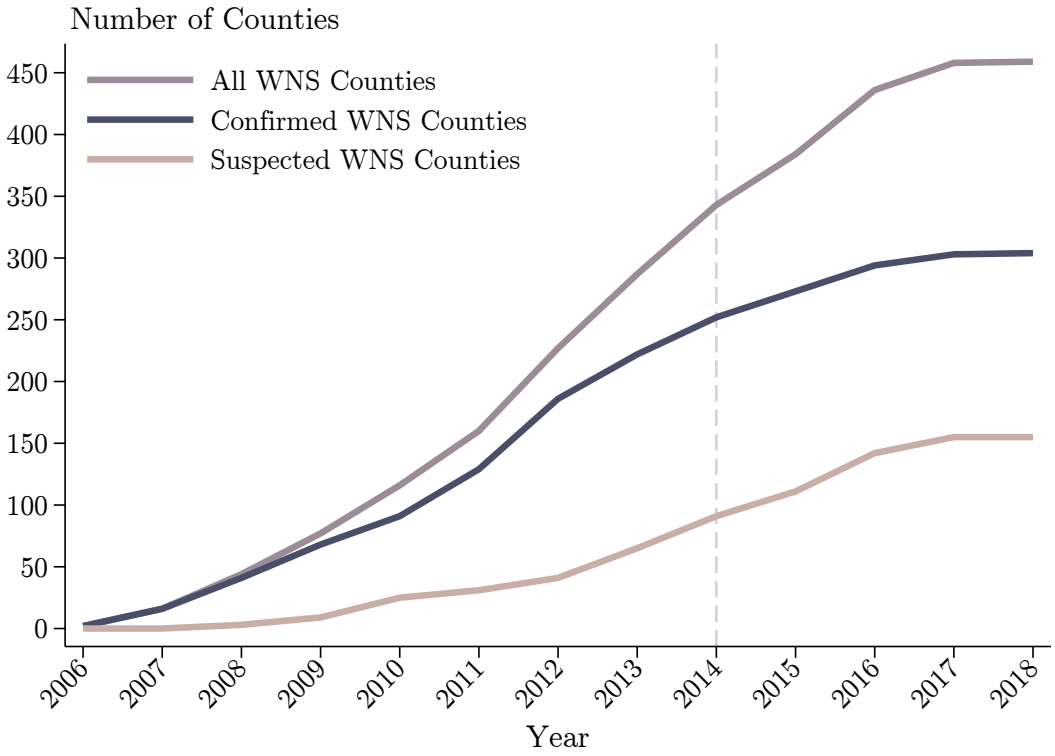
B.4 Classifying Counties According to the Ecological Regions of the United States, Level III

I use the data from the Environmental Protection Agency (EPA) on the Ecological Regions of the United States. The EPA reports the classifications at four different levels. Level I is the most coarse, while Level IV is the finest level of classification. I assign counties based on their area overlap with the Level III layer provided by the EPA. Specifically, for each county, I calculate the share of area overlap with each Level III ecological region, and assign the region with the highest share of overlap. This assignment results in 37 ecological regions assigned to the counties in the main analysis sample. In Figure B3, I plot the classification of counties by ecological region, and the names of the regions in Figure

B.5 State Agricultural Statistics Districts

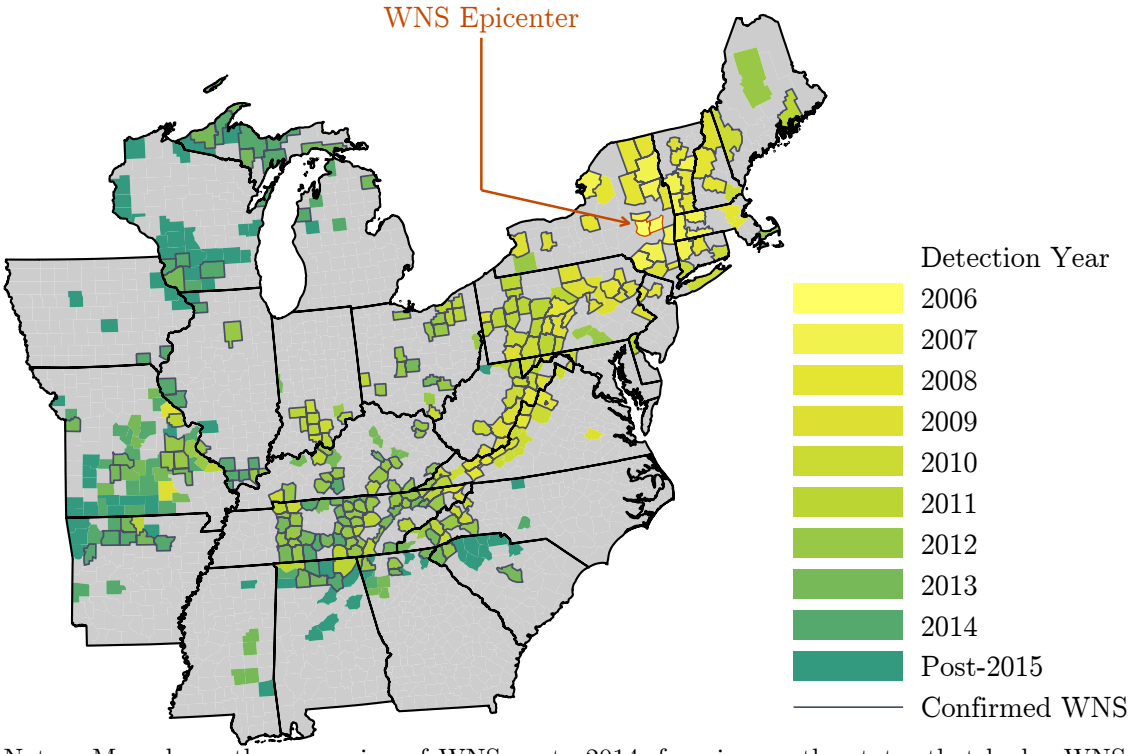
The US Department of Agriculture (USDA) divides each state up to 9 different Agricultural Statistics District (ASDs). The divisions are made such that the counties within each ASD are agriculturally homogeneous. In Figure B4, I plot the sub-state division into ASDs.

Figure B1: Number of WNS Counties, by Status, by Year



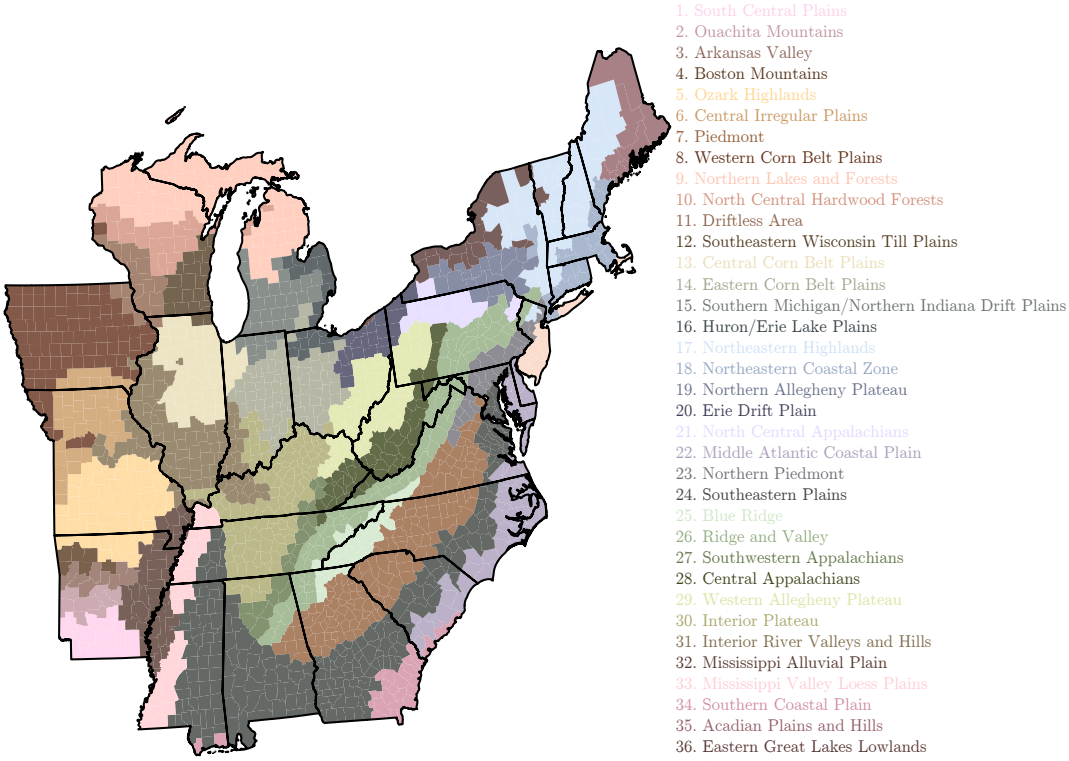
Notes: The cumulative number of counties each year with any WNS status, confirmed statues, or suspected status. Dashed line in 2014 highlights the set of counties used in the main analysis.

Figure B2: WNS Confirmed Counties



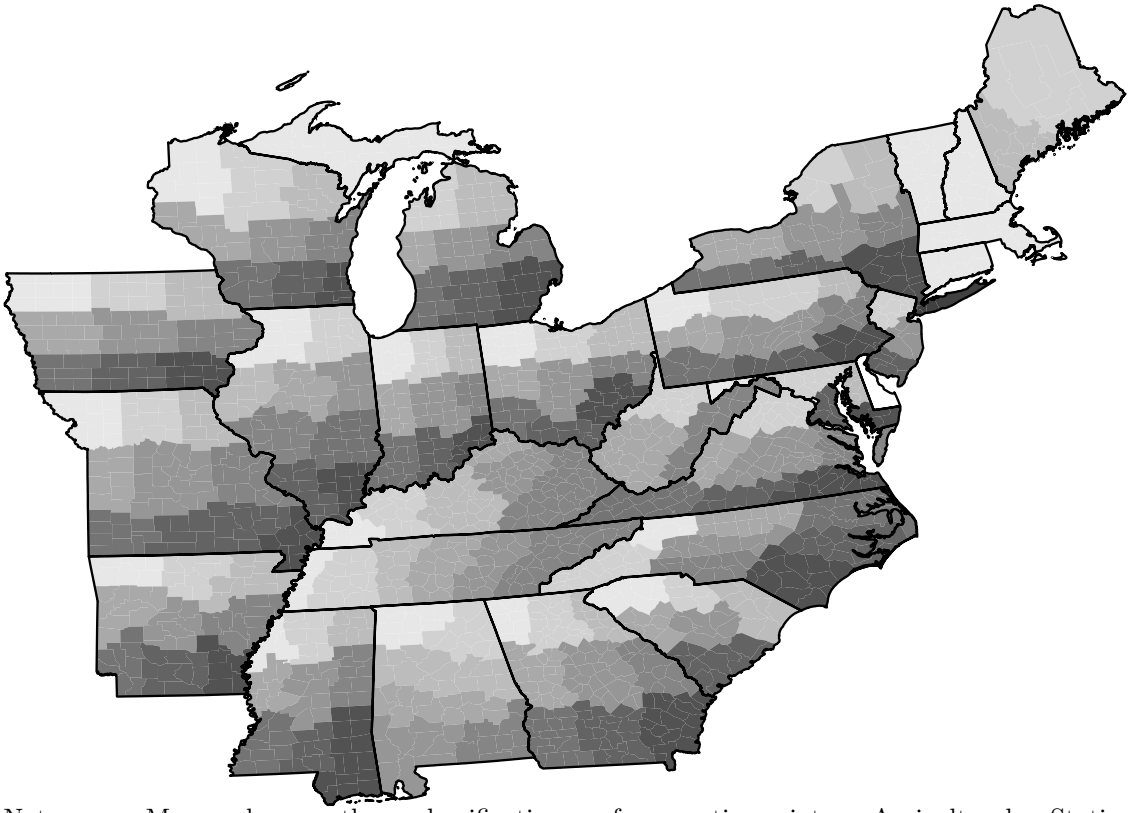
Notes: Map shows the expansion of WNS up to 2014, focusing on the states that had a WNS county by the end of 2014. Counties that are classified as WNS confirmed counties are highlighted with a gray border.

Figure B3: Ecological Regions Levels III & County Assignment



Notes: Map shows the classification of counties into ecological regions, focusing on the states that had a WNS county by the end of 2014. For more information on the EPA's classification, see: https://www.epa.gov/eco-research/ecoregions

Figure B4: Agricultural Statistics Districts



Notes: Map shows the classification of counties into Agricultural Statistics Districts, focusing on the states that had a WNS county by the end of 2014. For more information on the USDA's classification, see: https://www.nass.usda.gov/Data_and_Statistics/County_Data_Files/Frequently_Asked_Questions/index.php# and https://data.nal.usda.gov/dataset/nass-quick-stats/resource/5f1173c1-bcb4-4f88-aec0-3bbfee75657f

Table B1
WNS Detection by State by Year

State	Year of WNS Confirmed Classification by Fish and Wildlife Service									Total
	2006	2007	2008	2009	2010	2011	2012	2013	2014	
AL	0	0	0	0	0	1	0	2	2	5
AR	0	0	0	0	0	0	0	2	4	6
CT	0	1	1	0	2	0	0	0	0	4
DE	0	0	0	0	0	1	0	0	0	1
GA	0	0	0	0	0	0	3	2	0	5
IL	0	0	0	0	0	0	4	0	8	12
IN	0	0	0	0	3	6	0	0	0	9
IA	0	0	0	0	0	0	0	0	2	2
KY	0	0	0	0	1	2	13	1	1	18
ME	0	0	0	0	1	1	0	0	0	2
MD	0	0	0	1	2	0	0	0	0	3
MA	0	3	0	0	0	0	1	0	0	4
MI	0	0	0	0	0	0	0	5	5	10
MO	0	0	0	0	0	1	3	4	0	8
NH	0	0	2	1	0	0	0	0	0	3
NJ	0	0	2	0	0	0	0	0	0	2
NY	2	7	6	3	0	0	0	0	0	18
NC	0	0	0	0	4	1	2	1	0	8
OH	0	0	0	0	1	5	8	2	0	16
PA	0	0	4	10	3	10	0	0	0	27
SC	0	0	0	0	0	0	1	2	0	3
TN	0	0	0	2	1	8	21	14	4	50
VT	0	3	3	1	0	0	0	0	0	7
VA	0	0	6	3	1	1	1	0	0	12
WV	0	0	1	6	4	1	0	0	0	12
WI	0	0	0	0	0	0	0	1	4	5
Total	2	14	25	27	23	38	57	36	30	252

Notes: Each cell reports the number of counties in each state (row) that were classified as WNS confirmed by the Fish and Wildlife service between 2006 and 2014 (column).