Declining Bat Populations, Increased Pesticides Use & Infant Mortality*

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Abstract

Pesticides are a form of environmental pollution yet there are no well identified estimates for their effects. Using a natural experiment I find the first causally interpretable results for the adverse health effects of pesticides. This paper uses mortality shocks to bats - a major predator of insects - that result from the unexpected emergence of a wildlife disease known as White Nose Syndrome (WNS). WNS first emerged in 2006 and started to gradually spread across counties. I use a Difference-In-Differences strategy and find that farmers increase their use of insecticides by 39.6% relative to their mean use. Because insects carry fungi between plants the use of fungicides increases as well by 20.1% relative to the mean. Using linked birth and death certificates I focus on infant mortality due to non-violent causes for births that were conceived during the pesticides application season of April through July. I find that the infant mortality rate increases by 1.01 deaths per 1,000 births in the counties exposed to WNS. This is driven mostly by female infant mortality and represents an increase of 14.5% relative to the mean. These results suggest that mixtures of pesticide compounds can affect health even if each compound is used below its regulatory threshold.

**JEL Codes:** I10, Q53, Q57.

**Keywords:** environmental externalities, fetal health, pesticides, functional biodiversity.

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1 Introduction

When natural resources are finite, sustained economic growth depends on the substitutability between natural and man-made capital. This type of substitution is an important concept of sustainability in environmental economics (Solow 1993). However, we lack knowledge regarding the amount of man-made capital required to compensate for natural capital reductions, and the possible environmental externalities these substitution patterns might have. Pesticides can be used to substitute for natural pest-control provided by different species. Farmers in the U.S. spent about $7.9 billion on pesticides alone in 2006, $9.7 billion in 2007 and $14.5 billion in 2012 (in $2006) on agrichemicals which pesticides make a large share of (G. Arthur et al. 2011; United States Department of Agriculture 2016). Pesticides are a form of environmental pollution yet we do not have well identified estimates for its effect. In order to efficiently regulate pesticides and to determine the optimal level of conservation for the species that provide free pest-control, we need to know two key parameters: the environmental externalities caused by the use of pesticides, and the amount farmers use to compensate for reductions in naturally provided pest-control.

In this paper I exploit mortality shocks to bats and provide the first empirical validation that a reduction in the population of bats, which are a major predator of insects, causes farmers to compensate by using more pesticides. I use the quasi-experimental setting and report the first causally interpretable results which show an increase in the infant mortality rate, only for births conceived throughout the pesticide application season. This increase is driven mostly by the infant mortality rate of female infants. These results demonstrate that even when using each pesticide within its regulated limits there is an aggregate toxicological effect.

Identifying the causal effect of pesticides on health can be achieved with the ideal experiment of randomly increasing the use of pesticides across counties. Because this is not feasible I use a natural experiment in the form of a wildlife disease known as White Nose Syndrome which causes high mortality rates in bats that average at 73% (Frick et al. 2010). White Nose Syndrome emerged in the U.S. in 2006 and by 2012 has gradually spread to 188 counties, in 24 states (Blehert
et al. 2009). The pattern of disease dispersion follows both the migration paths of bats across counties as well as hiking trails that further disperse the spores of the fungus (Frick et al. 2010). This offers a quasi-random setting where the spread of the disease is neither a function of the pesticide use level, nor of the health outcomes in the county.

I use this setting of mortality shocks to bats - which lowers the predatory pressure on insects - and combine it with data on pesticides use on a county-year level. I demonstrate that counties exposed to White Nose Syndrome increase their use of insecticides and fungicides by 39.6%, and 20.1%, respectively. Use of fungicides increases because insects spread fungi across plants and further assist fungal dispersion by damaging the plant (Fennell et al. 1975; Kluth et al. 2002). I then use the quasi-random assignment of counties into higher pesticide use regimes and match them with data on birth outcomes. I use linked birth and death certificates and find that the infant mortality rate from non-violent causes for female infants goes up by 1.01 deaths per-1,000 births, representing an increase of 14.5% relative to the mean rate. I find evidence for a seasonal effect where only the births conceived during the pesticides application season of April-July are affected; further highlighting the importance of in utero exposure (Almond and Currie 2011).

An increased pest pressure should have adverse effects on crop yields. I find evidence that corn and soy yields go down by about 10% and 5%, respectively, right after the mortality shocks to bats. However, with the increased use of pesticides yields go back to their baseline levels prior to the mortality shocks. This suggests that the increased use of pesticides enables farmers to mitigate most, if not all, of the effects of the increased pest pressure. Results are robust to different specifications and sample definitions. The results for the use of insecticides and female infant mortality rate hold under randomization inference procedures.

White Nose Syndrome is caused by an invasive fungus and acts as a shock to the ecosystem. The fungus grows on the skin of the bats and essentially consumes it causing certain bat species to awaken during hibernation. They quickly deplete their fat reserves that were meant to sustain
them and do not survive the winter. Current estimates report an order of magnitude reduction in the population level of bats in affected sites (Frick et al. 2015). The research from recent years on White Nose Syndrome demonstrates that its emergence was unexpected and that the fungus was likely brought over as spores on the boots or backpacks of hikers from Europe (Frick et al. 2010). Since the fungus exists in Europe - where bats do not develop WNS - it provides strong support to the exclusion restriction that the fungus only affects pesticides use and fetal health outcomes through the channel of mortality shocks to bats. In addition, there are counties where the fungus has been detected but those counties only inhabit bat species that do not develop WNS. Controlling for the presence of the fungus without the disease results in no additional explanatory power. Combined, these features provide strong support for the necessary conditions for a causal interpretation of the results.

Experimentally manipulating the population level of some species of interest on a large scale is generally infeasible. This makes obtaining causal estimates of the effects of biodiversity loss a difficult challenge. Therefore, policy analysis often abstracts away from the economic importance of species. The unexpected introduction and effects caused by invasive species represent a source of exogenous variation. Under certain conditions, such shocks provide a natural experiment setting. I generalize this framework in a simple conceptual model which states the necessary conditions for identification and shows how these estimates can help to determine the optimal conservation policy. Theoretical work in the economic literature has addressed measuring biodiversity, valuing the marginal species, and setting the optimal conservation policy.¹ The optimal conservation policy is the solution to the optimization problem facing the central planner. It relies on knowing marginal direct and indirect costs and benefits from conservation. These are complicated functions and the current literature uses back of the envelope calculations, market and hedonic prices, replacement costs, and contingent valuation methods which do not accurately capture the associated trade-offs (Costanza et al. 1997; Brown 2000; Heal 2000; Atkinson et al. 2012).

Pesticides are regulated by the Environmental Protection Agency, the U.S. Department of Agriculture, and the U.S. Food and Drug Administration. Each pesticide is regulated individually and we do not know the effects of pesticide mixtures. Combining different pesticides can amplify their toxicity making their mixture effect a topic of great importance (Hernández et al. 2013; Cedergreen 2014). Pesticides are a non-source point pollutant that is susceptible both to wind and water erosion that lead to occupational and non-occupational exposures which could result in negative health effects.

Recent empirical work on the linkage between pesticides and health relies on relatively small and non-random samples which do not allow to draw causal estimates (Dolk and Vrijheid 2003; Andersson et al. 2014). Yet such papers find a seasonal effect where birth conceptions that took place during the period of the year when pesticides get applied suffer higher mortality rates (Regidor et al. 2004), and higher rates of birth defects (Winchester et al. 2009). However, these papers are unable to rule out any other seasonal effects other than the use of pesticides, and as a result, are only suggestive of a causal link between the two. Bretveld et al. (2006) review the literature on pathophysiological mechanisms through which pesticides can affect the reproductive system. They summarize that the main channel is disruption of hormonal activity, and in particular estrogen hormones.

The implication of these results relates to two main policy questions: the regulation of pesticides, and the conservation of bats. This paper provides evidence that pesticides have negative effects on fetal health which suggests that the current regulatory framework around pesticides is insufficient with respect of preventing such adverse health outcomes. This highlights that more attention needs to be dedicated to the mixture effects of pesticides and their presence in the air and water;
even at locations that are far from their application sites.

If bat populations decline and farmers use pesticides to compensate for the reduction of pest control then one approach to mitigate the health externality of pesticides is to preserve bat populations at sufficiently high levels. Other than White Nose Syndrome there are two additional threats to bat populations: land use change and wind energy (Fenton 1997; O’Shea et al. 2016). Land use change threatens to reduce the amount of habitat available to bats where they spend their hibernation period, and wind turbines are fatal to bats. If preserving bat populations is welfare improving then regulation could require additional steps to minimize the impact on bats, as it does for other endangered species. I elaborate on this issue in section 8.

The remainder of the paper is organized as follows: I cover the pest control provided by bats, the wildlife disease causing the mortality shocks, and the use of pesticides in the U.S. in section 2. In section 3 I review diversity theory in economics and introduce the framework of ecosystem shocks. I then present a conceptual model in section 4 which highlights the necessary assumptions for identification. I briefly describe the data in section 5, and detail the empirical strategy in section 6. I present the main results in section 7 and conclude in section 8.

2 Bats, White Nose Syndrome, Pesticides Use, & Birth Outcomes

2.1 Bats as Predators of Insects

Insectivorous bats consume 40% and above of their body weight in insects each night (Kunz et al. 2011). Both observational and experimental studies have demonstrated that bats limit the

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4 Bats die at wind farms as a result of either direct collision with the blades or abrupt changes in pressure around the edges of the blades. In response to the evidence on mortality of bats around wind farms the American Wind Energy Association announced in September 2015 that it will voluntarily stop the rotating of wind turbines on slow wind speed days during the fall migration season of bats (AWEA 2015).

5 Bats are nocturnal and spend their daylight hours at different roosting sites, ranging from caves to trees, before foraging for insects during the night. Bats forage for insects using either their echolocation abilities, olfactory senses, or passively monitoring noises made by insects to discern the location of their prey.

6 The dietary habits of bats have been a source of study since Poulton (1929).
growth of insects in forests (Kalka et al. 2008; Williams-Guillén et al. 2008) and in agricultural plots (Maine and Boyles 2015), and in particular insects that damage agricultural produce (Kunz et al. 2011).\textsuperscript{7,8} Results from field experiments show an increase in the densities of arthropods - of which insects are a sub-group of - on a range of 59%-84%, a 66% increase in fungal growth, and a 56%-68% increase in leaf damage (Williams-Guillén et al. 2008; Kalka et al. 2008; Maine and Boyles 2015). The connection between insects and fungal growth has been documented in the agronomic literature as far as 1975. The mechanism is that insects damage the crop in a way that opens pathways for fungal dispersion, as well as carrying the fungi themselves and spreading it from plant to plant (Fennell et al. 1975; Kluth et al. 2002).

2.2 White Nose Syndrome Causing Mortality Shocks to Bats

White Nose Syndrome (WNS) is an infectious wildlife disease which develops in certain bat species as a result of exposure to an invasive fungus species.\textsuperscript{9} The disease receives its name because the fungus grows around the nose of the bat creating a cluster of white flakes (Turner et al. 2011). The fungus spreads throughout the skin of the bat and essentially consumes the skin tissues.\textsuperscript{10} 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).\textsuperscript{11} The disease spread from 2 affected counties in New York state in 2006 up to 188 counties in 2012, in 24 states. Not all bat species develop symptoms such that there are counties where the fungus is present but there are no bats with

\textsuperscript{7} Observational studies examine the feces and stomachs of bats either by analyzing the fragments of such insects or by sequencing the DNA of insects in those samples.

\textsuperscript{8} The methodology employed by these experiments is similar: construct enclosures that prevent bats from passing through and place them over agricultural plots during the night (Williams-Guillén et al. 2008; Kalka et al. 2008; Maine and Boyles 2015).

\textsuperscript{9} Formally, the fungus is referred to as \textit{Pseudogymnoascus destructans}, previously referred to as \textit{Geomyces destructans}. The origin of the fungus is considered to be in Europe and it is thought that it was brought over as spores on the shoes or backpacks of travelers (Frick et al. 2010).

\textsuperscript{10} The symptoms of the disease involve 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 suppose to still be hibernating face an almost non-existent food supply and an increased caloric use rate due to the low temperatures, which is further worsened by the damage they suffer to their wings - and they generally do not survive the winter (Thomas et al. 1990; Blehert et al. 2009). For this reason many caves and mines used as hibernation sites have been sealed off with gates in order to prevent people from entering and awakening bats (Fenton 2012).

\textsuperscript{11} 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).
WNS. Figure 1 shows the gradual expansion of the fungus and the counties where bats develop the disease. Because the fungus can survive as spores even without an available bat host, no exposed counties return to non-exposed status.

The pattern of county contagion seems to follow the migration path of bats as well as hiking trails along the Appalachian. Yet ecologists are unable to predict exactly which counties will contract the fungus. 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).\textsuperscript{12}

Estimated bat mortality from WNS is extremely high making WNS to be considered by some wildlife ecologists as the worst wildlife disease ever documented.\textsuperscript{13} 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 (Ann and Jeremy 2012).\textsuperscript{14} The ecological literature argues that farmers will need to compensate for the decline in bat population levels by using more insecticides (Boyles et al. 2011; Kunz et al. 2011; Maine and Boyles 2015).

\subsection*{2.3 Pesticides Use & Their Environmental Externalities in the U.S.}

Farmers never know the true level of the pest population but use different tools such as pheromone traps to capture insects and decide on application intensity based on certain thresholds (Haynes et al. 1987; Blommers 1994; Way and Emden 2000). Pesticides are a form of non-point source pollution (Edwards et al. 1973; Lichtenberg and Zilberman 1988) that has the been the cause of

\textsuperscript{12} 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, and it appears that a radial dispersion pattern emerged around these new epicenters.

\textsuperscript{13} Thomas Risch, chair of the biological sciences department at Arkansas State University has said that “As far as we know, this is the worst wildlife disease [anyone has] been able to document.” Source: “A Killer in the Dark” http://arkansaslife.com/a-killer-in-the-dark/ Accessed: 05/03/2016. In addition, the Center for Biological Diversity states that “Biologists consider it the worst wildlife disease outbreak ever in North America.” Source: http://www.biologicaldiversity.org/campaigns/bat_crisis_white-nose_syndrome/ Accessed: 05/03/2016.

\textsuperscript{14} These large mortality shocks have resulted in the listing of the northern long eared bat as threatened under the Endangered Species Act in the U.S. (U.S. Department of the Interior and Service 2015). Two other species, the gray bat (\textit{Myotis grisescens}) and the Indiana bat (\textit{Myotis sodalis}) were already listed as endangered and are also negatively impacted by WNS.
environmental concern ever since “Silent Spring” by Carson (1962). The focus is centered around excess pesticides which are the pesticides that do not make contact with the target pest. It is estimated that only 0.1-0.3% of applied pesticides reach their target pests (Pimentel and Levitan 1986), while the rest are susceptible to wind and water erosion (Burkhard and Guth 1981; Pimentel and Levitan 1986; Hallberg 1989; Werf 1996). Persistence of pesticides in the environment is measured by half-lives: the time it takes for the original amount to reduce by half. Most insecticides have persistence values below 30 days, while other pesticides have values below 100 days. Few pesticides have values above 100 days (Howard 1991; Mahr, n.d.).

Using data on the concentrations of different agrichemicals, both fertilizer and pesticides, in surface water in the U.S. Winchester et al. (2009) find that elevated levels of agrichemicals during the period of April-July are correlated with higher birth defects prevalence rates. Regidor et al. (2004) find higher levels of still births and infant deaths within 24 hours. The effect is stronger during the months when pesticides are applied. Seasonal effects of non-point source pollution have also been documented with respect to fertilizer use in India (Brainerd and Menon 2014). The association of pesticides to fetal and infant mortality has been documented both in developing and in developed countries (Kristensen et al. 1997; Siqueira et al. 2010). The suggested mechanisms for the effects of pesticides on birth outcomes focus on changes to hormonal activity (Bretveld et al. 2006). Disruptions to the normal hormonal activity plays a crucial part during early stage of fetus development (Mnif et al. 2011).

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15 Surface runoff of pesticides is estimated to be between 2-10% of the amount applied and 94-97% of water samples in the U.S. between 1992-2001 had detectable levels of pesticides in surface water, while 33-61% of ground water samples had detectable levels of pesticides. Volatilization - where pesticide particles are picked up by the wind - is estimated to be the major cause of pesticide dispersion outside of the application field with erosion rates up to 90%.

16 However, that study aggregates all water measurements and all birth data across the U.S. and does not account for other seasonal effects such as higher ozone pollution levels in the spring (Monks 2000) and does not take into account spatial variation.
3 Diversity Theory, Invasive Species & Ecosystem Shocks

3.1 Diversity Theory

Empirical literature in economics and in ecology has reported different monetary values on the existence of species. However, these estimates are not informed by the decisions made by economic agents and are focused on cases where the species goes completely extinct; and therefore are unable to provide marginal estimates. These type of estimation techniques often end with raising the question of “is some number better than no number?” (Diamond and Hausman 1994; Kling et al. 2012).

Papers in the ecological literature suggest potentially large effects from biodiversity losses that are based on theoretical models or provide data from small scale experimental settings. However, they do not account for offsetting human behavior.\footnote{For the potential effects of biodiversity losses on socioeconomic system see Bianchi et al. (2006), Letourneau et al. (2009), Pereira et al. (2010), Hooper et al. (2012), and Meentemeyer et al. (2012); and Cardinale et al. (2012).} Diversity theory in the economic and the ecological literatures agree that: (i) precise estimates of the marginal benefits derived from biodiversity (or the marginal damages resulting from reductions in biodiversity) are greatly needed, and (ii) that estimating them is an intractable problem leading to a dearth of such estimates (Paulo and Jeroen 2001; Cardinale et al. 2012).

In his seminal paper from 1992, “On Diversity,” Weitzman (hereafter W92) suggests an objective function that takes into account both the direct benefits that species provide and their embedded evolutionary diversity. Maximizing such an objective function subject to a budget constraint that reflects the costs of conservation relies on knowing the marginal benefits that species provide. Because causally identified parameters are often not available W92 follows with developing a generalized diversity function which relies strictly on the hierarchical structure of the problem.\footnote{In the case of biodiversity this reduces to measuring the distance along an evolutionary (Phylogenetic) tree from the nearest neighbor. The intuition is that if we are interested in maximizing expected biodiversity then the value function decreases more with a loss of a species which has a more unique evolutionary history.}
3.2 Invasive Species & Ecosystem Shocks

We can rarely implement experiments in the field on a large enough scale such that they will have an effect on socioeconomic systems, yet we can use natural experiments which in this context I refer to as “ecosystem shocks.” Invasive species offer a source of such shocks. Non-native species or exotic species are new entrants that have managed to establish themselves in an ecosystem alongside the other incumbent species. They are considered to be invasive species if they cause damages to the incumbent species. The appearance of an invasive species is comparable to other forms of environmentally exogenous shocks (Dell et al. 2014).19

While it is not always clear why some species manage to invade and successfully colonize (Tsutsui et al. 2000) the fact that so many do creates a problem of a considerable magnitude. In the U.S. alone it is estimated that there are about 50,000 non-native species of which about 4,300 have been classified as invasive (Corn and Rawson 1999; Pimentel et al. 2005). In 2000 about 400 of the 958 species listed as threatened under the Endangered Species Act were listed due to their interactions with invasive species (Pimentel et al. 2000).

4 A Conceptual Model of Economic Production & Invasive Species

I describe a general structure connecting economic production, substitution between capital and a native species, an externality associated with the use of capital, an invasive species which negatively affects the native species, and a conservation policy. In this paper the native species are the bats, the invasive species is the fungus causing the wildlife disease, the capital is pesticides, economic production is agricultural activity, and the externality is negative birth outcomes. The conservation policy involves both land use and the expansion of wind energy, as wind turbines

19 Invasive species enter a new ecosystem either by natural processes of dispersal, unintentional human action, or intentional human action (Hulme et al. 2008; Pyšek and Richardson 2010). While the introduction of the non-native species might be intentional it is not expected it will cause severe damages and get classified as invasive (Hulme et al. 2008; Pyšek and Richardson 2010). In addition, the number of deliberate releases of non-native species has been declining over time (Hulme et al. 2008).
lead to mortality in bat populations.

First I begin with a simple formulation without the conservation policy. Population level of the invasive species determines the survival probability, $x$, of the native species - corresponding to the survival probability in W92. I begin by introducing the following assumptions:

1. **Substitutability of Native Species**: the contribution of the native species to the economic production can be substituted by capital, $K$, at a constant price $r$.

2. **Native Species Abundance is Increasing in Survival Probability**: the population levels of the native species, $N$, is a weakly increasing function of the survival probability, $x$. For now assume that survival probability is a function of the invasive species population level, $I$, such that $x \equiv x(I)$, and that $x$ is weakly decreasing in $I$.

3. **Emergence of Invasive Species is Unexpected**: the introduction of an invasive species is unintentional and was not known prior to its invasion.

4. **Exclusion Restriction**: the invasive species has no direct or indirect effects other than through the channel of the native species’ population level.

Next I outline the structure of the economy. Assume a production function of a final output, $Q$, as a function of capital and the population level of the native species. Under assumption (1) capital and the native species are substitutes such that:

$$Q \equiv Q(K, N), \frac{\partial K(Q)}{\partial N} \leq 0,$$  \hspace{1cm} (1)

The population level of the native species is a function the population level of the invasive species:

$$N \equiv N[x(I)] \equiv N(I), \frac{\partial N}{\partial I} \leq 0$$ \hspace{1cm} (2)

In addition to the direct costs borne by substituting $N$ with $K$, assume that there is an indirect cost of using $K$ in the form of an externality, $E$:

$$E \equiv E(K), \frac{\partial E}{\partial K} \geq 0$$ \hspace{1cm} (3)
Total welfare in the economy, $W$, is increasing in the final output, and decreasing in the cost of capital and the externality caused from using capital:

$$W \equiv Q(K, N) - rK - E(K) \quad (4)$$

We are interested in the marginal benefit to welfare from the increase in the abundance of the native species (see all derivations in Appendix B.):

$$\frac{dW}{dN} = \frac{\partial Q}{\partial N} (+) - \frac{\partial E}{\partial K} \frac{\partial K}{\partial N} (-) \geq 0 \quad (5)$$

The increase in production and the reduction in the externality are both welfare improving. Both are weakly increasing in $N$ such that the marginal increase in welfare from an increase in $N$ is weakly positive. While $\frac{dW}{dN}$ is the parameter of interest it is not feasible to experimentally manipulate the population levels of $N$ on a large scale. However, such an ideal experiment can be approximated by using an exogenous mortality shock resulting from an invasive species. The marginal change in welfare resulting from a marginal change in the population level of the invasive species is given by:

$$\frac{dW}{dI} = \frac{dW}{dN} \frac{\partial N}{\partial I} (+) \leq 0 \quad (6)$$

Under assumptions (3) and (4) the mortality shock is exogenous and only affects any outcome of interest through its effect on $N$. The natural experiment from the emergence of $I$ allows to identify the parameter of interest: $\frac{dW}{dN}$. Next I expand the model to include a conservation policy in order to demonstrate the policy implications of knowing the marginal contribution to welfare of species $N$. 

13
4.1 Optimal Conservation Policy

Assume there is a conservation policy, $P$, which has a direct effect on the survival probability of $N$ such that:

$$N \equiv N[x(P, I)] \equiv N(P, I), \quad \frac{\partial N}{\partial P} \geq 0, \quad \frac{\partial N}{\partial I} \leq 0$$  \hspace{1cm} (7)

Costs of the conservation policy are given by:

$$TC \equiv TC(P), \quad \frac{\partial TC}{\partial P} > 0$$  \hspace{1cm} (8)

Welfare in the economy is the same as before only now it includes the costs of the conservation policy:

$$W \equiv Q(K, N) - rK - E(K) - TC(P)$$  \hspace{1cm} (9)

The social planner’s problem is to set the optimal level of conservation policy which trades off direct net benefits in production and externality reductions, with increased costs associated with the policy. Taking the derivative of total welfare with respect to $P$ results in:

$$\frac{dW}{dP} = \underbrace{\frac{\partial Q}{\partial N}}_{\text{Marginal Benefits}} \cdot \frac{\partial N}{\partial P}^{(+)} + \underbrace{\frac{\partial E}{\partial K}}_{\text{Marginal Benefits}} \cdot \frac{\partial K}{\partial N}^{(-)} \cdot \frac{\partial N}{\partial P}^{(+)} - \underbrace{\frac{\partial TC}{\partial P}}_{\text{Marginal Cost}}^{(+)}$$  \hspace{1cm} (10)

Then the optimal conservation policy, $P^*$, solves for:

$$\frac{\partial Q}{\partial N} \frac{\partial N}{\partial P}^{(+)} + \frac{\partial E}{\partial K} \frac{\partial K}{\partial N}^{(-)} \frac{\partial N}{\partial P}^{(+)\text{}} - \frac{\partial TC}{\partial P}^{(+)\text{}} = 0$$  \hspace{1cm} (11)

$$\frac{dW}{dN} \frac{\partial N}{\partial P}^{(+)} = \frac{\partial TC}{\partial P}^{(+)}$$  \hspace{1cm} (12)

Assuming that the costs and efficacy of the conservation policy are known then knowing $\frac{dW}{dN}$ allows the central planner to set the optimal level of conservation. It is worth noting that observed changes in conservation do not provide a natural experiment setting because it is likely that both the exogeneity assumption and exclusion restriction do not hold. Generally, conservation will be responding to levels of $N$, and could have direct or indirect effects on $K$ and $E$. The contribution
of this paper can be seen as estimating the partial derivatives of interest. Explicitly: $\frac{\partial K}{\partial N}$, $\frac{\partial E}{\partial K}$, $\frac{\partial Q}{\partial N}$.

5 Data

Here I review in different data sources I use the paper. I provide more details in the online data appendix regarding how I combine them to create the main panel data set on a county-year level for pesticides use, White Nose Syndrome exposure, and birth outcomes. I report summary statistics for the main variables in Table 1.

5.1 Pesticides Use

Data from the U.S. Geological Survey (USGS) on estimated annual agricultural pesticide use for counties of the conterminous United States is available between 1999 and 2012 (Baker and Stone 2013; 2015). The USGS combines proprietary farm operation surveys from Crop Reporting Districts (CRD) with data from the U.S. Department of Agriculture National Agricultural Statistics Service (USDA NASS) on harvested crop area and calculates estimated pesticide use rates. This results in estimated used amounts for 489 different chemical compounds on a county-year level. I use data from the Environmental Protection Agency and multiple other sources to classify each active ingredient by type of product it is used in: insecticides, fungicides, herbicides, and other products.

I use the estimated pesticides data to construct three main variables of aggregated pesticide use, by pesticide type, on a county-year level. 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.

20 While data is available for 1992-2014 I end up using data from the period between 1999 and 2012 for two reasons: (i) the amounts used of pesticides in the 90s were much higher than they are around the period of exposure to WNS (see online data appendix), and (ii) data for 2013 and 2014 are still considered preliminary and are subject to revisions as more data become available. Results using the extended sample are similar and are available from the author by request.

21 A spreadsheet detailing the classifications and the references used to determine each classification is available by request from the author.

22 I also normalize by the size of mean harvested land in 1997 and 2002, available from the USDA Agricultural census. Harvested area is not reported in the annual USDA NASS reports and since the size of land used in agriculture is a likely outcome variable of interest it is endogenous to the emergence of WNS. Therefore I only
5.2 White Nose Syndrome Exposure Data

I use data from the Pennsylvania Game Commission on the detection of WNS across U.S. counties. Each county is classified as either confirmed, suspected, or not-detected. In Figure 1 I plot the gradual expansion of WNS across the Eastern U.S. Figure 2 shows the cumulative number of counties that are confirmed to have WNS each year. On average about 21 counties get exposed with WNS each year, with a minimum of 2 (conditional on post 2006) and a maximum of 54 counties.

As WNS expands across the Eastern U.S. more counties become exposed, and more counties have neighboring counties that are exposed. I exploit the spatial pattern of expansion in the following way: (i) I classify the neighboring relationship between all counties such that I know the first degree neighbors of each county, the second degree neighbors (the neighbor of the neighbor), up to the fifth degree; (ii) I then construct a variable, on a county-year level, with the share of first degree surrounding neighbors that are exposed to WNS, such that a value of 0 represents a county with no immediate exposed neighbors, and a value of 1 represents a county fully surrounded by exposed counties,23 (iii) finally I define subsamples of the full data set based on neighboring proximity to exposed counties such that each subsample has all the exposed counties and the first (or second, or third, etc.) degree neighboring counties.

5.3 Birth Outcomes

I use data from the National Center for Health Statistics - National Vital Statistics System (NCHS-NVSS) on birth and death certificates from 2003-2013. The data include all the birth certificates for each birth cohort as well as all the death certificates that can be linked with their birth certificates. I focus on the share of first degree surrounded counties because most bats have a nightly flight radius of 20-30km, and the maximum flight radius is 50km. The distance, measured as distance from county centroid to county centroid, of some of the immediate neighbors falls below that threshold while the distance of counties to their second degree neighbors falls almost exclusively beyond that threshold. In Figure A1 I plot the histograms of the centroid distances between counties and their first and second degree neighbors. While not all first degree neighbors fall below the 50km threshold, it is evident that most second degree neighbors are well above that threshold.

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use values prior to the onset of WNS which is 2006. For conciseness I do not report these results, which are similar, here. They are available by request from the author.
I use the data to calculate number of birth conceptions, infant mortality rates, fraction of low birth weights, and fraction of preterm births. Starting in 2003, birth certificates also record the last month of the mother’s menstrual cycle which serves as a proxy for the month of birth conception. I aggregate the fetal health outcome variable by county, year, birth conception month, and sex. Using data on the month of conception allows to compare pregnancies that start throughout the pesticides application season to those that begin earlier or later.

### 5.4 Crop Yields & Harvested Land Area Data

Data on crop yields for corn and soy yields, two of the most important crops in the U.S. are from the USDA NASS. Data on the amount of harvested land is reported in the agricultural census which is conducted in 5 year intervals. I use data on harvested land to normalize pesticide use values or to weigh observations. I use data from the Agricultural Census on chemical expenditure on the county-year level to interpolate for chemical expenditure between ag-census years. The idea is to use the data on the chemical use by compound from USGS and the expenditure on chemicals to develop a price index through which I calculate the chemical expenditure on years with data only for the chemical use. I cover in detail the construction of the interpolated chemical expenditure in the online data appendix.

### 5.5 Weather Data

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 between 10 degrees Celsius and 30 degrees Celsius, and number of degree days above 30 degrees Celsius, as well as data on precipitation, all on a county-year level. I construct weather variables for the agricultural growth period of April-September, as well as the nine month trajectory of degree days for each month-year. This allows to account both for the effects of temperature on agriculture (and the use of pesticides) as well as the direct effects of temperature throughout the pregnancy period.

---

24 I also construct different degree day bins for robustness checks. The choice of the cutoff around 30 degrees Celsius is based on the observed non-linear responses of crops to temperatures that appear above that threshold (2009).
6 Empirical Strategy

I run a Difference-In-Differences model which uses counties with bats that were exposed to White Nose Syndrome as the treatment group, and the counties with no exposed bats as the control group. The main outcome variables I am interested in are the use of pesticides and birth outcomes. The identifying assumption I make is that counties that were exposed to White Nose Syndrome would have had similar trends in pesticides use and birth outcomes to the non-exposed counties - had they not been exposed. The unexpected emergence of WNS and its gradual expansion such that counties phase into the treatment group results in a natural experiment (Meyer 1995; Greenstone and Gayer 2009) which approximates the ideal experiment of randomly selecting counties and manipulating the population levels of bats, and as a consequence, the pesticide use levels as well.

6.1 Average Treatment Effects

I estimate the generalized diff-in-diff model using the following specification:

$$y_{ct} = \beta_1 WNS_{ct} + \beta_2 SurroundingWNS_{ct} + f(Weather_{ct})\theta + \lambda_c + \lambda_{st} + \varepsilon_{ct}$$  \hspace{1cm} (13)

Where $y_{ct}$ is either the aggregated insecticides, fungicides, or herbicides normalized by county-area; the log of crop yields; chemical expenditure; or the birth outcome. I use levels of aggregated chemicals because farmers’ decisions regarding insecticides applications rely on whether the number of insects captured in a pheromone trap exceeds a certain subjective threshold. This lends towards more of a levels effect than a rate effect. I report results for a log specification in the appendix. The treatment variable, $WNS_{ct}$, is a dummy variable which receives the value of 1 for county $c$ at time $t$ if WNS has been detected in the county. Meaning once a county gets exposed to WNS this variable “turns on” and remains on throughout the sample. The parameter of interest is $\beta_1$ which captures the average effect of getting exposed to WNS on the outcome of interest.

The second treatment variable is $SurroundingWNS_{ct}$ which is the share of surrounding first degree neighboring counties, relative to county $c$, that are also exposed to WNS at time $t$. If none
of the counties that immediately surround county $c$ at time $t$ are exposed then this is equal to 0, if all those counties are exposed then this is equal to 1. I limit the interest to first degree counties because bats have a flight radius of up to 50km a night and most counties have their first degree neighbor within that distance, but almost all their second degree neighbors are beyond that distance threshold; which I demonstrate in Figure A1. This is used to capture heterogeneous effects because it is likely that an exposed county surrounded by other exposed counties is experiencing an even greater loss of pest control due to WNS than counties surrounded by non-exposed counties.\footnote{Bats from non-exposed counties might still forage for insects in exposed counties and provide pest control that could offset the effects of the mortality shock to bats in the exposed county.}

I control for weather by including degree days between 10 and 30 degrees Celsius and above 30 degrees Celsius - between April and September, as well as a quadratic function for precipitation - represented by the vector of variables and coefficients $f(\text{Weather}_{ct})\theta$.\footnote{I also run specifications with a more flexible bin construction for degree days which yield similar results but are not reported here.} Weather controls account for any variations to the application of pesticides that are governed by growing conditions such as rainfall and temperature. I include county and state-year fixed effects, controlling for any time-invariant unobserved county characteristics, and for flexible time-trends on the state level, respectively. Any remaining unobserved heterogeneity will be part of the error term, $\varepsilon_{ct}$, which I cluster at the county level.

### 6.2 Testing for Parallel Trends

The necessary assumption for a consistent estimate is that conditional on the observable variation the variable of interest and the unobserved heterogeneity are not correlated: $E(WNS_{ct}\varepsilon_{ct}|X_{ct}) = 0$.\footnote{where $X_{ct} = \{\text{SurroundingWNS}_{ct}, f(\text{Weather}_{ct}), \lambda_c, \lambda_{st}\}$} The necessary assumption for identification of a causal estimate is that the counties in the treatment group would have had parallel trends to those in the treatment group, had they not been exposed to the treatment. This assumption cannot be tested but parallel trends between treatment and control counties before exposure can be tested to strengthen the validity of the parallel trends (on the counterfactuals) assumption. I test this formally with a flexible generalized
difference-in-differences specification:

\[
y_{ct} = \sum_{r=-6}^{-1} \beta_r \mu_{rc} + \sum_{r=1}^{5} \beta_r \mu_{rc} + \alpha \text{SurroundingWNS}_{ct} + f(\text{Weather}_{ct})\theta + \lambda_c + \lambda_{st} + \varepsilon_{ct} \quad (14)
\]

Where \( \mu_{rc} \) is a dummy variable receiving the value of 1 whenever the county is \( r \) years away or from exposure.\(^{28}\) This results in a set of dummy variables which identifies for each exposed county whether it is 7 years or more away from exposure (\( \beta_{-6} \)) which is bottom coded as the longest pre-exposure period, ..., 2 years away from exposure (\( \beta_{-1} \)), year of exposure (\( \beta_1 \)), one year after exposure (\( \beta_2 \)), ..., 5 years after exposure (\( \beta_6 \)); with 5 top coded as the longest exposure period. Meaning that instead of an average treatment effect of getting exposed to WNS this regression model will estimate separate treatment effects as a function of the exposure length. Notice that the effects are all estimated relative to the omitted category of the year just prior to exposure (there is no \( \beta_0 \)) which is normalized to be 0. Control counties always have a value of 0 for each pre and post exposure dummy. This specification enables to compare counties in the treatment group versus the control group prior to exposure. If the identifying assumption holds we should expect that the coefficients on the years prior to treatment are close to zero. All remaining variables are the same as in Equation (13).

6.3 Exploiting the Seasonal Application of Pesticides

I exploit data on the month of conception in the linked birth and death certificates to further test whether conceptions that occur throughout the pesticides application season, April-July, experience a greater detrimental effect. I run a model of birth outcome on a county-year-conception month level with a full set of interactions between the calendar month and exposure to WNS. The formal specification is given by:

\(^{28}\) Formally: \( \mu_{rc} = I\{\text{Year-}(\text{Year Detected})_{ct} = r\} \)
\[
    h_{cym} = \sum_{m=1}^{12} \beta_m WNS_{cy} \times \mathbb{1}\{\text{Month} = m\} + \alpha SurroundingWNS_{ct} + g(W\text{eather}_{ct})\theta + \\
    + \lambda_c + \lambda_y + \lambda_m + \varepsilon_{cym}
\]

Where \( h_{cym} \) is the fetal health outcome for birth conceptions in county, \( c \), in conception year \( y \), and conception month \( m \). \( WNS_{cy} \) is the treatment dummy denoting exposed counties, \( c \), in year, \( y \); which is interacted with a dummy for each calendar month, \( \mathbb{1}\{\text{Month} = m\} \). Each \( \beta_m \) represents the average treatment effect of being exposed to WNS for conceptions that occur in month \( m \), relative to conceptions that occur in counties that are not exposed to WNS, also in month \( m \).

I control for degree days and precipitation as in equation (14) and add variables that control for the degree days trajectory nine months from month \( m \). The former degree days control for the effect for temperature on the pesticide use decisions (the indirect effect of temperatures on fetal health), while the latter control for the effect of temperature throughout the pregnancy (the direct effect of temperature on fetal health). I include a set of county, year, and calendar month fixed effects to average out any county time invariant effects, pooled shocks that occur in different years, and to remove any common seasonality within the year. \( \varepsilon_{cym} \) represents any unobserved heterogeneity. I cluster the standard errors by county.

### 6.4 Sample Definition

My preferred sample includes all the exposed counties and the subsample of their neighboring counties up to the 3\textsuperscript{rd} degree neighbor. Limiting the sample in this manner allows both the treatment and control counties to lie within similar agroecosystems. Results are not sensitive to the neighboring degree and for conciseness I report summary results for the standard model for

\[\text{Meaning that for conceptions that occur in January I include degree days between 10 and 30 degree Celsius, above 30 degree Celsius - for the period of April through September, and I add the number of degree days between 10 and 30 degree Celsius, above 30 degree Celsius for the period of January to September.}\]
the 3rd degree neighbor and the full sample, and results for the flexible generalized model for the 3rd degree neighbor only.30

6.5 Sample Weights

I choose sample weights depending on the outcome variable. Because the outcomes are averages on the county level I choose weights that represent the sample size on which they were calculated. For the pesticides data I use the amount of harvested land in 2002. Because the amount of harvested land is a likely outcome variable I use values prior to WNS exposure. However, because harvested land is reported in the agricultural census which takes place every 5 years I do not have values for 2005. Instead I use the values from the closest pre-exposure ag-census year which is 2002. This assigns a greater weight to counties that have a larger share of their area under agricultural cultivation.

For the birth outcomes I use either the number of birth conceptions or the estimated female population in the county between the ages of 15 to 49 which is considered in the demographic literature as the population which is exposed to pregnancy (H. Arthur et al. 2011). This assigns a higher weight to rates and fractions of counties with more birth conceptions, or with a larger exposed population in case conceptions are endogenous. This is especially important as there is heterogeneity between counties with respect to the number of conceptions. Weighting birth outcomes equally can potentially allow abnormal and unstable rates that are calculated based on a small number of cases to bias the analysis. For the log crop yields I use the baseline yield values between 1999 to 2005, in order to allow the rate effect to be determined more by the counties who had higher yields. This is because an increase from 1 to 2 has the same rate effect as an increase from 10 to 20, yet they are very different increases and weighting by baseline values takes that into account.

30 Results for any other sample definition are available upon request from the author.
6.6 Falsification Tests

I run two main falsification tests to further rule out any unobserved confounding factors that can contribute to a spurious effect. First I report results for herbicides use that according to the ecological and agronomic literatures should not respond to a mortality shock to bats. The second falsification test uses data from the death certificates regarding the cause of death of the infant. I compare the effect of WNS exposure on non-violent causes of death versus only violent causes of death (homicide and accidents). If the suggested mechanism of increased exposure to pesticides is indeed causing changes in mortality rates then it should only be captured by the naturally occurring deaths but not by the violently caused deaths.

7 Results

7.1 Pesticides Use

I report the results for the average effect of exposure to WNS on all three pesticide types in Table 2, across the 3rd degree neighbor sample (columns 1-3) and the full sample (columns 4-6). I first review the results from the 3rd degree neighbors sample which allows treatment and control counties to reside in a similar agroecosystem. On average, the use of insecticides and fungicides goes up by 2.54kg·km⁻² (column 1), and 1.66kg·km⁻² (column 2), respectively, while holding everything else constant. Results are statistically significant at the 1% and 5% levels and represent an increase, relative to the mean, of 39.6% and 20.1%, respectively. The use of herbicides appears to go down by 0.36kg·km⁻² (column 3) but is imprecisely estimated, and is only 0.7% relative to the mean amount used. Herbicides provide a falsification test as they should not respond to a mortality shock to bats.

Point estimates for insecticides and fungicides remain similar for the full sample estimation. However, the average use of insecticides and fungicides in the full sample is higher, such that the relative effects are smaller compared to those means. This suggests that states with no counties

that are exposed to WNS might serve as a weak control group to exposed counties in other states.

The coefficient for the share of first degree neighbors that are exposed to WNS is positive for both insecticides and fungicides, but is not statistically significant. That coefficient changes signs in the case of herbicides and becomes statistically significant at the 1% level for the 3rd degree neighbor sample. This could be explained if farmers are substituting away from herbicides, either because of budget constraints or regulatory constraints. However, the share of exposed surrounded counties is a crude measure of the heterogeneous treatment effect as it does not take into account the different number of roosting sites, different number of susceptible and non-susceptible bat species, and the location of bat sites relative for cropped areas, as well as the foraging behaviors of bats. These are all important channels that require much higher granularity of data in order to test them.

Results for the flexible diff-in-diff specification, in event time, for the 3rd degree neighbor sample are presented in Figures 3, 4, and 5 for aggregated insecticides, fungicides, and herbicides, respectively, in levels and in logs. There do not appear to be pre-exposure trends with respect to insecticides and fungicides. The use of herbicides appears to be trending down in levels, but not in logs. This means there are parallel pre-trends with respect to insecticides and fungicides use between the treatment and control counties; our two main pesticides of interest. Results in for the log of insecticides or fungicides replicate similar parallel trends but with imprecise point estimates that suggest smaller rate effects.

There appears to be a jump in the period right before exposure to WNS which could result from imprecise assignment of treatment status. I use treatment status assigned by the Fish and Wildlife Service which relies on collecting and analyzing samples in order to confirm the existence of WNS in the county. Several wildlife biologists from the different state agencies consider that exposure date to be earlier than the confirmed date. There are 15% of counties that have their presumed
WNS status at an earlier year (up to 4 years) than the confirmed WNS status.\textsuperscript{32}

There appears to be a cumulative effect such that with a longer duration of exposure the use of insecticides and fungicides within the treated counties increases more. The finding that the use of insecticides and fungicides correlate so well agrees with previous experimental evidence and agronomic knowledge, which was covered in section 2.1. As the use of insecticides increases and mitigates the problem of increased insect pressure then the spread of fungi by insects should be reduced as well. This can explain why the effect of fungicides appears to reverse its trend for the longer exposure periods. Results for herbicides show do not show a mean shift in post-exposure period to that in the pre-exposure periods, especially when measured in logs. This provides a falsification test as herbicide should not respond to mortality shocks to bats.

A possible explanation for the apparent lagged effect in exposure to WNS could result from two main reasons. First, it takes time for the disease to spread and cause a substantial mortality shock.\textsuperscript{33} A second reason for the lagged effect is that the true level of insect pressure is noisily measured by the farmers. Farmers observe the damages caused by insects and they use pheromone traps to monitor their population levels. It might be the case that the combined effects of slow dispersal of WNS and the time it takes farmers to update regarding the new environmental condition lead to the observed dynamic pattern.

### 7.2 Birth Outcomes

Number of births does not appear to be affected by the shock of WNS exposure. The results in Figure 6 show basically no change in the log of births conceptions in the April-July period for either males or females. The null effect on birth conceptions suggests that there is no increase on miscarriages. This allows to use the number of conceptions as weights for the different rates in

\textsuperscript{32}I am unable to show results using the WNS status assigned by local state agencies until that data is released to the public.

\textsuperscript{33}This will take longer in counties with a higher number of roosting sites and hibernacula as it will take longer for the disease to spread across the county. In addition, different counties have a varying number of bat species that are susceptible and that develop WNS. As WNS spreads in a county the mortality rate increases and the pest control provided by bats is reduced even more.
the following regressions. In Figure 7 I plot the effect of WNS exposure on the infant mortality rate (IMR) for both sexes, for all causes. IMR increases post exposure but appears to be falling after longer exposure. When separating the effects of WNS exposure on IMR by sex, Figure 8, it appears that the response of IMR is mainly driven by the effect on female IMR.

I report the results from the specification in Equation (15) in Figure 9b which shows the average difference between exposed and non-exposed counties with respect to their female infant mortality rates within the year. I plot the results from the separate regressions for non-violent and violent causes which demonstrate that there is only an effect for the non-violent deaths that occur between April and July. There appears to be no seasonal effect for the violent deaths. This seasonal effect has a causal interpretation because it is comparing mortality rates in each calendar month between treatment counties that are exposed to WNS and control counties that are not exposed.

I show the effect of WNS exposure on the female infant mortality rates, for non-violent and violent causes, per 1,000 births for the conceptions that take place during April-July in Figure 10. In table 3 I report the average effect of an increase of about 1.01 deaths per 1,000 births relative to non-exposed counties throughout the April-July period (column 2). This is significant at the 5% level and represents an increase of 14.5% relative to the mean female infant mortality in that period. In Table 4 I report small and imprecise effect for male IMR. Only the coefficients on the surrounding WNS is statistically significant, but has a negative sign (columns 2, 3, and 4).

In Tables 3 and 4 I also report the results for other periods of January-March (columns 1 and 4) and September-December (columns 3 and 6). I omit August as a buffer month as some pesticide applications might still take place in August. For female IMR from non-violent causes the January-March result (column 1) is positive but is only statistically significant at the 10% level while the September-December result is not statistically significant different from zero (column 3). In addition, I report the results for all three periods for the female infant mortality rates which are calculated using only the violent causes of death. Throughout the different periods the effect
is always small and non-significant (columns 4-6). These results help to rule out alternative unobserved causes for the increase in the female infant mortality rate. The results for male IMR are small and imprecise throughout the different estimated time-windows.

The results on the increased use of insecticides and fungicides, as well as the increase in female infant mortality can be summarized in an elasticity of pesticides on infant mortality on the range of 0.36 to 0.72, with an average of 0.48.\textsuperscript{34} This elasticity is comparable with similar estimated effects of other environmental pollutants on infant mortality rates. Chay and Greenstone (2003) find an elasticity of 0.3 for air pollution.\textsuperscript{35} Brainerd and Menon (2014) report an elasticity of 0.46 of infant mortality for birth conceptions that were exposed to fertilizer.\textsuperscript{36} However, Currie and Neidell (2005) report lower estimates that represent an elasticity of 0.09 with respect to CO concentrations on infant mortality.\textsuperscript{37} Overall the results are comparable in terms of magnitude to the effects of other forms of environmental pollution on infant mortality rates.

### 7.3 Crop Yields

I focus on corn and soy yields as those are the two crops grown in a large number of both treatment and control counties. I report the results for the generalized diff-in-diff model in Table 5, and the results for the flexible diff-in-diff model for corn and soy in Figures 11a and 11b, respectively. The results in Table 2 for the generalized diff-in-diff model suggest that corn yields drop by about 5% on average (column 1), which is significant at the 1% level. Soy yields do not change post WNS exposure (column 2). However, the results from the flexible diff-in-diff model for corn and soy in Figures 11a and 11b, respectively, suggest that there is an initial drop of about 10% and 5% in

\textsuperscript{34} An increase of 1% in the use of pesticides leads to a 0.48\% increase in the female infant mortality rate for birth conceptions that take place during April-July.

\textsuperscript{35} The authors use the 1981-1982 recession in the U.S. as a natural experiment for reductions in air pollution on the county level. They find that a 25\(\mu\)g·m\(^{-3}\) reduction in total suspended particulates leads to 1.25 fewer infant deaths per 1,000 births, which represents about a 10\% decrease.

\textsuperscript{36} The authors construct a measure for the presence of fertilizer chemicals in water in India on the state level and merge it with data from three rounds of Demographic and Health Surveys from 1992-1993, 1998-1999, and 2005-2006. They match each birth to its state, year, and month of conception and merge it with water quality data from the Central Pollution Control Board.

\textsuperscript{37} The authors use record level data on births in the state of California in the years 1989-2000 and match them with pollution levels for CO, PM10, and O3. The strongest effect they find is for CO concentrations where an additional unit increase in CO is associated with 0.1825 infant deaths per 1,000 births.
corn and soy yields in the 2nd period of exposure but yields appear to return to their pre-exposure levels over time. This appears to agree with the results on the use of insecticides and fungicides that appear to start going up after the 2nd period. This suggests that there is some delay in the effects of WNS on bats and insect populations, as well as an updating process by the farmers about the optimal application levels of pesticides. Chemical expenditure per-sq. km increases on average by $1,150 but is only significant at the 10% level (column 3). This represents a 12.6% increase relative to the mean. Given that insecticides and fungicides increase by more than 20% this suggests that farmers are able to change their pesticide inputs with an expenditure elasticity that is less than unity.

7.4 Robustness Checks

7.4.1 Validity of the Exclusion Restriction

To test for the validity of the exclusion restriction I use the counties where the fungus has been detected but there are no bats that develop WNS. If the fungus causes other direct or indirect effects that do not operate through the channel of bat mortality then estimating a model that controls for the fungus only counties should provide more explanatory power. Formally, I test:

\[ y_{ct} = \beta_1 WNS_{ct} + \beta_2 SurroundingWNS_{ct} + \beta_3 FungusOnly_{ct} + f(Weather_{ct})\theta + \lambda_c + \lambda_{st} + \varepsilon_{ct} \] (16)

Where \(FungusOnly_{ct}\) is equal to 1 for county, \(c\), at year, \(t\), once the fungus has been detected but WNS has not been confirmed in the county. All other variables are the same as in Equation 13. In table 7 I report the results that include the dummy for the presence of the fungus only. For all three outcomes of: insecticides use, fungicides use, and female IMR - the coefficient for having only the fungus, \(\beta_3\), is small and imprecise. The coefficients on WNS exposure and the share of surrounding WNS counties remain unchanged. This supports the necessary assumption for causal identification that the fungus operates solely through the channel of bat mortality.
7.4.2 Randomization Inference

To further rule out any spurious effects I use Randomization Inference methods (Fisher 1966; Young 2015). I randomly assign treatment status in the 3rd degree 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). 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.

In Figure 12 I report the result for the three types of randomization tests for the three main outcome variables for which I reject the null hypothesis of an average treatment effect of 0. Results for the aggregated use rate of insecticides and female infant mortality for birth conceptions that took place in April-July appear as right tail draws from the distribution of coefficients generated by the randomization inference procedure. This suggests that the previously reported models are not capturing a spurious effect or an effect generated by not properly controlling for time-trends of time-invariant properties. However, the result for the aggregated use rate of Fungicides does not appear to be in the tail of the distribution therefore the previously reported results regarding fungicides should be interpreted with caution.

7.4.3 Substitution Between Pesticide Types

To further rule out that the use of herbicides is increasing as well I test for substitution of more insecticides and fungicides relative to herbicides by constructing a sample on the county-year-pesticide type level and estimating the following specification:

\[
y_{ctk} = \beta_1 WNS_{ct} \times Insecticide_k + \beta_1 WNS_{ct} \times Fungicide_k + \\
+h(\text{SurroundingfWNS}_{ct})\pi + \lambda_{ct} + \lambda_{ck} + \lambda_{kt} + \varepsilon_{ctk} \\
\]  

(17)
Where $y_{ctk}$ is the aggregate pesticide type, $k$, at year, $t$, for county, $c$. $WNS_{ct} \times Insecticide_{k}$ and $WNS_{ct} \times Fungicide_{k}$ are the interactions between the dummy variable for WNS exposure with the dummy variable that is equal to 1 when the aggregate pesticide is the aggregated insecticides or fungicides, respectively. I control for the share of first degree surrounding counties that are exposed to WNS with $h(SurroundingWNS_{ct})$, which is either a simple interaction between that share and the pesticide type dummy, or the bins of that share in 20% intervals interacted with the pesticide type dummies. I control for county-year fixed effects that account for flexible time trends on the county level, pesticide type-year fixed effects which control for flexible time trends for the different pesticide types, and pesticide type-county fixed effects to capture any time-invariant heterogeneity with respect to the pesticide type on the county level. Any remaining unobserved heterogeneity is captured by $\varepsilon_{ctk}$ which I cluster by county.

Including a rich set of fixed effects non-parametrically controls for temporal effects. This allows to rule out any year effects, either on the county or the pesticide type level, that are not correlated with the gradual phasing of counties into the treatment group. The coefficients do not have an intuitive interpretation as those from Equation 13 because now the omitted category is the use of herbicides in non-exposed counties. However, the sign and precision of the coefficients supports the previous findings.

In Table 6 I report the results for the pesticides substitution specification. First, without controlling for the share of surrounding WNS counties the interactions between WNS exposure and the dummies for insecticide or fungicide are positive and significant at the 1% level (column 1). The coefficients for the continuous share of surrounding WNS counties interacted with the insecticide and fungicide dummies are positive and significant at the 1% level (column 2). The inclusion of the surrounding share of WNS counties makes the coefficients on the dummy for exposure significant.

38 In the simple interaction case: $h(SurroundingWNS_{ct}) = SurroundingWNS_{ct} \times Insecticide_{k} + SurroundingWNS_{ct} \times Fungicide_{k}$. In the bins case: $h(SurroundingWNS_{ct}) = \sum_{i \in 40\%,60\%,80\%,100\%} 1\{SurroundingWNS_{ct} \in [i - 20\%, i)\} \times (Insecticide_{k} + Fungicide_{k})$. Such that the share of surrounding WNS counties is binned as either between 20-40%, 40-60%, etc. and is interacted with the dummies for the pesticide types.
on the 10% level for insecticides but are no longer precise for fungicides (column 2).

Flexibly accounting for the share of surrounding WNS counties by using bins (column 3) reveals that the effect of becoming surrounded by counties that are exposed to WNS increases both in terms of magnitude and precision with the increase in the share of such surrounding counties. In column 4 I report the linear combinations for the average effects plus the effect of of each surrounding share bin. This allows to test for the joint significance of the effects. Insecticide and fungicide interactions are significant at 1% level across all the combinations.

7.4.4 Measurement Error

In the process of aggregating the different chemical compounds I am also summing any measurement errors in the estimation of the use rates of each compound. These errors are likely correlated and there is no reason to believe that they cancel each other out. To address this I identify the most used pesticide by pesticide type in each county and test its response to WNS exposure. This reduces the outcome variable to an estimated use rate of one compound and should alleviate the aggregation of measurement errors. Another reason to focus on the most used pesticide by pesticide type is that farmers in each county face different environmental conditions, different pest types, and are growing different crops. As a result farmers might have different optimal choices reflected in different rankings in terms of pesticide use.39

I report the results from the flexible diff-in-diff specification for the most used pesticides, by pesticide type, in Figure A2. The results appear to be similar to those of the aggregate pesticides: on average, counties exposed to WNS increase their use of insecticides compared to non-exposed counties and parallel trends are still evident; there do not seem to be systematic differences between treatment and control counties prior to exposure. Fungicides are again less precisely estimated

39 Specifically, I calculate the average use of each compound in a baseline period of 1999-2005 and assign for each county the most used, by volume, insecticide, fungicide, and herbicide. I then construct a most used insecticide variable by pulling the county-specific compound which was ranked as the most used insecticide for that county. I repeated this for fungicides and herbicides as well. It is important to note that not all compounds within each county are successfully matched in this manner, resulting in a smaller number of counties in both control and treatment groups.
but present a similar pattern of increasing with exposure length. Generally there seems to be no
response of herbicides to WNS exposure relative to pre-exposure periods.

7.4.5 Balancing on Composition

Because counties are exposed at different years this means that the composition in the treatment
group changes with the length of exposure. To clarify this point consider the following example.
Assume three different counties with some outcome \( Y_{ct} \) we are interested to measure as a result
of some treatment. Each county gets treated in a different calendar period denoted by \( T_c \) such
that in our data the first county has one period of post-exposure, the second county has two,
and the third county has three periods. For simplicity assume that each county has a 0 baseline
prior to treatment: \( Y_{ct} = 0 \forall c \in \{1,2,3\}, t < T_c \). If each county has a constant treatment
effect within county, but it is different across counties then it will appear that there is a dynamic
effect. Again, for simplicity assume that each county’s constant treatment effect is given by:
\( Y_{ct} = c \forall c \in \{1,2,3\}, t \geq T_c \).

As in similar settings I deal with the issue of compositional changes in the treatment group by
selecting an arbitrary treatment length cutoff, \( \bar{T} \), for the treatment group. Each county in the
treatment group has at least \( \bar{T} \) periods of being treated and any counties that have less than
that are omitted (Reber 2005). If the effect is driven solely by composition then we should
expect to see a flat treatment effect over time with only the last effect spiking up as it includes
information on exposure that is longer than \( \bar{T} \). If the treatment effect is indeed a dynamic one

\[40\text{This is not a threat to identification but if there is heterogeneity in the treatment effect such that counties}
\text{that get exposed earlier have a different response, either higher or lower, to WNS exposure then we might ob-
serve what appears to be a dynamic effect where in reality it is simply a heterogeneous one. By a dynamic}
effect I mean an effect that is a function of the treatment length. In this setting it translates to: counties that
are exposed to WNS for a longer duration end up increasing their use of insecticides more relative to those
with a shorter exposure period.}

\[41\text{Meaning that post-treatment the first county goes from 0 to 1, the second county goes from 0 to 2, etc.}
\text{I visualize this simple setting in Figure A3a. The average treatment effect at each post treatment period is the}
average of the constant treatment effects across the counties that are included in that post treatment period.
The first average treatment effect for the post treatment period is 2, followed by 2.5, followed by 3. This can
easily be interpreted as a treatment effect that is a function of treatment length.}

\[42\text{This means that the composition of the treatment group at each treatment period is the same. The treatment}
dummy for \( \bar{T} \) is now top coded such that any exposure period beyond \( \bar{T} \) is captured in that coefficient as well.}

32
then while holding the composition constant we should see an increase in the estimated effect at each exposure period, and not just the last one. This is summarized graphically in Figures A3b and A3c.

When balancing the sample on composition I choose the set of counties that are exposed for at least 4 periods and above.\textsuperscript{44} This ensures that each average treatment effect is estimated using the same group of counties. The results in Figures A4 and A5 demonstrate that the point estimates for the use of insecticides and fungicides are increasing for each post-exposure period, but not for herbicides. This supports the interpretation that there is a dynamic effect that it is not solely an artifact of treatment group composition.

7.4.6 Spatial Dependence of Standard Errors

Because the exposure to disease as well as the intensity of the treatment are partially a function of the spatial arrangement of counties the standard errors could be correlated such that it is not captured by clustering them at the county level. I relax this both by clustering at the state level and by correcting the covariance-variance matrix to include spatial dependence as a function of distance between county centroids as suggested by Conley (1999) and Bester et al. (2008).

In Table A1 I report the the adjusted standard errors. Clustering by state changes the significance level of insecticides from 1% to 5%, of fungicide from 5% to 1%, and of female IMR from 5% to 1%. Adjusting standard errors for spatial correlation tends to reduce the standard errors for the average treatment effect result for insecticides and fungicides. However, standard errors for the infant mortality result tend to be larger, suggesting that there are other potential determinants for infant mortality that co-vary across space. This tends to make the result on female IMR statistically significant at the 10% level.

\textsuperscript{44} Results for different cutoffs are available by request from the author.
8 Conclusions

In this paper I report findings from a natural experiment where a wildlife disease, known as White Nose Syndrome, caused large mortality shocks to bats. I demonstrate that farmers compensate for the reduction in pest control which is provided by bats by using more insecticides and fungicides; as suggested by the ecological and agronomic literatures. I use this quasi-experimental setting where WNS exposed counties increase their use of pesticides, which happens in different years for different counties, to test for the health effects of pesticides exposure. I find a large and significant increase in the female infant mortality rate, only for birth conceptions that take place during the April-July period when pesticides are applied, and only for non-violent causes of death.

On average the female infant mortality rate goes up by 1.01 per 1,000 birth conceptions for conceptions that occur between April-July. The number of post-exposure female births in the counties exposed to WNS is 104,356. This suggests an additional mortality of 105 female infants. Using the EPA value of mortality risk reduction (also referred to as the value of statistical life) of $7.4 million (in $2006) this represents a welfare loss of $931 million (in $2016).

Currently the regulation around pesticides focuses on regulating each compound separately. As the effects of different permutations are not tested we remain with observational data to alert us regarding the effects of chemical mixtures. The results here suggest that more work is needed on understanding the interacting effects of pesticides on health and that will require better monitoring of: pesticides applications, their contribution to air pollution, and the concentrations in rivers and underground water.

If the costs of conserving bat populations are low and the damages from the use of pesticides to substitute for their pest control are high then it is efficient and welfare improving to do so. Bat populations are under threat from land use changes that reduce the amounts of available habitat for them to hibernate in, and from the expansion of wind energy. Wind turbines are estimated to result in up to 888,000 dead bats each year in the U.S. (Smallwood 2013). Efficiency of wind
energy is going up and costs are coming down leading to an expected growth rate of 13\% in 2016 (IEA 2015). Further regulation might include measures limiting the location of onshore wind farms, restricting the height of wind turbines (Barclay et al. 2007), altering the rotational speed of the blades (Arnett et al. 2011), and the inclusion of devices that can deter bats (Joseph and Ed 2006; Baerwald et al. 2009; Khan 2014).

Further research can explore the health effects of pesticides as a function of distance from the application sites. As better data on the decline in bat populations as a result of WNS becomes available it will be possible to better estimate the marginal response of pesticides use with respect to losses in bat populations.
References


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Figure 1: Gradual Expansion of White Nose Syndrome Across U.S. Counties

Notes: map shows the counties in which the fungus, *Pseudogymnoascus destructans*, was detected and cross hashed counties identify the places in which White Nose Syndrome has been confirmed. Red colors represent earlier years of detection, corresponding to longer exposure intensities; whereas blue colors represent recent exposure.

Figure 2: Cumulative Number of Counties Exposed to White Nose Syndrome, 2006-2015
Notes: the cumulative number of counties detected with White Nose Syndrome.
Figure 3

Notes: flexible difference-in-differences regression results for the aggregate insecticides use on exposure to WNS in event time. Aggregated insecticides are measured on a county-year level and are normalized by the county’s area. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county FE, state-year FE, degree days and a quadratic function of precipitation. Observations are weighted by the harvested area in 2002 which serves as baseline of agricultural activity. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,382 clusters.

Source: data on pesticides use from the USGS (see Baker and Stone (2013, 2015)) for 1999 to 2012; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
Notes: flexible difference-in-differences regression results for the aggregate fungicides use on exposure to WNS in event time. Aggregated fungicides are measured on a county-year level and are normalized by the county’s area. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county FE, state-year FE, degree days and a quadratic function of precipitation. Observations are weighted by the harvested area in 2002 which serves as baseline of agricultural activity. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,382 clusters.

Source: data on pesticides use from the USGS (see Baker and Stone (2013, 2015)) for 1999 to 2012; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
Notes: flexible difference-in-differences regression results for the aggregate herbicides use on exposure to WNS in event time. Aggregated herbicides are measured on a county-year level and are normalized by the county’s area. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county FE, state-year FE, degree days and a quadratic function of precipitation. Observations are weighted by the harvested area in 2002 which serves as baseline of agricultural activity. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,382 clusters.

Figure 5

Source: data on pesticides use from the USGS (see Baker and Stone (2013, 2015)) for 1999 to 2012; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
Figure 6: Log(Number Births) for Conceptions during April-July
Notes: flexible difference-in-differences regression results for the log of birth conceptions on exposure to WNS in event time. Mortality rates are calculated using non-violent death causes for birth conceptions that occur during the pesticide application season between April and July. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the female population between 15 to 49. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,402 clusters.
Source: data on births is from the NCHS-NVSS linked birth and death certificates for birth cohorts 2003-2013; data on the estimated population is from the U.S. Census; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
Figure 7: Birth Conceptions April-July: Infant Mortality Rate, All Causes

Notes: flexible difference-in-differences regression results for the infant mortality rate per 1,000 births on exposure to WNS in event time. Mortality rates are calculated using birth conceptions that occur during the pesticide application season between April and July. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the number of female birth conceptions in each county throughout April-July. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,402 clusters.

Source: data on infant mortality is from the NCHS-NVSS linked birth and death certificates for birth cohorts 2003-2013; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
Figure 8: Birth Conceptions April-July: Infant Mortality Rate
Notes: flexible difference-in-differences regression results for the female infant mortality rate per 1,000 births on exposure to WNS in event time. Mortality rates are calculated using non-violent death causes for birth conceptions that occur during the pesticide application season between April and July. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the number of female birth conceptions in each county throughout April-July. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,402 clusters.
Source: data on infant mortality is from the NCHS-NVSS linked birth and death certificates for birth cohorts 2003-2013; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
Figure 9: Infant Mortality Rate in WNS Exposed Counties By Month of Conception

Notes: flexible difference-in-differences regression results for the infant mortality rate per 1,000 births on exposure to WNS interacted with calendar month, for the model in Equation (15). Mortality rates are calculated using either non-violent death causes (black diamonds) or violent death causes (gray squares) for birth conceptions in each month. Dashed black lines denote the months in which pesticides mostly get applied. Each coefficient represents the average difference of the female infant mortality rate in that month between WNS exposed counties to non-exposed counties. Blue lines represent 95% confidence intervals. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, year fixed effects, month fixed effects, degree days and a quadratic function of precipitation, as well as the nine months trajectory of degree days from each month. Observations are weighted by the number of female birth conceptions in each county in each month. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,393 clusters.

Source: data on infant mortality is from the NCHS-NVSS linked birth and death certificates for birth cohorts 2003-2013; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
(a) Female Infant Mortality Rate, Non-Violent Causes

(b) Female Infant Mortality Rate, Violent Causes

Figure 10: Birth Conceptions April-July: Female Infant Mortality Rate

Notes: flexible difference-in-differences regression results for the female infant mortality rate per 1,000 births on exposure to WNS in event time. Mortality rates are calculated using either (a) non-violent or (b) violent death causes for birth conceptions that occur during the pesticide application season between April and July. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the number of female birth conceptions in each county throughout April-July. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 1,402 clusters.

Source: data on infant mortality is from the NCHS-NVSS linked birth and death certificates for birth cohorts 2003-2013; data on the estimated population is from the U.S. Census; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
Figure 11: Log(Yield) & Chemical Expenditure

Notes: flexible difference-in-differences regression results for the natural log of (a) corn yields, (b) soy yields, and (c) chemical expenditure on exposure to WNS in event time. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the baseline yield values between 1999 to 2005 for crop yields. Chemical expenditure observations are weighted by the harvested area in 2002 which serves as baseline of agricultural activity. Standard errors are clustered at the county level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 23, 21, and 32 clusters, respectively.

Source: data on crop yields and chemical expenditure is from the USDA-NASS (see online data appendix about chemical expenditure); White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts (2009).
(a) Aggregate Insecticides

(b) Aggregate Fungicides

(c) Birth Conceptions April-July: Female Infant Mortality, Non-Violent Causes

Figure 12: Randomization Inference Results
Randomization took place either on the entire sample (full, in gray), across counties such that the temporal order of the treatment was preserved (block, in blue), or within treated counties such that only the temporal order was randomized (within, in green). Each histogram shows the distribution of the coefficients on exposure to WNS from the evaluations of Equation (13) for the 1,000 randomizations carried out per-randomization type. Vertical orange lines represent the value of the estimation on the non-randomized sample.
Table 1. Descriptive Statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Min.</th>
<th>Max.</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Treatment Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White Nose Syndrome (WNS) Dummy</td>
<td>0.027</td>
<td>0.163</td>
<td>0</td>
<td>1</td>
<td>20,160</td>
</tr>
<tr>
<td>Share of Surrounding WNS Counties</td>
<td>0.029</td>
<td>0.114</td>
<td>0</td>
<td>1</td>
<td>20,160</td>
</tr>
<tr>
<td><strong>B. Pesticide &amp; Crop Yield Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggregated Insecticides Used, in kg·km(^{-2})</td>
<td>6.404</td>
<td>16.454</td>
<td>0</td>
<td>393.383</td>
<td>19,686</td>
</tr>
<tr>
<td>Aggregated Fungicides Used, in kg·km(^{-2})</td>
<td>8.25</td>
<td>39.265</td>
<td>0</td>
<td>1139.142</td>
<td>19,573</td>
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<tr>
<td>Aggregated Herbicides Used, in kg·km(^{-2})</td>
<td>49.219</td>
<td>63.224</td>
<td>0</td>
<td>1009.864</td>
<td>19,741</td>
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<tr>
<td>Log(Corn Yields)</td>
<td>4.876</td>
<td>0.29</td>
<td>2.839</td>
<td>5.361</td>
<td>7,304</td>
</tr>
<tr>
<td>Log(Soy Yields)</td>
<td>3.666</td>
<td>0.284</td>
<td>1.792</td>
<td>4.159</td>
<td>7,042</td>
</tr>
<tr>
<td>Chemical Expenditure, in $1,000·km(^{-2})</td>
<td>9.193</td>
<td>105.956</td>
<td>0</td>
<td>9292.751</td>
<td>18,822</td>
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<tr>
<td>Agricultural Land, in km(^2)</td>
<td>350.834</td>
<td>389.479</td>
<td>0.348</td>
<td>2604.664</td>
<td>19,596</td>
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<tr>
<td><strong>C. Birth Outcomes Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male Birth Conceptions, April-July</td>
<td>205.485</td>
<td>530.358</td>
<td>0</td>
<td>12455</td>
<td>15838</td>
</tr>
<tr>
<td>Female Birth Conceptions, April-July</td>
<td>196.208</td>
<td>508.01</td>
<td>0</td>
<td>12054</td>
<td>15838</td>
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<tr>
<td>Male IMR, April-July, Non-Violent Causes</td>
<td>6.251</td>
<td>15.042</td>
<td>0</td>
<td>333.333</td>
<td>15832</td>
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<td>Male IMR, April-July, Violent Causes</td>
<td>0.456</td>
<td>3.316</td>
<td>0</td>
<td>95.238</td>
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<td>Female IMR, April-July, Non-Violent Causes</td>
<td>5.032</td>
<td>14.132</td>
<td>0</td>
<td>666.667</td>
<td>15831</td>
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<tr>
<td>Female IMR, April-July, Violent Causes</td>
<td>0.351</td>
<td>3.12</td>
<td>0</td>
<td>117.647</td>
<td>15831</td>
</tr>
<tr>
<td><strong>D. Weather Variables</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Degree Days, 10C-30C, April-September</td>
<td>2775.186</td>
<td>365.074</td>
<td>1784.922</td>
<td>3668.615</td>
<td>19,628</td>
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<tr>
<td>Degree Days, 30C, April-September</td>
<td>25.869</td>
<td>29.225</td>
<td>0</td>
<td>248.708</td>
<td>19,628</td>
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<td>Precipitation, in mm, April-September</td>
<td>627.024</td>
<td>162.058</td>
<td>34.724</td>
<td>1325.615</td>
<td>19,628</td>
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Table 2. Differences-In-Differences Pesticide Regressions Summary
Dependent Variables Measured in kg·km\(^{-2}\)

<table>
<thead>
<tr>
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<th>3(^{rd}) Degree Neighbor Sample</th>
<th>Full Sample</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Insecticides</td>
<td>Fungicides</td>
</tr>
<tr>
<td>WNS</td>
<td>2.54***</td>
<td>1.66**</td>
</tr>
<tr>
<td></td>
<td>(0.92)</td>
<td>(0.68)</td>
</tr>
<tr>
<td>Surr. WNS</td>
<td>4.12</td>
<td>0.84</td>
</tr>
<tr>
<td></td>
<td>(2.98)</td>
<td>(1.87)</td>
</tr>
<tr>
<td>% of Mean</td>
<td>39.6%</td>
<td>20.1%</td>
</tr>
<tr>
<td>% of SD</td>
<td>15.4%</td>
<td>4.2%</td>
</tr>
<tr>
<td>(R^2)</td>
<td>0.893</td>
<td>0.939</td>
</tr>
<tr>
<td>N</td>
<td>19,311</td>
<td>19,204</td>
</tr>
<tr>
<td>Clusters</td>
<td>1,382</td>
<td>1,381</td>
</tr>
</tbody>
</table>

Notes: *\(p < 0.1\), **\(p < 0.05\), ***\(p < 0.01\). Estimation results for the model in Equation 13. Results show the average treatment effect for counties exposed to White Nose Syndrome (WNS) relative to counties that were not exposed to WNS, for the three different response variables: aggregated insecticides, aggregated fungicides, and aggregated herbicides, normalized by county area and measured in kg·km\(^{-2}\). All results are shown for both the third degree neighbor subsample as well as the full sample. All results include county fixed effects, state-year fixed effects, degree days and a quadratic function for precipitation. Observations are weighted by the amount of harvested land in 2002. Standard errors are clustered at the county level. % of mean and % of sd report the size of the point estimate for WNS exposure relative to the mean or standard deviation of the dependent variable in the corresponding sample. Source: data on pesticides use from the USGS Estimated annual agricultural pesticide use for counties of the conterminous United States (Nancy_T_Baker2013-en; Nancy_T_Baker_and_Wesley_W_Stone2015-zw; Nancy2015-ab) for 1999 to 2012, White Nose Syndrome exposure data from the Pennsylvania Game Commission, weather data from Schlenker and Roberts 2009.
Table 3. Differences-In-Differences Female IMR Regressions Summary
Dependent Variables Measured in deaths per 1,000 birth conceptions

<table>
<thead>
<tr>
<th></th>
<th>Non-Violent Causes</th>
<th>Violent Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Jan-Mar</td>
<td>Apr-Jul</td>
</tr>
<tr>
<td>WNS</td>
<td>0.73*</td>
<td>1.01**</td>
</tr>
<tr>
<td></td>
<td>(0.41)</td>
<td>(0.40)</td>
</tr>
<tr>
<td>Surr. WNS</td>
<td>-0.40</td>
<td>0.84</td>
</tr>
<tr>
<td></td>
<td>(0.72)</td>
<td>(0.73)</td>
</tr>
<tr>
<td>% of Mean</td>
<td>12.6%</td>
<td>14.5%</td>
</tr>
<tr>
<td>% of SD</td>
<td>3.3%</td>
<td>3.2%</td>
</tr>
<tr>
<td>( R^2 )</td>
<td>0.228</td>
<td>0.223</td>
</tr>
<tr>
<td>N</td>
<td>14,006</td>
<td>14,013</td>
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<tr>
<td>Clusters</td>
<td>1,402</td>
<td>1,402</td>
</tr>
</tbody>
</table>

Notes: *\( p < 0.1 \), **\( p < 0.05 \), ***\( p < 0.01 \). Estimation results for the model in Equation 13. Results show the average treatment effect for counties exposed to White Nose Syndrome (WNS) relative to counties that were not exposed to WNS, for Female infant mortality rates by non-violent causes (columns 1-3) and violent causes (columns 4-6), for three different periods: pre-pesticides application season of Janauary-March (columns 1 and 4), pesticides application season of April-July (columns 2 and 5), and poest pesticides application season, September-December (columns 3 and 6). Mortality rates are calculated as the number of infants deaths divided by the number of birth conceptions, per 1,000 conceptions. All results are shown for the third degree neighbor subsample. Results for the full sample are available from the author by request. All results include county fixed effects, state-year fixed effects, degree days and a quadratic function for precipitation. Observations are weighted by the number of birth conceptions in the respective period. Standard errors are clustered at the county level. % of mean and % of sd report the size of the point estimate for WNS exposure relative to the mean or standard deviation of the dependent variable in the corresponding sample. Source: data on infant mortality is from the National Center for Health Statistics - National Vital Statistics System linked birth and death certificates for birth cohorts 2003-2013, White Nose Syndrome exposure data from the Pennsylvania Game Commission, weather data from Schlenker and Roberts 2009.
Table 4. Differences-In-Differences Male IMR Regressions Summary
Dependent Variables Measured in deaths per 1,000 birth conceptions

<table>
<thead>
<tr>
<th></th>
<th>Non-Violent Causes</th>
<th></th>
<th></th>
<th>Violent Causes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Jan-Mar</td>
<td>Apr-Jul</td>
<td>Sep-Dec</td>
<td>Jan-Mar</td>
<td>Apr-Jul</td>
</tr>
<tr>
<td>WNS</td>
<td>0.19</td>
<td>-0.06</td>
<td>0.21</td>
<td>-0.02</td>
<td>-0.06</td>
</tr>
<tr>
<td></td>
<td>(0.48)</td>
<td>(0.43)</td>
<td>(0.32)</td>
<td>(0.09)</td>
<td>(0.08)</td>
</tr>
<tr>
<td>Surr. WNS</td>
<td>0.30</td>
<td>-2.15***</td>
<td>-1.23*</td>
<td>-0.14</td>
<td>-0.40**</td>
</tr>
<tr>
<td></td>
<td>(0.86)</td>
<td>(0.73)</td>
<td>(0.64)</td>
<td>(0.19)</td>
<td>(0.16)</td>
</tr>
<tr>
<td>% of Mean</td>
<td>2.6%</td>
<td>.8%</td>
<td>3.4%</td>
<td>2.7%</td>
<td>15.3%</td>
</tr>
<tr>
<td>% of SD</td>
<td>.6%</td>
<td>.2%</td>
<td>1.3%</td>
<td>.2%</td>
<td>2%</td>
</tr>
<tr>
<td>$R^2$</td>
<td>0.248</td>
<td>0.251</td>
<td>0.301</td>
<td>0.140</td>
<td>0.151</td>
</tr>
<tr>
<td>N</td>
<td>14,013</td>
<td>14,013</td>
<td>14,014</td>
<td>14,013</td>
<td>14,013</td>
</tr>
<tr>
<td>Clusters</td>
<td>1,402</td>
<td>1,402</td>
<td>1,402</td>
<td>1,402</td>
<td>1,402</td>
</tr>
</tbody>
</table>

Notes: *p < 0.1, **p < 0.05, ***p < 0.01. Estimation results for the model in Equation 13. Results show the average treatment effect for counties exposed to White Nose Syndrome (WNS) relative to counties that were not exposed to WNS, for Male infant mortality rates by non-violent causes (columns 1-3) and violent causes (columns 4-6), for three different periods: pre-pesticides application season of January-March (columns 1 and 4), pesticides application season of April-July (columns 2 and 5), and post pesticides application season, September-December (columns 3 and 6). Mortality rates are calculated as the number of infants deaths divided by the number of birth conceptions, per 1,000 conceptions. All results are shown for the third degree neighbor subsample. Results for the full sample are available from the author by request. All results include county fixed effects, state-year fixed effects, degree days and a quadratic function for precipitation. Observations are weighted by the number of birth conceptions in the respective period. Standard errors are clustered at the county level. % of mean and % of sd report the size of the point estimate for WNS exposure relative to the mean or standard deviation of the dependent variable in the corresponding sample. Source: data on infant mortality is from the National Center for Health Statistics - National Vital Statistics System linked birth and death certificates for birth cohorts 2003-2013, White Nose Syndrome exposure data from the Pennsylvania Game Commission, weather data from Schlenker and Roberts 2009.
Table 5. Differences-In-Differences Crop Yield Regressions Summary
Dependent Variables are Log(Yield) or Chemical Expenditure (in $1,000·km\(^{-2}\))

<table>
<thead>
<tr>
<th></th>
<th>3(^{rd}) Degree Neighbor Sample</th>
<th>Full Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Corn</td>
<td>Soy</td>
</tr>
<tr>
<td>WNS</td>
<td>-0.05***</td>
<td>-0.03</td>
</tr>
<tr>
<td></td>
<td>(0.02)</td>
<td>(0.02)</td>
</tr>
<tr>
<td>Surr. WNS</td>
<td>-0.05</td>
<td>-0.04</td>
</tr>
<tr>
<td></td>
<td>(0.05)</td>
<td>(0.04)</td>
</tr>
<tr>
<td>% Of Mean</td>
<td>1.1%</td>
<td>.7%</td>
</tr>
<tr>
<td>% Of SD</td>
<td>18.9%</td>
<td>9.2%</td>
</tr>
<tr>
<td>(R^2)</td>
<td>0.832</td>
<td>0.853</td>
</tr>
<tr>
<td>N</td>
<td>7,290</td>
<td>7,014</td>
</tr>
<tr>
<td>Clusters</td>
<td>521</td>
<td>501</td>
</tr>
</tbody>
</table>

Notes: *p < 0.1, **p < 0.05, ***p < 0.01. Estimation results for the model in Equation 13. Results show the average treatment effect for counties exposed to White Nose Syndrome (WNS) relative to counties that were not exposed to WNS, for the natural log of corn and soy yields (columns 1, 2, 4, and 5), and chemical expenditure in $1,000·km\(^{-2}\). All results are shown for both the third degree neighbor subsample as well as the full sample. All results include county fixed effects, state-year fixed effects, degree days and a quadratic function for precipitation. Observations are weighted by the harvested land area in 2002. Standard errors are clustered at the county level. % of mean and % of sd report the size of the point estimate for WNS exposure relative to the mean or standard deviation of the dependent variable in the corresponding sample. Source: data on crop yields is from the US Department of Agriculture National Agricultural Statistical Service (see online data appendix for information about out of agricultural census years interpolation for for chemical expenditure), White Nose Syndrome exposure data from the Pennsylvania Game Commission, weather data from Schlenker and Roberts 2009.
Table 6. Differences-In-Differences Pesticide Substitution Regressions
Dependent Variable: Aggregated Pesticide Type Measured in kg km⁻²

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4) Linear Combinations</th>
</tr>
</thead>
<tbody>
<tr>
<td>WNS×Insecticide</td>
<td>8.34***</td>
<td>2.98*</td>
<td>3.79**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1.79)</td>
<td>(1.72)</td>
<td>(1.74)</td>
<td></td>
</tr>
<tr>
<td>WNS×Fungicide</td>
<td>6.74***</td>
<td>2.21</td>
<td>2.80</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(2.05)</td>
<td>(1.72)</td>
<td>(1.82)</td>
<td></td>
</tr>
<tr>
<td>Surr. WNS×Insecticide</td>
<td></td>
<td></td>
<td></td>
<td>18.83**</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(3.52)</td>
</tr>
<tr>
<td>Surr. WNS×Fungicide</td>
<td></td>
<td></td>
<td></td>
<td>15.92**</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(4.13)</td>
</tr>
<tr>
<td>Surr. WNS Bin [20%, 40%]×Insecticide</td>
<td>7.51***</td>
<td></td>
<td></td>
<td>11.30***</td>
</tr>
<tr>
<td></td>
<td>(2.14)</td>
<td></td>
<td></td>
<td>(2.62)</td>
</tr>
<tr>
<td>Surr. WNS Bin [40%, 60%]×Insecticide</td>
<td>6.56***</td>
<td></td>
<td></td>
<td>10.35***</td>
</tr>
<tr>
<td></td>
<td>(2.05)</td>
<td></td>
<td></td>
<td>(2.58)</td>
</tr>
<tr>
<td>Surr. WNS Bin [60%, 80%]×Insecticide</td>
<td>11.94***</td>
<td></td>
<td></td>
<td>15.73***</td>
</tr>
<tr>
<td></td>
<td>(1.90)</td>
<td></td>
<td></td>
<td>(2.37)</td>
</tr>
<tr>
<td>Surr. WNS Bin [80%, 100%]×Insecticide</td>
<td>12.90***</td>
<td></td>
<td></td>
<td>16.69***</td>
</tr>
<tr>
<td></td>
<td>(1.67)</td>
<td></td>
<td></td>
<td>(2.13)</td>
</tr>
<tr>
<td>Surr. WNS Bin [20%, 40%]×Fungicide</td>
<td>6.10***</td>
<td></td>
<td></td>
<td>8.91***</td>
</tr>
<tr>
<td></td>
<td>(2.25)</td>
<td></td>
<td></td>
<td>(2.96)</td>
</tr>
<tr>
<td>Surr. WNS Bin [40%, 60%]×Fungicide</td>
<td>5.56**</td>
<td></td>
<td></td>
<td>8.36***</td>
</tr>
<tr>
<td></td>
<td>(2.26)</td>
<td></td>
<td></td>
<td>(2.93)</td>
</tr>
<tr>
<td>Surr. WNS Bin [60%, 80%]×Fungicide</td>
<td>10.57***</td>
<td></td>
<td></td>
<td>13.37***</td>
</tr>
<tr>
<td></td>
<td>(2.09)</td>
<td></td>
<td></td>
<td>(2.74)</td>
</tr>
<tr>
<td>Surr. WNS Bin [80%, 100%]×Fungicide</td>
<td>11.88***</td>
<td></td>
<td></td>
<td>14.68***</td>
</tr>
<tr>
<td></td>
<td>(1.84)</td>
<td></td>
<td></td>
<td>(2.55)</td>
</tr>
</tbody>
</table>

$R^2$                         | 0.974   | 0.974   | 0.974   | 0.974                   |

N                             | 58,558  | 58,558  | 58,558  | 58,558                  |

Clusters                      | 1,400   | 1,400   | 1,400   | 1,400                   |

Notes: ∗p < 0.1, ∗∗p < 0.05, ∗∗∗p < 0.01. Estimation results for the model in Equation 17. Results show the average treatment effect on the use of insecticides and fungicides relative to herbicides (the omitted category) for counties exposed to White Nose Syndrome (WNS) relative to counties that were not exposed to WNS, in the third degree neighbor subsample. Linear combinations of the average effects and the bin dummy for the coefficients in column 3 are reported in column 4. e.g. column 4 row 5 is the linear combination for column 3 row 1 with column 3 row 5. All results include county-year, county-pesticide type, and pesticide type-year fixed effects. Observations are weighted by the amount of harvested land in 2002. Standard errors are clustered at the county level. Source: see Table 2.
<table>
<thead>
<tr>
<th></th>
<th>Aggregated Insecticides</th>
<th>Aggregated Fungicides</th>
<th>Female IMR April-July</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fungus Only</strong></td>
<td>0.09</td>
<td>-0.40</td>
<td>-0.76</td>
</tr>
<tr>
<td></td>
<td>(1.89)</td>
<td>(1.49)</td>
<td>(0.66)</td>
</tr>
<tr>
<td><strong>WNS</strong></td>
<td><strong>2.54</strong>*</td>
<td><strong>1.64</strong></td>
<td><strong>0.99</strong></td>
</tr>
<tr>
<td></td>
<td>(0.93)</td>
<td>(0.69)</td>
<td>(0.40)</td>
</tr>
<tr>
<td><strong>Surr. WNS</strong></td>
<td>4.11</td>
<td>0.86</td>
<td>0.82</td>
</tr>
<tr>
<td></td>
<td>(2.96)</td>
<td>(1.87)</td>
<td>(0.73)</td>
</tr>
<tr>
<td><strong>R²</strong></td>
<td>0.893</td>
<td>0.939</td>
<td>0.223</td>
</tr>
<tr>
<td><strong>N</strong></td>
<td>19,311</td>
<td>19,204</td>
<td>14,013</td>
</tr>
<tr>
<td><strong>Clusters</strong></td>
<td>1,382</td>
<td>1,381</td>
<td>1,402</td>
</tr>
</tbody>
</table>

Notes: *p < 0.1, **p < 0.05, ***p < 0.01. Estimation results for the model in Equation 16. Results show the average treatment effect for counties exposed to White Nose Syndrome (WNS) and counties that have only the fungus that causes WNS but no bats that developed WNS - relative to counties that were not exposed to WNS. All results are shown for the third degree neighbor subsample. All results include county fixed effects, state-year fixed effects, degree days and a quadratic function for precipitation. Observations are weighted by the amount of harvested land in 2002 for insecticides and fungicides, and by the number of birth conceptions for the female IMR. Standard errors are clustered at the county level. Source: see Table 2 and 3.
Appendix A.

Figure A1: Distribution of the Distance Between Counties and their Neighbors
Notes: each histogram shows the distribution of the distance from a county centroid to the centroid of its first degree neighbor, and to its second degree neighbor. The dashed line represents the maximal distance a bat can fly on a given night.
Figure A2: Most Used Pesticides

Notes: flexible difference-in-differences regression results for the most used pesticides, by pesticide type, on exposure to WNS in event time. Most used pesticides by type per-county are determined by the most used pesticide in a baseline period of 1999-2005, according to the amount used. In addition to reducing measurement error from the aggregation of different estimated pesticide use, this also reflects different optimal strategies chosen by the farmers conditional on different environmental factors, pest pressure, and crops grown. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the harvested area in 2002 which serves as baseline of agricultural activity. Standard errors are clustered on the state level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 32 clusters.

Source: data on pesticides use from the USGS Estimated annual agricultural pesticide use for counties of the conterminous United States (2013; 2015) for 1999 to 2012; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts 2009.
Figure A3: Sample Balancing: Dynamic Versus Compositional Effects

Notes: three examples illustrating the difference between dynamic and compositional effects, and how to test for them in the data. Figure (a) shows a simple case of three counties that have a baseline of 0 prior to treatment but have a constant treatment effect within county but it is different across counties. Counties also differ with respect to the length of treatment, which starts at period 1. The average treatment effect (solid line) appears to be going up which implies a dynamic effect but in reality is only a compositional effect because it is driven by the change in the composition of the treatment group. Figure (b) shows a version of Figure (a) with a longer treatment horizon. The black solid line shows the average treatment effect at each period, including all the treated counties. It increases over treatment time implying a dynamic effect but it only increases whenever a county exits the treatment group; i.e. when the composition changes. The blue thick line shows the average treatment effect if we only include counties that were treated above a certain threshold, in this case at least 4 periods (dashed orange line). It is higher than the black line, suggesting that composition plays a role, and only spikes up at the last period which is top coded to include all treatment periods above 4. Figure C shows a fully dynamic effect where all the counties have the same treatment effect which is linearly increasing with treatment length. Balancing the sample on counties exposed for only 4 periods (dashed orange line) and above results in average treatment effects that track the true effects and spike up at the final period which is top coded to include the later periods. Estimating both the balanced sample panel (black line) and the composition balanced panel (blue line) can help to distinguish between a dynamic or compositional effect.
Figure A4: Aggregated Pesticides - Balanced Composition

Notes: flexible difference-in-differences regression results for the aggregated pesticides, by pesticide type, on exposure to WNS in event time. The sample is balanced with respect to composition of the treatment group. Each treatment period has the same number of counties such that the composition of the counties is held constant when calculating the average treatment effect for each exposure period. Only counties with 4 exposure periods and above are include. The last exposure period is coded as 4 and above. This helps to determine whether the heterogeneous treatment effect is a result of different exposure lengths or different effects on the counties that were exposed earlier. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the harvested area in 2002 which serves as baseline of agricultural activity. Standard errors are clustered on the state level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 32 clusters.

Source: data on pesticides use from the USGS Estimated annual agricultural pesticide use for counties of the conterminous United States (2013; 2015) for 1999 to 2012; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts 2009.
Figure A5: Birth Conceptions April-July: Female Infant Mortality Rate, Non-Violent Causes

Notes: flexible difference-in-differences regression results for the female infant mortality rate per 1,000 births on exposure to WNS in event time. Mortality rates are calculated using non-violent death causes for birth conceptions that occur during the pesticide application season between April and July. Each coefficient represents the average difference, prior and post exposure, between counties that at some period get exposed to WNS versus counties that never get exposed; normalized relative to one period before exposure which is set to 0. Event time 1 is the first year in which a county gets exposed to WNS. Dashed gray lines represent 95% confidence intervals. The blue bars are the number of counties in the treatment group for each event time, which is not constant due to the gradual expansion of WNS. Each regression includes the share of first degree surrounding counties that are exposed to WNS, county fixed effects, state-year fixed effects, degree days and a quadratic function of precipitation. Observations are weighted by the number of female birth conceptions in each county throughout April-July. Standard errors are clustered on the state level. Sample includes all exposed counties and their neighboring counties up to the 3rd degree neighbor, resulting in 32 clusters.

Source: data on infant mortality is from the National Center for Health Statistics - National Vital Statistics System linked birth and death certificates for birth cohorts 2003-2013; data on the estimated population is from the U.S. Census; White Nose Syndrome exposure data from the Pennsylvania Game Commission; weather data from Schlenker and Roberts 2009.
Table A1. Adjusting Standard Errors for Spatial Correlation

<table>
<thead>
<tr>
<th></th>
<th>Aggregated Insecticides</th>
<th>Aggregated Fungicides</th>
<th>Female IMR April-July</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Coefficients &amp; Standard Errors Clustered by County</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WNS</td>
<td>2.54</td>
<td>1.66</td>
<td>1.01</td>
</tr>
<tr>
<td></td>
<td>(0.92)</td>
<td>(0.68)</td>
<td>(0.40)</td>
</tr>
<tr>
<td>A. Standard Errors Clustered by State</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WNS</td>
<td>(0.94)</td>
<td>(0.55)</td>
<td>(0.32)</td>
</tr>
<tr>
<td>B. Spatially Adjusted Standard Errors by Distance Cutoff</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance: 100km</td>
<td>(.31)</td>
<td>(.36)</td>
<td>(.73)</td>
</tr>
<tr>
<td>Distance: 200km</td>
<td>(.37)</td>
<td>(.34)</td>
<td>(.57)</td>
</tr>
<tr>
<td>Distance: 250km</td>
<td>(.4)</td>
<td>(.36)</td>
<td>(.48)</td>
</tr>
<tr>
<td>Distance: 300km</td>
<td>(.45)</td>
<td>(.4)</td>
<td>(.66)</td>
</tr>
<tr>
<td>Distance: 400km</td>
<td>(.47)</td>
<td>(.4)</td>
<td>(.57)</td>
</tr>
<tr>
<td>Distance: 500km</td>
<td>(.54)</td>
<td>(.44)</td>
<td>(.55)</td>
</tr>
<tr>
<td>Distance: 750km</td>
<td>(.5)</td>
<td>(.33)</td>
<td>(.64)</td>
</tr>
<tr>
<td>Distance: 1000km</td>
<td>(.48)</td>
<td>(.28)</td>
<td>(.64)</td>
</tr>
<tr>
<td>Distance: 1500km</td>
<td>(.5)</td>
<td>(.31)</td>
<td>(.57)</td>
</tr>
<tr>
<td>Distance: 2000km</td>
<td>(.51)</td>
<td>(.31)</td>
<td>(.62)</td>
</tr>
</tbody>
</table>

Notes: The original coefficient and their standard errors which are clustered by state are reported in panel A, for aggregated insecticides, aggregated fungicides, and female IMR for birth conceptions between April-July, for non-violent causes. Panel B reports the standard errors after adjusting them for spatial correlation with different distance cutoffs. Results are shown for the third degree neighbor subsample. All results include county fixed effects, state-year fixed effects, degree days and a quadratic function for precipitation. Observations are weighted by either the amount of harvested land in 2002 (columns 1 and 2), or by the number of female conceptions in April-July. Source: see tables 2 and 3.
Appendix B.

Derivation of Equation (5):
\[
\frac{dW}{dN} = \left( \frac{\partial Q}{\partial K} \frac{\partial N}{\partial N} \right) + \left( \frac{\partial Q}{\partial N} \right) - \left( r \frac{\partial K}{\partial N} \frac{\partial N}{\partial N} \right)
\]
\[
= 0
\]
\[
= \left( \frac{\partial Q}{\partial K} - r \right) \frac{\partial K}{\partial N} - \left( \frac{\partial Q}{\partial N} - \frac{\partial E}{\partial N} \right)
\]
\[
\geq 0
\]

From the Envelope Theorem, \( \frac{\partial Q}{\partial K} - r = 0 \) since \( K \) is chosen such that the marginal productivity of capital equates the price of capital.

Derivation of Equation (6):
\[
\frac{dW}{dI} = \left( \frac{\partial Q}{\partial K} \frac{\partial K}{\partial N} \frac{\partial N}{\partial I} \right) + \left( \frac{\partial Q}{\partial N} \right) - \left( r \frac{\partial K}{\partial N} \frac{\partial N}{\partial I} \right)
\]
\[
= 0
\]
\[
= \left( \frac{\partial Q}{\partial K} - r \right) \frac{\partial K}{\partial N} + \left( \frac{\partial Q}{\partial N} - \frac{\partial E}{\partial N} \right)
\]
\[
= \frac{\partial Q}{\partial N} - \frac{\partial E}{\partial N} \leq 0
\]

Derivation of Equation (10):
\[
\frac{dW}{dP} = \left( \frac{\partial Q}{\partial K} \frac{\partial K}{\partial N} \frac{\partial N}{\partial P} \right) + \left( \frac{\partial Q}{\partial N} \right) - \left( r \frac{\partial K}{\partial N} \frac{\partial N}{\partial P} \right)
\]
\[
= 0
\]
\[
= \left( \frac{\partial Q}{\partial K} - r \right) \frac{\partial K}{\partial N} + \left( \frac{\partial Q}{\partial N} - \frac{\partial E}{\partial N} \right)
\]
\[
- \frac{\partial T^C}{\partial P}
\]