Soy Intensification and Child Mortality in Argentina: an examination of Argentina's GMO led Soy boom and its effect on child health

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Abstract: Since 1996, Argentina has rapidly expanded its soy production and land area devoted to soy plantation. While the crop is a clear boon for the Argentina's economy, public officials have raised concerns regarding the safety of soy cultivation methods towards the communities that reside in major agricultural production areas. This study investigates effects the long-term impacts of soy cultivation on child health at the department level and finds that departments which increased their land area for soy production between 2001 and 2010 have child mortality rates 3.47 points higher than departments that did not. This finding is substantial from a policy perspective as Argentina's CMR in 2010 was 13.94 per 1000 births, meaning a 24% difference between the treatment and control groups. This study then discusses the policy implications of this effect, describes possible limitations to the data, and finally proposes alternative research possibilities that can be done on the subject by other researchers.

Introduction -

Soy is Argentina's most ubiquitous cash crop. In 2013, the nation recorded 19.4 million hectares of land harvesting soy, or about 51% of Argentina's total arable land. By comparison, the next three highest ranking crops were Maize, Wheat and Sunflower, with 3.7 million, 3 million and 1.8 million hectares harvested respectively (FAO, 2014). Despite the prominence of soy and its obvious importance on Argentina's economic life, the crop has not eluded widespread controversy. Since the early 2000's, when the crop expanded to new territories north and west of its native home in the Pampas, academics and public alike have condemned soy for its negative impacts on the environment and local communities through deforestation and displacement. But the most intense challenges have been ones related to the potential side effects of an agrochemical known as glyphosate, commonly used during soy cultivation.

Since 1996, agricultural producers throughout Argentina have used glyphosate extensively in industrial farming for the elimination of weeds. Patented in the 1970's by Monsanto under the brand name RoundUp, glyphosate is widely used by farmers of soy, corn and cotton and attributed with easier crop management and higher profit margins. Recent studies released raise doubts about the safety of the herbicide on human and embryonic health. Gasnier et al., 2009 concluded that glyphosate based herbicides are toxic endocrine disruptors in human cell lines. Carrasco et al., 2010 wrote that pre-natal glyphosate exposure could lead to birth defects in amphibian embryos. Numerous other studies published share similar findings: Yousef et al., 1995; Savitz et al., 1997; Daruich et al., 2001; Beuret et al., 2005; Dallegrave et al., 2007; Oliveira et al., 2007; Cavalcante et al., 2008.

Despite this research, there are also a number of studies that, on the contrary, find no causal link between glyphosate and negative impacts on human health. For instance, Williams et. al., 2000 conducted a systematic review of 188 prior studies on glyphosate and ultimately concluded that "under present and expected conditions of use, Roundup herbicide does not pose a health risk to humans." De Roos et. al. 2005 evaluated a cohort of 57,311 pesticide applicators who have used glyphosate and found that exposure to the chemical is not associated with cancer incidence overall. Acquavella et. al. 2004 show that average glyphosate levels detected in the urine of farmers are not high enough to warrant concern. Lastly, it should be mentioned that the US Environmental Protection Agency (EPA 1993, 1997), the World Health Organization (WHO 1994) and the European Commission (2002) have all concluded that regular glyphosate use does not result in adverse reproductive or developmental problems or birth defects. In short, there is not yet a firm academic consensus on the potential effects of glyphosate on human health, nor of GMO soy on community health.

This paper hopes to bring additional insight to this debate using historical cross-sectional data and social science techniques. Specifically, I examine Argentina's uniquely fast adoption of Ht soy and the country's dramatic increase in agricultural land devoted to the crop to evaluate the effect of increasing soy production on community health. I also exploit the fact that virtually 100% of the soy produced in Argentina since the early 2000's is genetically modified, heavily implying glyphosate usage for those hectares. Given that distinction, soy harvested hectares in Argentina becomes a reasonable proxy for relative agrochemical burden, allowing further investigation on the potential effects of glyphosate usage.

Using yearly cross-sectional data from the Sistema Integrado de Informacion Agropecuaria (SIIA) and Instituto Nacional de Estadísticas y Censos (INDEC) from 2001-2010,

I identify departments within Argentina (376 *departamentos* and 134 *partidos*) that increased soy harvested area between 2001 and 2010. Combining this data with Child Mortality data from Dirección de Estadísticas e Información en Salud (DEIS) over the same period, I run a differences-in-differences estimation controlling for time-varying elements related to child mortality, such as poverty and access to indoor toilets and tap water. The estimate suggests that departments which increased land devoted to soy production between 2001 and 2010 had 3.47 more child deaths per 1,000 live births than departments that did not. Later, I explore whether this increase was in fact due to higher levels of glyphosate or due to other factors related to intensification of soy, but obtain mostly inconclusive results.

The remainder of this paper is organized as follows. In Section I, I offer an overview of the growth of Argentina's soy industry and its rapid adoption of GM seeds. In Section II, I discuss potential epidemiological pathways for glyphosate to adversely affect human populations. I also discuss potential negative health outcomes and my reasoning for using Child Mortality Rate as the variable of interest. In Section III, I describe my data in further detail and discuss the identification strategy. In Section IV, I present the estimated impact of soy intensification on child mortality rate. Section V discusses further regressions that explore whether glyphosate is the main culprit behind the increased mortality rate. Section VI concludes with limitations to the data and opportunities for future study.

Section I - Literature

Soy has not always been Argentina's most dominant staple crop – throughout the 1950's and 1960's, as Argentina established itself as a major agricultural powerhouse, primary exports were

wheat and corn. The soybean sector did not emerge until the early 1970's, and at the time, was still a fledgling market with only 36,000 hectares under cultivation. Comparatively, Brazil had 1.7 million hectares under harvest at the time and the United States over 17 million. This small foothold of soy grew steadily through the 1980's and 1990's, spurred by favorable international soybean prices. During that period, soy expanded throughout the humid Pampean region of central Argentina, reaching over 6 million harvested hectares by the mid 1990's.

In 1996, Ministry of Agriculture approved the first formal registration of genetically modified soy. This act drastically changed the nature of soy plantation in Argentina in several key ways.

For one, it brought forth an unprecedented level of adoption of GMO technology. At the year of its release in 1996, approximately 370,000 hectares of land were cultivated with GM Soybeans, or only 6% of Argentina's total soy cultivation (Trigo, 2011). By 2000, nearly 90% of soy acreage would be converted over to the herbicide resistant variety (Penna & Lema, 2003). It cannot be understated how incredibly rapid this rate of adoption truly was – for comparison, it took the U.S. nine years to reach the same level of adoption for the same technology. Other GM varieties in Argentina, such as Bt-Cotton and Bt-Maize, both released in 1997, took 8 years and 13 respectively to reach similar rates of adoption. In the U.S., adoption rates of Bt-Cotton and Bt-Maize were even slower, with both GM varieties still hovering at between 60 and 70% adoption a full 15 years after their release (Duke & Powles 2009). In short, not only did Argentina accept GM technologies quickly as a whole, Ht Soy in particular was more readily adopted compared to other GM seeds.

The primary reason for this rapid adoption of GM Soy in Argentina was cost savings. A 2001 USDA report on soybean policy in Argentina notes that "cost savings attributable to

biotech soybeans are estimated at about $40 per metric ton, much larger than the $8-per-ton premium received by producers for non-biotech soybeans in Argentine markets" (USDA 2001). Penna & Lema, 2003 also list cost savings as a primary motivator for switching to GM soy, estimating an implied savings of $20 per hectare compared non-GM varieties. The same paper additionally mentions a 1999 survey on farmer's opinions of the advantages of transgenic soy and notes that 93% and 71% of farmers listed "lower costs" and "time savings" as their primary reasons for adopting GM soy, respectively. These savings essentially stem from much easier weed management. Glyposhate is a broad-spectrum, non-selective herbicide effective at eliminating virtually any organic plant matter. Paired with a genetically modified variety of soy that is meant to specifically withstand glyphosate, farmers can their spray fields indiscriminately, killing only weeds without fear of harming their primary cash crop. The simplicity of this Ht-soy/Glyphosate combination allowed farmers to control for virtually all weed species with just a single herbicide and eliminated the need for "consultants to provide prescription herbicide combination solutions dependent upon crop type, herbicide selectivity, and weed spectrum, even sometimes varying with different locations within a farm" (Duke & Powles 2009).

Another significant change resulting from the introduction of GM soy to Argentina was the rapid expansion of soy harvested area in the following years. Between 1996 and 2012, soy area under cultivation increased at an impressive compounded annual growth rate of 7.6%, more than tripling in size during that time span. Comparatively, soy harvested area grew only 3.5% for the period between 1971 and 1996 (Trigo, 2011), with this growth primarily only occurring in the Pampas. Meanwhile, Argentina's former agricultural staples actually lost land share – sunflower, for example decreased from a high of 4 million hectares under harvest to its present stock of 1.8 million today; wheat harvest decreased from 7.1 million hectares in 1996 to just 3

million hectares. Some of the increase in soy area came as a direct result of farmers substituting the new profitable crop for the older staples (Pengue 2005). In other cases, farmers intercropped GMO soy alongside wheat by sowing short-cycle Ht-soy immediately after harvesting a wheat crop, essentially doubling their income (Penna & Lema 2005). Lastly, much of the growth in cultivated hectares came from new land entirely. The cost reduction from transgenics introduced the possibility of extending the agricultural border for soy into marginal regions where climatic conditions were less favorable. Prior to the introduction of glyphosate, soy was grown mainly around the central Pampean region, including the provinces of Buenos Aires, Cordoba and Santa Fe. Afterwards, the crop spread into the northern and northwestern states, displacing other types of land use and increasingly at great cost to native forests (Goldfarb & Zoomers, 2010). The authors estimate that between 1996 and 2004, 34% of expanded soy area came from land previously used for other crops, 27% from former pastures and 41% on newly cleared forest and savannah.

The final significant impact that transgenic technology has had on the cultivation of soy in Argentina – and the one that is currently up for debate – is its effect on the livelihood of the communities that farm it. Qaim and Traxler, 2005 find mainly positive impacts from the adoption of Ht-soy. Their research, which primarily looks at aggregated farm-level welfare effects from 1996 to 2001, shows that total factor productivity increases an average of 10% for farmers using the new technology, and more importantly, that small farms (under 100 hectares) benefit more than their larger counterparts. Penna & Lema are of similar minds; they posit that cost savings ultimately help smallholder livelihoods through increased income. They state that small farmers account for 90% of the farmers using transgenic soy and that overall, farm size does not have an effect on adoption rates. Paredes and Martin discuss a more neutral effect. In

their study, of 120 smallholders in the Entre Rios province, they find that adopters of GM variety soy typically have larger farms, access to credit, higher education and a higher stock of farm machinery and equipment. The researchers do not specifically mention any positive financial impacts from accepting Ht-soy, though they do find a small income benefit related to Bt-cotton. Others are more forthrightly critical of soy's impacts. Garcia-Lopez & Arizpe, 2010 focus on the negative social implications of the expansion of soy, including displacement of indigenous and peasant populations, loss of livelihood and loss of food security. For instance, the rapid growth of soy led to the displacement of over 300,000 families in Argentina and resulted in 60,000 farms going out of business between 2000 and 2010. Pengue argues that intensification of soy creates a "huge ecological debt", including deforestation, water pollution, soil degradation, loss of agro-biodiversity, and health problems associated with increased use of pesticides and herbicides.

Outside of academia, public controversy surrounding the use of GM soy in Argentina runs rampant. Dr. Andres Carrasco's landmark finding, published in the journal Chemical Research in Toxicology in May 2010 (mentioned above), became the catalyst for a much wider debate on agrochemical usage in Argentina. Just three months after its publication, a group of physicians from crop-sprayed areas convened in Cordoba to discuss whether their medical caseloads showed any correlation between the arrival of intensive industrial agriculture and rising cancer rates and birth defects in their communities. Their report, coordinated by Dr. Medardo Avila Vazquez and Dr. Carlos Nota, showed an apparent link between spraying and reproductive problems, repeated miscarriages and serious birth defects. They also provide testimony that the number of birth defects per 10,000 live births in Chaco province has sharply increased, from 60 to 186, between 2001 and 2008. In 2012, further outcry against agrochemical usage fulminated when a group of farmers in Misiones filed a lawsuit against Monsanto claiming

that glyphosate based chemicals led to medical conditions including cerebral palsy, epilepsy, spina bifida, congenital heart defects, Down syndrome, missing fingers and blindness. Recently, the debate on herbicides reached international attention. A 2013 Associated Press report strongly links agrochemical usage to multiple organ problems, birth defects, cancer, miscarriages, respiratory illness, hydrocephalus and polyneuropathy (Warren, 2013).

The concern that agrochemical usage may lead negative health side-effects is certainly not unique to Argentina. Academic literature concerning the link between herbicide use and human health has been in existence since at least the 1970's, when the lingering effects of the military-grade defoliant "Agent Orange" were first brought to light. Arthur Galston's research on the potential carcinogenic effects of the chemicals compelled the Nixon administration to halt its usage during the Vietnam War. More studies published through the 1970's questioned its effects on American veterans (Bogen 1979) and many more have been published since, highlighting negative impacts on the health of Vietnamese communities.

But despite the lingering controversy and potential risk, response from lawmakers in Argentina has been tepid at best. In 2010, a regional court in Santa Fe banned agrochemical spraying within 500 meters of populated areas. However, this ruling is so far only limited to areas around San Jorge, a small town of about 17,000 inhabitants (Robinson, 2010). And, as Argentina has no national law pertaining to spraying of agrochemicals, provinces in the agricultural nation are free to set their own safety standards or none at all. The recent AP Report notes that "about one-third of the provinces set no limits [to spraying] at all, and most lack detailed enforcement policies." National attempts to ban the use of glyphosate have been unsuccessful. In 2009, a group of environmental lawyers petitioned the Argentinian Supreme

court to impose a six-month ban on the sale or use of glyphosate based chemicals, though the motion ultimately failed (Webber, 2009).

Section II - Pathways

Considering the current heightened level of scrutiny regarding the impact of transgenics in Argentina, an investigation into the rapid expansion of Ht-soy between 2001 and 2010 is warranted. There are ultimately a variety of pathways – both chemically related and not - for which soy intensification could reasonably bring about a higher rate of child mortality at the department level. In general, child mortality is most proximately caused by malnutrition, congenital malformations, dehydration, diarrhea, diseases such as malaria, and in some cases, toxic shock from environmental pollutants. Therefore, macroeconomic studies involving child mortality rate typically look at indicators such as poverty rates, unemployment, family income levels, education, access to health care and health services, and lastly community level infrastructure.

As for the pathways explored in this study, perhaps the simplest explanation, mentioned above by Garcia-Lopez & Arizpe, is that new soy growing areas may displace smallholders and indigenous communities. If it is true that soy expansion came as a result of large landholders and industrial farms forcing smallholders to relocate, this burden and sudden loss of family livelihood could have led to a higher rate of child mortality. Non-chemical environmental effects, such as deforestation, soil degradation, loss of agro-biodiversity could also foreseeably lead to higher instances of child mortality at a department level. This would be especially true if native communities depended on forests as a source of livelihood, if soil degradation led to a

decrease in yields of smallholders, or if lack of biodiversity of crops left farming communities more vulnerable to sudden price shocks.

Since community displacement and land degradation are both areas of interest in the social sciences, even without establishing a formal link to glyphosate, an effect of soy intensification on child mortality would still be interesting and significant. However, given Argentina's uniquely rapid adoption of bioengineered crops, data compiled for this study could even potentially be useful for settling the on-going debate on the negative effects of agrochemical use.

The pathways for soy intensification leading to higher rates of child mortality – this time through glyphosate – require additional assumptions than compared with above. Studies that discuss negative community level health impacts from agrochemicals traditionally cite one of two potential hazards: mishandling of the chemical by farmers and producers, and negligent storage and safekeeping of agrochemical containers leading to secondary level exposure. Both scenarios are very realistic possibilities in Argentina. For instance, the AP report notes that farmworkers are not always trained on how to properly handle pesticides and herbicides. This combined with more lax safety procedures in Argentina – preparing chemical solutions without the benefit safety gloves, masks and special clothing – may introduce high exposure risks not present in more industrialized nations like the U.S. However, for this exposure to directly impact child mortality, we would need to make the additional assumption that glyphosate stores itself inside farmworkers' bodies long-term, eventually leading to genetic defects and mutations in reproductive cells. Though there is a well-documented case for this with Agent Orange dioxin, there is little evidence that this occurs with glyphosate.

It is more likely that secondary level exposure, from negligent storage and spraying of the chemical, would manifest itself in higher rates of child mortality. The case for this pathway is strong – the intensity of agrichemical spraying in soy producing areas by itself introduces a hazardous potential for chemical runoff into nearby bodies of water. Aerial spraying from nearby fields can also drift over to neighboring households and communities, leading to health problems. Vazquez and Nota state that agrochemical usage per area has increased dramatically, from 2 liters per hectare in 1996 to more than 10 liters per hecature in 2010 – this is partly due to the growing resistance of weeds to glyphosate and the need to use more chemicals to achieve the same effect.[1] It may also be due to uneducated or uninformed farm workers using higher quantities than needed. Additional opportunities for exposure exist – for instance, empty chemical containers discarded haphazardly rather than properly disposed represent additional vectors for contagion (AP, 2013). In some cases, poor families re-use these containers to store water for flushing toilets, feeding chickens, and washing their clothes. These secondary-level exposures would then ultimately lead to higher child mortality rates if children are exposed to the chemical at a very young age, or if the mother is exposed to it during pregnancy.

Though numerous routes exist defining how soy intensification could lead to a higher child mortality rate, it is lastly important to mention that there are ways for soy intensification to have a negative effect as well. For instance, it is possible that growing soy could increase the income level of a particular community, reducing poverty levels and associated child mortality rates. Furthermore, it's feasible that soy production leads to greater access to infrastructure which may also affect mortality rates positively. Lastly, if it turns out that glyphosate is less toxic than market alternatives used for conventional soy, switching to Ht-soy may decrease child

[1] See Vila-Aiub et. al., 2007 for a discussion on glyphosate resistant weeds

mortality. These potential scenarios will be explored if it is shown that soy intensification leads to lower instances of child mortality.

Section III - Methodology

A. *Methods*

The first objective of this paper to find the average effect of soy intensification on child mortality in departments that expanded their hectares of soy under harvest. There are various pathways that could lead to a higher mortality rate in children, including population displacement, land degradation and toxins from agrochemical usage. If we are successful at finding a causal relationship between these two variables, we can next try to determine whether the rise or drop in CMR is specifically due to glyphosate, or whether other factors related to soy expansion are at play.

Ideally, we would like determine whether there is a causal effect between soy intensification and child mortality by directing a randomly chosen group of departments in Argentina to produce a certain quality of soy, and then comparing the outcome of the two groups. Given that a randomized control trial of this nature is not possible, we instead turn to non-experimental methods in order to determine our counterfactual case. Of course, one major initial concern is that departments with higher rates of soy adoption are innately different from ones with lower, and that these differences may be correlated with child mortality. For instance, departments that experienced larger increases in harvested land may be better endowed with fertile land and have lower mortality rates to begin with. In this case, the correlation between soy expansion and mortality would be confounded with soil fertility. If we assume that unobservable

confounding characteristics vary by department but are constant over time, one method for addressing these time invariant unobserved factors is to use panel data and estimate difference in differences models. With a difference in differences approach, we can compare the change in outcomes in a specified treated group before and after the rapid expansion of GM soy in the last decade to the change in outcomes in a control group. By comparing these changes, we control for observed and unobserved time-invariant department level characteristics that might be correlated with soy intensification as well as child mortality. The change in the control group gives us our best estimate for a counterfactual case – i.e. what would've happened to the treatment group without the expansion of soy.

I estimate the effect of soy on child mortality through two different methods. The first method uses data from only two time periods (2001 and 2010), while the second method uses data from all eleven time periods (2000-2010). The reason for this is because data from INDEC Censuses on household durables and infrastructure only exist for 2001 and 2010 (see Data Appendix for full list). Though these variables were not the primary dependent or independent variables of interest, I felt they were important covariates, so I wanted to conduct one set of regressions with only "pure" numbers as well as one using the full dataset but with imputed figures for the missing years' variables. For the two-period model, the change in hectares of soy harvested is not a binary intervention but rather a range of values, so this study deals with this scenario in two ways: I first run a regression using a dummy variable equal to 1 if the department increased the amount of soy cultivated between 2001 and 2010. Then, I use the soy harvested scalar figures as a measure for treatment *intensity*. Both methods yield similar results, though

different interpretations for the coefficients. The difference in differences models are specified below:

$$(1)\ y_{it} = \eta + \gamma d_i + \delta T_t + \alpha d_i * T + \theta_i + u_{it}$$

$$(2)\ y_{it} = \eta + \delta T_t + \alpha I_i * T + \theta_i + u_{it}$$

where y_{it} is the child mortality rate in a given department i in year t, η is a constant, d_i is a dummy indicating if the department converted at least 1% of their land to soy production between 2001 and 2010, T is a dummy for the year 2010, α is the difference in differences estimate for the average effect of treatment on child mortality rate, θ_i is a vector representing department level fixed-effects and u_{it} is a time-varying error term. In the next formula, all variables are as above except for I_i, which represents the intensity of soy expansion based on the number of hectares of soy harvested. For the panel regressions featuring all eleven time periods, I essentially the same model as written in equation (2), simply substituting the time dummy with the full vector of years and changing the intensity term so that it represents soy harvested by department by year.

In order for α to be in unbiased estimate of the true impact of soy intensification between 2001 and 2010, two critical assumptions must be made: first, we must assume that within departments which heavily intensified soy production – i.e. the treatment group in the absence of treatment – the effect of "time" on child mortality was identical to that of the control group. I test this parallel trends assumption in three ways: by discovering whether time trends in the control and treatment groups are the same in the pre-treatment phase, by finding an alternative treatment

group or outcome variable not effected by the program, and by finding an alternative comparison group. Secondly, we must assume the model controls for any unobserved, time-varying characteristics that are correlated with both mortality and soy harvest. For example, it could be the case that departments which significantly increased soy production happened to have cuts in the healthcare system or public welfare programs during the study period. I defend this assumption in two ways – first I show that the number of hectares cultivated in a particular department is largely dependent on fixed characteristics such as soil quality and general climate and not by observed time-varying factors. This suggests that intensification is also less likely to be correlated with unobserved time-varying department specific factors such as economic shocks and health care changes. Furthermore, I directly control for a number of observed time-varying economic characteristics such as income level, unemployment, household level durable assets and community infrastructure.

If it is established that there is a statistically significant effect of soy harvested hectares on child mortality rate, this study will then explore whether or not this effect is due specifically to the usage of the agrochemical glyphosate, or whether the effect is due to broader social and environmental factors such as forced migration or deforestation. I conduct this exploratory research in three different ways:

First, I use alternative crops such as maize, cotton and wheat as comparison groups against the soy findings. Out of all the crops grown in Argentina, soy is most intimately linked with glyphosate usage – virtually all soy grown in the nation since 2010 was genetically modified, and all genetically modified soy possessed the Ht-trait. The crops in the comparison groups had a much lower potential for glyphosate usage, or none at all, so exploiting this

variance among the crop types may give an indication of whether glyphosate was the main causal element for higher child mortality.[2]

Next, I look for interaction effects between soy harvested hectares and variables related to infrastructure. Since I hypothesized that a main epidemiological pathway for glyphosate to effect human health was through secondary level exposure – such as from chemical run-off or from negligent disposal of chemical containers – any interaction effect between soy harvested hectares and a variable such as access tap water or access to sanitation would be incredibly telling.

Lastly, I try to isolate other causes that may be behind the increased child mortality rate by specifically limiting the regression to certain departments. For example, since the soy expansion led to deforestation mainly in new soy harvest areas, I limit the regression to only the areas in the Pampean region, where soy intensification came from the substitution of wheat and maize growing areas, rather than expansion into new, formerly marginal lands. I repeat the limited regressions to isolate away alternative causes such as indigenous displacement as well.

B. *Data*

The data used in this paper come from a variety of national and local level Argentine surveys and censuses conducted at the department level.

Our dependent variable in this analysis is Child Mortality Rate derived from mortality incidence data tables provided by DEIS under the Argentine Ministry of Health. DEIS collects these statistics on a yearly basis from municipal level health registries and publishes an annual report aggregated by department and age group. I look specifically at the number of deaths per

[2] Though maize and cotton both had over 90% of hectares harvested as GM varieties by 2010, this shift took place gradually throughout the last decade. Furthermore, not all genetically modified maize and cotton seeds were glyphosate resistant – some had traits for lepidopteran and glufosinate resistance.

year under the age of five creating a database of 122,432 child deaths occurring between 2000 and 2010. Child Mortality Rate in this study are calculated through the traditional means, by dividing number of deaths of children less than five years old by number of children born in the same year. Throughout the duration of the study, the Child Mortality Rate in Argentina fell from a rate of 19.55 per 1000 to 13.94 per 1000.

Contextually, epidemiological studies related to agrochemical use typically look at a wide variety of health effects. For instance, Agent Orange studies consider low-birth weight, various forms of cancer, congenital birth defects, chronic respirational illness and higher rates of miscarriage as possible ailments related to chemical burden. Truthfully, any of these afflictions could be construed as variables of interest for glyphosate based on public indictments made against glyphosate in the past. Ultimately, child mortality was chosen as the dependent variable in this study based on availability of data and likelihood that that this variable would be responsive to GMO soy cultivation in the short term.

Data from Sistema Integrado de Informacion Agropecuaria were used for the independent variable of soy harvested. The SIIA is a clearinghouse for agricultural information that distributes information on the behalf of official government agencies such as Argentina's Ministry of Agriculture, Marketing Information Management, and Directorate of Information on Agriculture. This data is recorded yearly, at the department level, from 1993 to present and includes crop data on production, area sown, area harvested area and finally, total yield. The paper uses area harvested as opposed to other indicators for the regression equations based on the assumption that harvested area provides the best proxy for agrochemical usage. As of 2010, 264 departments of the 510 in the study had at least one hectare of land harvesting soy with a mean of 36,750 hectares. The mean of harvested soy area as a portion of total land size was 19.5% for

soy producing departments and 10.5% overall. Crop data on maize, cotton and wheat are additionally used, to provide comparison groups.

Instituto Nacional de Estadísticas y Censos (INDEC) data were used for additional background on households related to durables owned and household infrastructure – for instance, existence of a water main, access to sewage, and existence of an indoor toilet. INDEC coordinates Argentina's nation-wide population censuses, last conducted in 2001 and 2010, and publishes data at the department level for those years. The data for these variables are presented as number of households within a particular department that possess the durable good or infrastructure improvement. INDEC also conducts a yearly household census called Encuesta Permanente de Hogares (EPH), which collects microdata from households living in 31 different urban areas throughout Argentina. Microdata from EPH were used in this study for province level estimates of income, poverty rate and unemployment.

Lastly, I use a variety of geospatial databases for time-invariant bioclimatic factors that relate to the likelihood of expanding soy cultivated areas. Temperature, Altitude and Precipitation data were acquired from the WorldClim database as rasterized maps at the 5-arc minute resolution. These pixelated raster values were then averaged by department boundary vectors over the WGS84 datum, using the Zonal Statistics tool in ArcMap. Data from Global Administrative Areas Database were used for these boundary vectors as well as land area calculations, which allowed for a normalization of soy harvest data based on physical size of the department. There have not been any administrative boundary changes in Argentina for the duration of the study, so land area is constant across all years.

Section IV – Results of Soy Intensification on Child Mortality

A. *Baseline Comparisons*

The balance tests in Table 1 suggest that the treatment and the control groups were significantly different from one another in 2001, the baseline year. This was expected, as the "treatment" of expanding soy harvested area was not in any way randomized, and in fact, largely determined by the array of land and socioeconomic factors mentioned earlier. Climatically, departments in the treatment group had an average annual temperature of 2 degrees Celsius higher than the control and average annual rainfall amounts 190mm wetter than the control. Departments which would go on to plant more soy generally were closer to sea level, and had more temperate winters. The two groups also differed from each other economically. The treated departments generally had better infrastructure with 58% of households having access to indoor bathrooms and water supplies versus 52% in the control. Households in the treatment group were 7% more likely to own a refrigerator and computer, 10% more likely to have access to sewage and 3% more likely to own a cellphone in 2001. The treated group had slightly higher poverty and unemployment rates but interestingly, a lower child mortality rate in 2001. Control areas had an average CMR of 20.44 in the baseline versus 17.61 in the treatment. Based on these significant dissimilarities, it is clearly necessary to use an estimation method that controls for time invariant unobservable characteristics between departments. Otherwise, there would be no way to counter the hypothesis that differences in mortality rates are caused by innate, observed and unobserved differences between departments and not by soy.

After baseline comparisons, I look at factors that contributed to departments increasing or decreasing their land area for soy cultivation during the study years (Table 1a). Running a probit model regression on the likelihood of being included in the treatment group (i.e. those

departments which increased soy cultivation) I find that temperature, precipitation, land area and average population density are the biggest determinants to whether a department went gained soy hectares versus not.[3] This confirms the earlier postulate that time-invariant bioclimatic factors are primary determinants for being included in the treatment group. However, it should be noted that poverty and the existence of a water main were also statistically significant and positive while unemployment and having access to water within the house were statistically significant and negative, implying that socioeconomic conditions also had a role in whether soy was adopted between 2001 and 2010.

B. *Initial Regressions*

The simple difference-in-difference regressions for child mortality rate are shown in Table 2. For the first four equations, I use only a dummy variable representing whether or not the department increased its cultivation soy of hectares between 2001 and 2010. For the last four regressions, I look at the intensity of effect, interacting the actual numeric difference in soy hectares harvested between 2001 and 2010 with time. In each case, I run separate regressions using the variables from the Argentinian Census as raw figures – representing number of households in a department possessing a particular item – as well as proportionally, by dividing this number by the number of households in the department. Additionally, two of the Census variables – sewage and gas – had a significant number of entries missing, so I run regressions both including and excluding these variables.

The first two columns report the effect of soy on child mortality using raw census figures. The estimate in column 1 indicates that departments which increased their level of soy

[3] Temperature entered into the probit model as absolute value of the difference between the average annual temperatures in a department and the ideal for soy (17 degrees centigrade). Precipitation was also expressed as the absolute value of the difference between average annual precipitation and the ideal (850mm of rainfall).

production saw 3.94 more child deaths per 1,000 births. This amounts to a 24% difference compared to the mean. When sewage and gas are removed from the regression equation, the estimate on the impact of being treated drops to 3.47, though the statistical significance increases from the 90% to 95% level. Columns 3 and 4 repeat the above equations looking at the intensity of effect, represented by the difference in soy hectares harvested divided by land area. Column 3 reports that every 1% of total land area converted to soy cultivation increases the Child Mortality Rate by .12. Since the mean of the intensity score is 9.6 in treatment groups, this implies an increase of 1.15 for the mean department in the treatment group. This finding is statistically significant at the 95% level, though removing sewage and gas from the regression equation reduces the estimate for the impact down to .099 and the significance level down to 90%.

These initial regressions are not robust when using the proportional functional forms for census variables. Though this is somewhat concerning, when I run the same regressions this time dropping outliers, the estimates are once again statistically significant at the 90% level or higher regardless of functional form (not included in tables).[4]

Lastly, I run a regression estimating the impact of soy cultivation on Child Mortality Rate using all 11 time periods and a fixed effects model. For these equations, found in table 4, I use soy harvested area divided by land area as my independent variable. I continue to run the regressions allowing for both functional forms of the census variables as well as including and omitting sewage and gas from the regression equations. For the full panel regressions, all estimates are statistically significant at the 90% confidence level or higher, regardless of functional form or census variable inclusion. The estimate for column 4 reports that every 1% of land area used for cultivating soy in a department leads to a .063 increase in Child Mortality

[4] In this case, outliers were departments with very low population and birth rates. Since number of live births is the denominator in the Child Mortality Rate equation, low population municipalities tended to have erratic CMR estimates.

Rate. Since the mean percentage of land area devoted for soy was 19% in soy growing regions, this corresponds to a 1.2 point increase in CMR for the mean group. A Hausman test was able to reject the hypothesis that the fixed effect and random effect were the same, which suggests that the treatment variable is correlated with fixed department level unobservables.

C. *Tests for Parallel Trends*

Establishing parallel trends or using alternate comparison groups is a crucial part of this study - otherwise, there would be no reason to think that the difference-in-differences interaction is the true counterfactual for the treatment group had it not increased soy cultivation. Table 2a shows the results of the parallel trends estimation, which looks at the change in child mortality between the treatment and comparison group before the baseline (between 2000 and 2001). Looking at the interaction term, we cannot reject the null hypothesis that pre-baseline trends for child mortality rate are different between the control and treatment groups.

Admittedly, this is a rather weak defense of the parallel trends assumption: optimally, data on child mortality level would be available at the department for many years prior to 2000. Given that this information is unavailable, I next create a counterfactual case by using alternative treatment and comparison groups. In Table 2b, I perform the basic difference-in-differences estimates looking at mortality rates of different age cohorts, as well as overall mortality rates by department. Here, if we assume that harmful GMO soy cultivation methods have the biggest potential for negative impacts at the very earliest stages of life, we should see statistically significant impacts for our original cohort (under the age of 5), but not for other cohorts which

would've been too old to experience GMO soy as a child.[5] Equation 1 uses a slightly different definition for child mortality (dividing by the total population rather than just the number of live births), but still finds a statistically significant at the 95% level. Equations 2 through 4 look at different age cohorts as the dependent variable and find no impacts at any conventional level of significance. Interestingly, there is a statistically significant and positive impact on maternal mortality rates, implying that the negative effects of soy may be closely related to birthing and pregnancy.

I then use an alternate comparison group while keeping the same treatment group to see whether the differences-in-differences interaction term is truly representative of the counterfactual case. One obvious concern of the basic regression is that departments that expanded soy cultivation tend to be more "agrarian" than ones that didn't. For instance, they may have more fertile land, wider access to markets, lower population densities, and these innate differences may lead to different time trends regarding Child Mortality Rate and its covariates. In order to deal with this concern, I run the basic regression again, this time using the same treatment group but limiting the control group to only the departments which grew soy in 2001. In this way, I only look at the universe of samples where growing soy is ever actually suitable. The results, seen in table 2c, demonstrate that the impact of soy on child mortality is still statistically significant after narrowing down the comparison group. In any case, the statistically significant result on the earlier Hausman test should also allay concerns that differences *between* departments might lead to bias from confounding unobserved variables. Since the fixed effects model for the full panel regressions isolates the estimates to the variation within the same department, innate time-invariant differences will fall out.

[5] This is a valid assumption for several reasons: first, children exposed to the same level of chemical contamination as adults experience a higher dosage based on their smaller mass. Secondly, child populations are generally more vulnerable to socioeconomic shocks.

Lastly, I look at the impact on child mortality of different Argentinian staple crops. Maize, wheat and sunflower are the next highest ranked crops in Argentina in terms of land area devoted to harvest. Table 2d shows the results of the simple regression, using these crops as the treatment instead of soy. I also included cotton, which has historically been cultivated with glyphosate, though is grown in much lesser quantities compared to soybeans. The table shows that soy is the only one of the crops to have an impact on child mortality at any conventional level of significance. I also performed the crop comparisons using the full panel fixed effects and attained similar results – soy was the only crop that demonstrated a statistically significant increase on child mortality (not included in tables).

D. *Controls for unobserved, Time-Varying Characteristics*

Though I have presented a reasonable proxy for the parallel trends assumption using alternative treatment and control groups, the assumption that my regression models correctly control for all confounding and unobserved time-varying characteristics is unfortunately much more difficult to prove. For instance, while I directly control for a number of time-varying characteristics, such as literacy, poverty, unemployment and infrastructure, it is highly unlikely that I managed to control for all possible relevant variables. This raises the concern that certain omitted time varying characteristics would bias the estimates.

On one hand, I do argue that the probability of receiving treatment – i.e. increasing soy cultivation between 2001 and 2010 – is largely dependent on time fixed characteristics. This is a reasonable assumption to make when dealing with agricultural production, simply because the ability of crops to grow in particular regions are highly dependent on unchanging factors such as soil quality, yearly precipitation, average temperatures and altitude. However, my earlier probit

model indicates that some time-variant attributes, such as unemployment rate and access to an indoor water supply, affect the likelihood of being in the treatment group, implying that there may be other time-varying attributes that could be correlated to child mortality and also the treatment.

A separate reason why I believe my estimates are unbiased is the results from the above crop comparison tests, which show that only soy has a statistically significant impact on child mortality. These results are critical for my argument, since it essentially means that any time varying omission leading to spurious estimates on soy would have to be correlated with soy only and NOT with other crops. These results rule out the presence of a great deal of plausible explanations that diverge from my theoretical pathways.

Admittedly, my defense for adequately controlling time variant factors is far from irrefutable. Therefore, the difference-in-difference results above should be viewed only tentatively until further conclusions can be made about the likelihood that omitted time-varying characteristics biased the estimates.

Section V – Results of Glyphosate on Child Mortality

Though the above results on soy harvest and child mortality should still be viewed as tentative, the findings are alarming enough to justify further exploration into whether glyphosate was the primary cause of increased child mortality.

A. *Tests for interaction effects*

One feasible hypothesis is that glyphosate contaminated water sources, causing illness in infants and eventual death. In order to test for this, I use a triple differences-in-differences approach and

look for whether there was any interaction effect between soy and water related infrastructure variables such as access to water inside the house or access to a water main or sewage. I created dummy variables for the DDD-interaction term, equal to 1 if the department fell in the lower 50^{th} percentile of departments in terms of access and 0 otherwise. The results, reported in table 5a, show no evidence of an interaction effect of soy on water related infrastructure at any conventional level of significance. I also looked for an interaction effects between soy and non-water infrastructure variables, namely cell phone ownership and literacy, to provide some basis of comparison. The interaction estimate for literacy was not meaningful, but interestingly, the interaction for cell phone ownership was statistically significant at the 90% level. This implies that soy had a greater effect on child mortality in departments that lacked cell phone usage. It should be stipulated, however, that the above results should be viewed provisionally; aggregate department level data potentially lacks the high level of granularity needed to draw more definite conclusions. Microdata pertaining to water related infrastructure at the household level would've been a much more useful interaction term, but is unfortunately outside the scope of this study.

B. *Tests for "compelling heterogeneity"*

I next conduct a series of tests for whether soy had differing effects on child mortality rates, limiting on certain departmental categories. For instance, I look at high population density departments compared to low, as well has departments which grew soy in 2001 versus those that did not. My hope was that any differences found between these department types might indicate that the increase in child mortality was related to glyphosate, as opposed to other pathways to mortality such as deforestation and environmental degradation. The results in table 5b show that departments with population densities greater than 10 persons per square kilometer experience a

greater impact of soy on child mortality. Unfortunately, this finding can be interpreted in many different ways. For instance, if the increase in child mortality is truly due to glyphosate, it could be argued that departments with higher population densities have more people living in close proximity to areas that are contaminated, leading to higher rates of chemically induced illness and eventual death. However, it could also be that higher density departments have more cities or urban areas. Given that a person is less likely to be exposed to glyphosate in an urban area, this may imply that the effect of soy on child mortality has nothing to do with the agrochemical. Simply, there are a myriad of other potential explanations, so the findings really do not allow us to draw any meaningful conclusions about glyphosate itself.

The second set of results on table 5b show that, in the departments which did not cultivate soy in 2001 but would eventually go on to cultivate it in 2010, the impact of soy on child mortality tended to be greater. These "new soy" departments are generally north and west of the Pampean region where soy was originally grown in Argentina, and were areas to which soy expanded after the introduction of Ht-varieties after 1996. The positive results on these estimates imply that the increase in child mortality may be due to sources other than glyphosate. The reasoning is that old and new soy areas would've used glyphosate to the same extent, and therefore we would not expect different levels of impact when comparing the two groups.

C. *Tests looking at differences between soy planted and soy harvested*

The final set of tests that I conducted involved looking at departments which had considerably different numbers of hectares planted versus hectares harvested. This scenario occurs in cases where part of a crop is lost due to drought or flooding. I hypothesized that farmers who were able to bring their soy completely to harvest sprayed more than farmers who did not, simply because there is no incentive to spray crops that have already failed. I used the full panel fixed effects

regression, comparing departments based on the difference between total soy harvested and total soy planted throughout the 11 year period. The results (not included in tables) were that there was no statistically significant difference between the two regions. This result is not too surprising, however, given the small number of hectares that failed. On average, departments were able to harvest 96% of the amount originally sowed. Furthermore, it is impossible to determine how much less glyphosate these "non-harvested hectares" used. For instance, drought or flooding may have ruined the crop just right before harvest, meaning that there would be very little difference in the amount of glyphosate used. Ultimately, more granulated data, specific to farm level, would be helpful in establishing more confidence in the estimates, but this falls beyond the scope of this study.

Section VI – Conclusions, Limitations and Future research

The primary goal of this study was to see if soy cultivation had any impact on CMR at the department level over the last decade. Additionally, I wanted to determine whether glyphosate could be isolated as the primary cause of the rise in child mortality. The above results present a strong case for soy leading to higher rates of child mortality; however existing data do not allow me to draw any conclusions about this increase in child mortality being due to glyphosate specifically.

For the initial results on child mortality, I find that Departments which increased their land area for soy production between 2001 and 2010 have child mortality rates 3.47 points higher than departments that did not. This result is significant, as Argentina's CMR as of 2010 was just 13.94, meaning departments that heavily expanded soy had child mortality rates 24% higher than

the mean. This finding was robust across functional forms for variables relating to infrastructure and robust when accounting for variables in the dataset which had high rates of omission. Most importantly, I find a positive and statistically significant result for soy only, and not for other staple crops such as wheat, maize, cotton and sunflower.

Despite these results, the findings of soy on child mortality should be received only tentatively until certain limitations below can be adequately addressed:

1) The first limitation, already mentioned, is the possibility that the model failed to control adequately for time varying characteristics. Since fixed effects models only address time invariant heterogeneity, any department level characteristics that changed with time that are correlated with both soy hectares harvested and child mortality could bias estimates. Though the study provides some defense that essential variables have been controlled for, I cannot eliminate the possibility that remaining unobservables may bias estimates.
2) The second limitation of this study is that soy harvest was not simply a binary treatment that started contemporaneously with the initial years of this study. Instead, it had been grown in Argentina since the 1970's with GMO soy adoption starting in 1997, three years before the start of the health data. This means that the departments in the treatment group may have already begun experiencing the "effects" of soy from the outset of the study, meaning that estimates on the impact of soy could be biased. However, the bias could work in favor of the main hypothesis as well.
3) The last limitation is represented by weaknesses in the dataset itself. Though they study looked at the impact of soy at the department level, the dataset for variables for unemployment, income and poverty only included province level data. Though these covariates were not the main items of interest, finer grained data would've been desirable and may have potentially produced different results. Furthermore, information on education level was unavailable at the department

level. The study used literacy rate, which was available at the department level, as a proxy for education; but it should be noted that academic papers examining child mortality rate typically include a measure of maternal years of education received (Cruces 2008, Galiani 2003).

As for my secondary research question, this study unfortunately did not allow me to draw any conclusions relating to the causal effects of glyphosate on child mortality rate. One of the main limitations of this study was clearly the lack of availability of crop spraying data. Ideally, I would've been able to find sources specifically pertaining to where glyphosate was used by location, but this data is unfortunately not collected by state agencies and highly confidential to the companies that produce the chemicals. Inevitably, soy hectares harvested proved to be a far from perfect proxy for glyphosate usage overall:

- First, regional variations may exist in terms of how much glyphosate is used per hectare of land.
- Secondly, it is impossible to exclude other agrochemicals such as pesticides and insecticides that are sometimes used in conjunction with glyphosate.
- Lastly, temporal variations for GM soy adoption across departments may also weaken the ability of soy harvested hectares to act as a proxy.

The main cause of higher child mortality rates in soy producing departments may still be related to agrochemical use, though there are plenty of alternate explanations as well. As mentioned earlier, child mortality rates could be caused by hardship arising from land degradation or migration. Another scenario, not yet explored, is that soy may have undergone more volatile regional price shocks than other crops, which may have also contributed to higher mortality rates in children.

Though glyphosate could not be ruled as the primary cause of increased mortality rates, the general findings on soy intensification are indeed significant and troubling enough to warrant additional research on the subject matter. For one, scholars of human security issues may be interested in determining whether the higher child mortality rates in heavily soy intensified areas were due to factors such as indigenous population displacement. Another motivation for further research on the effects of GMO soy is that prior studies have focused on clinical methodologies conducted in sterile lab environments: e.g. in-vitro tests on amphibian and avian embryonic tissues. It can be argued that these conditions represent a poor proxy for the true epidemiological pathways of entry of glyphosate into human physiological systems. For obvious reasons, legal and morals restrictions prevent a direct medical study of the effects of glyphosate on human beings, but social science techniques like the ones employed in this study could help shed more light on the impacts of GMO technology. Promising avenues exist for detecting potential links between glyphosate and human health using alternate variables of interest such as cancer rates or miscarriages. For instance, one such source of incoming data comes from the UNICEF Multiple Cluster Indicator Survey (MICS4) of Argentina, due for release later this year. The fieldwork for the MICS4 survey of Argentina was conducted in 2011-2012 and includes microdata from 23,791 households throughout the nation, focusing on variables related to child and maternal health. Another possibility would be to consolidate cancer incidence rates from province level health registries, thus providing another key dependent variable for examination. These endeavors were outside the scope of this thesis but represent promising avenues for future research.

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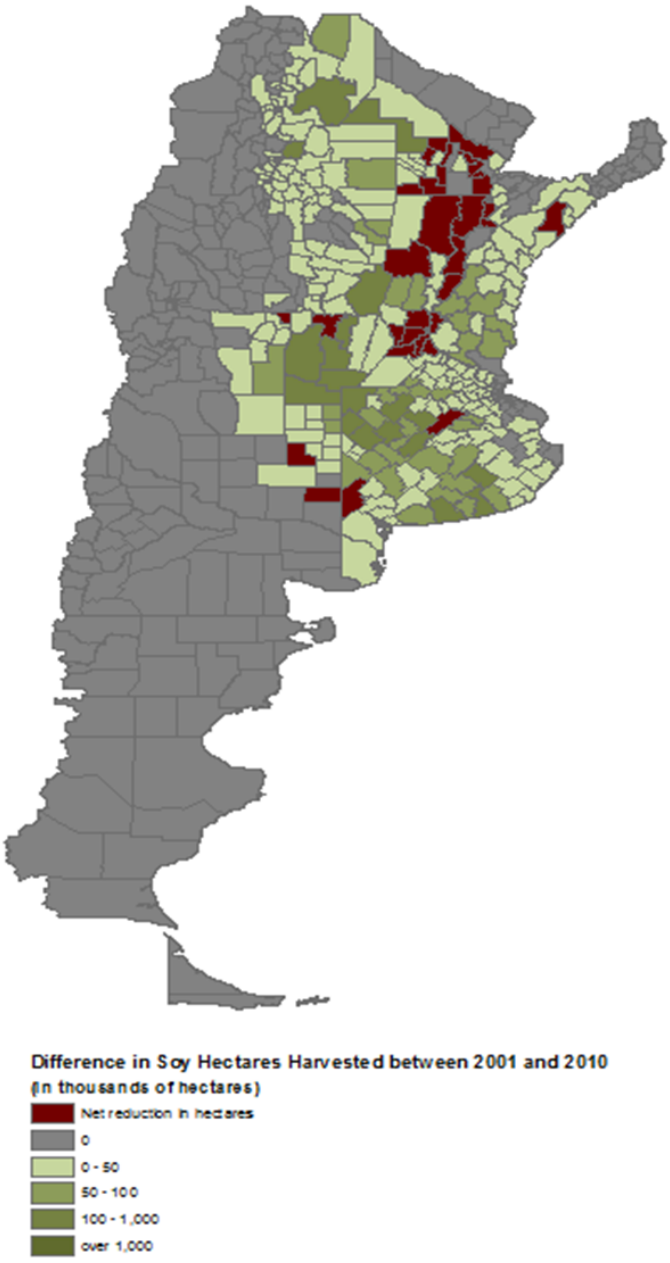
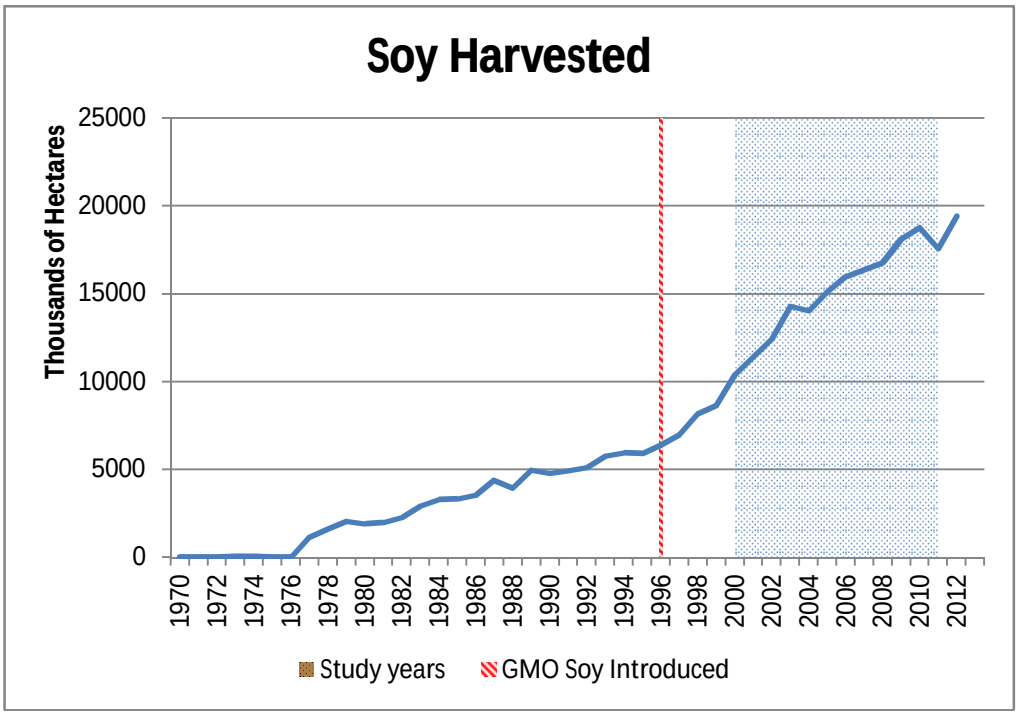
APPENDIX 1: Definitions and Sources

Variable	Definition	Source
Child Deaths	Number of deaths of children less than 5 years old by, department and year	Ministerio de Salud de la República Argentina
Live Births	Number of registered live births, by department and year	Ministerio de Salud de la República Argentina
Child Mortality Rate	= Child Deaths / Live Births	Ministerio de Salud de la República Argentina
Population	Total population of the department, by year	Ministerio de Salud de la República Argentina
Income	Income per capita, in dollars, by Province by year	Permanent Household Survey (EPH), INDEC
Poverty	Percent of the total population living under the poverty line, by Province by year	Permanent Household Survey (EPH), INDEC
Unemployment Rate	Unemployment rate by Province by year	Permanent Household Survey (EPH), INDEC
Literacy	Number of people over the age of 10 who have the ability to read and write, by department. (Also expressed as a %)	INDEC Decenial Census, 2001 and 2010
Water Main	Number of households with access to a water main, by department. (Also expressed as a %)	See Literacy
Water in House	Number of households with access to water inside the house, such as through a tap, by department. (Also expressed as a %)	See Literacy
Sewage	Number of households with access to sewage services, by department. (Also expressed as a %)	See Literacy
Toilet	Number of households with an indoor toilet, by department. (Also expressed as a %)	See Literacy
Gas Main	Number of households with access to a gas main, by department. (Also expressed as a %)	See Literacy
Refrigerator	Number of households with a refrigerator, by department. (Also expressed as a %)	See Literacy
Cell Phone	Number of households with a cell phone , by department. (Also expressed as a %)	See Literacy
Computer	Number of households with a computer, by department. (Also expressed as a %)	See Literacy
Land Area	Land area of department in square kilometers	GADM Database
Temperature	Average annual temperature, in degrees centigrade, by department	WorldClim GIS Data
Precipitation	Average annual precipitation, in degrees centigrade by department	WorldClim GIS Data
Coldest Quarter	The average temperature of the coldest 3 months, by department	WorldClim GIS Data
Driest Quarter	The average precipitation of the driest 3 months, by department	WorldClim GIS Data
Soy Harvested Area	Number of hectares of soy harvested, by department and year	Sistema Integrado de Información Agropecuaria
Cotton Harvested Area	Number of hectares of cotton harvested, by department and year	Sistema Integrado de Información Agropecuaria
Maize Harvested Area	Number of hectares of maize harvested, by department and year	Sistema Integrado de Información Agropecuaria
Wheat Harvested Area	Number of hectares of wheat harvested, by department and year	Sistema Integrado de Información Agropecuaria
Sunflower Harvested Area	Number of hectares of sunflower harvested, by department and year	Sistema Integrado de Información Agropecuaria

APPENDIX 2: Map of Soy 2001 and 2010 Harvested Differentials

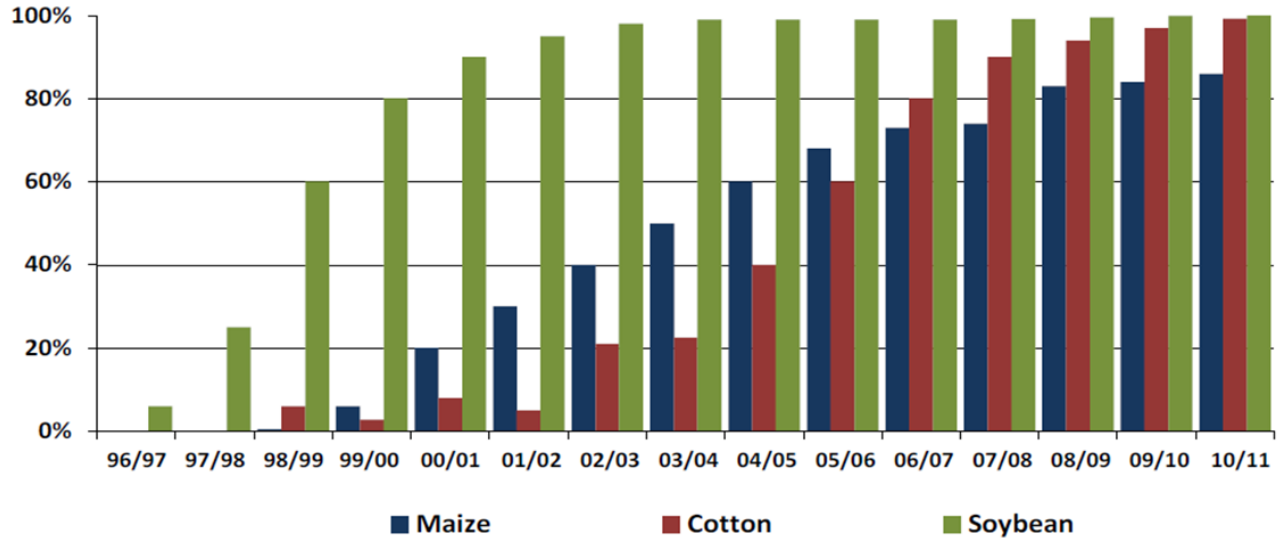


APPENDIX 3: Historical Chart of Soy Harvested Hectares for Argentina



APPENDIX 4: Argentina GMO crop share of total crop area for Maize, Cotton, Soy

Figure 1.4. Evolution of GM crop share in the total area for each crop



APPENDIX 5: Evolution of Mortality Rates for Departments which increased Soy Production vs. Departments which did not

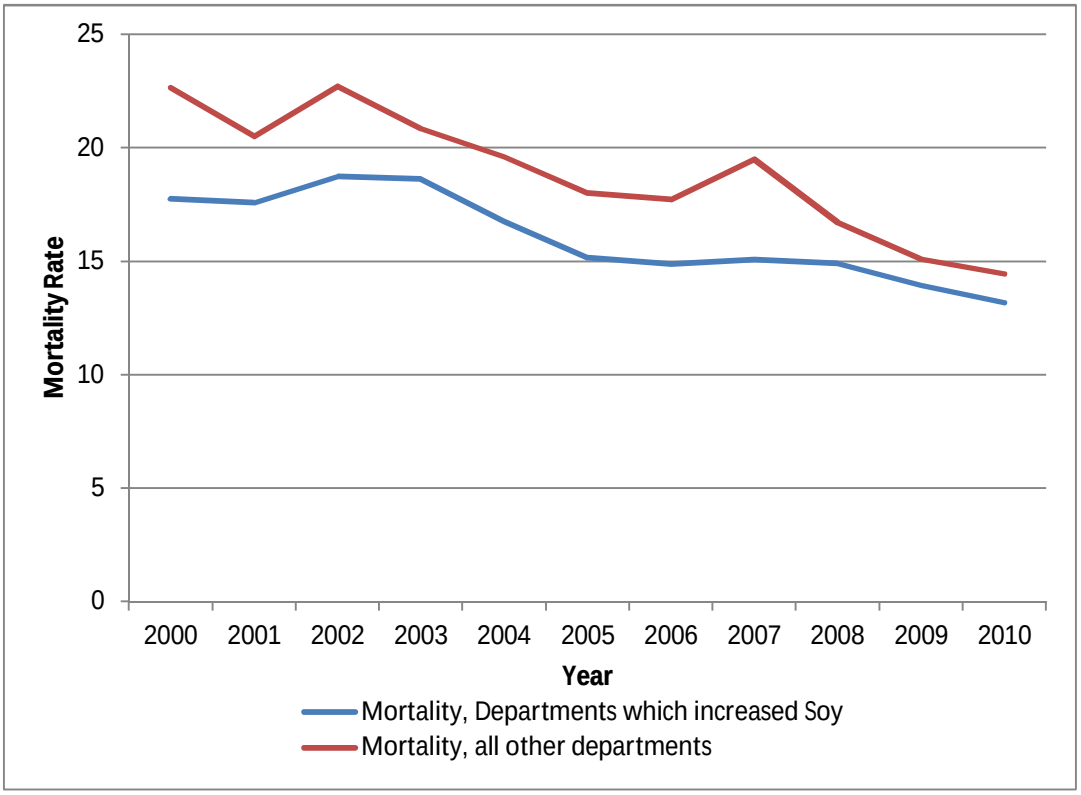


TABLE 1: BASELINE MEANS COMPARISON

Variable	Soy Expanded Areas		Control		Difference	Sample Size
	Mean	SE	Mean	SE		
child mortality rate	17.691	(1.113)	20.44	(0.754)	-2.749**	510
altitude	284.8	(73.65)	811.8	(50.42)	-527.0***	495
temperature	17.355	(0.358)	15.2	(0.245)	2.155***	495
precip	825.6	(33.08)	635.8	(22.65)	189.8***	495
coldest	11.193	(0.368)	9.112	(0.252)	2.081***	495
driest	91.012	(7.160)	84.24	(4.902)	6.772	495
sqkm	4224	(651.7)	6,715	(444.5)	-2,491***	501
watermainp	0.76741	(0.0174)	0.771	(0.0118)	-0.00359	510
waterinhousep	0.5824	(0.0164)	0.526	(0.0111)	0.0564***	510
sewagep	0.3734	(0.0254)	0.274	(0.0172)	0.0994***	510
toiletp	0.5913	(0.0164)	0.52	(0.0111)	0.0713***	510
gasp	0.3848	(0.0307)	0.339	(0.0208)	0.0458	510
fridgep	0.6741	(0.0136)	0.601	(0.00924)	0.0731***	510
cellp	0.1869	(0.00705)	0.157	(0.00477)	0.0299***	510
computerp	0.5913	(0.0164)	0.52	(0.0111)	0.0713***	510
poverty	0.4557	(0.00833)	0.425	(0.00571)	0.0307***	497
unemployment	16.81	(0.294)	15.54	(0.202)	1.270***	497
populationx	57903	(12,382)	75,679	(8,387)	-17,776	510
births	1083.2	(202.9)	1,378	(137.5)	-294.8	510
deaths	435.43	(93.77)	522.6	(63.52)	-87.17	510
watermainv	16090	(3,384)	18,180	(2,292)	-2,090	510
waterinhousev	12669	(2,778)	15,393	(1,882)	-2,724	510
sewagev	9916	(2,440)	11,184	(1,653)	-1,268	510
toileth	13032	(2,821)	15,655	(1,911)	-2,623	510
gasv	10712	(3,159)	14,782	(2,140)	-4,070	510
fridgeh	14170	(3,070)	17,616	(2,080)	-3,446	510
cellh	4213	(900.4)	4,779	(609.9)	-566.0	510
computerh	13032	(2,821)	15,655	(1,911)	-2,623	510

Notes: Treatment and control groups are highly unbalanced when using proportional (p) figures, though balanced with using raw (v/h) data. In 2001, the treatment group tended to have lower child mortality, have higher access to household durables and infrastructure, but also higher poverty and unemployment

TABLE 1a: PROBIT ESTIMATION FOR CAUSES OF SOY EXPANSION

Dependent Variable: Was Treated	
population density	-0.000368***
	(0.000100)
hectares	3.07e-07**
	(1.55e-07)
temperatured	-0.172***
	(0.0545)
precipd	-0.00457***
	(0.000486)
coldest	-0.0155
	(0.0410)
driest	-0.00297**
	(0.00142)
watermainp	0.793
	(0.560)
waterinhousep	-8.305**
	(3.776)
toiletp	9.834**
	(4.193)
fridgep	0.555
	(1.585)
cellp	1.307
	(2.087)
poverty	8.465***
	(1.688)
unemployment	-0.0775**
	(0.0334)
Constant	-2.559***
	(0.978)
Observations	482

Note: Soy growing is dependent on largely time invariant, department level factors such as rainfall and temperature, though there also seems to be a connection with poverty and unemployment

TABLE 2: SIMPLE DD ESTIMATION

Dependent: Child Mortality Rate	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Dummy Only				Intensity			
Soy effect	-4.960***	-5.011***	-3.171**	-3.190**	-10.71**	-11.04**	-2.798	-2.379
	(1.546)	(1.527)	(1.270)	(1.186)	(4.354)	(4.440)	(3.729)	(3.495)
Time effect	7.518**	8.415***	-6.600	3.589	6.595**	7.662***	-6.387	2.681
	(3.186)	(2.720)	(6.884)	(5.711)	(3.078)	(2.708)	(7.650)	(5.657)
Interaction Term	**3.936***	**3.469****	**2.485**	**2.362**	**12.25****	**9.969***	**5.787**	**5.970**
	(1.957)	**(1.566)**	**(1.536)**	**(1.409)**	**(5.217)**	**(5.595)**	**(4.035)**	**(5.144)**
Sewage	Yes	No	Yes	No	Yes	No	Yes	No
Porportional fuctional form	No	No	Yes	Yes	No	No	Yes	Yes
# of Observations	781	989	781	989	781	989	781	989
R^2	0.220	0.174	0.260	0.205	0.197	0.154	0.250	0.197

Note: Simple difference-in-difference regressions showed a statistically significant impact of soy on child mortality rate. Regressions were run using a dummy variable only (1 if the department grew more soy in 2010 than 2001, 0 otherwise) as well as a variable that measured intensity of soy expansion (the difference in soy hectares harvested between 2010 and 2001). These findings were not robust when using proportional figures for household durables and infrastructure. Two variables, gas and sewage, had a high percentage of missing data, so regressions were run with and without these variables.

TABLE 2a: TEST ON THE PARALLEL TRENDS ASSUMPTION

Effect of soy from 2000-2001	(1)
Soy effect	-4.841***
	(1.164)
Time effect	-2.198**
	(1.115)
Interaction Term	**2.092**
	(1.646)
# of Observations	1,020
R^2	0.024

Note: This test defends the parallel trends assumption by showing no statstically significant difference between the treatment and control trends for child mortality rate before the years of the study. However, this calculation is admittedly weak as data were only available for 2000-2001, and a longer time range would've been preferable.

TABLE 2b: ALTERNATE TREATMENT GROUP

Dependent:	(1) Child Mortality	(2) Overall	(3) 16 to 64	(4) 64+	(5) Maternal
Soy effect	-0.168***	1.291**	0.0874	1.392***	-0.606**
	(0.0498)	(0.511)	(0.0894)	(0.478)	(0.223)
Time effect	0.264**	1.506**	-0.0793	1.290**	0.106
	(0.103)	(0.661)	(0.311)	(0.534)	(0.726)
Interaction Term	**0.120****	**0.0853**	**-0.0740**	**-0.000432**	**0.599****
	(0.0539)	**(0.369)**	**(0.0835)**	**(0.313)**	**(0.216)**
# of Observations	781	781	781	781	781
R^2	0.309	0.347	0.047	0.418	0.038

Note: Given that the test for parallel trends was weak, these regressions show the effect of soy on alternate treatment groups. Here we find a statistically significant impact of soy on child mortality, but not on overall mortality rates nor adult and elderly mortality rates. Interestingly, there is a statistically and positive effect on maternal mortality rates.

TABLE 2c: ALTERNATE COMPARISON GROUP

Dependent: Child Mortality Rate	(1)	(2)	(3)	(4)
	Dummy Only			
Soy effect	-4.216**	-4.415***	-3.291*	-3.630**
	(1.515)	(1.483)	(1.575)	(1.539)
Time effect	3.922	4.640	-14.02	-5.002
	(5.408)	(4.789)	(10.01)	(11.36)
Interaction Term	**4.194****	**4.085****	**2.299**	**3.666****
	(1.840)	**(1.725)**	**(1.759)**	**(1.663)**
Sewage	Yes	No	Yes	No
Porportional fuctional form	No	No	Yes	Yes
# of Observations	465	572	465	572
R^2	0.249	0.220	0.296	0.246

Note: I further establish that the treatment group presents a strong counterfactual case by selecting an alternate comparison group. Here, the treatment group stays the same, but the control group is limited to ONLY areas that grew soy in 2001.

TABLE 2d: ALTERNATE CROPS

	(1)	(2)	(3)	(4)	(5)
Dependent:	Soy	Maize	Cotton	Wheat	Sunflower
Soy effect	-4.960***	-1.767	1.233	-0.0483	-0.877
	(1.546)	(1.214)	(3.044)	(1.463)	(0.783)
Time effect	7.518**	7.904**	7.493**	7.689**	7.728**
	(3.186)	(3.449)	(3.491)	(3.520)	(3.498)
Interaction Term	**3.936***	**0.142**	**-0.649**	**-0.619**	**-0.289**
	(1.957)	**(1.251)**	**(0.926)**	**(1.142)**	**(1.042)**
# of Observations	781	781	781	781	781
R^2	0.220	0.197	0.193	0.192	0.193

Note: I ran the same "Basic Difference-in-Differences" regression using alternate crops and found no statistically significant impacts other than soy

TABLE 3: DD ESTIMATION WITH FIXED EFFECTS

Dependent: Child Mortality Rate	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Dummy Only				Intensity			
Time effect	-3.420	6.346	-6.883	-15.22	-3.107	5.931	-7.145	-16.53
	(2.596)	(4.061)	(5.666)	(11.94)	(2.180)	(4.486)	(5.650)	(12.43)
Interaction Term	**-1.143**	**3.370****	**-1.149**	**2.024**	**0.178**	**0.516****	**0.106**	**0.428****
	(1.186)	**(1.554)**	**(1.219)**	**(1.313)**	**(0.110)**	**(0.195)**	**(0.122)**	**(0.207)**
Sewage	Yes	No	Yes	No	Yes	No	Yes	No
Porportional fuctional form	No	No	Yes	Yes	No	No	Yes	Yes
# of Observations	749	930	749	930	749	930	749	930
# of Cross Sectional Observations	469	471	469	471	469	471	469	471
R^2	0.184	0.245	0.190	0.285	0.183	0.236	0.188	0.283

Note: Results are statistically significant, but only when removing the two variables with high omission rates (sewage and gas).

TABLE 4: FULL PANEL ESTIMATION WITH FIXED EFFECTS

Dependent Variable:	(1)	(2)	(3)	(4)
	Child Mortality Rate			
Soy effect	**8.011****	**8.425****	**6.322***	**6.298***
	(3.247)	**(3.341)**	**(3.363)**	**(3.105)**
Sewage	Yes	No	Yes	No
Porportional fuctional form	No	No	Yes	Yes
# of Observations	5,511	5,511	5,511	5,511
# of Cross Sectional Observations	501	501	501	501
R^2	0.044	0.044	0.048	0.048

Note: Full panel regressions use imputed data based on 2001 and 2010 Census results. Data was only imputed for variables pertaining to household durables and infrastructure; data from the main dependent (child mortality) and independent (soy harvest) already existed for all the years of the study. Hausman and Breusch Pagan LM tests imply that fixed effects will yield the most consistent estimates for effect of soy on child mortality rate.

Hausman Test

Note: the rank of the differenced variance matrix (5) does not equal the number of coefficients being tested (15); be sure this is what you expect, or there may be problems computing the test. Examine the output of your estimators for anything unexpected and possibly consider scaling your variables so that the coefficients are on a similar scale.

	---- Coefficients ----			
	(b) fixed	(B) random	(b-B) Difference	sqrt(diag(V_b-V_B)) S.E.
soypart	8.010576	-1.01369	9.024267	3.42359
watermainv	-0.00026	0.000128	-0.00038	0.00021
waterinhou~v	0.000347	-0.00069	0.001033	0.0009
sewagev	0.000142	2.39E-06	0.00014	0.00024
toileth	-0.00113	0.000108	-0.00124	0.00129
gasv	9.52E-05	-6.6E-05	0.000161	0.0002
fridgeh	0.000223	-0.00015	0.000377	0.00104
telephoneh	6.03E-05	0.000408	-0.00035	0.00029
cellh	7.01E-05	0.000125	-5.5E-05	0.00023
computerh	-0.00024	-8.3E-05	-0.00016	0.00019
poverty	7.377032	10.81478	-3.43775	0.75925
income	-0.00074	-0.00059	-0.00015	0.00016
unemployment	0.187482	0.020633	0.16685	0.03686
literacy	0.000157	0.000102	5.55E-05	0.00017
density_n	-0.00029	-0.00045	0.000163	0.00028

b = consistent under Ho and Ha; obtained from xtreg
B = inconsistent under Ha, efficient under Ho; obtained from xtreg

Test: Ho: difference in coefficients not systematic

chi2(5) = (b-B)'[(V_b-V_B)^(-1)](b-B)
= 34.09
Prob>chi2 = 0.0000
(V_b-V_B is not positive definite)

Breusch and Pagan Lagrangian multiplier test for random effects

y[match3,t] = Xb + u[match3] + e[match3,t]

Estimated results:

	Var	sd = sqrt(Var)
y	167.2616	12.93297
e	120.7678	10.98944
u	30.93376	5.561813

Test: Var(u) = 0
chibar2(01) = 1149.20
Prob > chibar2 = 0.0000

TABLE 4a: SUMMARY STATISTICS

Variable		Mean	Std. Dev.	Min	Max	Observations	
Department	overall	283.5	147.2372	29	538	N =	5610
	between		147.3686	29	538	n =	510
	within		0	283.5	283.5	T =	11
Year	overall	2005	3.16256	2000	2010	N =	5610
	between		0	2005	2005	n =	510
	within		3.16256	2000	2010	T =	11
Child Mortality	overall	17.59951	12.88544	0	200	N =	5610
	between		7.23439	0	67.57045	n =	510
	within		10.66731	-42.4285	162.1217	T =	11
Soy Harvest/Hectares	overall	0.081556	0.166497	0	1.075061	N =	5511
	between		0.161316	0	0.88629	n =	501
	within		0.041779	-0.27136	0.501349	T =	11
Water Main	overall	17471.12	38657.34	-28.2222	413341.3	N =	5610
	between		38608.19	8	411264.7	n =	510
	within		2540.677	-54937.2	89879.46	T =	11
Water in House	overall	16269.4	35730.06	10.55556	411263	N =	5610
	between		35526.67	17.77778	355237.9	n =	510
	within		4091.776	-70806.2	103345	T =	11
Sewage	overall	10548.04	26646.55	-261	277408.9	N =	5610
	between		26617.31	0	260469.4	n =	510
	within		1679.402	-26635.9	47731.93	T =	11
Toilet	overall	16311.92	35463.24	26.66667	394337	N =	5610
	between		35324.36	52.11111	359305.9	n =	510
	within		3472.078	-57102.5	89726.36	T =	11
Gas	overall	12211.48	33196.9	-704	352877.3	N =	5610
	between		33133.15	0	317810.7	n =	510
	within		2487.228	-36759.6	61182.59	T =	11
Fridge	overall	17910.93	38490.08	17.88889	462862	N =	5610
	between		38352.25	53.44444	380049.2	n =	510
	within		3635.031	-64901.9	100723.7	T =	11
Telephone	overall	10320.34	26327.65	-1.44444	290968	N =	5610
	between		26273.76	0.444445	248973.3	n =	510
	within		2016.167	-37099.7	57740.34	T =	11
Cellphone	overall	10760.06	25257.93	-435.556	410658	N =	5610
	between		22577.74	27.44444	228089.3	n =	510
	within		11362.99	-182954	204474.5	T =	11
Computer	overall	12325.17	27889.15	22	342980.7	N =	5610
	between		27662.9	30.33333	287502.3	n =	510
	within		3732.704	-43153.2	67803.49	T =	11
Poverty	overall	0.369672	0.200853	0	0.8086	N =	5610
	between		0.086092	0.133521	0.508236	n =	510
	within		0.181503	0.008866	0.726761	T =	11
Income	overall	545.4806	510.4146	4.628521	3239.719	N =	5610
	between		153.421	375.9965	1207.492	n =	510
	within		486.8542	-656.464	2623.671	T =	11
Unemployment	overall	10.85185	4.980792	0.8	23.47987	N =	5610
	between		2.456771	2.313208	13.99623	n =	510
	within		4.333973	2.06317	21.44304	T =	11
Literacy	overall	57475.68	118148.3	353.2222	1426897	N =	5610
	between		118109.2	362.1111	1238703	n =	510
	within		5840.599	-130719	245670.1	T =	11
Density	overall	201.0922	958.035	0.036704	9664.239	N =	5610
	between		885.279	0.116549	7933.547	n =	510
	within		368.1502	-7109.89	1931.784	T =	11

TABLE 5a: INTERACTION EFFECTS

Interaction effect:	(1) WaterMain	(2) WaterInHouse	(3) Sewage	(4) Cell	(5) Literacy
Soy Interaction Term	3.119*	2.131	3.989*	1.448	3.868
	(1.739)	(1.607)	(2.072)	(1.519)	(2.372)
Triple DDD Interaction	**0.423**	**0.940**	**-0.922**	**4.815***	**0.485**
	(2.591)	**(3.253)**	**(2.135)**	**(2.656)**	**(2.741)**
# of Observations	781	781	781	781	781
R-squared	0.233	0.235	0.224	0.230	0.227

Note: The soy dummy was interacted with a dummy equal to 1 if the department was in the lower 50th percentile of departments in terms of aggregate access to the above variables. There is not yet strong evidence to claim that the increased child mortality rate in soy intensified regions is due to glyphosate.

TABLE 5b: HETEROGENEOUS EFFECTS TESTS

Dependent: Child Mortality Rate	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Density<3		Density>10		Old Soy		New Soy	
Soy effect	-7.047**	-6.837**	-2.119**	-2.081**	-2.42	-2.638*	-6.715**	-6.962**
	-2.933	-2.976	-0.902	-0.866	-1.39	-1.346	-3.022	-2.904
Time effect	6.801	7.684	12.60**	10.12**	5.906	4.144	8.238*	9.478**
	-7.863	-6.78	-5.018	-3.981	-4.578	-4.957	-4.155	-3.529
Interaction Term	**2.075**	**1.891**	**1.725**	**3.069***	**2.69**	**3.935**	**6.826***	**5.652****
	-3.301	**-3.225**	**-1.605**	**-1.526**	**-2.555**	**-2.434**	**-3.315**	**-2.539**
Sewage	Yes	No	Yes	No	Yes	No	Yes	No
# of Observations	197	288	366	414	357	420	424	569
R^2	0.223	0.177	0.318	0.276	0.264	0.242	0.209	0.157

Note: The basic regression was filtered based on population density as well whether soy was originally planted in that department in 2001. There is not yet strong evidence to claim that the increased child mortality rate in soy intensified regions is due to glyphosate.