

Long-Term Health Effects of Vietnam War's Herbicide Exposure on the Vietnamese Population

Nikolay Gospodinov^a, Hai V. Nguyen^b

^a Department of Economics, Concordia University and CIREQ

1455 de Maisonneuve Blvd. West Montreal, Quebec, H3G 1M8, Canada

Email address: nikolay.gospodinov@concordia.ca

^b Corresponding author. Faculty of Pharmacy, University of Toronto

144 College St, Toronto, M5S 3M2, Canada

Email address: vanhai.nguyen@utoronto.ca

Abstract

This paper investigates the long-term health impact of Agent Orange and other herbicides used in the Vietnam War on the Vietnamese population. Using health information of nearly 160,000 individuals and exploiting the variation in Agent Orange exposure by geographic areas and cohorts in a difference-in-difference setup, we analyze the consequences of Agent Orange and other herbicide exposure on the prevalence of cancer and hypertension. Our results indicate that exposure to Agent Orange increases the risk of cancer and hypertension with the largest burden falling on individuals in the cohort who lived in the most exposed areas during the spraying period.

Keywords: Agent Orange, difference-in-difference, herbicide exposure, Vietnam War.

1 Introduction

During the period 1961-1971 in the Vietnam War,¹ the US military used herbicides to clear dense forests where opposition forces were hiding and to destroy crops that those forces relied on. Consequently, civilians and members of both US and Vietnamese military forces were exposed to the herbicides. Among the chemicals used are the highly toxic Agent Orange and other herbicides including Agents Purple, Pink and Green, all of which were contaminated with varying levels of dioxin.

After the War, the long-term health effects of herbicide exposure have been a subject of debate and controversy. There have been many lawsuits brought by both US and Vietnamese veterans against the manufacturers of these chemicals. The importance of the issue is also reflected in the Act by the US congress that required the Institute of Medicine to review scientific evidences and publish its conclusions on the health effects of Agent Orange every two years.

Numerous medical and epidemiological studies establish a link between the exposure to Agent Orange and other herbicides (Agent Orange exposure, for short) and various types of cancer (lung, prostate, larynx, and prostate), type 2 diabetes (IOM, 2006), skin diseases (IOM, 2002) and cardiovascular disease mortality (Humblet *et al.*, 2008). The disease burden falls not only on the War veterans and civilians who were directly exposed but also on their children. It has been found that the children of veterans exposed to Agent Orange during the Vietnam War may have a higher risk of certain types of leukemia (IOM, 2000) and birth defects (Anh *et al.*, 2006).

¹ Vietnam War is between the communist Northern Vietnam and American-supported Southern Vietnam. It began in 1959 and ended on the 30th of April, 1975.

The results from the empirical studies of the effects of Agent Orange on hypertension are somewhat mixed. Kang *et al.* (2006) find that the US army veterans who were occupationally exposed to herbicides in Vietnam experienced significantly higher risks of diabetes, heart disease, hypertension, and non-malignant lung diseases than other veterans who were not exposed. In contrast, Calvert *et al.* (1998) do not uncover any significant association between dioxin and self-reported hypertension after controlling for hypertension risk factors. Bertazzi *et al.* (2000) study the dioxin accident in 1976 in Seveso, Italy, and report that while mortality from cancer has increased significantly among men in the highly exposed zones, hypertension risk does not appear to be significantly elevated. This result is also consistent with Chen *et al.* (2006) who show that serum concentrations of dioxins are not associated with an increased incidence of hypertension when major risk factors are controlled for.

Most of the existing studies of the effects of Agent Orange suffer from some common limitations. First, many medical studies utilize small samples of Vietnam War veterans (Kang *et al.*, 2006), chemical workers (Ott *et al.*, 1996; Fingerhut *et al.*, 1991; Manz *et al.*, 1991), pesticide manufacturers and applicators (Hooiveld *et al.*, 1998; Saracci *et al.*, 1991) and are often based on self-reported data. The small sample size and cross-sectional nature of these studies may lead to low participation rates and thus, sample selection bias and survivor bias (i.e. analyzing only disease prevalence among people who are still alive). Second, many studies are confined to detecting traces of dioxin in blood of affected people in Vietnam, rather than showing a causal link between Agent Orange exposure and the incidence of diseases such as cancer, mental underdevelopment, etc. In addition, potential confounders such as bombing intensity that are correlated with people's health anomalies are typically not accounted for.

Finally, while the treatment group is well defined in all medical studies, it is not clear who constitute the appropriate control group, especially when this control group is selected from exposed areas. This raises questions about the valid comparability between the treatment and control groups.

Despite the voluminous literature on the effects of Agent Orange, there are very few population-based studies on the long-term health consequences of the Agent Orange exposure on the Vietnamese population. We are only aware of one study by Do (2009) who independently studies the effects of Agent Orange on the prevalence of cancer in Vietnam. The analysis in our article and Do's (2009) paper, however, differs along several important dimensions. First, we study not only effects of herbicide spraying on self-reported cancer rates but also on objectively measured hypertension. Second, we capitalize on the fact that the North has not been exposed to herbicide spraying which allows us to use it as a valid treatment group in our difference-in-difference analysis. Third, Do's (2009) geographical unit of analysis is very small (commune level) and a failure to control for migration over the thirty years between the end of the War and the time of the survey raises concerns about the validity and the robustness of his findings. Finally, the causal interpretation of Do's (2009) results is not entirely clear.

The scarcity of the literature that use population based study is unfortunate for a number of reasons. First, until now all Vietnamese veterans' legal campaigns to claim compensation from US chemical companies that produced the toxic herbicides have been largely unsuccessful. Analysts observed that, political reasons aside, the lawsuits have failed because there is a lack of convincing evidence that the chemicals have caused disabilities and deadly diseases in exposed individuals. Second, it is reasonable to conjecture that the US veterans are relatively less exposed to Agent Orange than Northern Vietnamese veterans who in turn are probably less

exposed than the civilians in the sprayed areas. As a result, the health effects of Agent Orange estimated from samples of US veterans in Vietnam are likely to significantly underestimate the long-run consequences of the sprayed herbicides on the Vietnamese population. Third, Stellman *et al.* (2003) substantially revised up the amount of herbicides sprayed, which is almost double the previous estimates, and suggested that millions of Vietnamese were likely to have been sprayed upon directly. This raises the urgent need to investigate the long term health impacts of dioxin on the whole population.

More generally, there are very few studies on the long-term effects of wars and conflicts on the population health. Most studies have so far focused on the economic consequences - Davis and Weinstein (2002) for Japan; Brakman *et al.* (2004) for Germany; Miguel and Roland (2011) for Vietnam; and Bellows and Miguel (2009) for Sierra Leone - or on education and income outcomes as in Shemyakina (2006) and Akresh *et al.* (2011). One notable exception is Bundervoet *et al.* (2009) who studied the effects of the civil war in Burundi on the child's health outcomes. They find that a Burundi child exposed to the conflict will have a lower height compared to non-exposed children. Given that good health can improve households' welfare and income through improving the capacity to learn and work (Spence and Lewis, 2009) and is the main determinant of happiness (Easterlin, 2004), more research on the long-term health consequences of wars is clearly needed.

In this paper, we exploit a unique, nationally representative health survey to provide the first population-based evidence on the effects of herbicide exposure on the health of Vietnamese population. We focus on the effects of Agent Orange exposure on self-reported cancer prevalence and objectively measured hypertension. In addition to being objectively measured, our choice of looking at hypertension also reflects the considerable attention that this condition

attracted in the recent economic literature. Hypertension is found to be a costly health condition with an estimated annual cost of \$55 billion for the US and \$1,131 for an individual with hypertension (Balu and Thomas, 2006). Given its close relationship with happiness, hypertension has been suggested as a more objective measure of the well-being of a nation (see Blanchflower and Oswald, 2008).

The present study overcomes the limitations of the previous literature and offers several advantages. First, by using a nationally representative and large micro-data survey,² we circumvent the sample selection problem. Also, by exploiting the fact that the Northern part of Vietnam has not been exposed to Agent Orange spraying, we can provide a valid comparison between the treatment group (those who reside in the South regions) and the control group (those who reside in the North). Furthermore, the coverage of the whole Vietnamese population enables us to study the impact of Agent Orange not only on the people who were directly sprayed upon during the Vietnam War but also on the children who were born during and after the spraying period.

The rest of the paper is organized as follows. Section 2 outlines the empirical strategy. Section 3 discusses the main characteristics of the data. The empirical results and robustness checks are presented in Section 4. Section 5 concludes.

² A related strand of literature evaluates the long term effects of several important events on health and educational outcomes using microdata surveys. Examples include the effects of Chernobyl nuclear accident (Almond *et al.*, 2009), malaria eradication in India (Cutler *et al.*, 2007), the 1959-1961 famine in China (Almond *et al.*, 2007), income shocks (Banerjee *et al.*, 2009), etc.

2 Empirical Strategy

2.1 Difference-in-differences framework

The identification of the causal effect of Agent Orange exposure proves to be a nontrivial exercise. In order to estimate this causal effect, we combine some unique characteristics of our data with the difference-in-differences (DID) methodology that is often used to evaluate the treatment effect of a medical or policy intervention on a subset of groups in particular periods (see Rubin, 1974; Meyer, 1995; Athey and Imbens, 2006; among others).

First, the fact that only the Southern part of Vietnam was sprayed with herbicides while the Northern part was not, allows us to assign people who reside in the North to our control group and those who in the South to the treatment group. Second, we capitalize on the observation that herbicides were only sprayed for a specific period in the War, i.e. 1961-1971. This allows us to divide the population into three cohorts. The first cohort consists of those who were born before the Agent Orange and other herbicides were sprayed (i.e., before 1961) and thus, were exposed directly to the herbicides. The second cohort includes those who were born during the period of herbicides spraying (1961-1971). For this cohort, we are interested in “*in utero*” effects, i.e. effects of “*in utero*” exposure to the herbicides on later health outcomes of the child (Almond *et al.*, 2007). The third cohort is composed of those who were born after the spraying period and enables us to study whether Agent Orange effects are passed from one generation to another.

The DID method explicitly accounts for variations in treatment across groups and over time by calculating the change over time for each group (first difference) and then subtracting the resulting change in the control group from the treatment group (the second difference). The general specification for a DID regression is:

$$Y_i = a + b*G_i + T_i*c + (G_i*T_i)' \delta + x_i'\beta + e_i,$$

where, in our setup, Y_i is a dummy variable which is equal to 1 if person i has specific disease linked to Agent Orange such as cancer or hypertension; G_i takes a value of 1 if the person resides in the South and 0 otherwise; T_i are cohort dummies for cohorts born before 1961 and during 1961-1971 (thus, the unexposed cohort born after the spraying period is omitted dummy); G_i*T_i are interaction terms between area dummy and cohort dummies; and x_i is a vector of control variables that include individual characteristics (education, gender, marital status, being employed or not, whether the respondent migrated over the past 3 years, etc), well-established risk factors for various diseases and blood pressure (health-related behaviors such as smoking and drinking, and weight) as well as year-of-birth fixed effects that control for cohort-specific factors that affect both the North and the South. In order to capture the general health and economic conditions of the area in which the person lives, the set of control variables also includes urban or rural area and number of pharmacies available in the individual's residence area. Finally, we also incorporate the total provincial-level bombing amount used during the War to control for potential correlation between spraying intensity with the conflict and bombing intensity across areas.

2.2 Domestic migration, externalities and survival bias

The main threat to our identification strategy appears to come from possible migration of people within Vietnam during and after the spraying period. If the migration is large and correlates with health outcomes, then the estimated Agent Orange effects would be biased. For example, the migration of Agent Orange-exposed people from the South to the North would lead to under-estimating the effects of Agent Orange exposure. Below, we review the migration

process occurring in during and after the War and discuss its implications for our identification strategy.

Given that the Vietnam War was between the North and the South and that our identification strategy is based on South-North comparison, we distinguish two types of migration. The first type involves migration that occurred *within* the South and *within* the North. For example, those who were born in the poor central provinces of Vietnam that were sprayed by Agent Orange mainly migrate to the Southern cities in the South where the weather is more pleasant and jobs are more abundant. Or poor people in the Southern part of the North moved to Hanoi and Haiphong, two large provinces in the North, to settle down. In our South versus North DID comparison, this migration poses no threat as the comparison at North-South level enables us to internalize this migration. There were also scores of people in the Central area moving to the Highland area during 1990s in response to government policy to move people to new land areas. Consequently, in our sub-region analysis, where we break down the South into sub-regions (i.e. the Central, Highland, Mekong Delta, and Southeast) and compare them with the North, we will combine the Central and the Highland into one region.

The second pattern of migration that might threaten our identification is the migration *across* the North and the South. It turns out, however, that the number of migrants from the South (including the Central and the Central Highland) to the North is small and negligible. Total number of migrants migrating from the South (including the Central) to the North is only 5% of the number of the migrants from the North to the South in 1980s (Anh *et al.*, 1997). In 1990s, the same pattern prevails. A large number of people migrated from the unexposed North (including Red River delta, North East, and Northern Central Coast) to the Highland of the South following government policies that encouraged people to move to the underdeveloped

Central Highland (Loi, 2005).

For our analysis, the challenge that the Southbound migration presents is not serious. If the Agent Orange has effect on the health outcomes, then the inflow of unexposed people from the North to the South will dilute the effect of Agent Orange. In other words, the presence of more people from the unexposed North will make it less likely to find the effect of Agent Orange in the South. Thus, if we find any effect of Agent Orange on health outcomes, this would strengthen our results. We, however, also try to address this Southbound migration (and migration in general) by utilizing information on whether a person moved to her current residence area recently (within the past 3 years of the survey year).³ This information is quite useful for our purposes, because most of the migration from the Northern poor provinces to the South occurred after the economy and its transportation developed since the second half of 1990s.

The migration we have described, especially the migration from the North to the South, implies that analyses that exploit variation in Agent Orange exposure data at more disaggregated area levels (such as province, district or commune) to estimate the effect of Agent Orange are likely to generate biases. The reason is that such analyses cannot capture the large migration from the North to the South and also across provinces over a long time period.

Finally, we note that, in addition to internalizing the migration, conducting the comparisons between the North versus the South (and Southern regions) enables us to take into account the external effects of Agent Orange spraying. It is possible that dioxin was absorbed in the soil and found its way to the rivers that connect several geographical areas and contaminated

³ It seems reasonable to consider those who migrated for a long time as exposed people and include them in the treatment group because Agent Orange contamination is likely to persist over long periods.

areas that were not sprayed.⁴ As a result, spraying Agent Orange in one area could affect the health of people in the neighboring areas. Using data at a more disaggregated level (as in Stellman *et al.*, 2003) might underestimate the health impacts of Agent Orange.

Finally, we address a problem common to studies that investigate the long-term consequences of an event using survey data. The health survey used in this study is conducted in 2001, thirty years after the end of the Vietnam War, by interviewing respondents who have survived any damaging effects of Agent Orange to their health. While we do not have detailed data on mortality rates in the South and North of Vietnam over this period, numerous medical studies for American and Australian veterans who were deployed in Vietnam provide convincing evidence of higher overall mortality rates and cancer-related deaths than the rest of the male population (see, for example, Department of Veteran's Affairs, 2002). The possible increased mortality rates of the exposed population would give rise to a survivor treatment selection bias that acts in a direction of reducing the statistical significance of the results. Therefore, this should further strengthen the conclusions of this paper.

3 Data Description

3.1 Vietnam National Health Survey

The data used in the analysis come from the Vietnam National Health Survey (VNHS) conducted in 2001-2002. This is a multi-stage, complex survey that covers 158,019 individuals from 36,000 households. The survey has self-reported, individual-level information on various

⁴ Vietnam has an extensive network of rivers (2360 rivers with a length of more than 10 km). Eight of these rivers are large basins with a catchment area of 10,000 km² or more.

(<http://www.wepa-db.net/policies/state/vietnam/overview.htm>).

types of diseases related to Agent Orange exposure, including cancer and hypertension. More importantly, it contains valuable objective information on blood pressure of all respondents aged 16 and older.⁵

Moreover, the survey provides information on whether a person's family members fought in the War. The geographical residence of respondents is especially valuable since it enables us to assign a person to the control (North) or the treatment group (South).

Information on cancer is from respondents' answers. For hypertension, we follow the medical literature to classify a person as having hypertension if her systolic blood pressure readings consistently equal or exceed 140 mmHg in all three checks. There is some non-overlap between objectively measured hypertension and self-reported hypertension. This may be due to misreporting or unawareness of having hypertension. It is also possible that if those with hypertension were under medication at the time of blood pressure measuring, the three consecutive blood pressure tests administered on them may not detect hypertension. We therefore code someone as having hypertension if her blood pressure is 140 mmHg or above in all three tests, or self-reports having hypertension and scores 140 mmHg or above in one of the three blood tests.

Table 1 presents descriptive statistics of various health outcomes. Reporting both self-reported and objectively measured hypertension allow us to quantify the degree of underreporting in survey data which is expected to be even more severe for illnesses such as cancer. The cancer rate in the South is higher than that of the North although the average rate

⁵ During the survey, medical doctors interviewed each household and took blood pressure measurements three times according to the standard procedure which requires measuring blood pressure after the person had rested for at least 15 minutes.

for the whole country is very low. This suggests that the self-reported cancer rate is likely to be under-reported. There is a large difference between self-reported and measured hypertension rates: 13.8 per cent of the population aged 16 or older has hypertension, while only 7.6% of the population reported having hypertension. This points to the possibility of a potentially large bias in studies based on self-reported hypertension data. In what follows, we use only hypertension based on the objectively measured blood pressure.

Table 2 reports hypertension and cancer rates for different cohorts in the North and the South. For hypertension, we only consider people aged 70 or younger because this condition is likely to be affected by natural causes at older age. We observe higher hypertension rates for people in the South in cohorts born before 1961 and during the spraying period 1961-1971. In contrast, the cohort from the South born after the end of the spraying period in 1971 has lower hypertension rate than its Northern counterpart. Meanwhile, the cancer rate is much higher for the cohort born before 1961 than the other two younger cohorts. Furthermore, the cancer prevalence for cohort born before 1961 in the South is higher than that in the North but lower for cohorts born during 1961-1971 and after 1971.

3.2 Geographical and time distribution of herbicide sprays

The geographical distribution of Agent Orange and other herbicides is shown in Table 3. The Southeast (military region III) received the highest amount of spraying, with Agent Orange being the main chemical used. The next most heavily sprayed areas are the Highland (military region II) and the Central (military region I). The Mekong Delta area (military region IV) is the

least sprayed area.⁶

Table 4 shows the amount of herbicides sprayed in each year over the period. Agent Orange was mainly used between 1965 and 1970. Other herbicides including Agents Pink, Green and Purple, which are also contaminated with varying levels of dioxin (Stellman *et al.*, 2003), were sprayed throughout the 1961-1971 period. Note also that, as the quantity of herbicides used was revised up to almost twice the estimates in Table 4 (Stellman *et al.*, 2003), these geographical and time distributions of sprayed herbicide amounts are likely to be underestimated.

4. Estimation Results

4.1 Baseline regression results

4.1.1 South *versus* North comparison

As a baseline result, we first compare health outcomes across areas. We estimate a simple regression of the health outcome for cancer and hypertension on the South/North dummy and a vector of control variables described in Section 2. The results are reported in the first column for each health outcome in Table 5.

For cancer, the positive and statistically significant coefficient on the South dummy suggests that people in the South, holding all other factors fixed, have higher risk of cancer than the North. Majority of the other coefficients have the expected signs. It is interesting to note that the coefficient on the migration variable is insignificant lending support to our previous discussion that domestic migration is not expected to compromise the validity of our analysis.

⁶ It should be noted that our four geographical areas and four military regions do not coincide exactly but we use them interchangeably.

For hypertension, the South coefficient is positive and statistically significant at 1% significance level. Also, hypertension risks tend to be higher for single, female, unemployed, overweight, drinking, low income/education people from urban areas and with small families.

4.1.2 Southern areas *versus* North comparison

We next break down the South by smaller areas that vary in their herbicide exposure and compare them with the North. For now, we keep the Central and the Highland separate to give the broad sense of the differences across the Southern regions. The results are presented in the second column for each health outcome in Table 5. The Southeast and Mekong have higher rates of cancer than the North, with Southeast - the most heavily sprayed area - having the largest and highly significant coefficient. The results for hypertension indicate that all areas (except the Central) have higher risk of hypertension than the North, although only the Highland coefficient is statistically significant.

4.1.3 Cohort comparison

Further disaggregating the sample into three cohorts (before, during and after the spraying period) provides important information on Agent Orange impact on different groups of the population. The results from the cohort comparison are presented in Table 6. The first three columns in the table contain the South-North comparison by cohorts and the last three columns show the results from the comparison between the Southern areas and the North. The control variables are omitted from the table to preserve space.

The results for cancer suggest that direct exposure to Agent Orange and other herbicides poses the highest risk of cancer. More specifically, the cohort born before the spraying period in the South and Southeast has higher cancer prevalence than the corresponding cohort from the

North. The differences in cancer rates for this cohort, especially for the most sprayed Southeast region, are statistically significant.

The cohort 1961-71 (born during the spraying period) in the South has higher (and statistically significant) rate of hypertension than that in the North. This suggests that exposure to herbicides during “*in utero*” stage is harmful to blood pressure later in life. The cohort born before 1961 also has higher and statistically significant hypertension risk. Meanwhile, the cohort born after the spraying period 1961-1971 has a lower hypertension risk compared to the North.

Further comparisons between Southern areas versus the North suggest that for the cohort born before 1961, Highland’s population has the highest risk of hypertension, followed by Mekong’s and Southeast’s. For the cohort 1961-1971, the risks are highest for the Highland and the Southeast. These results are consistent with the fact that these two areas are the most heavily sprayed ones. In addition, people that belong to the cohort born after 1971 in all areas but the Highland do not exhibit higher risks of hypertension. This suggests that the hypertension burden of Agent Orange and other herbicides does not extend to the generations born after the spraying period.

4.2 Difference-in-difference analysis

So far we have studied effects of Agent Orange by comparing health outcomes between the South and the North, between the Southern areas and the North, and across age cohorts. We now combine the area and cohort comparisons in the DID analysis. As in Table 5, the first column (1) for each health outcome (cancer and hypertension) presents the results from the South-North comparison, and the second column (2) contains the results from the comparison between the Southern areas versus the North (We combine these two regions into one geographical area due

to migration from the Central to the Highland, as noted earlier). The parameters of interest are the coefficients on the interaction terms between the area and cohort dummies.

Consistent with Tables 5 and 6, the DID estimates indicate a higher risk of cancer for the cohort born before 1961 in the South and specifically, in the Southeast, Mekong and the combined 'Central and Highland' areas, than the same cohort in the North. In particular, the Southeast area - the most heavily exposed to Agent Orange – is characterized by the highest and statistically significant cancer risk. For hypertension, the coefficients on the interaction terms between the cohorts born during the 1961-1971 period and born before 1961 with the South are positive and statistically significant, suggesting that these cohorts in the South have higher hypertension risks than their counterparts in the North. In addition, all cohorts in all Southern areas (except the Central's cohort before 1960 and Mekong's cohort 1961-1971) have a higher rate of hypertension than the North. We also observe that the coefficients on the interactions terms for the Southeast and the combined 'Central-Highland' have the largest magnitudes, which is consistent with their status as the most heavily sprayed areas.

4.3 Robustness checks

4.3.1 Placebo tests

In our first robustness check, we conduct placebo tests to examine the robustness of the DID results. The idea is that if Agent Orange does not have effect on certain health outcomes, then we should not see the effects on these outcomes when applying our DID method. Following the IOM's list of diseases related to Agent Orange (IOM Updates, 2006) and other research on Agent Orange, our placebo tests look at three health outcomes: BMI, height and 'having difficulty to speak and/or being deaf.' The DID estimates from these placebo regressions are presented in Table 8. The estimates

indicate that the cohort born before 1961 has a lower BMI but this difference is not statistically significant. The cohort born during spraying period 1961-1971 even has a higher BMI than that of the North. Regarding height, the before 1961 cohort has higher height while the cohort born during spraying period has lower height, but this effect is not statically significant. Finally, the regression with difficult speech and/or deafness as an outcome indicates that both cohorts have lower likelihood of having these health conditions (statistically significant for the cohort born before 1961). Thus, all the estimates in the placebo regressions indicate that Agent Orange does not have any negative effect on these outcomes. These findings confirm the placebo tests of not detecting effects of Agent Orange where they are not expected to be found. If our previous estimates for cancer and hypertension were driven by some unobservables, we would have seen at least some negative effect of Agent Orange on these health outcomes.

4.3.2. War effects *versus* Agent Orange effects

Our next robustness check involves estimating the War effects and compares them with the Agent Orange effects we have estimated. To obtain the War effect, we still employ the same DID framework, but focus on the period 1972-1975 where the Agent Orange was no longer used but the War still continued. The control period for this exercise is 1976-1980 (after the War).

We focus on three health outcomes: cancer, hypertension and height. For cancer and hypertension, finding no significant effects would mean that our previously documented Agent Orange effect on these two health outcomes was not driven by the War intensity. For height, while there is no scientific evidence that links Agent Orange and height, there is evidence that the War affects adversely one's height due to lack of nutrition (see, for example, Bundervoet *et al.*, 2009, in this *Journal*). If our DID framework is credible, it should confirm this effect.

The results are shown in Table 9. The interaction term coefficients are not statistically significant in the cancer and hypertension regressions, indicating that those born after the spraying period (but still in the War period) have no elevated risk of having cancer and hypertension. Meanwhile, the interaction term is negative and statistically significant in the height regression, indicating that those born during 1972-1975 have lower height than the same cohort in the North. These results suggest that our earlier findings of Agent Orange effects on cancer and hypertension are not driven by war effects (such as war trauma and lack of nutrition).

4.3.3 Difference-in-difference analysis within the South

Our final robustness test is to conduct a DID analysis within the South only. This aims to further address the concern that the South and North differ in some unobservable ways which drive our results. In this within-South DID estimation, identification of the effect is based on the variation in the exposure to Agent Orange across sub-regions in the South. Because the Southeast is the highest receiving area, we focus on comparing the Southeast with the rest of the South. The results are expected to indicate that the disease burden in the Southeast is heavier than that for the rest of the South.

Table 10 displays the results which confirm this prediction. The estimates in the cancer regression suggest that the cohort born before 1961 in the Southeast has a higher likelihood of having cancer than the same cohort born in other regions of the South. For the hypertension regression, although the coefficients on the interactions terms are not statically significant (the cohort born before 1961 in only marginally insignificant at the 10% significance level), their positive signs indicate that both cohorts (born before 1961 and during 1961-1971) also have elevated risk of having hypertension. Together, these estimates point to the conclusion that the

burden of cancer and hypertension are higher for those in southeast than those in the rest of the South.

5 Concluding Remarks

In this paper, we studied the long-term health effects of Agent Orange and other herbicides on the Vietnamese population. Our main findings, based on a comprehensive self-reported and objectively measured data for nearly 160,000 individuals, can be summarized as follows. First, we find that people who are directly exposed to herbicide spraying have a higher risk of developing cancer. Second, the Agent Orange exposure appears to raise significantly the risk of hypertension for those who lived as well as those who were born during the spraying period. Finally, the results suggest that the most harmful effects of Agent Orange occur in areas that received the largest amounts of herbicide spray. We also argue that some potential limitations of our data set such as unaccounted migration and survivor treatment selection bias are expected to further strengthen our findings.

While the focus of this study is not only on the health consequences of direct exposure to Agent Orange but also “*in utero*” and post-spraying contamination effects on the Vietnamese population, some of our findings and conclusions can also be used to infer the harmful effects inflicted by herbicide exposure on the Vietnam War veterans. The group of Vietnam War veterans would roughly correspond to our cohort that was born before 1961 (with direct exposure to herbicides) and resided in the most exposed areas during the spraying period. Our findings of elevated risk of cancer and hypertension for this cohort can complement the small-sample studies conducted for the Vietnam War veterans.

Table 1. Summary Statistics for Health Outcomes

	Vietnam	South	North
Measured hypertension	0.138 (0.0011)	0.132 (0.0015)	0.146 (0.0018)
Self-reported hypertension	0.076 (0.0009)	0.084 (0.0013)	0.067 (0.0019)
Cancer	0.000965 (8.93e-05)	0.00104 (0.000127)	0.000885 (0.000126)
Observations	158,019	71,705	86,314
(Hypertension)	(100,442)	(45,833)	(54,609)

Notes: Weight-adjusted standard errors in parentheses.

Table 2. Hypertension Condition and Cancer by Cohorts

Cohort	Hypertension			Cancer		
	Vietnam	North	South	Vietnam	North	South
Before 1961 (and ≤70 years old)	0.236 (0.00248)	0.232 (0.0035)	0.240 (0.00349)	0.0028409 (0.0002952)	0.0022797 (0.0003695)	0.0034094 (0.0004615)
During 1961-1971	0.052 (0.0015)	0.0495 (0.0022)	0.0541 (0.00209)	0.0005926 (0.0001668)	0.0008284 (0.0003125)	0.0003882 (0.0001536)
After 1971 (and ≥16 years old)	0.016 (0.00073)	0.0187 (0.0011)	0.0138 (0.00088)	0.0001545 (0.0000446)	0.0001614 (.0000751)	0.0001485 (0.0000519)

Notes: Weight-adjusted standard errors in parentheses.

Table 3. Herbicide Use by Military Region, 1965-1971 (Million Gallons)

Military Region	Agent Orange	Agent White	Agent Blue	TOTAL (%)
I	2.25	0.36	0.30	2.91 (16.5)
II	2.52	0.73	0.47	3.72 (21.0)
III	5.31	3.72	0.29	9.32 (52.7)
IV	1.23	0.44	0.06	1.73 (9.8)
Total	11.31	5.25	1.13	17.68
(%)	63.9	29.7	6.4	100

Source: Tschirley (1992); Institute of Medicine (1994).

Table 4. Quantity of Herbicides Recorded on the Services HERBS Tape (Gallons)

Year	Agent Orange	Agent White	Agent Blue	Other	TOTAL
1962				3,700	3,700
1963				4,885	4,885
1964				14,560	14,560
1965	34,025			244,725	278,750
1966	242,800	45,900		182,161	470,861
1967	167,085	33,835	25,401	23,795	250,116
1968	77,259	80,245	36,846	72,977	267,327
1969	79,922	29,745	17,917	71,460	199,044
1970	27,805	10,655	26,623	28,495	93,578
1971			11,063	2,400	13,463
Total	628,896	200,380	117,850	649,158	1,596,284

Source: U.S. Army and Joint Services ESG (1986); Institute of Medicine (1994).

Table 5. Geographical Area Comparison

Variables	Cancer		Hypertension	
	(1)	(2)	(1)	(2)
South	0.0377* (0.0199)		0.629*** (0.240)	
Central		-0.0034 (0.0247)		-1.20*** (0.342)
Highland		-0.0056 (0.0423)		4.15*** (0.549)
Southeast		0.0881** (0.0364)		0.515 (0.354)
Mekong		0.0429* (0.0238)		0.369 (0.295)
Income	-0.0131 (0.0249)	-0.0221 (0.0253)	-2.08*** (0.257)	-2.09*** (0.263)
Male	-0.0056 (0.0196)	-0.006 (0.0196)	-1.85*** (0.299)	-1.83*** (0.298)
Hholdsize	0.0005 (0.0064)	-5.01e-04 (0.006)	-0.34*** (0.0571)	-0.34*** (0.0573)
Married	0.007 (0.0369)	0.007 (0.0370)	-2.11*** (0.309)	-2.18*** (0.309)
Elementary	0.0623* (0.0340)	0.0665* (0.0341)	-0.218 (0.317)	-0.200 (0.319)
Secondary	0.0616 (0.0399)	0.0693* (0.0406)	-0.426 (0.332)	-0.488 (0.338)
High School	0.0299 (0.0450)	0.0399 (0.0458)	-0.793** (0.400)	-0.819** (0.408)
Smoking	-0.12*** (0.0279)	-0.13*** (0.0279)	-0.225 (0.326)	-0.236 (0.326)
Urban	0.0670** (0.0277)	0.0685** (0.0270)	0.587** (0.260)	0.477* (0.260)
Migrated	-0.0003 (0.0371)	-0.0149 (0.0384)	0.780 (0.484)	0.747 (0.493)
Province bombing	-4.93e-8 (4.97e-8)	-3.88e-8 (5.34e-8)	-2.8e-06*** (5.60e-07)	-1.03e-06* (5.80e-07)
Employed			-3.19*** (0.395)	-3.19*** (0.395)
Body Weight			0.450*** (0.0172)	0.450*** (0.0173)
Drinking			2.56*** (0.324)	2.54*** (0.324)
Constant	0.0731 (0.226)	0.145 (0.230)	56.4*** (4.41)	57.1*** (4.43)
Observations	157459	157459	92062	92062
R-Squared	0.003	0.003	0.176	0.176

Notes: Linear probability models are estimated. All regressions include year-of-birth dummies, War veteran status, number of pharmacies in the residence area, and province's area and population in 1999. In addition, hypertension regressions control for timing of blood pressure checks. Robust standard errors in parentheses: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. All coefficients and standard errors are multiplied by 100.

Table 6. Cohort Regressions

Variables	Cohort	Cohort	Cohort	Cohort	Cohort	Cohort
	<1961	1961- 1971	>1971	<1961	1961- 1971	>1971
	(1)	(2)	(3)	(4)	(5)	(6)
CANCER						
South	0.177** (0.0746)	-0.0620 (0.0450)	0.006 (0.008)			
Central				0.0284 (0.0919)	-0.0424 (0.0815)	-0.006 (0.0126)
Highland				0.155 (0.149)	-0.0785 (0.0537)	-0.0262 (0.0365)
Southeast				0.323** (0.133)	-0.0938 (0.0624)	0.0240 (0.0150)
Mekong				0.160* (0.0855)	-0.0500 (0.0515)	0.0117 (0.009)
Observations	41264	25687	88889	41264	25687	88889
R-Squared	0.004	0.001	0.001	0.004	0.001	0.001
HYPERTENSION						
South	1.36** (0.597)	0.853** (0.378)	-0.44** (0.172)			
Central				-1.67* (0.884)	-0.952* (0.531)	-0.78*** (0.224)
Highland				6.55*** (1.32)	4.28*** (0.891)	1.67*** (0.505)
Southeast				0.837 (0.860)	1.22** (0.548)	-0.79*** (0.244)
Mekong				1.23* (0.727)	0.188 (0.464)	-0.71*** (0.205)
Observations	33832	24563	33667	33832	24563	33667
R-Squared	0.108	0.024	0.019	0.109	0.026	0.020

Notes: Linear probability models are estimated. All regressions control for the same covariates as in Table 5. Robust standard errors in parentheses: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. All coefficients and standard errors are multiplied by 100.

Table 7. Difference-in-Difference Regressions

Variables	Cancer		Hypertension	
	(1)	(2)	(1)	(2)
South_6171	-0.0311 (0.0392)		1.01*** (0.344)	
South_60	0.135** (0.0613)		1.48*** (0.512)	
South	0.008 (0.0128)		-0.16 (0.193)	
Central&Highland		5.76e-04 (0.0182)		-0.0513 (0.260)
Mekong		0.0231 (0.0152)		-0.106 (0.227)
Southeast		2.56e-03 (0.0235)		-0.270 (0.303)
Central&Highland_6171		-0.0269 (0.0506)		1.152*** (0.487)
Central&Highland_60		-0.0180 (0.0701)		0.184 (0.701)
Southeast_6171		-0.0606 (0.0419)		1.04** (0.509)
Southeast_60		0.353*** (0.125)		2.43*** (0.792)
Mekong_6171		-0.0160 (0.0455)		0.570 (0.435)
Mekong_60		0.0743 (0.0722)		1.65** (0.642)
Observations	157459	157459	92064	92064
R-squared	0.003	0.004	0.176	0.177

Notes: Linear probability models are estimated. All regressions control for the same covariates as in Table 5. South_60 denotes the interaction term between South and the cohort of people born before 1961; other interaction terms are defined similarly. Robust standard errors in parentheses: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. All coefficients and standard errors are multiplied by 100.

Table 8. Placebo Tests: DID Regressions for BMI , Height, and Difficult Speech or Deafness

Variables	(1)	Height	(2)
	BMI		Difficult speech or deafness
South_6171	0.145*** (0.0220)	-0.0688 (0.106)	-0.00106 (0.00127)
South_60	-0.00167 (0.0204)	1.077*** (0.0995)	-0.00345** (0.00172)
South	-0.0723*** (0.0144)	-0.107 (0.0765)	-0.00179** (0.000826)
Observations	101,023	85,252	121,008

Notes: Linear probability models are estimated. All regressions control for the same covariates as in Table 5. South_60 denotes the interaction term between South and the cohort of people born before 1961; other interaction terms are defined similarly. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1.

Table 9. War Effect *versus* Agent Orange Effect: DID Regression for 1972-1980

	(1)	(2)	(3)
Variables	Height	Cancer	Hypertension
South_7275	-0.357* (0.190)	-0.000341 (0.000349)	-0.000626 (0.00519)
South	-0.0497 (0.144)	0.000356 (0.000253)	-0.00645* (0.00351)
Observations	16,117	17,343	16,052
R-squared	0.492	0.001	0.022

Notes: Linear probability models are estimated. All regressions control for the same covariates as in Table 5. South_7275 denotes the interaction term between South and the cohort of people born during 1972-1975; Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1.

Table 10. Within-South Difference-in-Difference Analysis

	(1)	(2)
Variables	Cancer	Hypertension
Southeast_6171	-0.000321 (0.000309)	0.000498 (0.00515)
Southeast_60	0.00332*** (0.00124)	0.0123 (0.00792)
Southeast	1.64e-05 (0.000277)	-0.00963*** (0.00310)
Observations	86,095	50,877
R-squared	0.007	0.183

Notes: Linear probability models are estimated. All regressions control for the same covariates as in Table 5. Southeast_60 denotes the interaction term between Southeast and the cohort of people born before 1961; other interaction terms are defined similarly. Robust standard errors in parentheses: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

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