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

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Abstract

Background: Long-term health effects of exposure to Agent Orange have been a subject of debate and controversy. Most studies on Agent Orange health effects were based on small samples. The objective of this population-based study is to determine whether Agent Orange exposure increases the risks of cancer and hypertension for the Vietnamese population. Methods: This study employs a quasi-experiment research design to estimate the causal long term effect of Agent Orange on incidences of cancer and hypertension for Vietnamese population. Specifically, difference-in-differences regressions are estimated which compute the difference between the Agent Orange-affected cohort versus the unaffected cohort in a treated area (where the Agent Orange was sprayed) and compare that difference with the similar difference computed for the control area (where the Agent Orange was not used). Results: People who were directly exposed to Agent Orange spraying have a higher risk of developing cancer. 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. The most harmful effects of Agent Orange occur in areas that received the largest amounts of herbicide spray. Interpretation: The results provide statistical evidence for the harmful effects inflicted by herbicide exposure on the Vietnamese population. Our findings of elevated risk of cancer and hypertension complement the small-sample studies conducted for the Vietnam War veterans and raise warnings for the use of Agent Orange and other herbicides in populated areas.

Keywords: agent orange; difference-in-differences; herbicide exposure; Vietnam war

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Introduction

During the period 1961-1971 in the Vietnam War, 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.

The long-term health effects of the exposure to Agent Orange and other herbicides (Agent Orange (AO), for short) 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 AO every two years. Recently, a renewed interest in the health effects of AO has been prompted by a U.S. Department of Agriculture's approval in September 2014 of two new genetically modified crops that are immune to ingredients of AO. These crops, which allow farmers to spray fields with herbicide containing 2,4-D (a component of AO) to kill pests and weeds, and its approval have been met with strong resistance from numerous advocacy groups.¹⁹⁻²¹ In the latest development, the International Agency for Research on Cancer (IARC), the specialized cancer agency of the World Health Organization, have just classified the 2,4-D as a "possible carcinogen to human" while noting that this decision was based on "inadequate evidence in humans and limited evidence in experimental animals" and that "epidemiological studies did not find strong or consistent increases in risk of non-Hodgkin lymphoma or other cancers in relation to 2,4-D exposure".²² This makes research on the long-term health effects of AO (especially on cancer at the population level) timely.

Numerous medical and epidemiological studies established a link between the AO exposure and various types of cancer¹, type 2 diabetes², skin diseases³, cardiovascular disease mortality⁴ and hypertension^{1,2,5-7}. The disease burden falls not only on the War veterans and civilians who were directly exposed but also on their children who may have a higher risk of certain types of leukemia²³ and birth defects²⁴.

Most of the existing studies on AO's health effects suffer from some common limitations. First, many medical studies utilized small samples of Vietnam War veterans^{7,8}, chemical workers⁹⁻¹¹, pesticide manufacturers and applicators^{12,13} and were often based on self-reported data. The small sample size and cross-sectional nature of these studies might lead to low participation rates and thus, sample selection bias. Second, many studies were confined to detecting traces of dioxin in blood of affected people in Vietnam¹⁴, rather than showing a causal link between AO exposure and the incidence of diseases. In addition, potential confounders such as bombing intensity that was correlated with people's health anomalies were typically not accounted for. Finally, while the treatment group was well defined in all medical studies, it is not clear who constituted the appropriate control group, especially when this control group was selected from exposed areas.

In this paper, we exploit a unique, nationally representative health survey to provide population-based evidence on the effects of herbicide exposure on cancer and hypertension incidence of the Vietnamese population. In addition to circumventing the sample selection problem and ensuring a valid comparison between the treatment group (those who resided in the South regions and were exposed to AO) and the control group (those who resided in the North and were not exposed to AO), the coverage of the whole Vietnamese population enables us to study the AO effects on people who were directly sprayed upon during the Vietnam War as well as on children born during the spraying period.

Methods

Differences-In-Differences strategy

To estimate the causal effect of AO exposure, the difference-in-differences (DID) methodology^{15–17} (also known as “before-after analysis with control group”¹⁸) is employed. The DID exploits the variation in the timing of AO spraying and whether (and by how much) geographical areas were sprayed. Specifically, as the South was sprayed AO while the North was not, people residing in the North are assigned to the control area and those residing in the South form the treatment area. Further, because AO was only sprayed for a specific period in the War, i.e. 1961-1971, the population was divided into three cohorts: i) exposed cohort of people who were born before AO was sprayed (before 1961); ii) exposed cohort of people who were born during the spraying period (1961-1971) (to study effects on later health outcomes of “*in utero*” and childhood AO exposure²⁵), and iii) unexposed cohort of people who were born after the spraying period. The DID method computes the difference between the AO-affected cohorts versus the unaffected cohort in a treated area where AO was sprayed (first difference) and compares that difference with the similar difference computed for the control area where AO was not used (second difference).

Formally, we estimate a DID regression of the following form, using linear probability model:

$$Y_i = a + b * AREA_i + (AREA_i * COHORT_i)'\delta + YOB_i'c + x_i'\beta + e_i$$

where Y_i is an indicator variable equal to 1 if person x_i has specific disease linked to AO and 0 otherwise; $AREA_i$ takes a value of 1 if the person resides in a sprayed area and 0 otherwise; $COHORT_i$ are indicators for cohorts born before 1961 and during 1961-1971 (thus, the unexposed

cohort born after 1971 is omitted category); the covariates of interest are $AREA_i * COHORT_i$, the interaction terms between area dummies and cohort dummies; YOB are year-of-birth fixed effects that control for cohort-specific factors affecting both the North and the South; and x_i is a vector of control variables that include individual characteristics (income, education, gender, marital status, being employed or not, past 3 year migration) and risk factors for blood pressure (smoking, drinking, and weight). To control for economic conditions which might drive migration into and out of the area in which the person lives, urban status, number of pharmacies available in the residence area, and provincial size and population are included. Provincial-level bombing amount used during the War is also included to control for potential correlation between AO spraying intensity and bombing intensity across areas.

Two types of regional comparisons are considered in the DID analysis: the South versus the North and Southern sub-regions versus the North. The latter comparison exploits the variation in herbicide spraying level across Southern sub-regions. As shown in Table 1, among the Southern sub-regions, the Southeast area (military region III) received the highest herbicide amount, with Agent Orange being the main chemical used, followed by the Highland (military region II) and the Central (military region I) and lastly, the Mekong Delta area (military region IV). We note that although the amount of each herbicide in Table 1 is slightly lower than the recent data²⁶, the ranking of the Southern areas by amount of herbicides received remains valid.

Addressing domestic migration and externalities

As the DID strategy assumes that the composition of people in each area remains unchanged before and after the AO spraying, migration of people can bias the DID estimates. The first type of migration is the migration *across* the North and the South. While the number of migrants

from the South to the North is very small²⁴, there were large numbers of migrants from the North and the Central to the underdeveloped Highland in the South following government policies in 1990s²⁷. Although this Southbound migration should dilute AO effects in the South and strengthen any finding of AO's health impacts on the Vietnamese population, we address this by utilizing information in the survey on whether a person moved to his current residence within the past 3 years (of the survey year). We also combine the Central and the Highland into one area to internalize the migration from the Central to the Highland.

The second type of migration is the migration *within* the South and *within* the North. However, our North South comparison can internalize this migration. It can also accounts for external effects of AO spraying. (Agent Orange could be absorbed in the soil and found its way to the rivers that connect several geographical areas and contaminated areas that were not sprayed). The implication is that analyses that use AO exposure data at a disaggregated level might under- estimate AO's health impacts.

There was also migration out of Vietnam. In fact, large numbers of people fled South Vietnam to other countries after the War. The migration of these people (known as "boat" people) would bias upwards the estimates if they were among the healthiest in the society. However, cancer rates were found to be higher for U.S. Vietnamese who left the South after the Vietnam War compared to residents in the North²⁸. This would introduce downward biases and strengthen any findings of AO effects.

Data and study outcomes

The data are 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 cancer as well as both self-reported hypertension and objectively measured blood pressure. The geographical residence of respondents is especially valuable since it enables us to assign a person to the control (the North) or the treatment group (the South and its sub-regions).

The study focuses on two health outcomes, i.e., cancer (any type) and hypertension. Cancer is constructed as an indicator variable based on respondents' self-reports. The objectively measured blood pressure was used to classify a respondent's hypertension status. We follow the medical literature to classify a person (aged 16 and older) as having hypertension if her systolic blood pressure readings consistently equal or exceed 140 mmHg in all three checks. Only people aged 70 or younger are included in the analysis on hypertension because this condition is likely to be affected by natural causes at older age.

Results

Descriptive analysis

Table 2 presents unconditional descriptive statistics of the two health outcomes (hypertension and cancer) for different cohorts in the North and the South. Cancer rate is higher for the cohort born before 1961 than the other two younger cohorts. Furthermore, it is significantly higher in the South than in the North for the cohort born before 1961 but not for the cohorts born during 1961-1971

or after 1971. Overall, the cancer rate for the whole country is low which suggests that it is likely to be under-reported.

The overall hypertension rate is lower in the South than in the North. By cohort, hypertension rates in the South are higher for the 1961-1971 cohort but lower for the cohorts born before 1961 and after 1971. Figure 1 plots cancer and hypertension rates by one-year cohort defined by individual birth year for the North and the South. These trends follow each other closely, suggesting that the North and the South do not have differential time trends in the rates of these two diseases.

Differences-in-differences regression results

The estimates from our DID regressions are presented in Table 3. In column 1, the coefficient estimate for the interaction term involving the cohort born before 1961 is positive and statistically significant. This indicates that the pre-1961 cohort in the South has a higher risk of cancer relative to the reference cohort (born after 1971) and relative to the North. Meanwhile, the coefficient on the 1961-1971 cohort is not statistically significant. Given this cohort's relatively young age (40 or less in the survey year), it may be hard to detect any adverse impact of AO on cancer. Column 2 presents estimates from breaking down the South by its region. The coefficient estimates are positive for the interactions involving the Southeast and Mekong areas. In particular, the Southeast area - the most heavily exposed to AO - is characterized by the highest and statistically significant cancer risk.

For the hypertension regressions, the coefficients on the interaction terms involving the cohorts born before 1961 and during 1961-1971 in column 3 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 for the pre-1961 cohort in the Central-Highland) have a higher rate of hypertension than the North. Also, the coefficients on the interactions terms for the Southeast and the Central-Highland have the largest magnitudes, which is consistent with their status as the most heavily sprayed areas (column 4).

Sensitivity analyses

War effects *versus* Agent Orange effects

Our first sensitivity check involves testing if the AO effects we have estimated were confounded by the War effects (such as war trauma and lack of nutrition). We employ the same DID framework, but focus on the period 1972-1975 where AO was no longer used but the War still continued. The control period for this exercise is 1976-1980 (after the War). Finding no significant effects on cancer and hypertension would mean that our previous AO effects on these two health outcomes were not driven by the War effects. The results are shown in Panel 1 of Table 4. 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 (i.e. during 1972-1975) have no elevated risk of having cancer and hypertension.

Difference-in-differences analysis within the South

In another robustness check, we address the concern that the South and the North might differ in some unobservable ways which drive our results. We exclude the North and run the DID regressions using the South only. Identification of the effect is now based on the variation in the AO spraying level across sub-regions in the South. We focus on comparing the Southeast with the rest of the South because the Southeast is the highest AO receiving area. The disease burden was expected to

be heavier in the Southeast than in other Southern areas.

The results in Panel 2 of Table 4 confirm this prediction. The estimates from the cancer regression suggest that the pre-1961 cohort in the Southeast has a higher likelihood of having cancer than its counterpart in other Southern regions. For the hypertension regression, the pre-1961 cohort has a significantly higher risk of having hypertension while the coefficient on the 1961-1971 cohort is positive but not statistically significant. Together, these estimates indicate that the burden of cancer and hypertension are higher in the Southeast than in the rest of the South.

Discussion

In this paper, we studied the long-term health effects of AO and other herbicides on the Vietnamese population. Our main findings can be summarized as follows. First, people who were born before the spraying period 1961-1971 have a higher risk of developing cancer. Second, the AO 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 AO occur in areas that received the largest amounts of herbicide spray. We have shown that our estimated AO effects on cancer and hypertension were not driven by war effects or by some unobservable differences between the South and the North.

While the estimated coefficients of the AO effect on cancer in Table 3 seem small, they actually represent a large effect. Compared with the cancer rate of 0.28% for the unexposed North, the estimated increase of 0.135 percentage points represents a 48% increase in the cancer risk for the South. Meanwhile, the magnitudes of the increases for hypertension risk in the South are estimated to be 3.4% and 22% for the cohort born before 1961 and during 1961-1971, respectively (i.e. increases

of 0.9 and 1 percentage points divided by the North's hypertension rates of 27.8% and 4.6% for the pre-1961 and 1961-1971 cohorts, respectively).

We are aware of only one population-based study by Do (2009)²⁹ who independently studied the effects of AO on the prevalence of cancer in Vietnam. The analyses in our paper and his paper, however, differ along several important dimensions. First, we studied not only effects of herbicide spraying on self-reported cancer rates but also on objectively measured hypertension. Second, we capitalized on the fact that the North has not been exposed to herbicide spraying which allowed us to use it as a valid treatment group in our DID analysis. Third, our analysis at regional levels controlled for possible population migration over the thirty years between the end of the War and the time of survey while his analysis, whose geographical unit of analysis is very small (i.e. at commune level), does not. Finally, the causal interpretation of his results is not entirely clear.

Our study has a number of limitations. First, while blood pressure was objectively measured, cancer was self-reported, and thus subject to reporting biases. However, if reporting biases were similar between the North and the South, they would be cancelled out in the DID strategy and thus, would not bias our AO estimates. Second, the use of younger cohort born after 1971 as the comparison group may be limited in that their health outcomes might also be affected by AO if the chemicals remain in the environment for decades or there is intergenerational transmission. However, this would lead the AO effects to be underestimated and thus strengthen our findings of AO effects. Another limitation is the survivor selection bias. That is, the study population includes respondents who have survived any damaging effects of AO. However, 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 their higher overall mortality rates and cancer-related deaths than the rest of the male

population³⁰. The possible increased mortality rates of the exposed population would act in a direction of reducing the statistical significance of the results and thus, should further strengthen the conclusions of this paper.

To conclude, our study showed that AO significantly increase the risks of cancer and hypertension for AO-exposed Vietnamese population. Our findings can 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 can complement the small-sample studies and raise warnings on the spraying of AO in the environment. In particular, the finding of elevated risk of cancer found in Vietnamese subpopulation who were exposed to AO in the Vietnam War provides direct evidence that supports the WHO's recent classification of 2,4-D as probably carcinogenic to humans.

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Appendix: Tables and Figures

Table 1: 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

Note: Data are from Tschirley (1992)³¹; Institute of Medicine (1994)³². Military regions I, II, III, and IV correspond to the Central, Highland, Southeast, and Mekong Delta described in the text.

Table 2: Summary statistics for hypertension and cancer

Cohort	Cancer				Hypertension			
	Vietnam	North	South	p-value	Vietnam	North	South	p-value
Born before 1961	0.00284 (0.00029)	0.00228 (0.00037)	0.00341 (0.00046)	0.056	0.274 (0.002)	0.278 (0.0034)	0.270 (0.0033)	0.104
Born during 1961-1971	0.00059 (0.00017)	0.00083 (0.00031)	0.00039 (0.00015)	0.21	0.048 (0.001)	0.0462 (0.0021)	0.0497 (0.002)	0.22
Born after 1971	0.00015 (0.00004)	0.00016 (0.00007)	0.000075 (0.00005)	0.88	0.015 (0.001)	0.0184 (0.0012)	0.0133	0.000 (0.0008)
All cohorts	0.000965 (0.00009)	0.00088 (0.00013)	0.00104 (0.00013)	0.39	0.132 (0.001)	0.141 (0.0018)	0.124 (0.0015)	0.000
N	158,019	71,705	86,314		100,442	45,833	54,609	

Notes: Weight-adjusted means and standard errors are reported. For hypertension, subsample of individuals aged between 16 and 70 is used.

Table 3: Difference-in-Differences Regression Results

Variables	Cancer		Hypertension	
	(1)	(2)	(3)	(4)
South * Cohort 1961-1971	-0.0311 (0.0392)		0.936*** (0.333)	
South * Cohort before 1961	0.135*** (0.0613)		1.02** (0.00497)	
South	0.008 (0.0128)		-0.223 (0.190)	
Central-Highland * Cohort 1961-1971		-0.0269 (0.0506)		1.47*** (0.475)
Central-Highland * Cohort before 1961		-0.0180 (0.0701)		-0.0237 (0.685)
Southeast * Cohort 1961-1971		-0.0606 (0.0419)		1.17** (0.495)
Southeast Cohort before 1961		0.353*** (0.125)		2.10*** (0.776)
Mekong Cohort 1961-1971		-0.0160 (0.0455)		0.356 (0.418)
Mekong Cohort before 1961		0.0743 (0.0722)		0.927 (0.624)
Central Highland		5.76e-04 (0.0182)		-0.0617 (0.257)
Mekong		0.0231 (0.0152)		-0.163 (0.224)
Southeast		2.56e-03 (0.0235)		-0.382 (0.296)
N	157459	157459	93129	93129
R-squared	0.003	0.004	0.176	0.177

Notes: Linear probability models are estimated. Robust standard errors clustered at province level in parentheses. All coefficients and standard errors are multiplied by 100. All regressions include individual-level control variables (income, gender, household size, marital status, education, smoking status, urban status, employment, body weight, drinking status, migration, and year-of-birth dummies), and area-level variables (number of pharmacies in the residence area, and province's size and population in 1999). In addition, hypertension regressions control for timing of blood pressure checks. South*Cohort 1961-1971 denotes the interaction term between South and the cohort of people born during 1961-1971; other interaction terms are defined similarly. Significance level: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 4: Sensitivity analysis

I. War Effect versus Agent Orange Effect: Difference-in-Differences Regression for 1972-1980		
	(1)	(2)
	Cancer	Hypertension
South * Cohort 1972-1975	-0.0341 (0.0349)	-0.0810 (0.510)
South	0.0356 (0.0253)	-0.707** (0.348)
N	17,343	16,055
R-squared	0.001	0.021
II. Difference-in-Differences Regression, South only		
	Cancer	Hypertension
Southeast * Cohort 1961-1971	-0.0321 (0.0309)	0.555 (0.496)
Southeast Cohort before 1961	0.332*** (0.124)	2.11*** (0.785)
Southeast	1.64e-03 (0.0277)	-1.39*** (0.298)
N	86,095	51,200
R-squared	0.007	0.161

Notes: Linear probability models are estimated. All regressions control for the same covariates as in Table 3. South*Cohort 1972-1975 denotes the interaction term between South and the cohort of people born during 1972-1975; other interaction terms are defined similarly. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1.

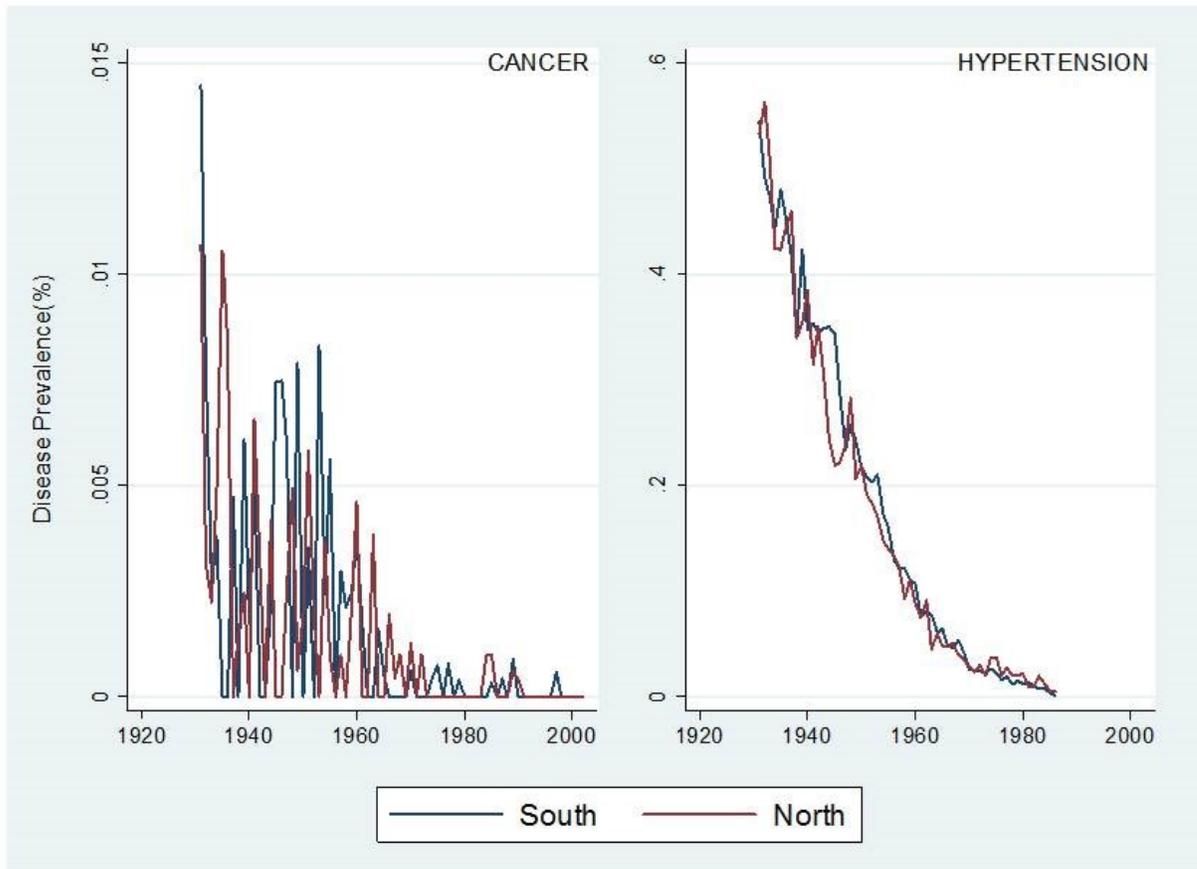


Figure 1: Trend in disease rates of birth year cohorts