The Persistent Health Effects of Defoliating Vietnam

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Abstract

This study investigates the long-run unintended environmental effects of conflicts on health and socioeconomic outcomes by focusing on dioxin exposure from herbicidal warfare deployed in the Second Indochina War. To address the omitted variable bias caused by the non-randomness of dioxin exposure, I leverage the difference in dioxin contamination, unknown during the war, between different herbicides. The estimation results show a higher prevalence of disability in more exposed areas, with one standard deviation increase in the exposure index associated with about 5%-20% of the disability prevalence in the unexposed communes. My methods allow me to uncover two main channels contributing to impaired health: direct dioxin exposure during wartime and cascading intergenerational effects from the initial exposure. I also find that dioxin exposure affects educational, labor, and economic outcomes.

Keywords: conflict, chemical warfare, health, Vietnam
JEL codes: I18 013 Q53

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1 Introduction

Even though wars and conflicts leave behind decimated populations and annihilated economies, the existing economics literature points out that economic activities converge to the pre-war levels, even with big shocks like World War II (Davis and Weinstein, 2002; Brakman, Garretsen and Schramm, 2004).[1] The recovery of economic forces, in the long run, could be explained by the neoclassical thoughts on convergence. However, war and conflicts may have unintended consequences on human capital accumulation through environmental factors, which might not recover within a couple of generations. In this paper, I study the long-run environmental impact of conflicts on health and socioeconomic outcomes by focusing on the Second Indochina War.

The Second Indochina War[2] was one of the most devastating conflicts of the 20th century. Not only did the US forces and allies drop about 7.6 million tons of bombs, three times the amount of ordnance used in World War II and the Korean War combined (Clodfelter, 1995), they also deployed herbicidal warfare, which sprayed twenty million gallons of herbicides (Stellman and Stellman, 2018). In 1961-1971, Operation Ranch Hand used defoliants, including Agent Orange and Agent White, to destroy the foliage cover of insurgent forces, as well as crops potentially supplying these forces. An unintended consequence of the operation was contamination by 2,3,7,8-Tetrachlorodibenzo-P-dioxin (TCDD, hereafter "dioxin") in Agent Orange, which affected 2.1-4.8 million Vietnamese civilians (Stellman et al., 2003). Because dioxin is a toxic and persistent organic pollutant White and Birnbaum (2009), the effect of Agent Orange is one of the long-lasting legacies of the Second Indochina Wars.

In this project, I study the long-run impact of dioxin exposure on health and socioeconomic outcomes. The econometrics exercise compares the outcome variables between areas with different levels of dioxin exposure. However, the endogeneity of exposure to dioxin-contaminated herbicides arises because of the non-randomness of spray paths, which causes an omitted variable bias. For instance, herbicide was sprayed where the guerilla and North Vietnamese forces were based Le, Pham and Polachek (2022). Because these areas were more likely to be remote and mountainous, the sprayed areas would have had worse socioeconomic outcomes than

[1] In the context of this paper, Miguel and Roland (2011) does not find any evidence for the long-run impact of US bombing on poverty or population in Vietnam. However, Yamada and Yamada (2021) and Riaño and Caicedo (2021) found opposite results in Laos.

[2] It is commonly known as the 'Resistance War against America' in Vietnam or the 'Vietnam War' in the US

non-sprayed ones, even in the absence of herbicide sorties. In addition, herbicides were applied around military bases to avoid surprise attacks. Given that the estimated parameter consists of the effects of i) dioxin exposure, ii) herbicides, and iii) confounding factors, the bias from the confounding factors could underestimate or overestimate the impact of Agent Orange.

To solve the endogeneity issue, I use the total exposure to Agent Orange and Agent White as a control variable. This method leverages the difference in dioxin contamination between Agent Orange and White to estimate the effect of dioxin exposure. Both were herbicides used against broadleaf plants and trees (Institute of Medicine, 2011). However, Agent Orange was dioxin-contaminated, while Agent White was not. Even though Dow Chemical and other herbicide producers knew about the dioxin contamination in Agent Orange and its biological impact, high-ranking government officials were unaware of the issue (Burnham, 1983). Therefore, exposure to dioxin could be considered a random shock after accounting for the non-randomness of herbicide sorties. The total exposure to Agent Orange and Agent White in the regression model is a continuous matching variable to match communes with similar herbicide exposure, implying similar characteristics. Therefore, this method purges the omitted variable bias.

This project uses two datasets. One is information on exposure, which I measure using data from Stellman and Stellman (2011). Using the records on spray paths, herbicide type, and amount in each sortie in Stellman and Stellman (2011), I calculate the amount of dioxin-containing herbicides dropped within a five-kilometer (km) radius of commune centroids and discount it by the distance to the centroids. This measurement is the Stellman Exposure Opportunity Index proposed by Stellman et al. (2003). The second dataset is the 2009 Census of Population and Housing that the General Statistics Office of Vietnam conducted on April 1, 2009. The dataset includes demographic, educational, labor, migration, and health information for about 15% of the population.

The baseline results show that individuals born by 1975 living in areas with higher exposure to dioxin-containing herbicides were more likely to report visual, auditory, mobility, and cognitive difficulties in 2009. I find that the magnitude of the impact is non-trivial. For instance, for the birth cohorts of 1955-1964, a unit increase in dioxin exposure level raises the chance of reporting difficulties in seeing, hearing, walking, and memory by 0.247, 0.052, 0.145, and 0.091 percentage points, respectively. One standard deviation increase in exposure level is associated with 5-20 percent of the disability prevalence in areas with zero exposure to dioxin. Using the regional difference in population composition, I find that the wartime exposure to dioxin and the intergenerational effects of that exposure are the two channels for the persistent effects of dioxin.

This paper contributes to a growing literature evaluating the impact of pollution on human capital accumulation. Early-life exposure to pollution has a persistent effect on health outcomes. For instance, Rosales-Rueda and Triyana (2019) found the impact of forest fires on children's stature and lung capacity, which could last 17 years after exposure. Children living near the heavy metal mines are at a higher risk of stunted growth by five percentage points (von der Goltz and Barnwal, 2019). Even among infants, the effect is also observable (Currie and Neidell, 2005; Currie, Neidell and Schmieder, 2009). In addition to health, early-life exposure to pollution affects cognitive function and educational outcomes (Aizer et al., 2018; Persico, Figlio and Roth, 2020; Persico and Venator, 2021; Rau, Urzúa and Reyes, 2015; Currie et al., 2009). Continuing in this line of research, I find the long-lasting adverse effects on health, educational, and socioeconomic outcomes, even in the generations born after the war.

The insights gained from this project remain relevant outside the context of the Second Indochina War. For instance, dioxin and similar chemical substances are byproducts of industrial processes. As thermal industrial activities, including combustion engines, emit these substances (Dopico and Gómez, 2015), dioxin exposure is a problem in both developed and developing countries (Schecter et al., 1991). The results in this paper are also relevant to other persistent organic pollutants. A closely related case is Dichlorodiphenyltrichloroethane (DDT) and other organochlorine pesticides. Studies have found that DDT, a once-popular insecticide, increases the risk of cancer and disorders of the reproductive, nervous, and immune systems (Beard, 2006; Eskenazi et al., 2009). Another case is the "forever chemicals" Perfluoroalkyl and polyfluoroalkyl (PFAS), which had numerous applications, including household products. Evidence shows the potential health issues caused by PFAS contamination (Beans, 2021), which implies its effect on human capital accumulation.

This study also contributes to the literature on the socioeconomic impact of conflicts. I find the Second Indochina War, through dioxin exposure, reduced human capital stocks, and the effects are still observable decades after. This result aligns with the existing literature (Chamarbagwala and Morán, 2011; León, 2012; Akbulut-Yuksel, 2014; Grimard and Laszlo, 2014; Islam et al., 2016; Akbulut-Yuksel, 2017). By crippling human capital, warfare distorts local economies in the long run, as in Feigenbaum, Lee and Mezzanotti (2022), Yamada and Yamada (2021), and Riaño and Caicedo (2021). However, the long-term effects of wars and conflicts on affected populations are not unforeseeable. On the other hand, the impact of dioxin in the Second Indochina War was unintended, as dioxin affected both sides. Therefore, this project is distinct from the existing literature.

In addition, the results provide evidence for the long-lasting effect of conflicts that persist through the environmental channels. The economics literature on wars and conflicts seems to overlook the chemical side of these shocks, as many similar contexts have not been studied. For instance, the British used rainbow herbicides, including Agent Orange, during the Malaya Emergency (1947-1960). Another related context is the efforts against drug production. In Colombia, glyphosate, which may cause cancer, has been used to destroy coca crops (Massey, 2001). The US government made similar efforts to erase poppy fields in Afghanistan (Whitlock, 2019) that funded insurgency.

This is not the first project working on the long-run impact of the Second Indochina War. Do (2009), Appau et al. (2021), Yamashita and Trinh (2022), Le, Pham and Polachek (2022), Bui (2023) and Ito, Tran and Yoshida (2023) find the adverse impact of dioxin exposure on health, agricultural, educational outcomes and population size. On the effect of US bombing, interestingly, Miguel and Roland (2011) find that bombing intensity does not affect the local economic development in Vietnam. However, Palmer et al. (2019), Singhal (2019), Appau et al. (2021) and Vuong, Chang and Palmer (2021) provide evidence that US bombing lowers agricultural productivity and health outcomes in Vietnam, which could imply a contradiction to Miguel and Roland (2011). In the context of Laos, Yamada and Yamada (2021) and Riaño and Caicedo (2021) also find that heavily bombed areas have worsened economic outcomes.

The literature on the impacts of the Second Indochina War is fascinating. However, my criticism is that the literature relies excessively on the geography instance instrumental variables for causal inference. An issue with the instrumental variable approach is the non-transparency in its estimation. For example, Miguel and Roland (2011), Singhal (2019), Palmer et al. (2019) and Appau et al. (2021) use the distance to the 17th parallel as an IV for bombing intensity. Without considering any control variables, the instrumental variable estimation would put more positive weight on the southmost and northmost areas and put more negative ones on the areas around the 17th parallel. As a result, the method compares outcomes between communes around the 17th parallel and the southmost and northmost ones and ignores the economic hub surrounding Ha Noi and Ho Chi Minh City. Since this comparison is arbitrary, whether the geographic distance IV could solve the endogeneity of the war is still unambiguous.

The rest of the paper is organized as follows. Section 2 describes the data and Section 3 explains the econometric model. Section 4 presents the health impact of dioxin exposure. Section 5 estimates the economic impact. Section 6 concludes.

2 Data

2.1 Spray missions

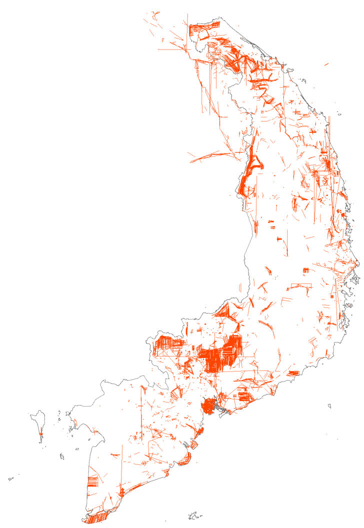
I retrieve records of spray missions in Operation Ranch Hand (ORH) from Stellman and Stellman (2011). For each mission, Stellman and Stellman (2011) includes the date, flight path, herbicide type, volume, and delivery vehicles. The dataset includes 8,959 missions from 1961 to 1971, which, in total, sprayed about 19 million gallons of defoliants. Figure 1a maps spray missions that sprayed dioxin-contaminated herbicides, of which 96% were Agent Orange. Figure 1b maps missions that used the dioxin-free Agent White. Figure 1c plots the monthly herbicide use, showing that the deployment of defoliants rose significantly after the mass arrival of US and allied forces in 1965. The number of spray missions by month also shows a similar trend as in Figure 1d. To the best of my knowledge, this is the best source of information on ORH.

I use the Stellman Exposure Opportunity Index (EOI) as the explanatory variable because it accounts for the amount of dioxin-contaminated herbicides and the locations of the spray paths relative to the centroid. Appendix A describes how to calculate the Stellman EOI, and Figure 2a maps the calculated EOI at the commune level. To check whether the calculated EOI is reliable, I construct two other measurements of the exposure to Operation Ranch Hand. The first measurement is the number of missions that sprayed herbicide on any location within a five-km radius of commune centroids. Since each would consist of different paths, which could be either a point or a straight line, I break them down into spray paths and calculate the second measurement by counting the number of spray paths that sprayed defoliant on any location within a five-km radius of commune centroids.

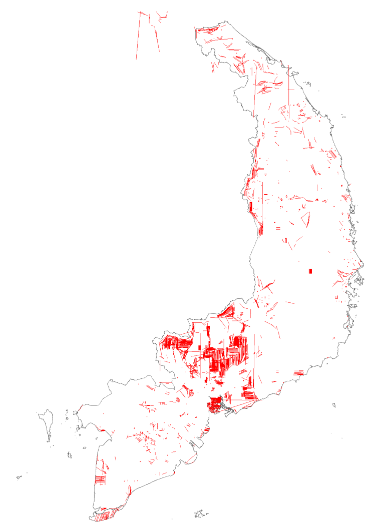
Figures 2b and 2c map the numbers of missions and spray paths within a five km radius of commune centroids. These maps show a similar feature to the map of calculated EOI in Figure 2a. Furthermore, these two measures and EOI show a strong correlation. The correlation coefficient between EOI and the number of missions within five km is 0.779. The correlation coefficient between EOI and the number of spray paths is 0.701. Interestingly, these figures show a consistent pattern that the US military sprayed heavily on the outskirts of Saigon and the mangrove

Figure 1: Data on spray missions, 1961-1971

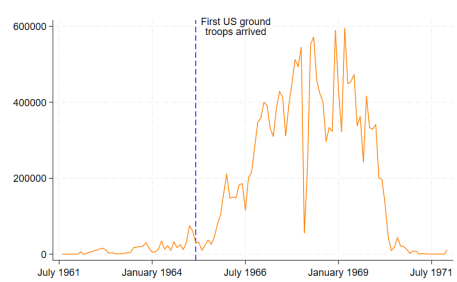
(a) Dioxin-contaminated spray missions



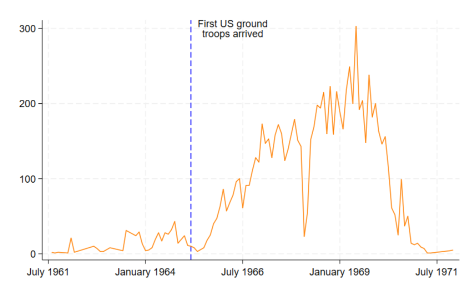
(b) Missions sprayed Agent White



(c) Monthly herbicide use (gallons)



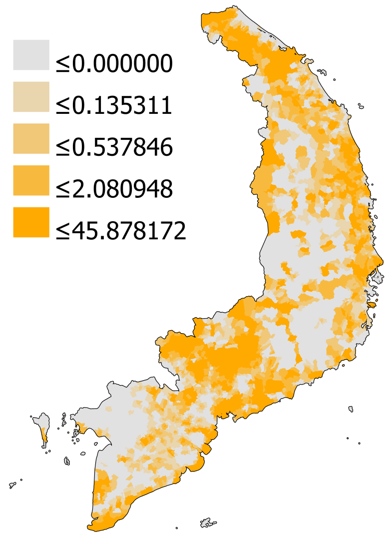
(d) Number of missions by month



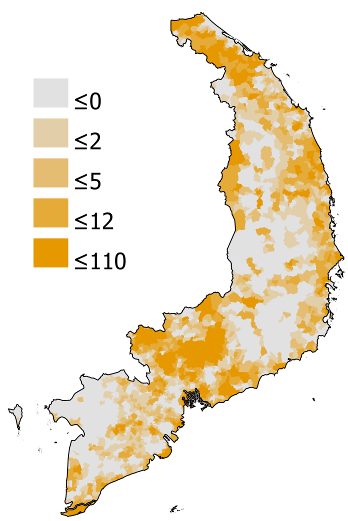
Note: Records of spray missions are from Stellman and Stellman (2011). Figure 1a maps spray paths of Agent Orange and other dioxin-contaminated herbicides. Figure 1b maps paths that used the dioxin-free Agent White. Figure 1c plots monthly herbicide use in gallons. Figure 1d plots the number of spray missions each month. Figures 1c and 1d mark March 8, 1965, when the first U.S. troops arrived in Danang as the escalation in the Second Indochina War.

Figure 2: Dioxin exposure

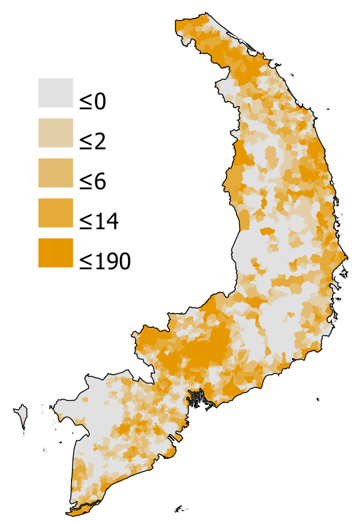
(a) Stellman Exposure Opportunity Index



(b) Number of spray missions



(c) Number of spray paths



Note: Figure 2 maps different measures for dioxin exposure at the commune level. Figure 2a maps the Stellman Exposure Opportunity Index from dioxin-contaminated spray missions within a 5 km radius of commune centroids, which is the explanatory variable in this paper. Figure 2b maps the number of dioxin-contaminated missions sprayed in all locations within a 5 km radius of commune centroids. Figure 2c maps the number of paths, either points or lines, sprayed dioxin-contaminated herbicides in any location within a 5 km radius of commune centroids.

forests on the southernmost tip of the country. These were also the locations of the Vietminh bases during the First Indochina War (1946-1954) against the French colonial forces. Other heavily defoliated areas are the Central Coast, which was and still is a population center, and along the Ho Chi Minh trail.

2.2 The 2009 Census of Population and Housing

Table 1: Summary statistics of EOI and health outcomes

	Obs. (1)	Mean (2)	Std. Dev (3)	Min (4)	Max (5)
Outcome variables at the commune level					
Stellman Exposure Opportunity Index	4,316	1.42	3.86	0	45.88
Estimated population in 2009	4,265	10,587	8,199	536	91,037
Estimated non-migrated population in 2009	4,265	8,653	5,542	451	51,551
Binary outcome variables at the individual level					
Reporting visual difficulties	5,819,714	0.05			
Reporting auditory difficulties	5,819,362	0.03			
Reporting mobility difficulties	5,819,380	0.03			
Reporting cognitive difficulties	5,818,181	0.04			
Being able to read and write	5,818,819	0.90			
Finished middle school	5,818,819	0.22			
Participating in the labor force	4,619,930	0.26			
Working in agricultural sector	3,440,923	0.59			

Note: The Stellman Exposure Opportunity Index measures the exposure to dioxin-contaminated herbicides. I calculate using the Agent Orange Data Warehouse built by Stellman and Stellman. The description of the calculation is in Appendix A. Other variables are from the 2009 Vietnamese Census of Population and Housing conducted by the General Statistical Office of Vietnam. Individual-level outcome variables are binary.

For health outcomes, I use the self-reported disability prevalence from the Census of Population and Housing as the dependent variables. The dataset is a sample of 15% of the population from the 2009 census. The outcomes are binary variables of whether an individual reports difficulties in seeing (even with glasses), hearing, memory, and walking. To minimize the noise that comes from migration, I restrict the sample to individuals who did not migrate within five years before the census. Also, since most spray missions happened in South Vietnam, the analysis does not include those living north of the 17th parallel at the time of the survey. The summary statistics of the outcome variables are in Table 1. Figure C1 plots the disability rate by age, which shows the increase in self-reported disability starting at 40 years old.

In addition to the health outcomes, the 2009 Census provides information on educational attainments, labor participation, and population size. The summary statistics of these variables are also in Table 1.

3 Econometric model

3.1 Cross-sectional comparisons

To estimate the correlation between health outcomes and dioxin exposure, I start with an OLS estimation with the following specification.

$$y_{icpt} = \beta EOI_{cp} + \delta X_{icpt} + \gamma_p + \theta_t + \varepsilon_{icpt} \tag{1}$$

In Equation 1, y_{icpt} is the health outcome of individual i living in commune c of province p born in year t. The explanatory variable is the commune-level Exposure Opportunity Index EOI_{cp} that measures exposure to dioxin-containing defoliants. I also control for individual and household-level covariates X_{icpt}, including gender, age, ethnicity, marital status, and urban status. Other parameters are province FE γ_p, birth cohort FE θ_t, and error terms ε_{icpt}.

One concern of the OLS estimation is the nonrandom exposure to dioxin and Operation Ranch Hand. The operation had two targets: foliage cover and crops that could supply the North Vietnamese forces. Therefore, areas around the bases belonging to the North Vietnamese had higher exposure to dioxin and other defoliants. As noted above, since these areas were often mountainous or remote, and therefore on a relatively slow development trajectory, the model would overestimate the human capital effects of dioxin exposure. On the other hand, there also could be underestimation, because herbicides also were sprayed around the South Vietnamese and US military bases and communication lines to reduce the chance of being ambushed. Including the province fixed-effect γ_p does not fully solve the problem because it only accounts for the cross-province variation in exposure level. Without accounting for the within-province variation in exposure level, the model suffers from omitted variable bias.

To address this omitted variable bias, I exploit the usage similarity between Agent Orange and Agent White. These herbicides were broad-leaf defoliants and were a compound of 2,4-D and another chemical. However, Agent Orange had dioxin, and

Agent White did not because 2,4,5-T Agent Orange was contaminated by dioxin. Since the US military was unaware of the dioxin contamination in 2,4,5-T, dioxin exposure from the Second Indochina War was unintended, as it was not supposed to hurt US personnel. Since both herbicides had the same functions and dioxin contamination was unexpected, using the total exposure to Agent Orange and Agent White as a control variable could create a continuous matching variable to match communes with similar characteristics. Therefore, the method accounts for the non-randomness of spray missions by purging the omitted variable bias.

Under the lens of Borusyak and Hull (2023), this method assumes the perfect substitution between Agent Orange and Agent White. Under this assumption, I could simulate the dioxin exposure by randomly choosing the spray paths in the pool of the ones that sprayed Agent White and Agent Orange herbicides and assigning the herbicide type randomly as dioxin-containing or not. With the spray paths and chance of spraying dioxin herbicides similar to the real-life version, I calculate the average Exposure Opportunity Index of dioxin across multiple simulations and use it as a control variable to account for the difference in communes' characteristics. Since the simulated dioxin exposure converges to be proportional to the total exposure to Agent Orange and Agent White, denoted as EOI'_{cp}, the below regression model labeled "Adjusted OLS" fits the framework proposed by Borusyak and Hull (2023) given the assumption of perfect substitution between the two herbicides.

$$y_{icpt} = \beta EOI_{cp} + \beta' EOI'_{cp} + \delta X_{icpt} + \gamma_p + \theta_t + \varepsilon_{icpt} \quad (2)$$

There are other sources of bias that I cannot overcome with available data. First, the sample consists of those who survived the Vietnam War and the hardships afterward. Without the individuals who were so severely affected that they did not survive, the estimated effect of dioxin exposure is biased downward. Second, the sample does not have migration information older than five years. Specifically, in the 2009 Census, the data only had the location of an individual in 2004. Restricting the sample to those who did not relocate in 2004-2009 only partly solves the issue because of the possibility of migrations between areas with different exposure levels before 2004. Again, the estimated effect of dioxin exposure would be biased downward. Third, sorting in migration leads to an upward bias.

3.2 Mechanism

Operation Ranch Hand could have affected current health outcomes through several channels. In this study, I focus on the effect of dioxin exposure. Without considering the sorting in migration, there are several possible channels. First, some people might have been directly exposed to dioxin during Operation Ranch hand. This could have happened through inhalation, dermal absorption, the food chain, or even famine caused by defoliants. Direct exposure to dioxin also affected those who were in utero. Second, people might have been exposed indirectly to dioxin residue in the environment after the operation. The third channel is the intergenerational effect, which could transmit the impact of dioxin exposure from older generations to younger ones by worsening socioeconomic conditions. This channel could happen to cohorts born after and even during the operation. To understand the size of these channels, I make two comparisons; one is between regions with different in-migration trends, and another is between those exposed directly during the utero period and those that were not.

3.2.1 Indirect exposure to dioxin

The Kinh ethnic group, which currently accounts for 85% of the Vietnamese population, were settled on the Central Coast and in the Mekong Delta for a few centuries before the Vietnam War. On the other hand, the Central Highlands area had significant settlements of the Kinh ethnic group starting in the late 19th century after French colonialism arrived, and most of the population arrived after 1975 (Evans, 1992; Hardy, 2000). As shown in Figure C2, the US and South Vietnamese governments estimated that most of the Central Highlands area had fewer than 50 people per square mile, while a significant share of the Central Coast and Mekong Delta had more than 500 people per square mile.

Interestingly, the majority of migrants to the Highlands are from above the DMZ. Table C1 shows the origins of the migrants to regions in South Vietnam in 1989, 1999, and 2009. North Vietnam was the dominant source of migrants in these years. However, in 1989, migrants from North Vietnam since 1984 accounted for only 1.63% of the Central Coast population and 0.80% of the Mekong Delta population. This number is 9.34% in the Central Highland. The numbers from the 1999 and 2009 censuses show that migration to these regions became smaller. Still, the inflow of 2004-2009 migrants to Central Highlands was larger than inflows to the Mekong Delta and Central Coast in all three censuses. Meanwhile, the former capital city of South Vietnam and surrounding areas, which are the Southeast region, received a constant flow of migrants.

One of the initial causes of this migration pattern was the New Economic Zone policy (1976-1980) that encouraged Southern urban residents and Northerners to move to remote and mountainous areas in South Vietnam (Desbarats, 1987). Even though migration inflows to the Central Coast were insignificant, the policy caused internal migration from coastal areas to the mountainous ones in the region. The difference in migration between regions implies heterogeneity in the effect of dioxin exposure. For instance, since the population of Central Highlands and the Southeast consists mostly of migrants, they were only exposed to dioxin residue after the war. On the other hand, the operation persistently affected the non-migrant Mekong Delta and Central Coast populations through all three channels described above. However, I expect the impact of wartime dioxin exposure and the intergenerational effects of that exposure to be significant in the Mekong Delta but not on the Central Coast because of the internal migration.

3.2.2 In utero exposure vs. intergenerational effects

Since most herbicides were sprayed from 1965 to the end of 1969, the estimated effects in the baseline model of dioxin exposure on the health outcomes of birth cohorts born by 1965 could be attributed to direct exposure. Similarly, the impact of dioxin exposure is intergenerational for the sample of those born after 1972, the last birth cohort that could have been directly exposed. However, for those born in 1965-1972, who were children and newborn babies during the operation, it is unclear whether intergenerational or in utero effects are the dominant force. To address this question, I use the records of herbicide sorties to identify the time of the last spray on each commune. Then, I use this information to separate the sample of those born in 1965-1975 into treatment and control groups. For instance, the last spray mission that might have used dioxin-containing herbicides was in November 1971. In communes affected by that sortie, people born by the end of August 1972 would be in the treatment group, and those born in September 1972 and after in the control group.

With birth cohort treatment, the second variation is the dioxin exposure level, which Exposure Opportunity Index measures. Since dioxin exposure is nonrandom, I use the simulated Exposure Opportunity Index and its interaction with the birth cohort treatment as control variables. The econometric specification would be

$$\begin{aligned} y_{icpt} = &\beta_1 EOI_{cp}.treat_{cpt} + \beta_2.EOI_{cp} + \beta_3.treat_{cpt} \\ &+ \beta_1'.\overline{EOI}_{cp}.treat_{cpt} + \beta_2'.\overline{EOI}_{cp} + \delta X_{icpt} + \gamma_p + \theta_t + \varepsilon_{icpt}. \end{aligned} \tag{3}$$

In Equation 3, y_{icpt} is the health outcome of individual i living in commune c of province p born at time t. Two treatment variables are EOI_{cp}, which measures dioxin exposure level, and $treat_{cpt}$, 1 for those born within nine months since the last spray on commune c and 0 otherwise. Control variables are simulated exposure level $\overline{EOI}_{cp}$ and individual and household level covariates X_{icpt}, including gender, age, ethnicity, marital status, and urban status. Other parameters are province FE γ_p, birth year cohort FE θ_t, and error terms ε_{icpt}. The error term ε_{icpt} is clustered at the commune level.

One concern over the generalized difference-in-differences method is the parallel trend assumption, as Callaway, Goodman-Bacon and Sant'Anna (2021) point out. The assumption is that the trend between age and health outcomes has a similar slope between areas with different exposure levels. If this assumption holds, the estimate $\hat{\beta}_1$ is the effect of direct dioxin exposure, including during the in utero stage, and $\hat{\beta}_2$ is the intergenerational effect. However, this assumption would be violated if health deterioration due to aging might happen faster in areas with higher dioxin exposure. Therefore, $\hat{\beta}_1$ might also capture the aging effect of dioxin exposure. To reduce the estimation bias, I minimize the age variation in the sample by only including those born between January 1965 and December 1974. Since people born from 1965 to 1974 would be 35-44 years old in 2009, the aging effect on health outcomes should be trivial in this group.

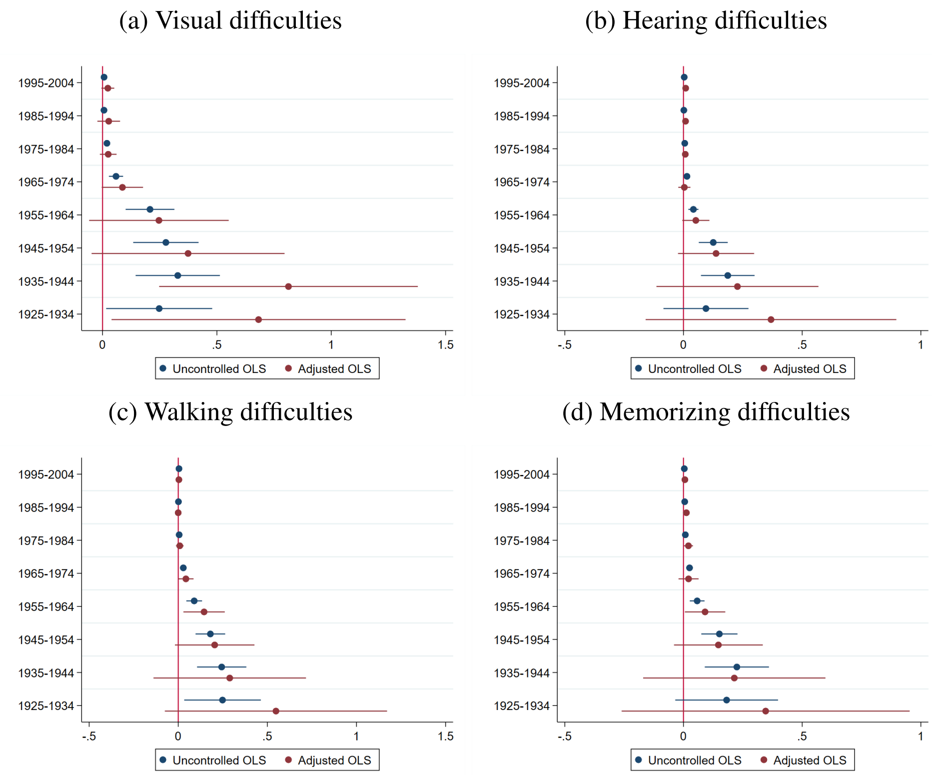
4 The health effects of dioxin exposure

4.1 Baseline results

Using the regression model following Equations 1 and 2, I compare the health outcomes between areas with different dioxin exposure. I apply models on different samples separated by birth cohorts to reveal the effect of dioxin exposure and eliminate the effects of aging. Figure 3a plots the estimated effect of dioxin exposure on the chance of having eyesight problems. The plot shows an increase in effect size as people age. It is statistically significant for those born before 1975. As the unit of the horizontal axis is percentage points, the results show that a one-unit increase in Exposure Opportunity Index would raise the chance of having eyesight problems by 0.087 percentage points for those born in 1965-1974. The numbers are 0.247, 0.374, 0.813, and 0.682 for those born in 1955-1964, 1945-1954, 1935-1944, and 1925-1934. In 2009, individuals in these five birth cohort groups were from 35 to 84 years old. Since the effect magnitude becomes larger in the older generations, the results suggest that aging amplifies the effect of dioxin exposure. Details of these estimations are in Table C2.

Compared to eyesight issues, the effect of dioxin exposure on hearing difficulties has a smaller magnitude. A one-unit increase in Exposure Opportunity Index

Figure 3: Dioxin exposure and disability prevalence, 2009 Census



(a) Visual difficulties

(b) Hearing difficulties

(c) Walking difficulties

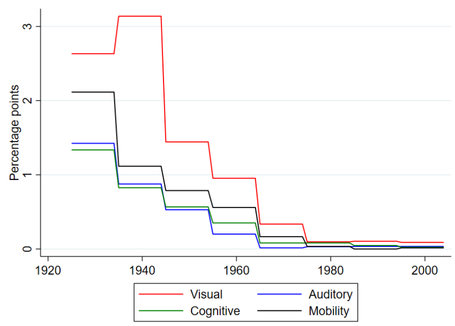
(d) Memorizing difficulties

Note: Figure 3 shows the impact on disability prevalence (in percentage points) of a one-unit increase of the Exposure Opportunity Index at the commune level. These are regression results from different samples of birth cohort groups from the 2009 Census of Population and Housing. The samples consist of 15% of the population who were living below the 17th parallel and who had not migrated within five years previous to the survey. Unadjusted OLS does not control for the simulated EOI, but the controlled OLS does. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

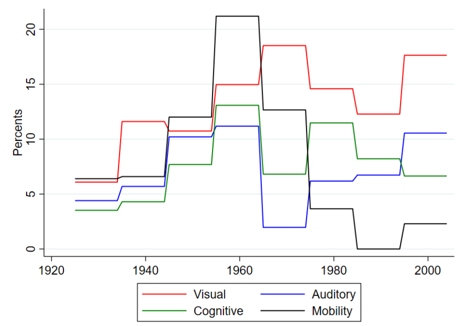
would raise the chance of having hearing difficulties by 0.052 and 0.137 percentage points for those born in 1955-1964 and 1945-1954. The number for the 1935-1944 and 1925-1934 birth cohorts are 0.227 and 0.369. Even though it is consistent that the effect magnitude is larger in the older generations, the effect on these two birth cohort groups is not statistically significant. A possible explanation is that hearing issues are common in these age groups, so that dioxin exposure has little additional effect. The results are in Table C3, which I plot in Figure 3b.

Figure 4: Impact of one standard deviation increase in dioxin exposure

(a) In percentage points



(b) Relative to sample mean



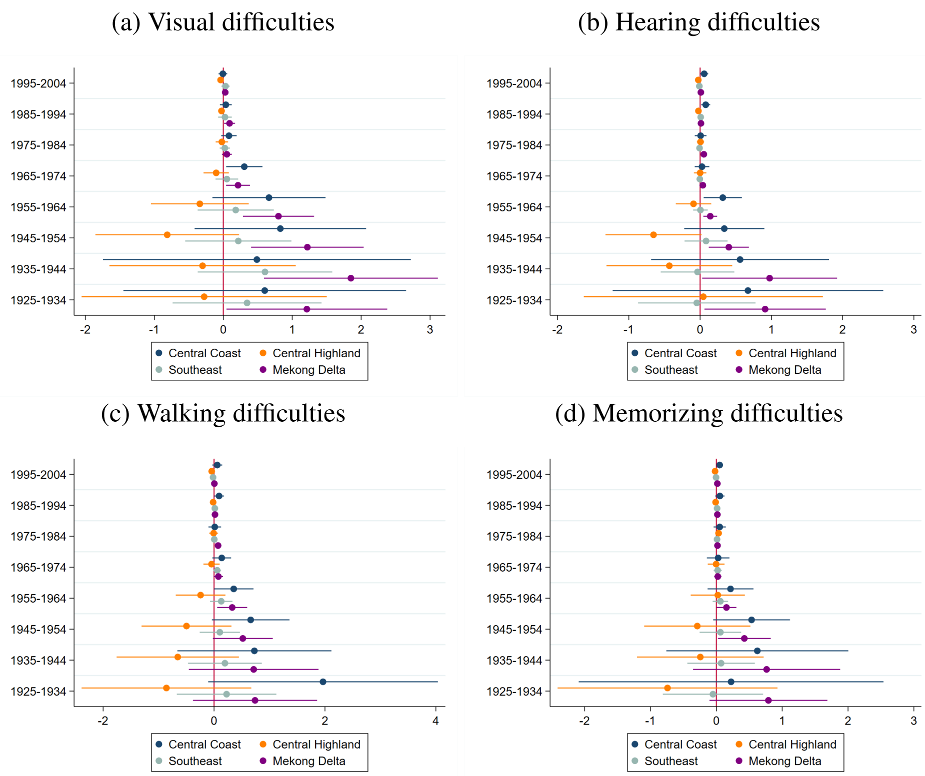
Note: Figure 4 shows the impact of a one-standard-deviation increase in EOI on disability prevalence by birth cohorts. Figure 4a shows the change in percentage points of the disability probabilities. Figure 4b compares the impact magnitude in Figure 4a to the prevalence in unexposed communes.

The estimated effects of dioxin exposure on walking and memory difficulties show similar patterns, in that the point estimates are larger in the older generations. A one-unit increase in EOI would increase the chance of having walking problems by 0.043 percentage points in the 1965-1974 birth cohorts. These numbers would jump to 0.145, 0.204, and 0.548 in the 1955-1964, 1945-1955, and 1925-1934 birth cohorts. The effects on walking are shown in Table C4 and Figure 3c Unlike the other health outcomes, the impact of dioxin exposure on memory becomes statistically significant in the younger generation, as shown in Table C5 and Figure 3d. The effect of a one-unit increase in EOI increase the chance of having memory difficulty by 0.012, 0.021, and 0.091 percentage points in the 1985-1994, 1975-1984, and 1955-1964 birth cohorts.

To understand the magnitude, I multiply the estimated coefficient by the standard deviation of the Exposure Opportunity Index to obtain the health effect of a one-standard-deviation increase in exposure level. Figure 4a plots all point estimates by age group regardless of statistical significance. The figure shows that a one-standard-deviation increase in EOI could raise the disability prevalence by one to three percentage points for those born before 1945 and less than one percentage point for those born between 1945 and 1975. Figure 4 compares the effect magnitude to the disability prevalence in zero-exposure areas. For the cohort born by the end of the Vietnam War in 1975, the effect of one standard deviation of EOI would be around 5-20% of the disability prevalence. In short, the persistent health effect of dioxin exposure is non-trivial.

4.2 Indirect exposure to dioxin

Figure 5: Estimation results by regions, 2009 Census



Note: Figure 5 shows the impact on disability prevalence (in percentage points) of a one-unit increase of the Exposure Opportunity Index by region. The data is from the 2009 Census of Population and Housing. The sample consists of 15% of the population who were living below the 17th parallel and who had not migrated within five years previous to the survey. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

As discussed in Section 3.2.1, I estimate the effect of dioxin exposure by regions to understand the magnitude of the effects of indirect exposure. Because migrants who came after the Vietnam War account for a big part of the Central Highlands and

Southeast populations, those populations are less likely to have experienced direct exposure to dioxin during the war or to have inherited adverse effects from older generations. Therefore, estimations using the sample of the Central Highlands and Southeast populations would reveal the health effects of indirect exposure, resulting from dioxin residue in soil and decreased agricultural productivity.

Figure 5 plots the Adjust OLS estimations with samples from the Central Coast, Central Highland, Southeast, and Mekong Delta. The results suggest that exposure to dioxins has not affected the current Central Highlands and Southeast populations. Surprisingly, in the Central Highland, the statistically insignificant point estimates show better health outcomes in a more exposed commune in certain age groups. From the current data, I do not have enough evidence to conclude that dioxin residue left from the Vietnam War has persistent health effects on the contemporary population. The extreme level of dioxin concentration in areas such as Bien Hoa airbases, Da Nang airbases, or A Sau Valley still harms the local communities. But, nationwide, the dioxin residue might not have any effect.

The Mekong Delta sample shows larger and more statistically significant estimates of the effect of dioxin exposure than the one in Figure 3.1. However, the Central Coast sample shows smaller estimates with higher standard errors than the Mekong Delta sample. This result suggests that the persistent health effect of dioxin exposure is weaker in the Central Coast. A possible explanation is the internal migration within the Central Coast. Figure fig:population1968 shows high population density in coastal plains but low density in mountainous areas. Moreover, provinces in the southern tip of the Central Coast from Phu Yen to Binh Thuan have low population density.

To check whether the estimation results are robust, I conduct the same analysis with samples from rural areas. Figure C3 plots the estimated parameters and shows a similar trend as Figure 5. These results suggest direct exposure and intergenerational effect as the primary channels.

4.3 In utero exposure vs. intergenerational effects

Table 2 presents the impact of direct dioxin exposure and intergenerational effect in the sample of those born in 1965-1974. The coefficients of the interaction of birth cohort treatment and dioxin exposure represent the effect on those conceived before the last spraying. On the other hand, the coefficient of dioxin exposure represents the effect on the whole sample. I interpret these two coefficients as the impacts of in utero exposure and intergenerational effects. Because data on birth-

Table 2: Utero exposure vs. intergenerational factor in 1965-1974 birth cohorts

	Central Coast	Central Highlands	Southeast	Mekong Delta
	(1)	(2)	(3)	(4)
Panel A: Visual difficulties				
EOI/100	0.164	-0.02	0.004	0.133**
	(0.105)	(0.091)	(0.045)	(0.067)
treat × EOI/100	0.22	-0.072	0.074	0.163*
	(0.154)	(0.107)	(0.094)	(0.098)
Observations	243,926	115,274	192,293	361,048
R-squared	0.008	0.009	0.007	0.008
Panel B: Hearing difficulties				
EOI/100	0.046	-0.027	-0.013	0.047**
	(0.059)	(0.037)	(0.017)	(0.022)
treat × EOI/100	0.005	0.039	-0.006	-0.028
	(0.070)	(0.064)	(0.024)	(0.027)
Observations	243,923	115,272	192,266	361,034
R-squared	0.005	0.005	0.004	0.003
Panel C: Walking difficulties				
EOI/100	0.099	0.021	0.044	0.037
	(0.088)	(0.061)	(0.030)	(0.034)
treat × EOI/100	0.068	-0.029	-0.009	0.073
	(0.078)	(0.090)	(0.028)	(0.048)
Observations	243,878	115,242	192,243	360,955
R-squared	0.022	0.014	0.01	0.01
Panel D: Memorizing difficulties				
EOI/100	-0.003	-0.011	-0.001	0.017
	(0.088)	(0.043)	(0.026)	(0.029)
treat × EOI/100	0.053	0.048	0.038	0.031
	(0.095)	(0.100)	(0.030)	(0.038)
Observations	243,923	115,272	192,274	361,038
R-squared	0.01	0.006	0.006	0.007

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Table 2 shows the results of the diff-in-diff regressions with two layers of variation. One is dioxin exposure measured by Exposure Opportunity Index, and another is direct exposure, including the in utero period. The sample consists of those born in 1965-1974. Other variables are simulated EOI, birth cohort treatment, and the interaction of the simulated EOI and birth cohort treatment. I also control for gender, age, ethnicity, marital status, urban status, birth year cohort FE and province FE. The error term is clustered at the commune level.

place is unavailable, the estimates rely on the assumption that a person living in a commune was born in the same commune. However, this assumption is unlikely to hold; therefore, the coefficients underestimate the in utero and intergenerational effects.

As discussed in the previous section, because the Central Highlands and Southeast have been two popular destinations for migrants from all over the country, the in utero and intergenerational effects in these regions are small and noisy. This is consistent with Columns (2) and (3), in which estimates are statistically insignificant. The Central Coast has a similar situation because of the internal migration to mountainous and western areas. On the other hand, the results from the Mekong Delta show the in utero and intergenerational effects of dioxin exposure on visual impairments, which have similar magnitudes. A one-unit increase of EOI increases the chance of having visual difficulties by 0.133 and 0.163 percentage points due to intergenerational effect and in utero exposure, respectively. Since only 1.8% of the 1965-1974 birth cohorts in unexposed areas reported eyesight problems, the magnitudes of both channels in terms of one standard deviation of dioxin exposure, which is 3.8, would be 28% and 34% of the visual difficulty prevalence. However, only the intergenerational channel impacts hearing impairments. Since 0.78% of the 1965-1974 birth cohorts report hearing difficulties, the coefficient of 0.047 means that the impact of one standard deviation would be 23% of the hearing impairment prevalence.

The results show that both in utero exposure and intergenerational effects of dioxin affect health outcomes, even though the estimated coefficients for in utero exposure are statistically significant only for visual difficulties. There are many explanations. Firstly, the data on birthplace is unavailable, leading to underestimation of the effect. Secondly, the sample consists of those who survived direct dioxin exposure. Lastly, the 1965-1974 birth cohorts were too young in 2009 to have health issues. The self-reported disability prevalence in these birth cohorts in the 2009 Census does not exceed 2%. Even though the results underestimate the effect of dioxin exposure, they still show the non-trivial impacts of intergenerational and in utero effects.

4.4 Robustness checks

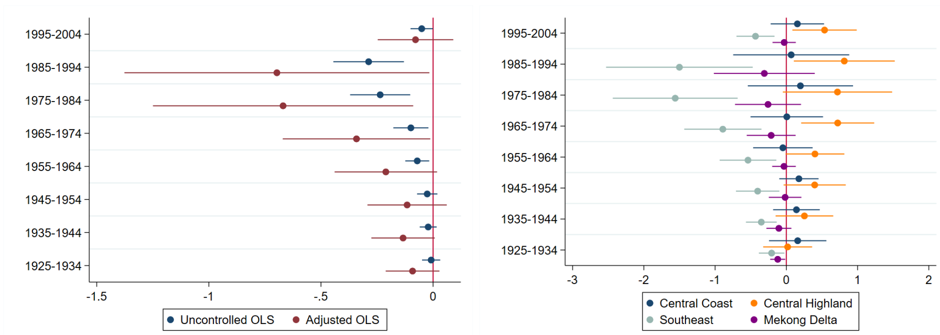
4.4.1 Sorting by migration

One potential source of bias is sorting by migration. If the information on dioxin contamination were publicly available, there would be migration from the exposed areas to the unexposed ones. Even when the information is unavailable, if the associated effects were noticeable, it would result in the same migration pattern. Since migration is costly, the characteristics of migrants would be different from those who choose to stay. For instance, healthy and productive members of exposed communities might be more likely to move to unexposed areas. If such sorting happens, the econometric model overestimates the impact on health and labor outcomes.

To test whether migrants were more likely to move to unexposed areas, I use the regression models with Equations 1 and 2, in which the binary outcome variable is one if a person moved to a commune within the previous five years and zero otherwise. Given that the data is from the 2009 census, a person was an immigrant if he lived in a different commune in 2004. The estimation would show the change in migration status for a one-unit increase in exposure index. An insignificant estimated coefficient implies indifference in migration inflow between exposed and unexposed communes. A disadvantage of this outcome variable is the inability to capture migration that happened before the five-year threshold.

Figure 6: Migrant inflows and dioxin exposure, 2009 Census

(a) Whole sample (b) By regions



Note: Figure 6 the correlation between migrant inflows and dioxin exposure at the commune level. Figure 6a plots the estimates with the whole samples. Figure 6a plots the Adjusted OLS results by regions. The data is from the 2009 Census of Population and Housing. The sample consists of 15% of the population who were living below the 17th parallel at the time of the survey. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

Figure 6 plots the correlation between the inflow of migrants and exposure level by age groups. Figure 6a plots the estimations with and without simulated exposure level. Both specifications show that areas exposed to dioxin have been less likely to receive migrants. However, this trend is not homogeneous among regions, as shown in Figure 6b. The Central Coast and Mekong Delta do not show any correlation between migration inflows and exposure to dioxin. However, migrants were more likely to come to areas with higher dioxin exposure in the Central Highland. This correlation explains the negative coefficients in the Central Highlands estimates in Figure 5. The Southeast shows an opposite trend, in that migrants were more likely to come to areas with lower dioxin exposure, meaning that the Southeast estimates overestimate the health impacts of dioxin exposure. Even though these results suggest sorting by migration, the bias caused by this phenomenon seems small because the estimates for the health impact of dioxin in the Southeast are not statistically different from zero, as shown in Figure 5.

I conduct a similar test for emigrant outflows. However, information on the district of origin is available only for individuals who migrated within a province. To test this hypothesis, I use district-level average exposure as the explanatory variable and exclude those who migrated across provinces from the sample. The regression model is

$$y_{idpt} = \beta.EOI_{dp} + \beta'.\overline{EOI}_{dp} + \delta X_{idpt} + \gamma_p + \theta_t + \varepsilon_{idpt}. \tag{4}$$

In Equation 4, y_{idpt} is a binary variable if person i from district d of province p born in year t migrated out of district d. The explanatory variable is the average dioxin exposure of all communes within district d, EOI_{dp}. I also control for the average simulated exposure level $\overline{EOI}_{dp}$, individual characteristics X_{idpt}, province fixed-effects γ_p, and birth cohort fixed-effects θ_t. The standard error ε_{idpt} is clustered at the district level.

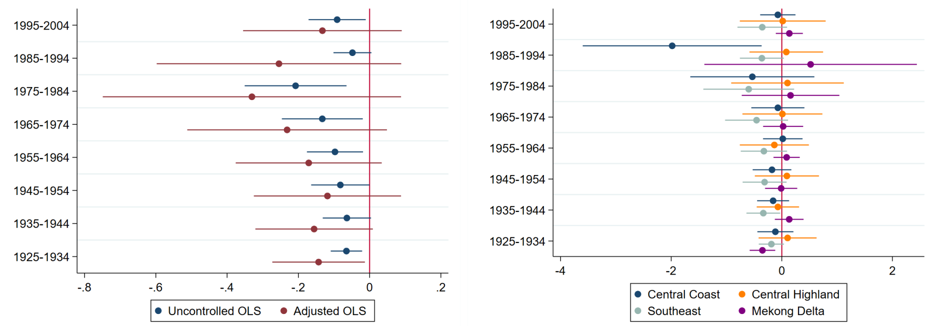
Figure 7 plots the correlation between emigrant outflows and dioxin exposure. Using the whole sample, Figure 7a shows that districts with higher average dioxin exposure would have smaller migration outflows, even though the estimates are not statistically significant. This trend might happen in the Central Coast or Southeast, but not in the Central Highlands or Mekong Delta, as shown in Figure 7b. Based on these results and those on migrant inflows, dioxin exposure does not strongly correlate with migration flows. Therefore, it should not be a concern in this project.

4.4.2 Skewness of EOI

The distribution of the Exposure Opportunity Index is heavy-tailed because of the extreme level of dioxin exposure in the Ma Da forest of Dong Nai province. To test

Figure 7: Within-province emigrant outflows and dioxin exposure, 2009 Census

(a) Whole sample (b) By regions



Note: Figure 7 plots the correlation between within-province emigrant outflows and district-level dioxin exposure. Figure 6a plots the estimates with the full samples. Figure 6a plots the Adjusted OLS results by regions. The data is from the 2009 Census of Population and Housing. The sample consists of 15% of the population who were living below the 17th parallel. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the district level.

whether the health outcomes of these extreme locations manipulate the estimation results, I transform the value of EOI and use it as the explanatory variable. The transformations that I use in this exercise are modified logarithm $\log(EOI_{cp} + 1)$ and inverse hyperbolic sine $\log(EOI_{cp} + \sqrt{EOI_{cp}^2 + 1})$. The results in Figures C4 and C5 show a trend that is similar to the one in Figure 3. It suggests that the results are not driven by the health outcomes of those living the areas with extreme levels of dioxin exposure.

4.4.3 Bombing and Agent Blue

A potential source for omitted variable bias is bombing intensity. However, because the spatial distributions of bombing and herbicide sorties that used Agent Blue are non-random and endogenous, controlling these two variables alone would not solve the issue. In the case of bombing intensity, I calculate the total weight of bombing dropped within a 10-km radius of commune centroids and restrict the sample to communes that received less than 300,000 tons of ordnance throughout the Vietnam War, which is about the average bombing intensity at the commune level. As in Figure C6a, this group of communes is on the very left tail of the distribution, which limits the variation in bombing intensity and the related endogeneity issue. Figures C7 and C8 plot the estimation results that control the modified logarithm of bombing intensity, which show patterns similar to the baseline results in Figures 3 and 5.

Another source is Agent Blue, which is another type of herbicides, which purpose is for To account for exposure to Agent Blue, I only use communes that have an Exposure Opportunity Index to Agent Blue of less than 0.2, which is the average exposure to Agent Blue at the commune level. As in Figure C6b, these communes are on the very left tail of the distribution, and therefore using this sample would reduce the endogeneity of Agent Blue. Figures C9 and C10 plot the estimation results with exposure to Agent Blue as an additional control variable. The results do not change much, except that estimations with the Central Highlands sample in Figure C9 are negative and statistically significant. These results mean that, in the Central Highland, people living in communes with higher exposure to dioxin would have better health outcomes. The best explanation is suggested by Figure 6, which shows that migrants to the Central Highlands chose to stay in areas with higher exposure. To sum up, not including bombing intensity and exposure to Agent Blue does not change the results substantially.

5 Dioxin exposure and long-term economic development

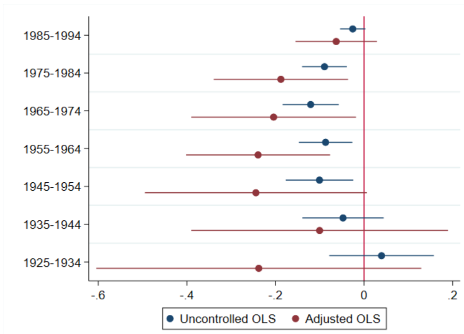
In a context that is similar to Vietnam, Yamada and Yamada (2021) and Riaño and Caicedo (2021) found that Laotian areas with higher bombing intensity during the Second Indochina War emit less light at night, meaning that these areas have less economic activity. Riaño and Caicedo (2021) attributed this in part to unexploded ordnance and in part to the persistent impact of bombing intensity on human capital accumulation and structural transformation. Similarly, the persistent health effects of herbicidal warfare in Vietnam may have affected educational attainment and, thus, local labor markets. Therefore, dioxin exposure would have long-term economic effects. In this section, I test these hypotheses.

5.1 Dioxin exposure and educational outcomes

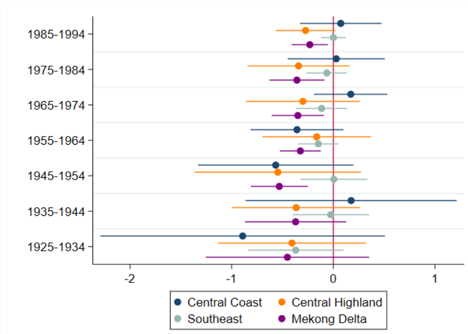
To study the impact of dioxin exposure on educational attainment, I use two binary outcome variables, namely being able to read and write and finishing lower secondary education. Figure 8 plots the regression results, in which the ability to read and write is the dependent variable. Figure 8a shows that people living in highly exposed communes are less likely to be able to read and write. A one-unit increase in exposure level reduces the channel of being able to read and write by

Figure 8: Dioxin exposure and the ability to read and write

(a) Whole sample



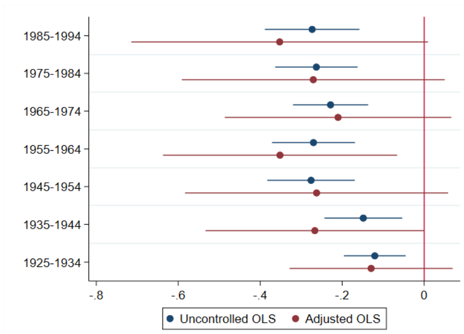
(b) By regions



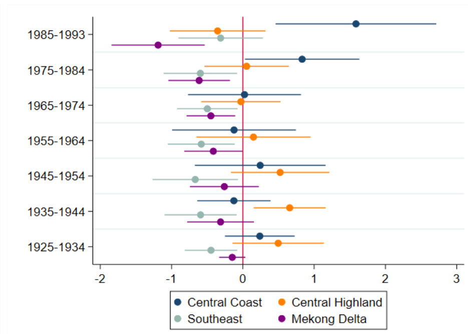
Note: Figure 8 plots the impacts of one unit of the Exposure Opportunity Index of dioxin-containing herbicides on the chance of being able to read and write (unit: percentage points). The samples consist of 15% of the population who were living below the 17th parallel and who had not migrated within five years previous to the survey. The control variables in Uncontrolled OLS are age, gender, marital status, ethnicity, religion, urban status, and province FE. Adjusted OLS control for these variables and simulated dioxin exposure. Regressions in Figure 8b are Adjusted OLS. Standard errors are clustered at the commune level.

Figure 9: Dioxin exposure and lower secondary education

(a) Whole sample



(b) By regions



Note: Figure 9 plots the impacts of one unit of the Exposure Opportunity Index of dioxin-containing herbicides on the chance of finishing lower secondary education (unit: percentage points). The samples consist of 15% of the population who were living below the 17th parallel and who had not migrated within five years previous to the survey. The control variables in Uncontrolled OLS are age, gender, marital status, ethnicity, religion, urban status, and province FE. Adjusted OLS control for these variables and simulated dioxin exposure. Regressions in Figure 9b are Adjusted OLS. Standard errors are clustered at the commune level.

about 0.2 percentage points for cohorts born from 1945 to 1984. Even though the ability to read and write is an educational outcome, it also reflects cognitive ability, which explains the impact on the cohorts born in 1945-1955. Figure 8b plots the Adjusted OLS estimations by region. I do not find any evidence for the impact of dioxin exposure on the ability to read and write in in the Central Coast, Central Highland, and Southeast. However, in the Mekong Delta, which was populated during the war and received a tiny inflow of migrants after the war, I find that people in highly exposed communes are less likely to be able to read and write.

Figure 9 plots the impact of dioxin exposure on education, using completion of lower secondary education as the outcome variable. Figure 9a shows that people in highly exposed communes are less likely to have finished middle school, even for those born long before the war. To understand the source of this odd result, I run the regressions by regions and plot them in Figure 9b. Similarly to Figure 8b, I do not find any evidence for the impact on educational attainment in the Central Coast and Central Highlands areas. However, in the Southeast, the communes with higher dioxin exposure have lower educational attainment. One explanation for this pattern is that communes with higher dioxin exposure attract fewer migrants, as shown in Figure 6b. On the other hand, in the Mekong Delta, the impact of dioxin exposure on education is observed only for those born after 1955. Thus, dioxin exposure distorts human capital accumulation, not only through health effects, but also by adversely affecting educational outcomes.

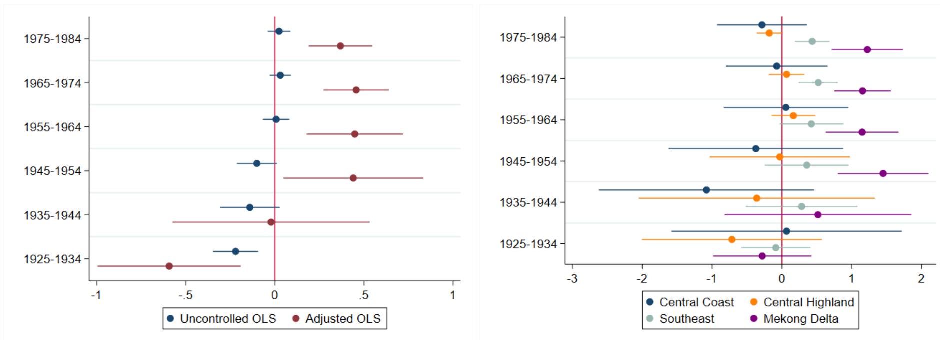
5.2 Dioxin exposure and labor outcomes

Since dioxin exposure adversely affects health and education attainments, it would affect the labor supply. Using the 2009 census, I define a person as unemployed if that person did not work or receive any payment within the previous seven days and was not returning to work in the next 30 days. Using this outcome variable, I run regressions by age group and plot the results in Figure 10. Figure 10a plots the estimation with the whole sample, showing that one increase in exposure level increases the chance of unemployment by about 0.5 percentage points for the group born after 1945. Figure 10b plots the regression results by region. Dioxin exposure does not affect employment in Central Coast and Central Highland. But, dioxin exposure increases the chance in Southeast and Mekong Delta. In the Mekong Delta, one increase in exposure level raises the probability of unemployment by more than one percentage point for the post-1945 birth cohorts.

Riaño and Caicedo (2021) found that bombing intensity pushes back structural transformation by keeping exposed individuals from moving out of the agricultural

Figure 10: Dioxin exposure and unemployment

(a) Whole sample (b) By regions



Note: Figure 10 plots the impacts of one unit of the Exposure Opportunity Index of dioxin-containing herbicides on the chance of being unemployed (unit: percentage points). The samples consist of 15% of the population who were living below the 17th parallel and who had not migrated within five years previous to the survey. The control variables in Uncontrolled OLS are age, gender, marital status, ethnicity, religion, urban status, and province FE. Adjusted OLS control for these variables and simulated dioxin exposure. Regressions in Figure 10b are Adjusted OLS. Standard errors are clustered at the commune level.

sector. I conduct the same test for dioxin exposure and plot it in Figure C11. The results do not support the hypothesis that dioxin exposure has distorted structural transformation in Vietnam.

5.3 Dioxin exposure and population size

Figure 11 provides evidence for the impact of dioxin exposure on population size. As in Figure 11a, the Adjusted OLS estimates with the whole sample show that a one-unit increase in exposure level decreases the population size by about four percent. However, in Figure 11b, no region shows a statistically significant impact of dioxin exposure on population size, except for the Mekong Delta sample. Still, it is worth noting that the point estimates for the Central Coast sample are similar to those for the Mekong Delta. The effects of dioxin exposure on non-migrant population in Figure C12 have the same patterns.

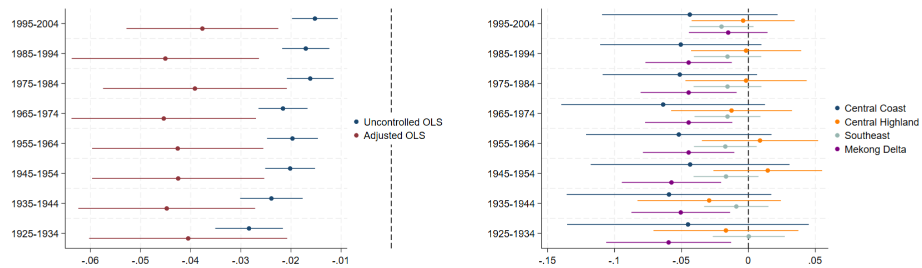
5.4 Dioxin exposure and luminosity

Because dioxin exposure affects human capital accumulation, unemployment, and population size, it is likely to distort economic activities. To test this hypothesis, I

Figure 11: Dioxin exposure and log of population

(a) Whole sample

(b) By regions



Note: Figure 11 plots the impacts of one unit of the Exposure Opportunity Index of dioxin-containing herbicides on the log of population. The samples consist of 15% of the population who were living below the 17th parallel. The control variables in Uncontrolled OLS are age, gender, marital status, ethnicity, religion, urban status, and province FE. Adjusted OLS control for these variables and simulated dioxin exposure. Regressions in Figure 11b are Adjusted OLS. Standard errors are clustered at the commune level.

use the nightlight data acquired from NOAA called Version 4 DMSP-OLS Night-time Lights Time Series as a proxy for the economic outcome. In this test, I use both Uncontrolled and Adjusted OLS specifications.

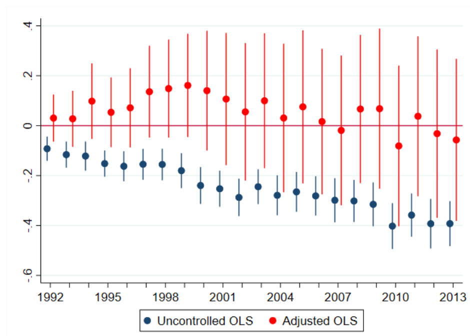
$$y_{cp} = \beta.EOI_{cp} + \gamma_p + \theta X_{cp} + \varepsilon_{cp} y_{cp} = \beta.EOI_{cp} + \overline{\beta}.\overline{EOI}_{cp} + \gamma_p + \theta X_{cp} + \varepsilon_{cp}$$

In the above equations, y_{cp} is the average luminosity level in commune c of province p. The main explanatory variable is the Exposure Opportunity Index EOI_{cp}, which is a proxy for dioxin exposure level. $\overline{EOI}_{cp}$ is the simulated exposure level. Other variables are province fixed effects γ_p and control variable X_{cp}, which includes urban status, longitude, and latitude. The standard error ε_{cp} is bootstrapped.

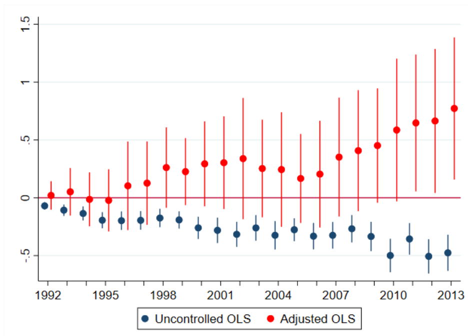
Figure 12 separately plots the estimation results for the cross-sectional nightlight data from 1992 to 2013. The results show that dioxin exposure adversely affects luminosity only in the Mekong Delta. These estimates are consistent with the previous findings that the adverse effect of dioxin exposure persists in the Mekong Delta. The reason is that the Mekong Delta was populated before the war and, therefore, received fewer migrants after the war. By limiting the inflow of migrants, the Mekong Delta perpetuates the impact of the Vietnam War.

Figure 12: Dioxin exposure and luminosity

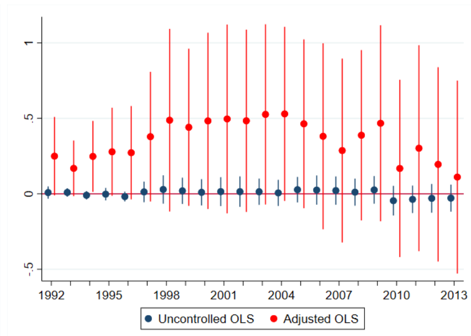
(a) Whole sample



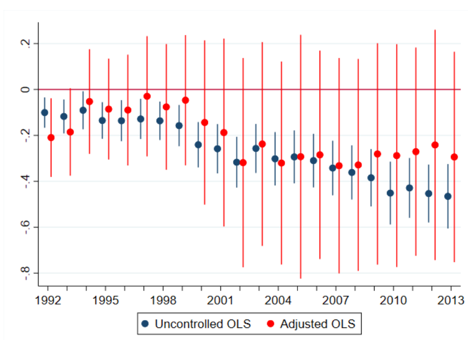
(b) Central Coast



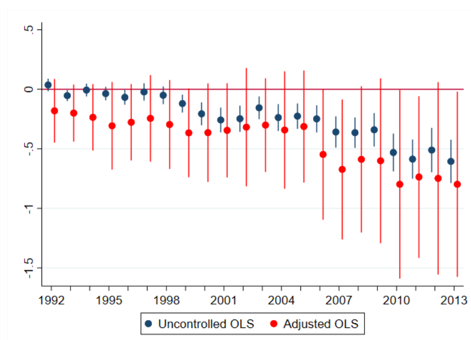
(c) Central Highland



(d) Southeast



(e) Mekong Delta



6 Conclusion

In this study, I investigate the long-term impact of dioxin exposure from the Vietnam War on human capital accumulation. Using the method proposed by Borusyak and Hull (2023), the results show that individuals living in exposed areas are more likely to report having difficulties in seeing, hearing, memory, and walking. The magnitude of these effects is non-trivial; the impact of one standard deviation of exposure index is about 5-20% of the baseline disability prevalence. My methods allow me to uncover two main channels contributing to impaired health: wartime dioxin exposure and cascading intergenerational effects from the initial exposure. However, I found no conclusive evidence regarding the health effects of post-war exposure to dioxin residue, despite dioxin being a persistent organic pollutant.

I also found that herbicide warfare has long-term impacts on local economies. By affecting health, dioxin exposure distorts human capital accumulation, as well as reducing educational attainment and population size. I also found that communes with higher dioxin exposure have a high unemployment rate. It is unclear whether the cause of higher unemployment is impaired health or lower levels of development. In terms of economic activities, communes with higher exposure emit less luminosity, which means less economic activity. In conclusion, even though the Second Indochina War ended in 1975, its socioeconomic legacy remains persistent.

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A Exposure Opportunity Index

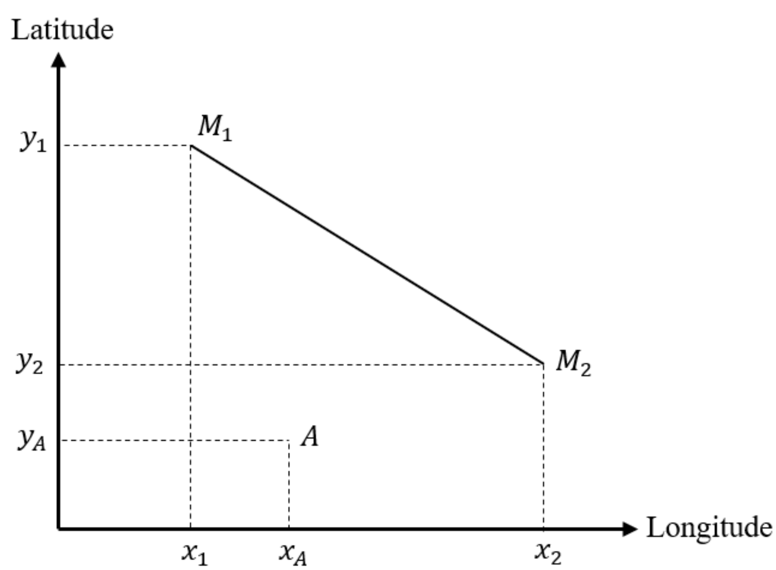
This section describes the Exposure Opportunity Index (EOI) proposed by Stellman and Stellman (2004). In the records, two types of spray patterns are point and straight line. For the point source of dioxin, the calculation is straightforward. Denote that i is the mission number, h_i is the amount of sprayed herbicides in gallons on point $M(x, y)$, and $d(A, M)$ is the distance from point $A(x_A, y_A)$ to point M. Then the EOI on A from mission i would be

$$EOI_i^A = \frac{h_i}{d(A, M)} = \frac{h_i}{\sqrt{(x - x_A)^2 + (y - y_A)^2}} \tag{5}$$

In other missions, Stellman and Stellman (2004) calculated the exposure index from all points along the flight path. As the flight path in other missions could be broken down into straight lines, let's just consider mission i sprayed h_i gallons from point $M_1(x_1, y_1)$ to point $M_2(x_2, y_2)$ as in Figure A1. The amount of herbicide sprayed in a point $M_j(x_j, y_j)$ along M_1M_2 line is $h_i/d(M_1, M_2)$. The exposure from point M_j to A is

$$EOI_{ij}^A = \frac{h_i}{d(M_1, M_2)} . \frac{1}{d(M_j, A)} = \frac{h_i/\sqrt{(x_1 - x_2)^2 + (y_1 - y_2)^2}}{\sqrt{(x_j - x_A)^2 + (y_j - y_A)^2}} \tag{6}$$

Figure A1: The case of a straight line spray mission



Denote $m = (y_2 - y_1)/(x_2 - x_1)$ and $n = y_1 - mx_1$, the linear function of M_1M_2 is $y = mx + n$. The exposure from point M_j to A could be rewritten as

$$EOI_{ij}^A = \frac{h_i/\sqrt{(x_1 - x_2)^2 + (y_1 - y_2)^2}}{\sqrt{(x_j - x_A)^2 + (mx_j + n - y_A)^2}} \tag{7}$$

The exposure from mission i to A would be the sum of exposure from all points along M_1M_2.

$$\begin{aligned}
EOI_i^A &= \sum_j EOI_{ij}^A & (8)\\
&= \int_{x_1}^{x_2} \frac{h_i/d(M_1, M_2)}{d(M(x,y), A)} dx & (9)\\
&= \int_{x_1}^{x_2} \frac{h_i/\sqrt{(x_1 - x_2)^2 + (y_1 - y_2)^2}}{\sqrt{(x - x_A)^2 + (mx + n - y_A)^2}} dx & (10)
\end{aligned}$$

Since $h_i/\sqrt{(x_1 - x_2)^2 + (y_1 - y_2)^2}$ is a constant, we only need to calculate

$$\begin{aligned}
&\int_{x_1}^{x_2} \frac{1}{\sqrt{(x - x_A)^2 + (mx + n - y_A)^2}} dx & (11)\\
=& \int_{x_1}^{x_2} \frac{dx}{\sqrt{(m^2+1)x^2 + 2((n - y_A)m - x_A)x + x_A^2 + (n - y_A)^2}} & (12)\\
=& \frac{1}{\sqrt{m^2+1}} \int_{x_1}^{x_2} \frac{dx}{\sqrt{x^2 + 2\frac{(n-y_A)m - x_A}{m^2+1}x + \frac{x_A^2 + (n-y_A)^2}{m^2+1}}}. & (13)
\end{aligned}$$

Since $\int dx/\sqrt{x^2 + c} = \ln(x + \sqrt{x^2 + c})$, then

$$\begin{aligned}
&\int_{x_1}^{x_2} \frac{1}{\sqrt{(x - x_A)^2 + (mx + n - y_A)^2}} dx & (14)\\
=& \frac{1}{\sqrt{m^2+1}}.\ln\left[x + \frac{(n - y_A)m - x_A}{m^2+1} + \frac{d(M(x,y), A)}{\sqrt{m^2+1}}\right]_{x_1}^{x_2}. & (15)
\end{aligned}$$

The right-hand side of Equation 15 could be rewritten to look like Equation 3 in Stellman and Stellman (2004).

$$\frac{1}{\sqrt{m^2+1}} \ln\left[\frac{(m^2+1)x + (n - y_A)m - x_A}{\sqrt{m^2+1}} + d(M(x,y), A)\right]_{x_1}^{x_2}$$

Using Equations 10 and 15, the exposure of mission i spraying through $M_1 M_2$ on point A would be

$$EOI_i^A = \frac{h_i/d(M_1, M_2)}{\sqrt{m^2+1}} \ln \left[x + \frac{(n - y_A)m - x_A}{m^2+1} + \frac{d(M(x,y), A)}{\sqrt{m^2+1}} \right]_{x_1}^{x_2}. \quad (16)$$

The total exposure on point A would be the sum of the exposure index from all missions.

$$EOI^A = \sum_i EOI_i^A \quad (17)$$

B Additional tables and figures

Figure C1: Disability prevalence by age, 2009 Census

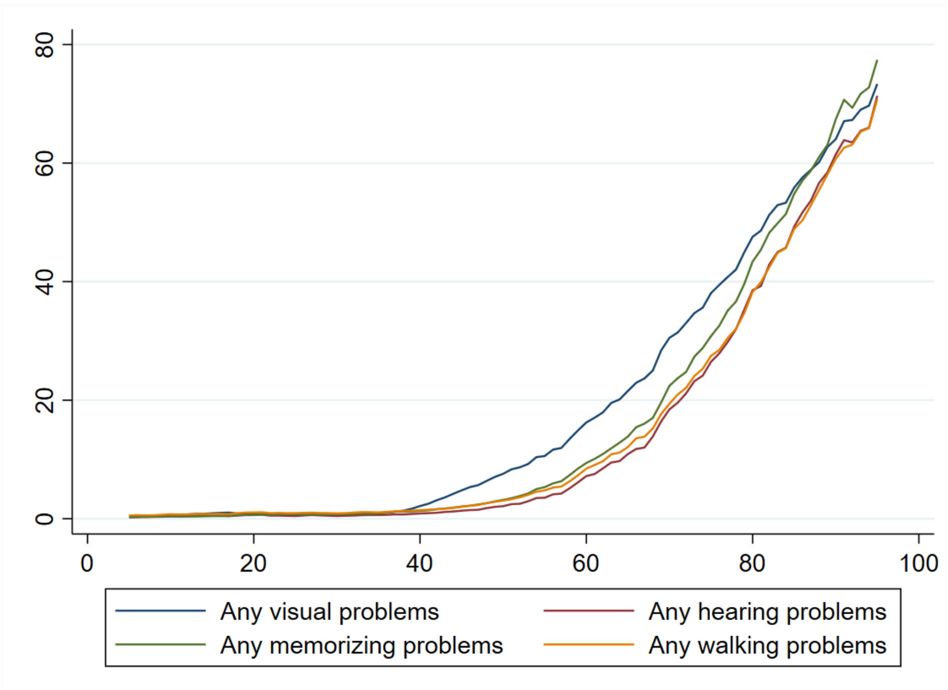


Table C1: Migration flows in South Vietnam in 1989, 1999 and 2009

	"Where were you five years ago?" (%)											
	Central Coast			Central Highland			Southeast			Mekong Delta		
	1989	1999	2009	1989	1999	2009	1989	1999	2009	1989	1999	2009
North Vietnam	1.63	0.71	0.69	9.34	5.41	2.22	4.33	3.34	3.06	0.80	0.15	0.11
Central Coast	97.79	98.67	98.77	2.44	2.00	0.72	0.65	0.86	1.41	0.08	0.04	0.04
Central Highland	0.19	0.26	0.24	87.06	91.77	96.41	0.14	0.20	0.49	0.01	0.01	0.01
Southeast	0.33	0.29	0.23	0.86	0.55	0.51	93.90	94.13	91.19	0.34	0.30	0.25
Mekong Delta	0.07	0.06	0.08	0.30	0.27	0.14	0.99	1.47	3.84	98.77	99.50	99.58

Note: The numbers are calculated based on the 1989, 1999 and 2009 Censuses of Population and Housing conducted by the General Statistics Office of Vietnam. Data are retrieved from Minnesota Population Center (2020)

Table C2: Impact of dioxin exposure on visual difficulties by birth cohorts

	Birth cohorts							
	1995-2004		1985-1994		1975-1984		1965-1974	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.007	0.023	0.006	0.027	0.019***	0.025	0.059***	0.087*
	(0.005)	(0.014)	(0.007)	(0.025)	(0.006)	(0.019)	(0.016)	(0.046)
Observations	1,046,344		1,030,283		977,967		912,541	
R-squared	0.002	0.002	0.003	0.003	0.002	0.002	0.008	0.008
	1955-1964		1945-1954		1935-1944		1925-1934	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.208***	0.247	0.277***	0.374*	0.329***	0.813***	0.248**	0.682**
	(0.054)	(0.156)	(0.073)	(0.215)	(0.094)	(0.288)	(0.118)	(0.328)
Observations	680,368		345,856		182,705		105,800	
R-squared	0.012	0.012	0.018	0.019	0.025	0.025	0.028	0.028
Simulated EOI	✗	✓	✗	✓	✗	✓	✗	✓
Province FE	✓	✓	✓	✓	✓	✓	✓	✓
Birth year FE	✓	✓	✓	✓	✓	✓	✓	✓

Robust standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table C3: Impact of dioxin exposure on hearing difficulties by birth cohorts

	Birth cohorts							
	1995-2004		1985-1994		1975-1984		1965-1974	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.003**	0.009*	0.002	0.009	0.005**	0.008	0.015***	0.004
	(0.002)	(0.005)	(0.002)	(0.006)	(0.002)	(0.007)	(0.005)	(0.013)
Observations	1,046,292		1,030,247		977,919		912,495	
R-squared	0.002	0.002	0.002	0.002	0.003	0.003	0.004	0.004
	1955-1964		1945-1954		1935-1944		1925-1934	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.042***	0.052*	0.125***	0.137*	0.187***	0.227	0.095	0.369
	(0.011)	(0.029)	(0.031)	(0.082)	(0.058)	(0.174)	(0.091)	(0.269)
Observations	680,323		345,825		182,686		105,789	
R-squared	0.006	0.006	0.013	0.013	0.023	0.023	0.03	0.03
Simulated EOI	✗	✓	✗	✓	✗	✓	✗	✓
Province FE	✓	✓	✓	✓	✓	✓	✓	✓
Birth year FE	✓	✓	✓	✓	✓	✓	✓	✓

Robust standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table C4: Impact of dioxin exposure on walking difficulties by birth cohorts

	Birth cohorts							
	1995-2004		1985-1994		1975-1984		1965-1974	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.004	0.004	0.001	0	0.005	0.009	0.028***	0.043**
	(0.003)	(0.008)	(0.002)	(0.008)	(0.004)	(0.011)	(0.008)	(0.022)
Observations	1,046,009		1,030,048		977,751		912,318	
R-squared	0.004	0.004	0.006	0.006	0.008	0.008	0.013	0.013
	1955-1964		1945-1954		1935-1944		1925-1934	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.090***	0.145**	0.180***	0.204*	0.244***	0.289	0.248**	0.548*
	(0.023)	(0.059)	(0.042)	(0.114)	(0.071)	(0.218)	(0.110)	(0.318)
Observations	680,193		345,765		182,656		105,764	
R-squared	0.011	0.011	0.013	0.013	0.021	0.021	0.03	0.03
Simulated EOI	✗	✓	✗	✓	✗	✓	✗	✓
Province FE	✓	✓	✓	✓	✓	✓	✓	✓
Birth year FE	✓	✓	✓	✓	✓	✓	✓	✓

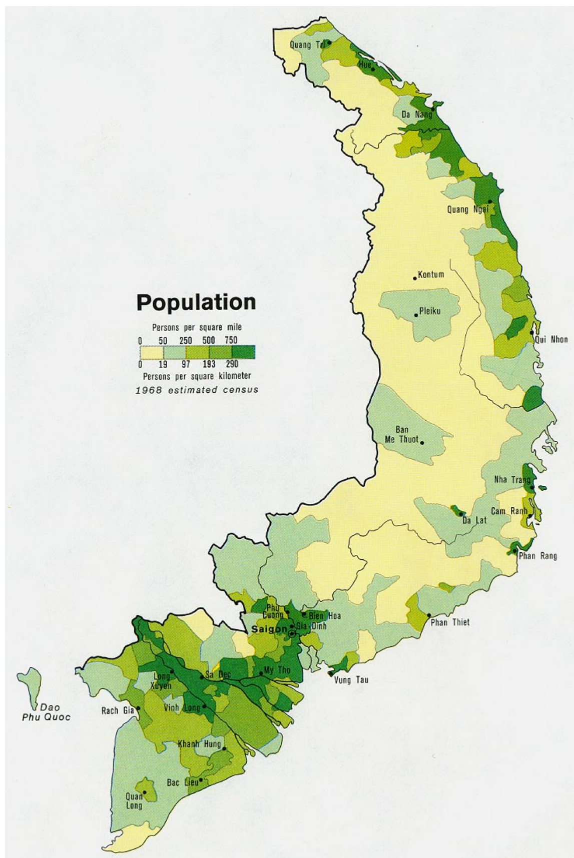
Robust standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table C5: Impact of dioxin exposure on memorizing difficulties by birth cohorts

	Birth cohorts							
	1995-2004		1985-1994		1975-1984		1965-1974	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.004**	0.006	0.005***	0.012*	0.008**	0.021**	0.026***	0.021
	(0.001)	(0.004)	(0.002)	(0.006)	(0.003)	(0.010)	(0.007)	(0.022)
Observations	1,046,288		1,030,240		977,933		912,507	
R-squared	0.001	0.001	0.002	0.002	0.004	0.004	0.007	0.007
	1955-1964		1945-1954		1935-1944		1925-1934	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EOI/100	0.058***	0.091**	0.151***	0.147	0.225***	0.214	0.182*	0.346
	(0.016)	(0.043)	(0.039)	(0.095)	(0.069)	(0.196)	(0.110)	(0.309)
Observations	680,328		345,828		182,690		105,785	
R-squared	0.008	0.008	0.012	0.012	0.023	0.023	0.034	0.034
Simulated EOI	✗	✓	✗	✓	✗	✓	✗	✓
Province FE	✓	✓	✓	✓	✓	✓	✓	✓
Birth year FE	✓	✓	✓	✓	✓	✓	✓	✓

Robust standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

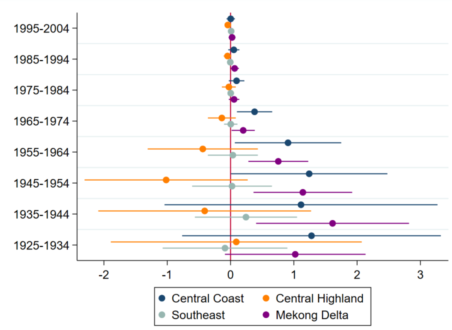
Figure C2: Population density in South Vietnam in 1968



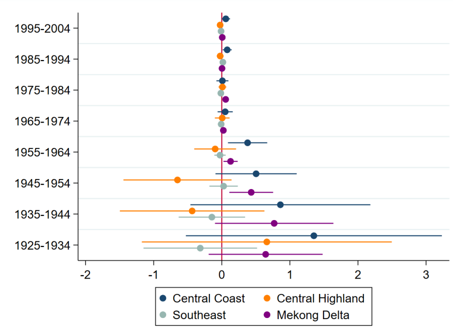
Note: The map was produced by the US Central Intelligence Agency. I retrieved it from Perry-Castaneda Library Map Collection, the University of Texas at Austin.

Figure C3: Estimation results on rural areas by regions, 2009 Census

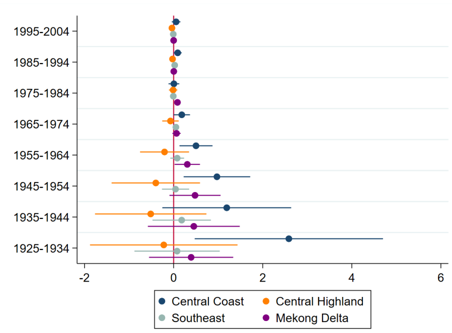
(a) Visual difficulties



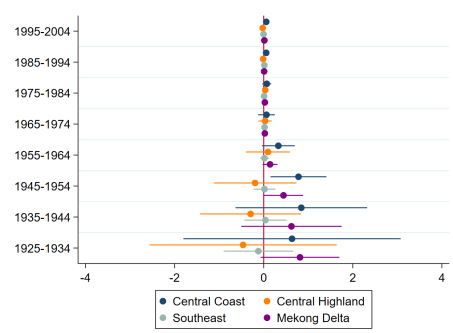
(b) Hearing difficulties



(c) Walking difficulties



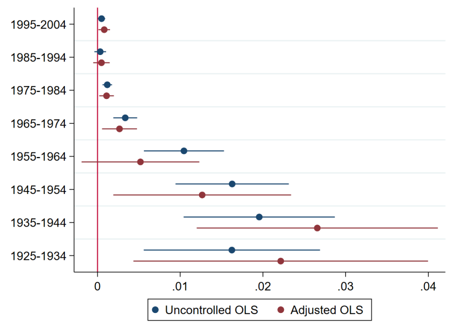
(d) Memorizing difficulties



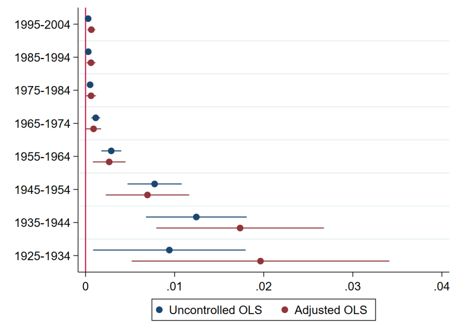
Note: Figure C3 compares the Adjusted OLS results between regions. The data is from the 2009 Census of Population and Housing. The sample only consists of 15% population living in rural areas below the 17th parallel that did not migrate within five years. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

Figure C4: Modified logarithm transformation of EOI, 2009 Census

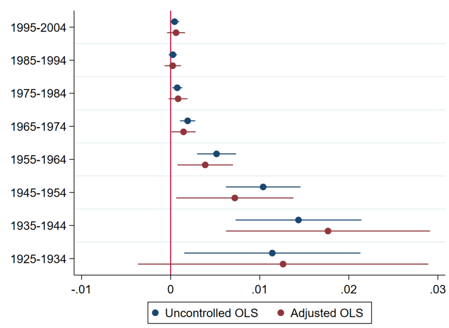
(a) Visual difficulties



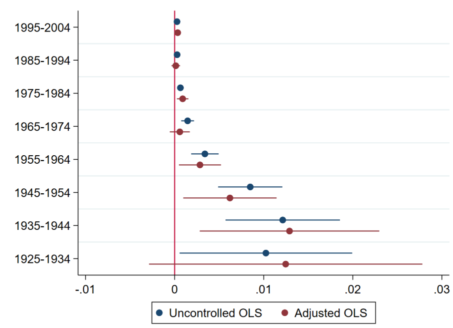
(b) Hearing difficulties



(c) Walking difficulties



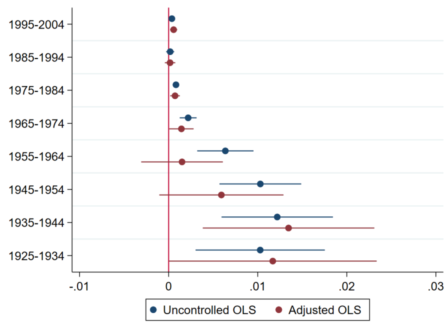
(d) Memorizing difficulties



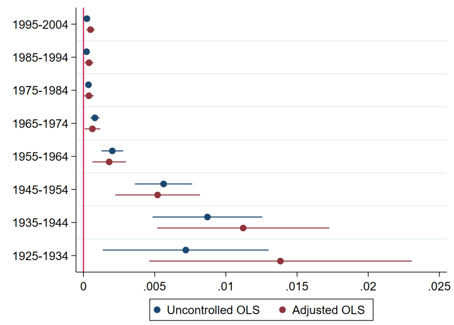
Note: Figure C4 compares the Adjusted OLS estimates with the modified logarithm transformation of the Exposure Opportunity Index by region. The data is from the 2009 Census of Population and Housing. The sample only consists of 15% population living in rural areas below the 17th parallel that did not migrate within five years. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

Figure C5: Inverse hyperbolic sine transformation of EOI, 2009 Census

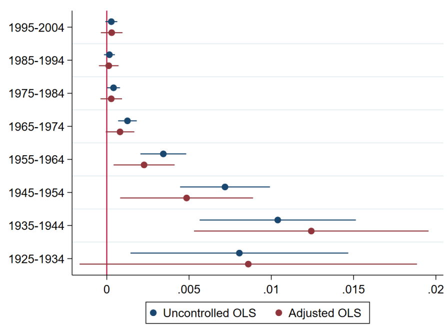
(a) Visual difficulties



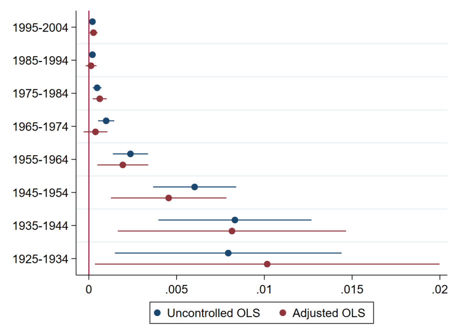
(b) Hearing difficulties



(c) Walking difficulties



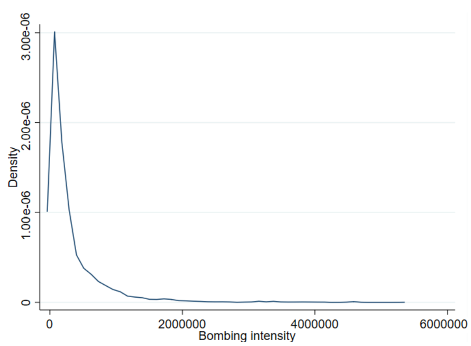
(d) Memorizing difficulties



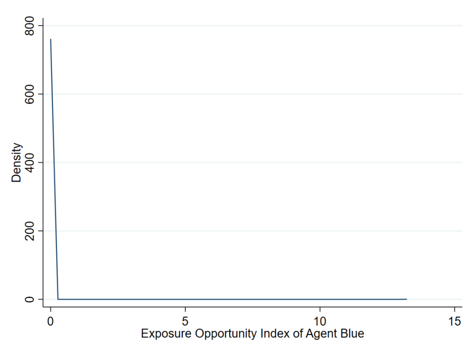
Note: Figure C5 compares the Adjusted OLS estimates with the inverse hyperbolic sine transformation of the Exposure Opportunity Index by region. The data is from the 2009 Census of Population and Housing. The sample only consists of 15% population living in rural areas below the 17th parallel that did not migrate within five years. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

Figure C6: Kernel density plots

(a) Bombing intensity



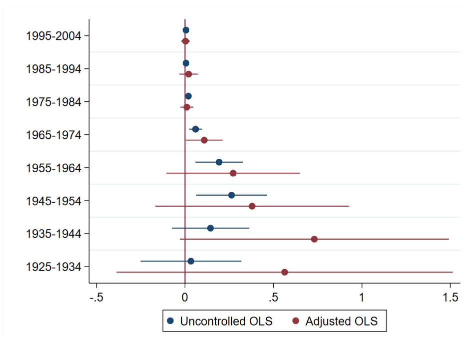
(b) Exposure to Agent Blue



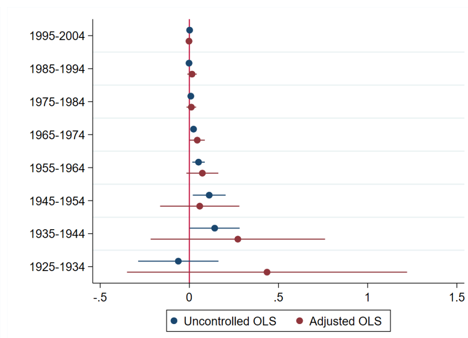
Note: Figure C6a plots the Kernel density of bombing intensity at the commune level. Bombing intensity is measured by the total weight of ordnance dropped within the 10-kilometer radius of commune centroids. Figure C6b plots the Kernel density of exposure to Agent Blue, which is measured by Exposure Opportunity Index.

Figure C7: Estimation results after controlling bombing intensity, 2009 Census

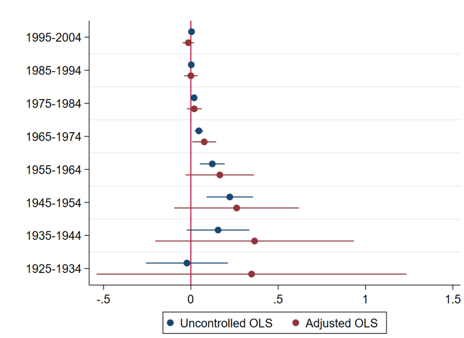
(a) Visual difficulties



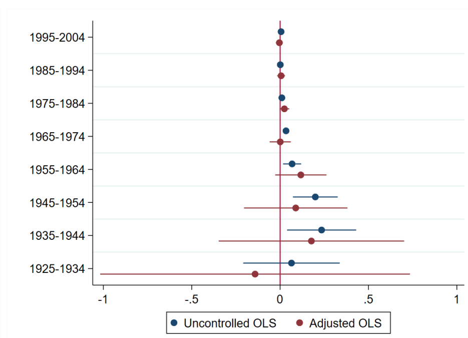
(b) Hearing difficulties



(c) Walking difficulties



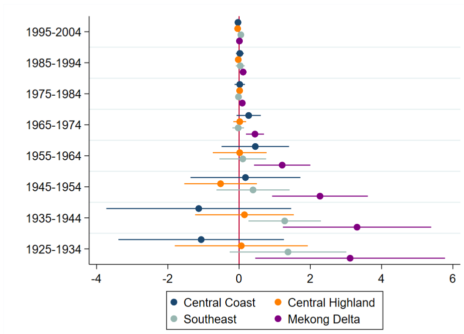
(d) Memorizing difficulties



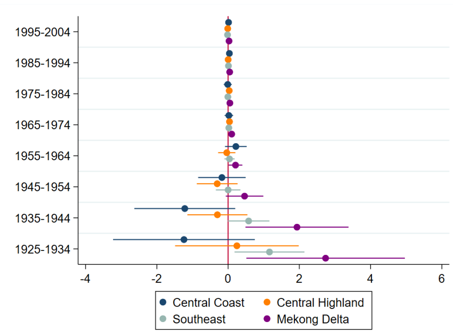
Note: Figure C7 plots the estimation results after controlling for bombing intensity. The data is from the 2009 Census of Population and Housing. The sample only consists of 15% population living in rural areas below the 17th parallel that did not migrate within five years. Since the spatial distribution of bombing intensity is not random, I only include communes that received less than 300,000 tons of bombs within their 10-kilometer radius. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

Figure C8: Estimation results by regions after controlling bombing intensity, 2009 Census

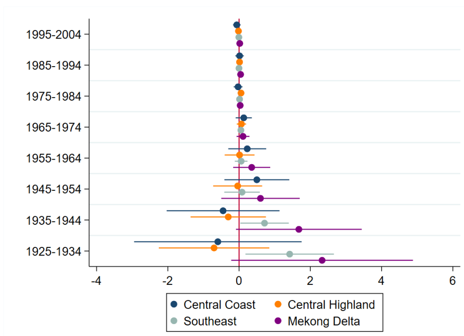
(a) Visual difficulties



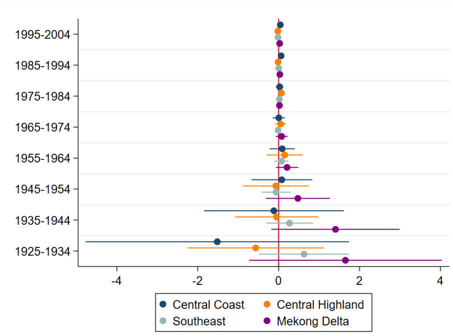
(b) Hearing difficulties



(c) Walking difficulties



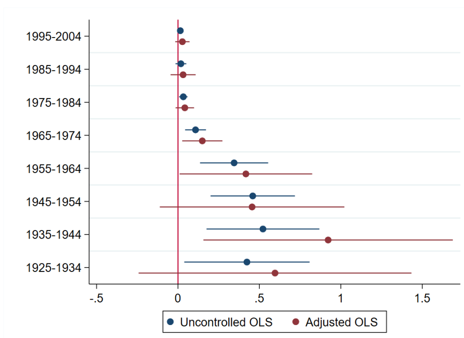
(d) Memorizing difficulties



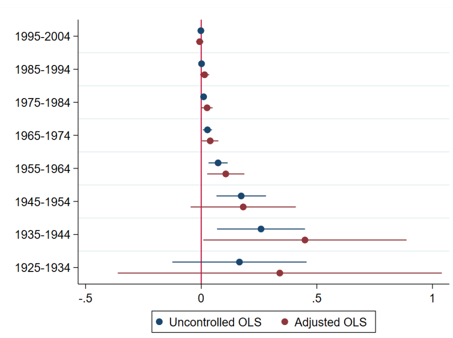
Note: Figure C8 plots the estimation results after controlling for bombing intensity by region. The data is from the 2009 Census of Population and Housing. The sample only consists of 15% population living in rural areas below the 17th parallel that did not migrate within five years. Since the spatial distribution of bombing intensity is not random, I only include communes that received less than 300,000 tons of bombs within their 10-kilometer radius. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

Figure C9: Estimation results after controlling Agent Blue exposure, 2009 Census

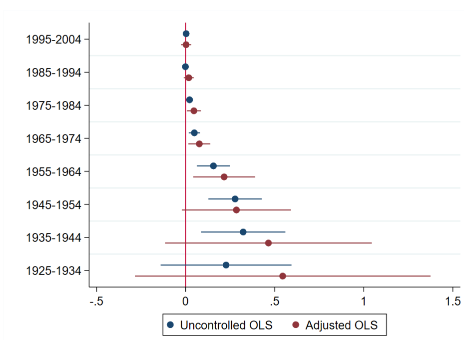
(a) Visual difficulties



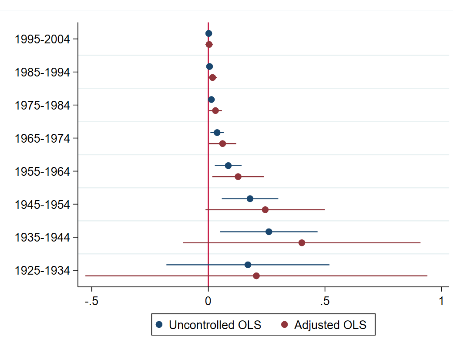
(b) Hearing difficulties



(c) Walking difficulties

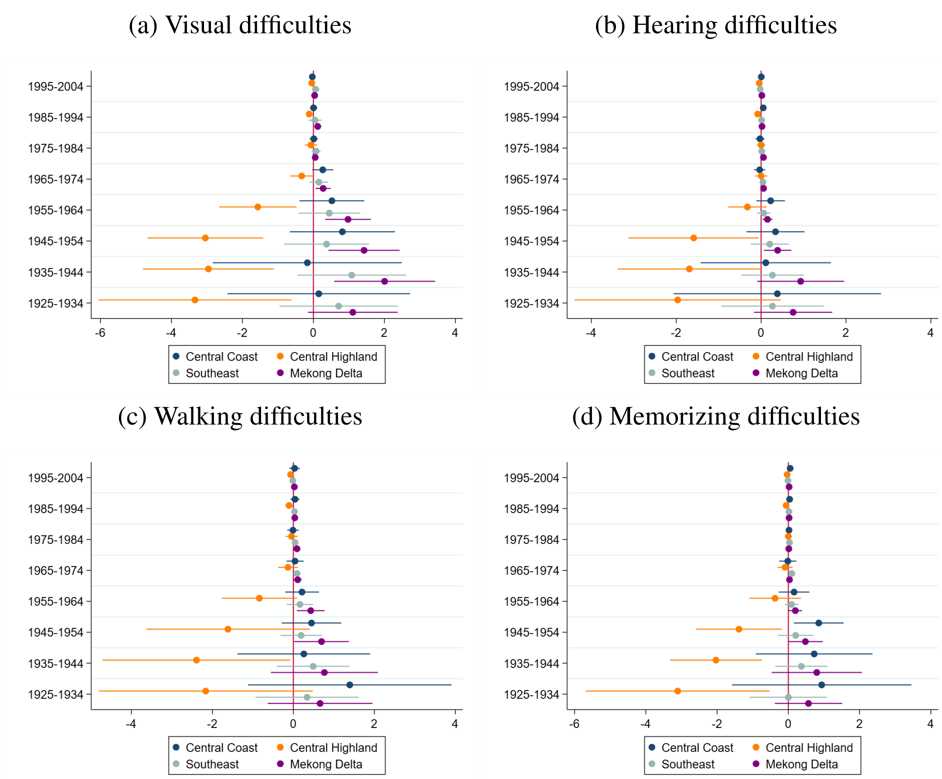


(d) Memorizing difficulties



Note: Figure C9 plots the estimation results with the sample with zero exposure to Agent Blue. The data is from the 2009 Census of Population and Housing. The sample only consists of 15% population living in rural areas below the 17th parallel that did not migrate within five years. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

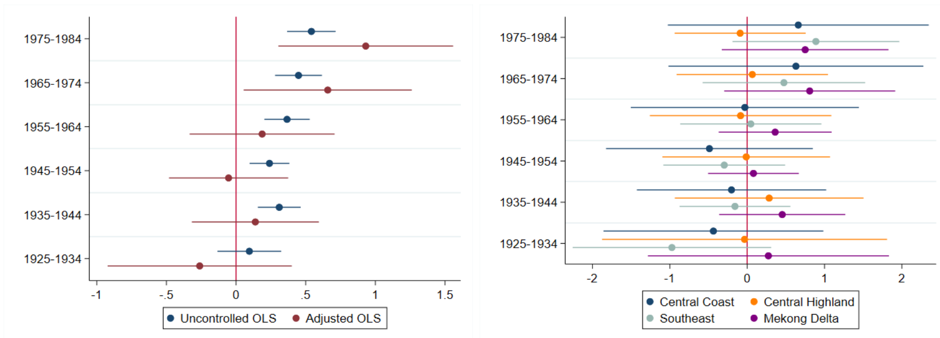
Figure C10: Estimation results by regions after controlling Agent Blue exposure, 2009 Census



Note: Figure C10 plots the estimation results after controlling for bombing intensity by region. The data is from the 2009 Census of Population and Housing. The sample only consists of 15% population living in rural areas below the 17th parallel that did not migrate within five years. Since the spatial distribution of bombing intensity is not random, I only include communes that received less than 300,000 tons of bombs within their 10-kilometer radius. The control variables are age, gender, marital status, ethnicity, religion, urban status, and province FE. Standard errors are clustered at the commune level.

Figure C11: Dioxin exposure and working in the agricultural sector

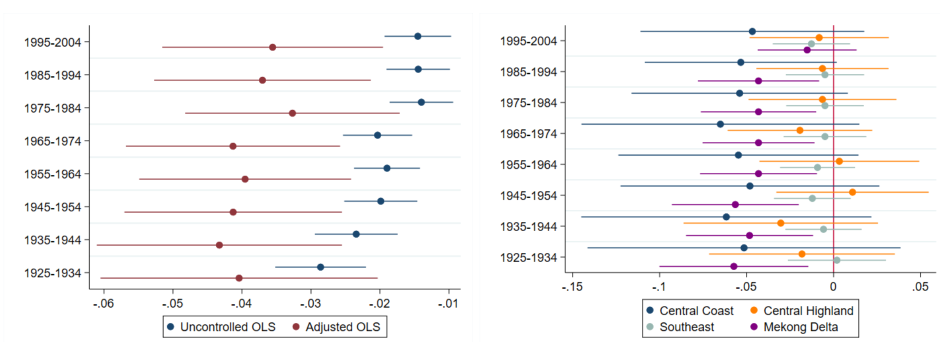
(a) Whole sample (b) By regions



Note: Figure C11 plots the impacts of one unit of the Exposure Opportunity Index of dioxin-containing herbicides on the chance of working in the agricultural sector (unit: percentage points). The samples consist of 15% of the population who were living below the 17th parallel and who had not migrate within five years previous to the survey. The control variables in Uncontrolled OLS are age, gender, marital status, ethnicity, religion, urban status, and province FE. Adjusted OLS control for these variables and simulated dioxin exposure. Regressions in Figure C11b are Adjusted OLS. Standard errors are clustered at the commune level.

Figure C12: Dioxin exposure and log of non-migrant population

(a) Whole sample (b) By regions



Note: Figure 11 plots the impacts of one unit of the Exposure Opportunity Index of dioxin-containing herbicides on the log of population. The samples consist of 15% of the population who were living below the 17th parallel and who has not migrated within five years previous to the survey. The control variables in Uncontrolled OLS are age, gender, marital status, ethnicity, religion, urban status, and province FE. Adjusted OLS control for these variables and simulated dioxin exposure. Regressions in Figure 11b are Adjusted OLS. Standard errors are clustered at the commune level.