# Pre-Analysis Plan

# Evaluating the Long-Run Effects of Water Fluoridation: Evidence from Community Water Fluoridation Programs

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December 3, 2020

Community water fluoridation has been named one of the 10 greatest public health achievements of the 20th century and led to vast improvements in dental health. Leveraging county level variation in the timing of fluoride adoption in a difference-in-differences design, I will estimate the causal effect of childhood fluoride exposure on long run health and labor market outcomes. I will do this using restricted census data linking individuals' adult outcomes to their county of birth.

This pre-analysis plan documents sources of original data, construction of variables, and the analysis plan. The analysis plan includes the specifications, outcomes, controls, robustness checks, and a discussion of the heterogeneity analysis that will be used in the study. At the time of writing, I have not created the analysis dataset and no part of the analysis has been conducted. I am pre-committing to this outline both to improve research transparency and to increase confidence that the results of this study do not reflect statistical noise hand-picked from repeated hypothesis testing.

# 1 Project Background

In the 1930's two dentists named Dr. Frederick McKay and Dr. G.V. Black discovered that exposure to fluoride in drinking water protects teeth against decay. Guided by robust supporting evidence from health and epidemiology literatures, community water systems in the U.S. commonly add low doses of fluoride to the public water supply to prevent tooth decay. The first U.S. city added fluoride to their public water supply in 1945 and today 73% of individuals using public water systems drink fluoridated water. Water fluoridation is especially relevant during childhood, as fluoride exposure during tooth development prior to tooth eruption has a large impact on susceptibility to decay (Singh, Spencer and Armfield, 2003). Despite improving trends in dental health in the U.S.,[1] tooth decay is still one of the most common chronic childhood diseases and one in four children below the poverty line have untreated tooth decay (Newacheck et al., 2000; Dye, Li and Thornton-Evans, 2012). Reducing childhood tooth decay alleviates pain and can result in fewer school absences and improved academic performance (Jackson et al., 2011). Because of its role in improving dental health in the U.S., the CDC named community water fluoridation as one of the 10 greatest public health achievements of the 20th century (Gooch, 2020).

The impact of fluoride on dental health varies based on both the amount and timing of fluoride exposure. The largest benefits occur when individuals are exposed to fluoride during the early stages of tooth development, which begins in utero and is completed by age 8. This childhood exposure directly affects the tooth structure making it more resistant to decay. Cases of tooth decay decrease as fluoride levels increase but the marginal benefits shrink at levels above 0.7 mg/L and plateau by 1.2 mg/L (Heller, Eklund and Burt, 1997). While early research found that fluoride exposure was most beneficial for children, additional studies have shown moderate benefits for adults as well (DHHS, 2015).[2] Despite these advantages, water

[1] The prevalence of any tooth decay in adult teeth among adolescents decreased from 90% in the 1960's to 60% by 2004 (DHHS, 2015)

[2] Adult fluoride exposure reduces the production of tooth-damaging acid by mouth bacteria and simultaneously fortifies teeth making them more resistant to acid. Some evidence suggests that the benefits of adult exposure are concentrated among individuals who were also exposed to water fluoridation during childhood

fluoridation continues to be a controversial topic worldwide and the vast majority of Europe does not fluoridate their water.[3]

While the majority of U.S. communities, scientists, and public health experts view low levels of fluoride as beneficial, we have little evidence of the long-run effects of fluoride beyond its impact on dental health. Glied and Neidell (2010) provide the best evidence in the U.S. context, leveraging variation in the timing of community water fluoridation programs to estimate long run labor market impacts of water fluoridation in the National Longitudinal Survey of Youth – 1979 (NLSY79). Unfortunately, the narrow group of birth cohorts in their sample (individuals born 1957-1964) provided insufficient variation for within county comparisons or a difference-in-differences analysis. Their results instead rely on the relatively strong assumption that all unobservable county characteristics affecting labor market outcomes are uncorrelated with fluoridation status. Perhaps due to the small sample size and limited identifying variation available among the NLSY79 cohorts, they find positive but insignificant effects in the full sample. The positive effects are driven by a statistically significant 4% increase in income among women, which the authors interpret as evidence of appearance based discrimination. The income point estimate for males is zero. In contrast, a more recent working paper leverages natural variation in fluoride levels in Sweden and finds positive effects on labor force participation and income, with larger effects for men (Aggeborn and Öhman, 2017). It is difficult to interpret whether differing results between

(Singh, Spencer and Armfield, 2003).

[3]Opponents of water fluoridation frequently express concerns about potential negative side effects. Exposure to fluoride during tooth development (in utero through age 8) increases the risk of dental fluorosis. The frequency and severity of dental fluorosis both increase with exposure level. Cases of mild fluorosis affect about 23% of people in the U.S. and are correlated with decreases in tooth decay (Beltrán-Aguilar, Barker and Dye, 2010). Mild fluorosis is characterized by lacy white markings on teeth but does not negatively impact dental health (DHHS, 2015). Severe cases of fluorosis are rare in the U.S., less than 1% according to Beltrán-Aguilar, Barker and Dye (2010), but can include pitting of the teeth that damages tooth structure. Risk of sever fluorosis increases at fluoride levels above 2mg/L. Fluoride exposure at levels above 4 mg/L (4x standard water fluoridation rates) can cause skeletal fluorosis resulting in weaker bones and joints, increased joint pain and risk of fractures (DHHS, 2015). A number of studies have also argued that fluoride may negatively impact IQ but these studies suffer from small sample sizes and are typically focused on populations exposed to extremely high fluoride levels (see Choi, Zhang and Grandjean (2012) for a review). There is no established biological channel connecting fluoride exposure to IQ and no evidence of negative I.Q. effects at common community water fluoridation levels.

these studies are driven by statistical noise, different time periods, different locations, or differences in the source of fluoride variation.[4] While the Swedish data provide significant precision and measurement advantages over the NSLY79, fluoride exposure is low; over 90% of Swedish observations were exposed to fluoride levels less than those typically added in the United States (.8-1.2 mg/L).[5] Childhood exposure to water fluoridation has the potential to influence long run outcomes, but its actual impact is unclear.

I will provide the first large sample evidence on the long run health and labor market effects of water fluoridation programs. The samples available in restricted Census data sets will allow for vast increases in precision; a much weaker identifying assumption via inclusion of birth county fixed effects; expansions in the set of outcomes, birth cohorts, and fluoride variation studied; detailed heterogeneity analysis, and exploration of mechanism. The Census and American Community Survey (ACS) will support analytical samples of millions (versus thousands in the NLSY79).[6] I will provide the first causal estimates of the impact of childhood exposure on health and mortality for adults aged 30 and older, while also mapping out impacts on labor market outcomes across the life course for several decades of birth cohorts.

## 2 Data

The primary data source is restricted individual-level U.S. Census, American Community Survey (ACS), and Current Population Survey (CPS) data linked to the NUMIDENT file (U.S. birth and death records), housed in the Census Research Data Centers. This includes ACS years 2001-2016. The NUMIDENT file contains each individual's date and location of birth as well as date of death for those who are deceased. Water fluoridation

[4] Aggeborn and Öhman (2017) also estimates effects on cognitive ability and health, finding no effect on either.

[5] Sweden also has a meaningfully different health care environment than the United States. Currently, Swedish dental care is provided by the government for everyone under the age of 24 and is subsidized at all other ages.

[6] As is shown in Appendix C of Anders, Barr and Smith (2019), this type of large sample administrative data reduces the likelihood that statistically significant results are false positives, improves precision, and reduces publication bias.

data comes from the 1992 Fluoride Census (a public record provided by the CDC).[7,8] The locations from both the NUMIDENT file and fluoridation records are recorded as strings at the city or county level. These locations are matched to their county level FIPS codes.

My analysis sample is limited to all individuals born in a U.S. county that was included in the 1992 fluoridation census and successfully linked to its county FIPS code. The sample will be restricted to include individuals of prime working age (between 25-54) at the time of the survey. For computational ease, I will collapse the data to birth-year X birth-county X survey-year level separated by both gender and race. Each cell will be weighted by the number of observations in that cell.

**Defining Fluoride Exposure:** I will define treatment for each birth-county by birth-year cohort as the fraction of life exposed to community water fluoridation from conception through age 8.[9,10] By this definition, a fully treated county-birth-cohort would have been exposed to fluoride for 104 months of childhood (9 months in utero + 12 months * 8 years)./footnoteBecause counties may have multiple public water systems with different water fluoridation policies and because some households source drinking water from private wells not all individuals in a treated county will drink fluoridated water. This definition fails to account for the resulting variation in the fraction of each county drinking fluoridated water. Section 4.3 outlines alternative treatment definitions and specifications that allow for heterogenous effects by the percent of each county exposed to fluoridated water. Additional

[7]Matthew Neidell has also generously provided the cleaned version of the 1992 Fluoride Census used in Glied and Neidell (2010).

[8]Via a Freedom of Information Act request to the CDC, I have obtained current natural fluoride levels for each community water system. I expect these levels to be relatively stable over time and may use this data in robustness checks or other ancillary analysis.

[9]This treatment definition is consistent with other early childhood interventions where years of exposure is the most relevant parameter. Specifically, both Hoynes et al. (2016) and Barr and Smith (2019) use the fraction of early childhood with access to food stamps in order to estimate long run effects in a difference-in-differences setting.

[10]This definition intentionally does not account for differences in fluoridation levels. Fluoride levels were determined at the local level but CDC guidelines adjusted recommended rates relative to average local temperatures which may affect rates of water evaporation and consumption. Because of this, variation in fluoride level between .8-1.2 may not reflect actual increases in individual fluoride intake but simply the difference in the level of fluoride communities were adding in order to reach the same expected level of fluoride exposure.

event studies exploring the potential for non-linear treatment affects by age at first exposure will also be explored, as described in section 3.

**Outcome Variables:** The purpose of this research is to identify the net labor market and health effects of community water fluoridation. Using a construction similar to Bailey et al. (2020), I will examine two indices that best capture these outcomes in the ACS: (i) economic self-sufficiency, and (ii) physical ability and health. These indices average across standardized component variables, reversing signs when necessary such that a more positive value implies a better outcome. The Economic Self-Sufficiency Index includes variables indicating whether or not an individual was in the labor force, worked last year, weeks worked last year, usual hours worked per week, labor income, other income not from public sources, income-to-poverty ratio, not in poverty, reverse coded income from welfare, and supplemental security. The Physical Ability and Health Index includes reverse coded information on the presence of a work disability, an ambulatory difficulty, a cognitive difficulty, an independent living difficulty, a vision or hearing difficulty, and a self-care difficulty.

The index approach alleviates concerns about multiple hypothesis testing and improves statistical power (Kling, Liebman and Katz, 2007). To obtain results more directly comparable to the existing literature, I will directly estimate the effects on labor income, mortality (from NUMIDENT) and self-reported health status (in the CPS). Additional variables relating to assortative mating, educational attainment, career choice, incarceration, and mortality will be examined as supplementary analyses as explained in section 4.3 to explore mechanisms and the broader effects of water fluoridation.[11] The next section outlines the details of my analytical approach.

[11] Given the structure of the data, it may also be possible to link individuals to the childhood fluoride exposure of their parents. If this proves feasible, I may also include estimates on second generation outcomes using the same strategy as for the first generation.

## 3 Proposed Analyses

I will use a difference-in-differences strategy leveraging the staggered adoption of community water fluoridation across the United States. This design compares outcomes of county-birth-cohorts with exposure to fluoridated water those without any, while controlling for county and year of birth. This strategy does not rely on the exogeneity of fluoride levels conditional on observables, but on the weaker assumption that the shift in health and labor market outcomes of untreated individuals across time effectively proxies for the shift in outcomes that would have occurred for individuals drinking fluoridated water in the absence of fluoride treatment. The basic reduced form difference-in-differences specification is as follows:

$$Y_{cbt} = \theta_c + \delta_{s(c)b} + \mu X_{cbt} + \beta(Exp8)_{cb} + \epsilon_{cbt} \tag{1}$$

In this specification, $(Exp8)_{cb}$ represents a county-birth cohort's cumulative exposure to fluoridated water during childhood. The coefficient of interest is $\beta$ and, conditional on the identifying assumption, can be interpreted as the causal effect of childhood fluoride exposure. The long run health and labor market outcomes are represented by $Y_{cbt}$; while $\theta_c$ and $\delta_{s(c)b}$ respectively represent birth county and state-by-birth-cohort fixed effects; and $X_{cbt}$ contains a vector of covariates including sex, age, age squared, and race. Although not necessary for identification, the controls for sex, age, and race are included to increase precision. All specifications will cluster standard errors at the county of birth level.

To explore how fluoride exposure affects children of different ages, I will estimate an additional specification where $(Exp8)$, the cumulative exposure measure, is replaced with a set of timing variables indicating the first year of water fluoridation relative to a person's birth. This dynamic difference-in-difference specification is as follows:

$$Y_{cbt} = \theta_c + \delta_{s(c)b} + \mu X_{cbt} + \sum_{a=-5\,[a\neq 8]}^{15} \beta_a P_c * 1[Fl - b = a] + \epsilon_{cbt} \tag{2}$$

In this specification, $Fl$ and $b$ represent the first year that an individual's birth county fluoridated their water and that individual's birth year. The timing variable $a$ represents each individual's age in the first year of water fluoridation and covers the period from 5 years before birth through age 15 with age 8 as the omitted year.[12] The dynamic treatment effects are captured in $\beta_a$ and represent the effect of receiving fluoridated public water beginning at age $a$. All other terms are equivalent to those in equation 1.

Given that water fluoridation prior to tooth eruption makes teeth permanently more resistant to decay, I expect the largest effect to be for individuals whose counties adopted water fluoridation prior to their birth, with similar effects regardless of how many years before birth the water fluoridation began. Initial exposure occurring later in childhood should result in reduced effect sizes, as fewer teeth are exposed during formation, with much smaller benefits of exposure after the age of 8.[13] This pattern of expected results is shown in Figure 1.

## 4 Ancillary Analysis

This section outlines some of the additional tests and analyses I will perform to explore the internal and external validity of the results as well as the policy implications. First, I describe alternative specifications that will account for county level variation in treatment intensity. Second, I describe potential threats to internal validity and how I will test for evidence of these threats. Third, I provide motivation for and a description of the heterogeneity analyses that I plan to conduct to explore how effects differ by subgroup. I conclude with a brief discussion of the potential mechanisms and some initial thoughts on how I will test for them.

[12] Those first exposed to fluoride outside of this range will be binned in either $a = -5$ if the county-birth-cohort was born more than 5 years after initial water fluoridation or $a = 15$ of they were older than 15 when fluoride was first adopted in their county.

[13] It is worth noting that only county of birth is observed, not counties of residence throughout childhood. The likelihood of an individual residing in their birth county decreases over time, so estimates will be attenuated toward zero when estimating the effect of exposure in later years.

## 4.1 Variation in Treatment Intensity

The county-birth-cohort specifications described in Section 3 fail to account for heterogeneity in the fraction of each county exposed to fluoride. Because counties may have multiple public water systems with different water fluoridation policies, and because some households source drinking water from private wells, not all individuals in a treated county will drink fluoridated water. This means that my main estimates represent the average treatment effect in county-birth-cohorts where anyone is exposed to fluoride which will understate the true effect of fluoride exposure at the individual level. Because treatment intensity is so closely tied to understanding the individual level impacts of fluoride exposure, I will present results from several methods intended to account for this variation.

The most direct route available is to incorporate county level treatment intensity into the treatment definition. I will alternative results from both Equation 1 and 2 where the treatment variables ($Exp8_{cb}$ and $P_c$ respectively) are scaled by the fraction of the county receiving fluoridated public water during the treatment period. Unfortunately, this method may introduce bias if counties with different treatment intensities were on different outcome trajectories prior to treatment that are unrelated to fluoride exposure. For example, this may be the case if urban counties, where private well use is less common, have a higher percentage of individuals drinking fluoridated water and also income and employment trends that are improving faster than those in rural areas. To account for this, I will also present results from otherwise identical specifications that add related county-level controls interacted with a time trend to capture variation caused by any differences in the type of counties with high or low treatment intensity. These county level control variables will include the percent of the county that is urban, the percent of the county with private wells, and the number of public water supply plants within the county.

As a second method to account for heterogeneity in treatment intensity, I will create bins of treated counties that have a similar fraction of their population drinking fluoridated water and estimate effects separately by these treatment intensity bins. I expect coefficient

estimates to be the largest for counties with full exposure to fluoridated water and expect these counties to best represent the effect of fluoride on treated individuals. The final approach I will take is to estimate effects in a triple differences framework, interacting my treatment definition with the percent of each county receiving fluoride. This approach has the advantage of allowing heterogenous effects by treatment intensity without constraining the model by directly incorporating them into the treatment variable.

## 4.2 Testing Identifying Assumption

The key identifying assumption is that, conditional on birth cohort and county fixed effects, the non-fluoride factors that influence an individuals' long run health and labor market outcomes are orthogonal to the presence or level of community water fluoridation in their county of birth at a particular age. This means that, conditional on birth cohort and county fixed effects, any difference in outcomes among those exposed to fluoridated water is the result of the fluoride itself and not any other factor. In this setting, the main assumption is that the shift in health and labor market outcomes of untreated individuals across time effectively proxies for the shift in outcomes that would have occurred for individuals drinking fluoridated water in the absence of fluoride treatment. It is impossible to observe the counterfactual outcomes of individuals exposed to water fluoridation, but I will conduct several tests to explore how reasonable this assumption is.

First, as shown in Equation 2, I will estimate dynamic effects relative to an birth-cohort's age at the time of initial water fluoridation in their county. If treatment and control groups have differential trends unrelated to fluoride treatment, then we would expect individuals born after the beginning of water fluoridation to continue to trend apart despite the fact that water fluoridation is not changing for these groups. On the other hand, consistently sized effects for these birth cohorts would provide suggestive evidence that the identifying assumption holds. Additionally, because treatment is likely to have the strongest effect on children age 0-8, smaller treatment effect size at older ages is consistent with differences being driven by water fluoridation rather than some other factor. This expected pattern of

results is shown in Figure 1.

My identifying assumption might also fail if there are meaningful shifts in the composition of people being born into treated and untreated counties across the sample period. This could occur if demographic shifts between counties happened simultaneous to water fluoridation or if individuals migrate between counties in response to water fluoridation. Aggeborn and Öhman (2017) suggest that migration in response to water fluoridation is unlikely because fluoride in water is colorless, odorless, and tasteless, meaning that changes in water fluoridation are not salient to the affected populations. Additionally, decisions regarding water fluoridation are frequently made with little or no input from local residents, making it even more unlikely that water fluoridation levels are salient enough to drive migration across counties.[14] It is however still possible that migration patterns happened to coincide with water fluoridation. I will test this by estimating the effect of water fluoridation on the racial composition of births. Although county composition could change in ways not reflected in race, the lack of any changes in racial composition would support the plausibility of my identifying assumption.

Another potential concern is misspecification. Because my primary analysis uses a difference-in-differences design with staggered treatment timing, my estimates only represent the average treatment effect of water fluoridation under the implicit assumption of consistent effect sizes across cohorts. If the effect of water fluoridation changes over time, then counties that were early adopters of water fluoridation are not valid controls for later adopters. If early adopters are used as controls, any changes in fluoride effects across time would influence the difference-in-differences estimates and bias the estimated average treatment effect (Goodman-Bacon, 2018). This is of particular concern in this setting, where a wide range of birth cohorts are being studied and several potential reasons for heterogenous cohort effects exist.[15] To explore the likelihood of this type of misspecification, I will decompose my main

[14] While some referendums were held allowing individuals to vote on community water fluoridation, roughly two-thirds of early water fluoridation decisions were made by government administrators without citizen input (Crain, Katz and Rosenthal, 1969).

[15] Potential drivers of heterogenous cohort effects could include increased exposure to fluoride from other

difference-in-difference estimates following Goodman-Bacon (2018) specifically checking for the prevalence of negatively weighted 2x2 estimations, an indicator of heterogenous cohort effects. If the decomposition suggests that heterogenous cohort effects are present, I will re-estimate results using a stacked difference-in-difference approach following Cengiz et al. (2019). This stacked design prevents early fluoride adopting counties (which may be contaminated by time varying treatment effects) from acting as controls for counties that adopted fluoride later. This method effectively estimates the unbiased average treatment effect even in the presence of heterogeneous cohort treatment affects.[16] If the evidence suggests that heterogenous treatment effects are present, this stacked difference-in-differences design will become my preferred specification.

## 4.3 Heterogeneity Analysis and Mechanism

Because exposure to water fluoridation was determined at the local level and was not a targeted intervention based on family income or other demographic characteristics, a wide variety of people were affected. While the main estimation results represent the average impact of water fluoridation, I will also explore how effects vary across population subgroups, including socioeconomic status, race, age at exposure, and gender. The large sample available in the ACS will allow me to obtain relatively precisely estimates of treatment effects even while exploring subgroup heterogeneity. For each potential source of heterogeneity I will estimate overall differences as well as dynamic effects. This heterogeneity analysis will also help disentangle the mechanism through which dental health affects long run outcomes.

There are two likely channels through which dental health could improve long run outcomes. First, dental health is an aspect of overall health and individuals with poor health (dental or otherwise) can be expected to miss days of school or work, have decreased productivity due to pain or discomfort, and face increased financial burden and stress from

sources, like the 1970's popularization of commercial fluoride toothpaste and other products, changing access to dental care over time, or generational differences in the importance of dental health.

[16] I will also present effect sizes by cohort. To the degree that treatment effects vary over time, effects for more recent birth cohorts are the most policy relevant, as their childhood sources of fluoride exposure will be more similar to those of children today.

associated health care costs. Second, dental health is also a readily visible component of individual appearance and has been shown to be an important factor in individual attractiveness (Eli, Bar-Tat and Kostovetzki, 2001). Overall attractiveness is positively correlated with income (Mobius and Rosenblat, 2006), suggesting that improving oral health may result in an increased beauty premium for treated individuals.

The results of the dynamic event studies described in Equation 2 will allow me to disentangle whether or not effects are likely to be driven by the health or beauty mechanisms. Because teeth that are visible components of a smile develop earlier, beauty effects should result in larger impacts of exposure at younger ages while I would expect general health effects to be more linear across time (with approximately equal effects on visible and hidden teeth). A set of potential dynamic effects are outlined in Figure 1.

I will also explore differences in the effect of water fluoridation between men and women. If beauty is the primary channel, then I would expect there to be smaller impacts for men because women have traditionally been held more harshly to beauty norms and may face more appearance-based discrimination than men in the labor market.[17] I will also estimate dynamic results from Equation 2 separately by gender. Because I expect beauty effects to be relatively large for women and for children exposed in early childhood, I would expect to see the largest beauty driven labor market benefits for women exposed to fluoride at young ages and the smallest effects for men exposed during late childhood. However, if effects sizes are consistent across gender and tooth visibility, that would support general improvements in health and productivity as the primary mechanism.

Whether driven by improved health or beauty, improvement in an individual's characteristics also has the potential to impact their marriage market outcomes through assortative mating. I will test for assortative mating impacts by estimating the effect of fluoride exposure on the health and labor market outcomes of partners, which has not previously been

[17]It may be worth noting that beauty effects do not necessarily indicate labor market discrimination, as changes in appearance may also affect individual confidence and subsequent behavior separate from any appearance-based discrimination on the part of employers or others.

explored in the literature.[18] If childhood exposure to water fluoridation does impact partner characteristics, the breakdown of dynamic effects on assortative mating from Equation 2 as well as the effect on men vs. women will again shed light on the primary mechanism behind changing partner characteristics. As before, I expect impacts of beauty to have larger effects on women and early childhood exposure while general health effects are likely to be more consistent across gender and years of water fluoridation during childhood.

Separate from any attempt to understand the mechanisms involved, I will also explore heterogeneity by socioeconomic status. The goal of water fluoridation is to improve dental health. We can expect these improvements in dental health will be largest for individuals and groups who had the worst dental health prior to treatment. Because individuals with high incomes and socioeconomic status are more likely to have access to dental care, both preventative care and treatment, water fluoridation for this group is less likely to impact severe tooth decay or visible elements of oral health. On the other hand, minor dental issues are more likely go untreated and progress into tooth decay or tooth loss for low socioeconomic status individuals. While no one dimension perfectly captures socioeconomic status, I will explore differential effects by race, and birth county characteristics of median income and education level.

[18] Glied and Neidell (2010) mention in a footnote that they found no effect of fluoride on marriage markets, but they were likely underpowered to detect some reasonable effects.

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# 5 Figures

Figure 1