Essays on Dairy Farm Productivity, Tax Policy, and Health Outcomes

by

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To my loved ones.

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ABSTRACT

Technology and policies serve as two effective tools for enhancing human production and well-being over time. Technological advancements contribute to increasing productivity and enrich the material consumption of individuals. Governments utilize policies aimed at influencing human behavior to improve social welfare surplus. Understanding the impacts of technology and policies on various aspects of human society empowers policymakers and individuals to make informed decisions. Through studying these effects, policymakers and individuals can develop strategies to maximize benefits and mitigate negative consequences.

In the three chapters of my dissertation, I investigated genetic improvements in dairy breeding animals and productivity growth in Wisconsin dairy farms. I explored the long-term effects of in utero cigarette tax exposure on adult health. Finally, I examined the long-term health and behavioral effects of early childhood exposure to childcare subsidy programs.

In the first chapter, I examine the relationship between investments in animal breeding and productivity growth on Wisconsin dairy farms using a control function approach. I incorporate farm-level annual investment in breeding and genetics into the law of motion of productivity as in De Loecker (2013) to test the relationship between these investments and realized productivity. Our unique dataset also allows us to look at the effect of choosing bulls with high milk yield potential on productivity. Our results indicate that breeding investments made three years prior are associated with higher productivity of the current cohort. However, the farms with the highest level of productivity reap the lowest benefits from breeding investments, suggesting that there are diminishing returns to investing in genetics. When milk output is not quality-adjusted, the contribution of breeding to productivity is undetectable, suggesting that breeding and investments in milk quality are related. I conclude that investments in breeding and genetics significantly contribute to dairy farm productivity, especially in terms of milk quality.

In the second chapter, I explore the long-term effect of in-utero cigarette tax exposure. Cigarette taxes have been shown to reduce maternal smoking and enhance birth outcomes. However, it is still uncertain whether these effects persist into adulthood. This study investigates the effects on adult health outcomes of exposure to higher cigarette taxes while in utero. Utilizing a generalized difference-in-difference methodology and analyzing a rich dataset spanning births from 1968 to 1994, I find that a 10-cent higher cigarette tax while individuals were in utero leads to a significant 1.8 percentage point reduction in the likelihood that the treated individuals (evaluated at ages 25 to 35) ever experienced health conditions such as asthma, lung disease, heart disease, or heart attacks. The examination of mechanisms underscores pathways through parental smoking behavior during pregnancy, birth outcomes, childhood health, smoking behavior in adolescence and adulthood, cognitive ability, educational attainment, and age of first childbirth for treated individuals. The study contributes to the burgeoning literature on early-life determinants of health and enriches our understanding of the complex interplay between cigarette policies and long-term health, with implications for policymakers and public health interventions.

In the third chapter, I investigate the health impacts of early-life exposure to Child and Dependent Care Tax Credits (CDCTC). This research leverages variation in state generosity regarding the CDCTC to explore how the accumulated exposure in the first four years of life affects children's health. I suggest that the CDCTC may influence child health through both income and substitution effects, as it boosts household income and encourages the substitution of parental childcare with purchased child care services. Our findings reveal intriguing patterns: children born to mothers with less than high school education reap benefits from CDCTC exposure, whereas those born to mothers with more than high school education experience poorer health outcomes. This discrepancy is attributed to the lower quality of purchased child care services compared to the higher quality of maternal childcare provided by highly educated mothers. The findings from our research on Child and Dependent Care Tax Credits have direct policy relevance. They challenge conventional

wisdom on the utility of such subsidies, providing policymakers with nuanced insights that can inform future reforms.

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1.1 Introduction

Milk yield has experienced significant growth in the past decades. Figure 1.1 charts the growth path of average cow milk yield from 2003 to 2019, which grew on average 1.4% a year during this period. At the same time, the productivity of dairy bulls available on the market has grown at a higher rate: milk yield, butterfat yield, and protein yield of dairy bulls grew on average 3.3%, 5.1%, and 4% per year over the same period. When combined into a genetic production index,[1] its growth path is similar to that of milk yield. The dairy science literature attributes as much as 50% of the growth in milk yield in the past decades to genetic improvement in dairy cows (Pryce and Veerkamp 2001; Shook 2006; Van Raden 2004). While its precise contribution is difficult to quantify, it is clear that genetic improvement is an essential vector of productivity growth in dairy.

Choosing genetics is critical for productivity improvement of a dairy herd, and yet investments in herd genetics has been largely ignored when estimating dairy farm production functions (e.g., Jang and Du 2019; Mukherjee et al. 2013; Njuki et al. 2020). By omitting this decision, conventional production function estimation may misattribute this productivity growth to other input factors, which results in biased input coefficients (De Loecker 2013). Moreover, not understanding this vital vector of productivity growth in dairy farming makes it difficult to understand the effect of new genetic improvements on the future of

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[1]This index is a weighted average of milk production, fat production, and protein production traits of dairy bulls for sale. The precise weights are determined by Principal Component Analysis, the details of which are found in the supplementary Appendix A.

the dairy industry.

This paper investigates the effect of genetics investment on dairy farm productivity in Wisconsin. Dairy farms choose bull genetics using artificial insemination each year in order to change the future productivity of the herd. These investments are reflected in both how much farms spend on breeding each year and the genetic indices of the bulls they choose in each year. In this paper, we leverage both of these measures to better understand how investments in genetic improvement impact the evolution of productivity on dairy farms.

Using two rich data sources on observed farm- and animal-level decisions, we integrate investment in genetic improvements into the productivity dynamics, which is modeled by a first-order Markov process in the style of Levinsohn and Petrin (2003) and De Loecker (2013). Since investments in genetics impact productivity three years from the date of investment, variation in breeding investment helps identify the parameters in the farm production function. Using this method, we analyze both the relationship of genetic investment to productivity growth as well as the extent to which omitting investment in genetics biases estimated factor shares in the production function.

We find that both breeding expenditures and the production potential of the genetics farmers choose are significant factors in the dynamic evolution of productivity. In our sample of Wisconsin dairy farms, farmers with higher breeding expenditures three years prior had higher productivity, as did farms that chose bulls with a higher genetic production index. We also find that dairy farms with high productivity reap the smallest gains from increasing investment in high-yield genetics, suggesting diminishing returns to investment in genetics. The magnitude of the relationship between breeding investments and productivity depends on whether the output we use is adjusted for milk quality, which suggests that breeding investments and milk quality improvements are related. In addition to providing evidence on how genetics impact productivity, our analysis demonstrates the importance of adjusting for output quality when analyzing dairy farm performance.

Our work expands on the productivity literature concerning dairy by modeling breeding

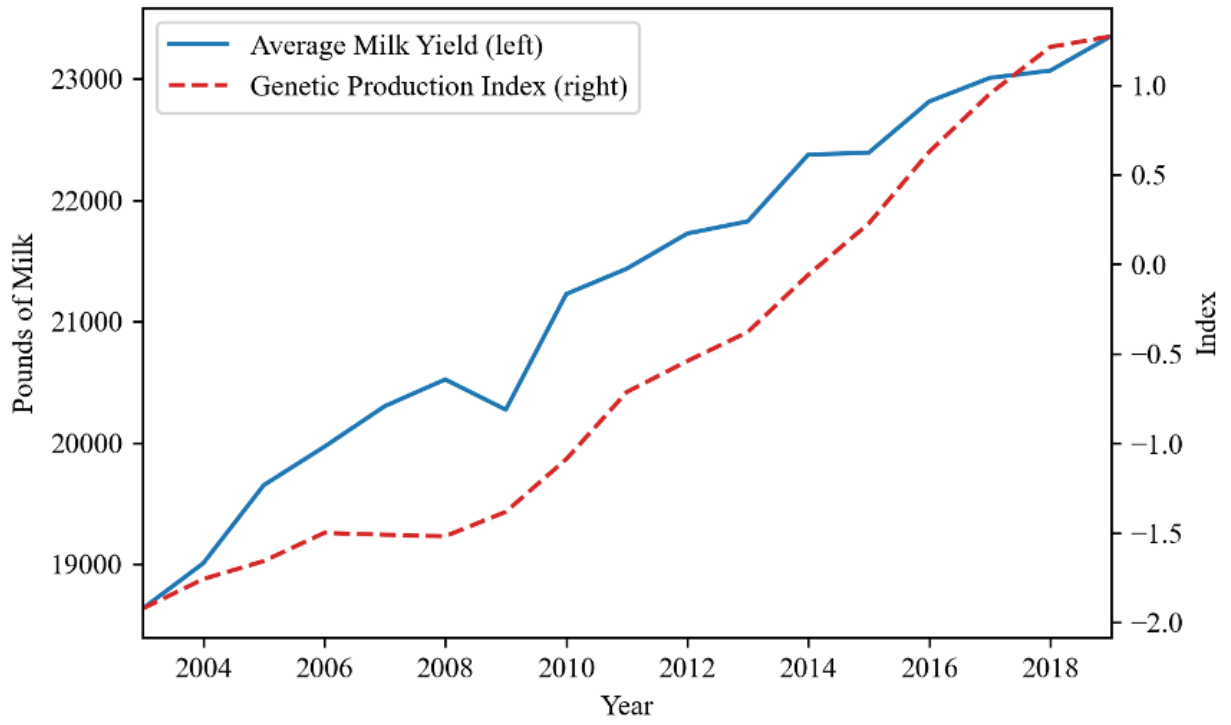


Figure 1.1: Average Milk Yield and Genetic Productivity, 2003-2019

as an endogenous investment into future productivity. The effect of breeding on productivity growth has thus far only been explored as an exogenous factor in technological progress and not as an investment decision. Townsend and Thirtle (2001) calculate that animal breeding research has a 35% return on investment in South Africa using time series data on R&D expenditures. Studies examining the returns to breeding with farm-level data are even more rare. Like the majority of the dairy economics literature, these studies use the distance function approach (Bravo-Ureta et al. 2021). Using a modified Malmquist productivity index, Atsbeha et al. (2012) find that 19% of the productivity growth rate in the Icelandic dairy sector is due to changes in the average sire "breeding value," or estimated productivity. Importantly, Atsbeha et al. (2012) view genetics not as an input that farmers invest in but rather feature of the technology, similar to technological progress. Roibas and Alvarez (2010) use a similar, frontier based method and find that the most technically efficient dairy farms experience the highest returns to genetics.

The weakness of these approaches is that it does not control for the endogeneity of the

genetics of the herd. The genetic productivity of a herd is not an exogenous factor, but rather a result of investment decisions made by the farm in the past. The decision to adopt improved genetics is an investment into future productivity based on current conditions including productivity of the existing cohort. Much like the framework of Olley and Pakes (1996), dairy farms make investments every year in the genetics of their herd. Every year, farmers choose bulls to breed with their cows to obtain replacement cows roughly three years in the future. These investments in bull quality provide the proxy variable we can use to identify production function coefficients (e.g., Levinsohn and Petrin 2003; Olley and Pakes 1996). Our work joins other studies which use the control function approach to correct for the endogeneity of input decisions in dairy farming (Frick and Sauer 2018; Jang and Du 2019; Kirwan et al. 2012; Läpple et al. 2021). Using the control function approach, our work represents animal breeding more accurately as an endogenous investment made by the farmer to improve future productivity and not simply as a exogenous feature of the technology. Our work also takes advantage of cow-level data which provide more detailed and granular data for understanding the economics of dairy operations, especially the selection of genetics (Hutchins and Hueth 2021; Hutchins et al. 2021).

Our work builds on previous control function analyses of dairy by modeling the relationship between genetics investment and productivity by including genetic investments in the productivity law of motion. De Loecker (2013) demonstrates that, in the case of exporting firms, neglecting critical variables in the law of motion leads to poorly estimated factor shares. In the case of dairy, by omitting inter-temporal breeding investments we may be misattributing output to various factors such as labor, feed, or even capital. Our study is also the first to examine the impact of genetic investments in health on productivity, a potentially important but so far unexplored aspect of productivity growth (Townsend and Thirtle 2001). Finally, our paper contributes to the production function literature by demonstrating an intuitive use for supervised machine learning in model estimation. We correct for survival bias using the method outlined in Olley and Pakes (1996) but

significantly improve the probability prediction step by using a random forest algorithm.

The paper proceeds as follows. First, we explain our empirical model which draws on De Loecker (2013) to incorporate genetic investments into the production function. We then describe our three data sources which provide farm- and cow-level information to estimate our model. We present the results of our analysis in the fourth section before concluding with a discussion of the significance of the results.

1.2 Empirical Model

In our model, farm i produces Q_{it} units of quality-adjusted milk in period t using these inputs: cows (denoted by C hereafter), labor (L), capital (K), feed (F) and intermediate inputs (M). The output is adjusted with the percentage composition of fat, the percentage composition of protein, and the average somatic cell count (SCC).[2] Labor corresponds to hired workers, capital refers to building, machinery and equipment, and intermediate inputs refer to electricity. Feed includes total amount of purchased feed.[3] The Cobb-Douglas production function is specified as[4] :

$$Q_{it} = C_{it}^{\beta_c} L_{it}^{\beta_l} K_{it}^{\beta_k} F_{it}^{\beta_f} M_{it}^{\beta_m} exp(\omega_{it} + \epsilon_{it}), \tag{1.1}$$

where ω refers to the Hicks-neutral farm-level productivity measure, which implies that technical change affects milk yield only by changing ω and not the balance of other inputs.[5] The term ϵ is an i.i.d. measurement error term that accounts for random productivity

[2]We discuss the detailed procedure of quality adjustment in a later section.

[3]The construction details of the output and input variables are described in the data section.

[4]We follow the Cobb-Douglas specification of the production function mainly because it is a predominant specification in the industrial organization literature (De Locker and Syverson 2021). Other specifications include, for example, translog (e.g., De Loecker and Warzynski 2012) and the constant elasticity of substitution (e.g., Grieco et al. 2016).

[5]Hicks-neutral productivity is defined as a non-input-related or factor-neutral shift of the production function. It measures the changes in output that are not explained by changes in inputs. Therefore, a higher productivity here means more output can be produced from a fixed set of inputs. This is a predominant setup in the industrial organization literature (De Locker and Syverson 2021).

shocks. The logarithmic form of Equation 1.1 is:

$$q_{it} = \beta_c c_{it} + \beta_l l_{it} + \beta_k k_{it} + \beta_f f_{it} + \beta_m m_{it} + \omega_{it} + \epsilon_{it}, \tag{1.2}$$

where lower-case letters denote the log form of the upper-case letters, that is, $q_{it} \equiv log(Q_{it})$, $c_{it} \equiv log(C_{it})$, $l_{it} \equiv log(L_{it})$, $k_{it} \equiv log(K_{it})$, $f_{it} \equiv log(F_{it})$, and $m_{it} \equiv log(M_{it})$. To correctly estimate the parameters of this equation, we must address three things: input endogeneity, the quality of output, and survival or selection bias.

Input Endogeneity

Input endogeneity is a result of the correlation between farmers' input decisions and unobserved productivity ω_{it}. Therefore, OLS estimates of the input coefficients are biased. There are some approaches proposed in the early literature to deal with endogeneity such as the fixed effect model (e.g., Hoch 1955), utilizing the first order conditions of flexible inputs (e.g., Hall 1988), and using input prices as instrumental variables (e.g., Griliches and Mairesse 1998). The fixed effect model assumes a time-invariant productivity shock, i.e., $\omega_{it} = \omega_i$, which does not work well in practice. Similarly, the static first-order condition typically does not hold in the choices of flexible inputs. Using input prices as IVs is also questionable for reasons like not capturing input quality and/or lack of variations across firms.[6] The caveats of the prior approaches motivate the Olley-Pakes/Levinsohn-Petrin (OP/LP; Levinsohn and Petrin 2003; Olley and Pakes 1996) method that we adapt to deal with the endogeneity problem.

OP/LP methods use a proxy variable that is monotonically increasing in productivity, such as investment (in OP) and intermediate inputs (in LP), to control unobserved productivity. In our study, we use the investments in breeding and genetics, g_{it}, as the proxy for

[6]We refer readers to Section 2.1, Ackerberg et al. (2015) for a detailed discussion of the related methods in the early literature.

unobserved productivity: $g_{it} = g_t(\omega_{it}, c_{it}, k_{it})$.[7] If there is a positive, monotonic relationship between the amount of investment g_{it} and productivity ω_{it} for any combination of c_{it} and k_{it}, meaning ω_{it} is increasing in g_{it}, then the inverse, $\omega_{it} = g_t^{-1} = h_t(g_{it}, c_{it}, k_{it})$, can be used as a valid proxy for productivity. We use two variables to represent for genetic investment: breeding expenditures and the average productivity and health indices of the bulls chosen for breeding. By using g_{it} as the proxy for unobserved productivity, we replace ω_{it} in Equation 1.2 with $\phi_t(g_{it}, c_{it}, k_{it}) = h_t(g_{it}, c_{it}, k_{it}) + \beta_k k_{it} + \beta_c c_{it}$ and rewrite the production function as follows:

$$q_{it} = \beta_l l_{it} + \beta_f f_{it} + \beta_m m_{it} + \phi_t(g_{it}, c_{it}, k_{it}) + \epsilon_{it}, \tag{1.3}$$

where ϕ_t is a third-order polynomial approximation of productivity which includes the linear terms $\beta_k k_{it}$ and $\beta_c c_{it}$. Based on Equation 1.3, we obtain the predicted output by regressing observed output on all of the inputs and the proxy variable of breeding investment. Using the predicted output $\hat{q}$, we estimate the productivity shock $\omega_{it} = q_{it} - \hat{q}_{it}$ to estimate the parameters of the law of motion of productivity.

We depart from the standard OP/LP model by following De Loecker (2013) and amending the law of motion for productivity. Instead of a first-order Markov process for productivity defined in the standard OP/LP model, we assume that genetic investments in year $t-3$ impact productivity in year t. This time lag is a result of the biological constraints of dairy farming. When a cow becomes pregnant after breeding, it gives birth ten months later. That offspring is then bred at about one year old so that it can begin producing at two years old. Altogether, the breeding decision and the productivity resulting from that decision are about three years apart. If ξ_{it} denotes the productivity shock, the corresponding law of motion becomes:

$$\omega_{it} = \varphi_t(\omega_{i,t-1}, G_{i,t-3}) + \xi_{it}. \tag{1.4}$$

[7] Note that the input factors, number of cows (c) and capital (k) are fixed and thus affected only by information at $t-1$. Also, cow number is relevant in the sense that genetic investment g is the total investment on the herd.

For Equation 1.4, we consider two cases. In the first case, productivity follows an AR(1) process and is a linear function of genetic choice three years ago and a productivity shock. It is represented as:

$$\omega_{it} = \rho\omega_{i,t-1} + \gamma G_{i,t-3} + \xi_{it}. \tag{1.5}$$

In the second case, we add an interaction between the level of productivity last period ($\omega_{i,t-1}$) and the level of investment that will be realized next period ($G_{i,t-3}$)[8]:

$$\omega_{it} = \rho\omega_{i,t-1} + \theta_1 G_{i,t-3} + \theta_2 G_{i,t-3}\omega_{i,t-1} + \xi_{it}. \tag{1.6}$$

In De Loecker (2013), the interactions allow firms with different levels of productivity to have different returns to exporting. In our case, it captures heterogeneous returns to breeding investments by allowing returns to vary with farm productivity. In essence, $\omega_{i,t-1}$ reflects the productivity of last year's cow cohort and ρ reflects how much those effects persist. The genetic investment $G_{i,t-3}$ reflects the productivity of the incoming cohort, with θ_1 measuring the incoming cohort's impact on productivity. The effect of adding the new cohort to the existing cohort is measured by θ_2. Since the new cohort is almost always genetically related to the current cohort, the interaction between current and incoming cohorts is a significant part of how productivity evolves on dairy farms. The sign of θ_2 also sheds light on whether there are decreasing or increasing returns to genetic investments in dairy. A positive sign would indicate that the most productive dairy farms get the most out of genetics investment (increasing returns), whereas a negative sign would indicate that the returns to genetics are lowest for the most productive farms (decreasing returns).

Following De Loecker (2013), we assume $E[G_{i,t-3}\xi_{it}] = 0$ to obtain identification. This assumes that breeding decisions made three years prior are independent of the evolution of current productivity. This assumption gives a moment condition we can use to identify

[8]This requires the assumption that the new cohort is genetically related to the current cohort. This is reasonable as a majority of Wisconsin dairy farmers raise replacement heifers by themselves for obtain replacements cheaper than in the market and have more control over genetics and disease. We thank a reviewer for pointing this out.

the parameters of interest by applying the general method of moments (GMM):

$$E\{\xi_{it}(l_{i,t-1}, k_{i,t-1}, c_{i,t-1}, f_{i,t-1}, m_{i,t-1})\} = 0. \tag{1.7}$$

This moment condition highlights the importance of including $G_{i,t-3}$ when identifying the coefficients of the production function. If we omit breeding from the evolution of productivity, that is $\omega_{it} = \phi_t(\omega_{i,t-1}) + \xi_{it}$, then the productivity shock ξ_{it} contains the impact of genetic investment on productivity. If genetic investment correlates with any of these inputs, omitting genetic investment from the law of motion would bias the factor shares. The estimation is conducted in two steps. Using the inputs and the proxy variable, the first stage regression generates predicted output with the estimated input coefficients.[9] Together with the law of motion, the output in the first step is used to construct the productivity shock ξ_{it}. In the final stage, the production function parameters are estimated using the moment conditions mentioned above.

With the method, we address two questions concerning the role of genetic improvement in dairy productivity growth. First, we examine whether farmers' choice in genetics affects the law of motion of productivity. In contrast to previous work, we explicitly model genetic improvement as an investment in future productivity made by the farmer and not solely an exogenous feature of the technology. Second, we examine the extent to which ignoring genetic improvement biases estimated factor shares in the production function when studying dairy farming.

[9] Ackerberg et al. (2015) discusses the "functional dependence problem" in identifying the coefficients of the labor input β_l in the first stage of the OP and LP methods. The concern is that after controlling investment (or intermediate input) in a non-parametric function for unobserved productivity, there is no variation left to consistently estimate β_l. In our setting, the unique features of the control variable, the investment in breeding and genetics, enable us to break the functional dependence problem and identify the parameters of β_l, β_f, and β_m. As the data generation processes of l_{it}, f_{it}, and m_{it} are different from that of G_{it}, the "optimization errors" (Ackerberg et al. 2015) in the choices of l_{it}, f_{it}, and m_{it} induce variations for identification. Variation also comes from the different timings of the choices of l_{it}, f_{it}, m_{it}, and G_{it}. While l_{it}, f_{it}, and m_{it} are chosen at t as functions of ω_{it}, the choice of G_{it} partially depends on the information of G_{t-3} (Equation 1.4). We thank a reviewer for pointing this out.

Quality Adjustment of Output

As quality and quantity jointly determine milk price, dairy farmers take both into consideration when making genetic choices. Therefore, we adjust milk production by quality attributes to measure productivity and its growth accurately. Following Atsbeha et al. (2012), we generate a milk quality index with three key attributes affecting milk price: nutrient component percentage, including the butterfat percentage (C_{it}^{fat}) and protein percentage ($C_{it}^{protein}$), and hygienic quality attribute represented by somatic cell count in unit milk (SCC_{it}). The higher the butterfat and protein percentage, the better the milk quality; the lower the SCC, the better the milk quality.

Let I_{it} denotes the unit milk value represented by milk price received by farm i at time t. Assuming that the unit value is approximately linear in protein percentage, fat percentage, and SCC (Atsbeha et al. 2012), we estimate the following hedonic price equation:

$$I_{it} = \alpha_0 + \alpha_1 C_{it}^{fat} + \alpha_2 C_{it}^{protein} + \alpha_3 SCC_{it} + \eta_{it}, \tag{1.8}$$

where the term η_{it} is a normally distributed random error. With the estimated parameters $\hat{\alpha}_0$, $\hat{\alpha}_1$, $\hat{\alpha}_2$, and $\hat{\alpha}_3$, we calculate the average unit milk value of all sample farms $\hat{I}$ with average milk quality attributes C^{fat}, $C^{protein}$ and SCC as follows:

$$\hat{I} = \hat{\alpha}_0 + \hat{\alpha}_1 C^{fat} + \hat{\alpha}_2 C^{protein} + \hat{\alpha}_3 SCC. \tag{1.9}$$

The term $\hat{I}_{it}/\bar{\hat{I}}$ is specified as the milk quality index, which equals one when a farm's milk quality is equivalent to the sample average, greater (or lower) than one when a farm's milk quality is better (or lower) than average. The quality-adjusted milk output of farm i, $\tilde{Q}_{it}$, is then calculated as:

$$\tilde{Q}_{it} = \frac{\hat{I}_{it}}{\bar{\hat{I}}} Q_{it}. \tag{1.10}$$

Survival Bias

Firms dropping out of the data presents a problem for accurately estimating production functions, especially if firms drop out for having too low of productivity. This is especially a concern when analyzing the dairy sector because there is a long-run trend towards farm consolidation as small farms go out of business and the remaining farms grow larger (MacDonald et al. 2020). If the farms dropping out of our sample have lower productivity than those "surviving," our production function estimates would be biased.

We follow Olley and Pakes (1996) and assume that firm i stays in the market ($\chi_{it} = 1$) instead of exiting the market ($\chi_{it} = 0$) if its productivity exceeds some threshold $\bar{\omega}_{it}(K_{it}, C_{it})$ which makes the sample selection rule:

$$\chi_{it} = \begin{cases} 1 & \omega_{it} \geqslant \bar{\omega}_{it}(K_{it}, C_{it}), \\ 0 & \text{otherwise.} \end{cases} \tag{1.11}$$

Since we know that our investment measure G_{it} is also a function of current productivity ω_{it}. The practical implication of this is that the probability of a firm i exiting the market in time t, $P_{it} = P(\chi_{i,t-1} = 0)$, is a function of last year's inputs: $K_{i,t-1}$, $C_{i,t-1}$, and $G_{i,t-1}$. To correct the production function estimates, the probability has to be estimated using these three variables and then included in the final estimation.[10]

Olley and Pakes (1996) uses probit and a kernel estimator to estimate these probabilities and includes a range of non-linear interactions of the three variables. We improve on their method by using a supervised machine learning algorithm, random forest, to estimate the probabilities as a function of capital, the number of cows, and genetic investment. The

[10]Details of how P_{it} is incorporated into the estimation are found in Olley and Pakes (1996). Effectively, instead of just approximating a proxy function ϕ_{it} as would be ordinary in a control function approach, another proxy function, ψ_{it}, has to be approximated as a function of $\hat{\phi}_{i,t-1}$, $K_{i,t-1}$, $C_{i,t-1}$, $G_{i,t-1}$, and the probability estimate $\hat{P}(K_{i,t-1}, C_{i,t-1}, G_{i,t-1})$. The function ψ_{it} is included in the final estimation of the production function to correct for the sample selection bias.

advantage of the random forest is that it naturally takes into account non-linearities when predicting since it iteratively splits the features in random places to improve model fit. Our random forest model is trained using 5-fold cross-validation and the details of the estimation can be found in the supplementary materials in Appendix B. When benchmarked against probit, OLS, and kernel estimation, the random forest performs significantly better even without training. Our analysis in the supplementary materials in Appendix B demonstrates that supervised machine learning can greatly improve this step of estimating production functions which have sample selection. We next turn to our data sources for estimating the production function and understanding the role of genetic investment in dairy farm productivity.

1.3 Data

The main data used in this study consist of 372 farm-level observations of output and inputs from 60 farms in Wisconsin over 2008-2018. The data is collected by the University of Wisconsin Center for Dairy Profitability (CDP). The CDP data is collected by two farm records associations which provide record-keeping, tax preparation, and consulting services to dairy farms throughout Wisconsin. Compared to the agricultural census, farms that participate in CDP benchmarking are larger: the average in the CDP data is about 177 cows whereas the averages in the 2012 and 2017 census were closer to 120. Given that more than 60% of Wisconsin farms in the census are less than 100 cows, our sample represents slightly larger operations but would still be considered small-scale dairies. In both the CDP and census, less than 10% of farms are larger than 500 cows. Our CDP sample is still broadly representative of small- to medium-scale dairies with between 50 and 500 cows.

We also combine the CDP data with records of breeding decisions from Dairy Herd Improvement Associations (DHIAs) over 2012-2018 to measure the genetic quality of bulls each farm selects. DHIAs collect cow-level records for the purpose of benchmarking and

genetic evaluation, as well as details on which bulls bred to which cows on the farm. By matching bull identities to their publicly available market traits, we can obtain a measure of genetic quality that is based on which bulls the farm chooses to breed with each year rather than expenditure.

For the dairy farms in the CDP data set, we observe extensive output and input information in each year. On the output side, we observe revenue from selling milk, the quantity of sold milk, butterfat, and protein, and the somatic cell count (SCC) of unit milk. On the input side, we observe expenditure on hired labor, feed, fuel, and utility, the number of cows, and the value of buildings, machinery and equipment owned by the farms. Critically, we also observe breeding expenses, which is expenditure on bull semen and breeding services to produce replacement offspring in the future. To obtain the indirect quantity measures of output and inputs, the revenue and the expenditure are deflated with national level price indices obtained from the USDA Quick Statistics (NASS 2022).[11][12] The price indices used to deflate each factor are found in the bottom of Table 1.1.

Table 1.1 presents the descriptive statistics of the ouput and inputs, including mean, standard deviation, and percentage change over 2008-2018. During the sample period, the average farm size measured by average milk revenue and herd size shows a growing trend. The average milk revenue increases from 74 million in 2008 to 133 million in 2018 and grows by 80 percent. The average herd size grew by 64 percent, from 177 in 2008 to 290

[11]We do not use the observed milk quantity as we want to adjust milk quality as discussed above.

[12]This is the common practice in the literature to obtain quantity measures of output and inputs (De Locker and Syverson 2021). This generates the so-called revenue-based total factor productivity (TFPR). Given the lack of farm specific output and input prices, aggregate price indices (national level indices in our case) necessarily generate measurement errors in the output and inputs measures, although we focus only on the dairy farms in Wisconsin. One possible solution is to decompose revenue to price and quantity and then add in a demand structure for price (e.g., De Loecker 2011). But this solution also requires assumptions on which the literature has not settled. The decomposition method does not work for measurement errors in inputs. We refer the readers to a detailed discussion in Section 4 of De Locker and Syverson (2021).

With regards to milk quality, farmers are paid on their components using Class III prices. However, since prices change month to month there is no way to apply this pricing scheme to milk output measured at the annual level. Also, farms receive individual premiums and incentives that we do not observe. To adjust for milk quality, we use regression-derived weights calculated from Equation 1.8 to perform the quality adjustment as Atsbeha et al. (2012) does. This procedure necessarily introduces some measurement error in the case that the estimates are inaccurate. Still, we believe this approach is highly preferred to not quality adjusting output and so the risk of measurment error is justified.

Table 1.1: Summary Statistics of CDP Sample

Variable	All	2008	2013	2018	Change % over 2008-2018
Milk Sold Income[1]	91,215 (104,043)	73,788 (86,610)	88,498 (97,403)	132,895 (142,901)	80
Milk (1,000 Pounds)	5,469 (6,152)	4,347 (5,080)	5,463 (5,992)	7,786 (8,195)	79
Butter Fat (1,000 Pounds)	202 (224)	161 (186)	202 (214)	297 (312)	84
Protein (1,000 Pounds)	165 (185)	131 (154)	166 (181)	234 (246)	79
Somatic Cell Count Index (1,000 Cells/mL)	186 (88)	224 (100)	180 (69)	120 (33)	-46
Herd Size	213 (213)	177 (183)	208 (204)	290 (282)	64
Capital $(1,000)$[2]	376 (462)	326 (523)	357 (398)	516 (527)	58
Feed $(1,000)$[3]	306 (356)	225 (313)	282 (305)	391 (391)	74
Hired Labor $(1,000)$[4]	117 (154)	105 (146)	122 (156)	136 (187)	30
Utility (100 kWh)	19 (16)	18 (18)	18 (13)	24 (20)	33
Breeding Expenditure $(1,000)$[5]	20 (20)	19 (21)	19 (19)	27 (23)	42
Observations	372	45	35	19	-

Note: The numbers in the table are means and standard deviations (in parentheses). [1] Milk sold income is adjusted with dairy product price index. [2] The market value of building is adjusted with building material price index. The market value of machinery and equipment is adjusted with machinery price index. [3] The expenditure on feed is adjusted with forage feed price index. [4] The expenditure on hired labor is adjusted with wage rate price index. [5] The breeding expenditure is adjusted with CPI.

Table 1.2: Summary Statistics of Milk Quality Indexes

Symbol	Description	Unit	All	2008	2013	2018	Change % over 2008-2018
C_{it}^{fat}	Butterfat	%	3.75 (0.24)	3.76 (0.26)	3.80 (0.19)	3.84 (0.14)	2
$C_{it}^{protein}$	Protein	%	3.04 (0.19)	3.09 (0.37)	3.06 (0.11)	3.04 (0.08)	-2
SCC_{it}	Somatic Cell Count Index	1,000 Cells/mL	200 (90)	218 (93)	180 (70)	134 (40)	-39
$\frac{\hat{I}_{it}}{\hat{I}}$	Linear Adjuster	#	0.91 (0.03)	0.92 (0.03)	0.88 (0.02)	0.90 (0.01)	-2
Q_{it}	Milk Production	1,000 Pounds	4,514 (5,448)	4,087 (4,751)	4,707 (5,504)	7,334 (7,937)	79
$\tilde{Q}_{it}$	Adjusted Milk Production	-	4,080 (4,916)	3,791 (4,436)	4,120 (4,786)	6,565 (7,093)	73
	Observations		738	60	46	28	

Note: The numbers in the table are means and standard deviations are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

Table 1.3: Quality Adjustment Parameters

Variable	Parameters
Butterfat Percentage	1.057*** (0.186)
Protein Percentage	0.572** (0.244)
Somatic Cell Count Index	-0.003*** (0.001)
R-square	
Within Herd	0.3188
Between Herd	0.6161
Overall	0.4138
Observations	738

Note: The parameters are estimated with fixed-effects regression.

in 2018. During the same period, the average breeding expenditure grows by 42 percent. Table 1.2 shows the descriptive statistics such as mean, standard deviation, and % change over 2008-2018 of milk quality indices containing butterfat percentage, protein percentage,

and SCC. Table 1.3 shows the estimates of Equation 1.8 which we use to construct quality-adjusted output and the "linear adjuster." The linear adjuster is about .9, meaning it revises output slightly lower due to farms producing volume without necessarily producing more components. The linear adjustment stays fairly constant as there is only a minor change in butterfat percentage and protein percentage from 2008 to 2018. However, SCC decreased by around 40 percent during the period. SCC is typically used as a proxy for the incidence of mastitis, so its decrease implies increasing milk quality and cow health.

In this study, we focus on the breeding technology of artificial insemination (AI), which is the most widely used breeding technology in the US dairy sector (Khanal and Gillespie 2013; Nehring et al. 2021). In AI, semen from selected sires are artificially introduced to cows. AI has wide adoption in the dairy industry because it allows for more precise control over the genetic quality of future replacements and lowers the risk of venereal disease (Foote 1996). According to data from the Agricultural Resources Managment Survey (ARMS), it was used by more than 80% of dairy farms in 2016 (Nehring et al. 2021). Farms tend to avoid buying replacements from outside the farm due to the higher cost and the risk of disease. Especially in the Wisconsin context, AI is the preferred method for choosing the future genetics of the herd.

Figure 1.2 shows the distribution of breeding expenditure, our main investment proxy, across farms in our whole sample. Breeding expenditure is made up of two components: genetic quality and technician fees. Technician fees are usually more or less uniform across cows, but the remaining variation in expenditure is due to the cost of the genetics itself. Hedonic analyses of dairy genetics markets show that the most expensive genetics tend to be those with high production traits and, to a lesser extent, bulls with better health traits (Richards and Jeffrey 1996; Schroeder et al. 1992).

To adjust for herd size, Figure 1.2 shows breeding expenditure per cow. On average, farms spend about $90 per cow. As a reference point, the average price for a dairy bull was

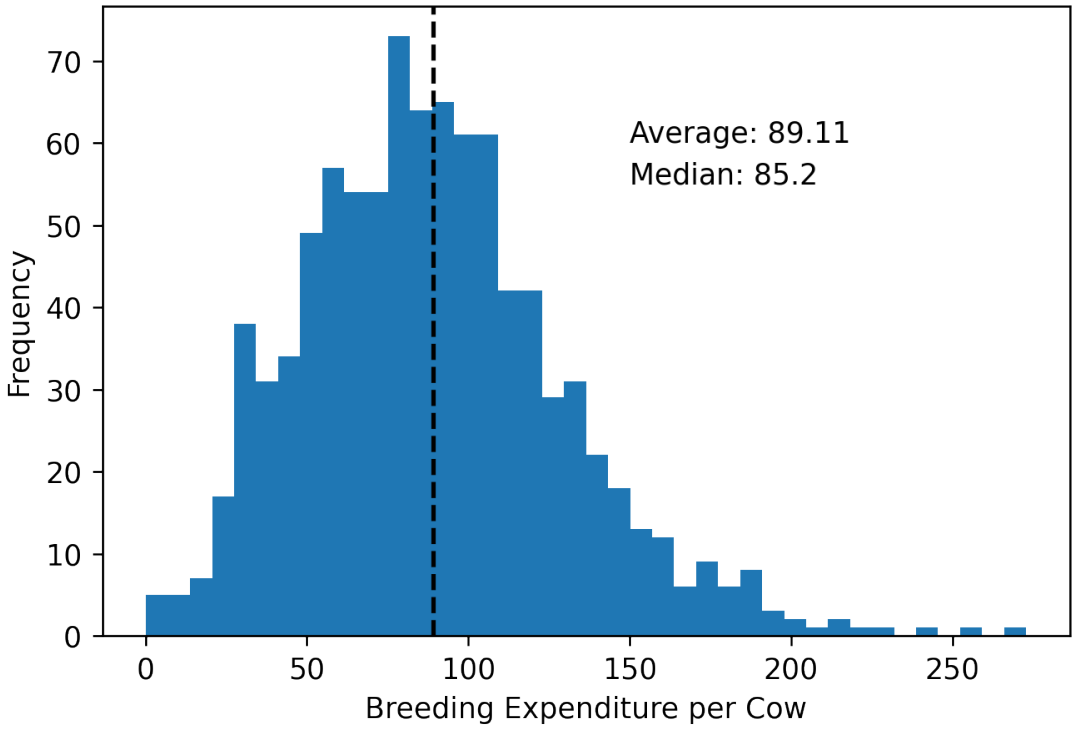


Figure 1.2: The Distribution of Breeding Expenditure per Cow

20-30 dollars per service in the period 2010-2020.[13] Since some cows do not get pregnant the first time, we may expect that in one year a farmer may purchase two bull services, meaning an annual cost between 40 and 60 dollars per cow per year. The remaining cost can be explained by additional service costs charged by the breeding company which do not scale with genetic quality in the majority of cases.[14] The median breeding expenditure is $85 due to the large right tail of farms that spend more than $150 per cow on breeding. While not much data is available on the costs of individual genetics on dairy farms in general, the price of bulls is primarily be explained by genetic quality (Richards and Jeffrey 1996). For this reason, the breeding expenditure variable is a suitable proxy for investment in future productivity through genetic improvement.

[13] Authors' calculations using posted sire price data from NAAB (2022).

[14] Large outliers may be explained by embryo transfer, a more expensive delivery method for genetics used in a minority of cases.

One potential component of breeding expenditure that can attenuate its impact on productivity is repeated breeding attempts. If a cow does not conceive after the farm pays for breeding services, the farm must purchase the genetics again to repeat the attempt. To the extent that some infertility may be a result of management, breeding expenditure may be high in some cases because of persistently poor fertility. If firms with poor fertility are also less productive, then this attenuates the positive impact of breeding expenditure on productivity towards zero. We should also expect that firms with high productivity farms get more productivity out of their breeding expenditure, meaning the interaction between productivity and breeding expenditure (θ_2 in Equation 1.6) would be positive.

To complement our analysis using breeding expenditure, we merge CDP data with our DHIAs data to obtain a more direct measure of genetic investments: the genetic traits of the bulls that farms choose. Dairy farms make investments into the genetics of their herd by purchasing bulls with certain traits. Farms can choose bulls with higher production traits, for example a higher fat or protein yield, in order to have replacement cows in the future that yield more fat or protein. Fat and protein yield are especially important to Wisconsin dairy farms because they are located in the Upper Midwest Milk Marketing Order (Federal Milk Marketing Order 30) and are paid based on the amount of protein and fat they produce in pounds. Given these incentives, most dairy bull selection indices do not include milk yield independent of fat and protein (that is increasing volume). Instead, fat and protein yield are the primary trait indices using in dairy bull selection.[15] Farms can also choose to invest in health by choosing bulls with a lower "somatic cell count" (a measure that correlates to the incidence of mastitis) or a higher "daughter pregnancy rate" (a measure that correlated to fertility).

Our data on genetic indices comes from the Council on Dairy Cattle Breeding (CDCB). The CDCB estimates genetic indices for dairy sires called "predicted transmitting ability"

[15]See, for example, the weights for the Net Merit index used widely in the dairy industry. In this index, fat and protein are valued at $4 a pound whereas milk yield is valued at less than $.01 a pound. Milk yield is given a low weight because, independent of fat and protein yield, there is little market value to producing high volume.

(PTAs) three times a year which are publicly available through the National Association for Animal Breeders (NAAB 2022). The indices measure how much a sire will "transmit" performance relative to a base bull whose PTA is equal to zero. For example, if a bull has a butterfat PTA score of 50, then the farmer can expect that offspring from that bull will produce 50 more pounds of butterfat than the base bull. The indices are used by companies to price bulls and by dairy farmers to help inform their breeding choices.

We use around 140,000 dairy bull evaluations from the period 2012-2019 and focus on five traits: milk yield, fat yield, protein yield, somatic cell count, and daughter pregnancy rate. The first three are production related, the fourth is related to health, and the last trait is a measure of fertility. To construct production and health indices from these five traits, we use a technique called Principal Component Analysis (PCA). PCA is an orthogonal, linear transformation of data into a new coordinate system whereby the first axis, called the "first component," explains the most variance in the data possible, the second axis the second most variance, and so on. PCA was introduced to the economics and psychology literature by Hotelling (1933) and has since been applied in economics in a variety of ways (Aït-Sahalia and Xiu 2019; Manyong et al. 2006; Nieuwoudt 1972). One popular and relevant application of PCA is constructing indices of several different variables (Vyas and Kumaranayake 2006). Since each component is a linear combination of variables in the data, the components can be interpreted as a weighted index of the variables. This is especially useful when there is correlation between different variables and we would prefer one index that represents them all.

In the case of genetic traits, PCA helps us construct two indices that represent our three production traits and two health traits. In our calculations, the first two components captured 83% of the variance in these five traits. The first component was highly correlated with the three production traits, while the second component was highly correlated with somatic cell count and daughter pregnancy rate. We use the first component as our

"production index" and the second as our "health index."[16]

After estimating these indices, we match this data to DHIAs data of farm breeding choices. By matching the bull ID in DHIAs to its evaluation from the CDCB, we know the bull's production and health index scores at the time it was chosen. This gives us two measures of genetic investment: breeding expenditure and genetic indices. The first of these measures is simply a cost measure, how much dairy farms spend on breeding, while the second more accurately measures *what* dairy farms are investing in. Unfortunately, there are only a few farms both in DHIAs and CDP so the genetic index scores can only be used in a subset of our data. Still, by examining the subset of data belonging to both the CDP data and the DHIAs, we have a never-before-seen look into how dairy farmer breeding choices translate into productivity.

The summary statistics for this merged sample are in Table 1.4. The merged CDP-DHIAs sample has 88 observations over 2012-2018. The average herd size in the merged sample is 275 cows per farm, larger than the average herd size in the CDP sample, 213 cows per farm. The average output and input are also larger for dairy farms in the merged CDP-DHIAs sample.

Combining both CDP and DHIAs data, we obtain two proxy measures for genetic investments: breeding expenditure and the average production and health indices of their bull choices. These two measures capture different aspects of genetics investments. Total breeding expenditure contains the cost of the bull semen, labor fees, and management fees. According to hedonic analyses of dairy bull prices, bulls with the most productive genetics in terms of milk production tend to be the most expensive, so we can assume breeding expenditure is weakly increasing in genetic traits measuring productivity (Richards and Jeffrey 1996; Schroeder et al. 1992). Our genetic indices measure the average traits chosen in that year, which helps us measure the type of investments being made. This measure also

[16]Table A1 in the supplementary appendix reports the loading scores for the production index and shows that milk yield, fat yield, and protein yield are correlated to the first component in a similar way. This indicates there is no direct trade-off between milk yield and component yield. We refer the readers to the supplementary Appendix A for a more detailed discussion.

Table 1.4: Summary Statistics for CDP-DHIAs Sample

Variable	Mean	Std. Err.	Minimum	Maximum
Milk Sold Income (1,000)[1]	127,665	137,428	10,140	632,094
Milk (1,000 Pounds)	7,469	7,867	640	35,528
Butter Fat (1,000 Pounds)	279	288	24	1,305
Protein (1,000 Pounds)	225	236	20	1,062
Somatic Cell Count Index	148	58	61	374
Herd Size	275	259	38	1,177
Capital (1,000)[2]	517	536	4	2,401
Feed (1,000)[3]	424	446	13	2,150
Hired Labor (1,000)[4]	147	191	0	855
Utility (100 kWh)	23	19	4	84
Breeding Expenditure (1,000)[5]	26	24	1	92
Genetic Production Index	0.80	0.56	-1.30	1.91
Genetic Health Index	0.30	0.44	-1.32	1.40
Observations		88		

Note: [1] Milk sold income is adjusted with dairy product price index. [2] The market value of building is adjusted with building material price index. The market value of machinery and equipment is adjusted with machinery price index. [3] The expenditure on feed is adjusted with forage feed price index. [4] The expenditure on hired labor is adjusted with wage rate price index. [5] The breeding expenditure is adjusted with CPI.

partially alleviates concerns about potential distortion on total genetic investment caused by herd size. It is not a per-cow based measure as it is averaged over chosen bulls. However, it reflects the average quality of genetics incorporated into a herd, and should be highly correlated with the average level of investment, especially for large herds. We analyze both production traits and health traits using our created indices. Milk producing traits have received the most attention (Atsbeha et al. 2012; Roibas and Alvarez 2010), yet health traits can also have an important relationship to productivity. Many health conditions such as mastitis directly impact milk production, making it feasible that farms may invest in these health traits to increase productivity.

Figure 1.3 shows the growing trend of average breeding investment and the production ability of the chosen sires in our sample. These Wisconsin dairy farms made breeding

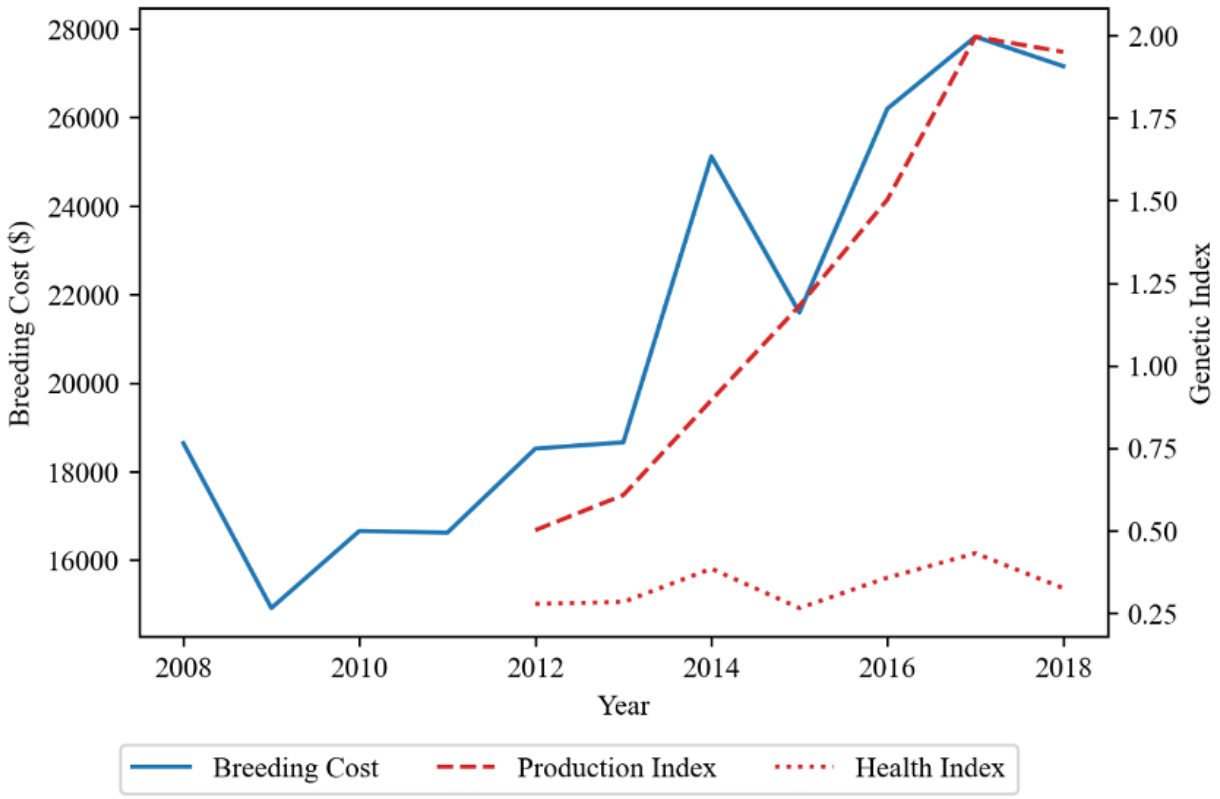


Figure 1.3: Average Breeding Investment and Genetic Production Index, 2008-2018

decisions similar to the rest of the country: the genetic production index is growing rapidly over time just as in Figure 1.1. In contrast, the health index scarcely grew at all during this period. There seems to be little variation in the health index during this period, reflecting a tendency for dairy farmers to invest in milk production more than in health traits.

In the next section, we present our estimation results from this unique dataset to explore the role of breeding in accelerating productivity growth in dairy. Comparing across methods, we also determine whether omitting genetic investment leads to biased estimates of factor shares.

1.4 Results

Panel A of Table 1.5 presents the parameters estimated with four models: an OLS model, a standard LP model in which breeding expenditure is the proxy variable for unobserved

productivity (but not in the law of motion), a LP model with breeding expenditure in the law of motion (Equation 5), and an LP model where breeding expenditure is interacted with productivity in the law of motion (Equation 6). In these last two models breeding expenditure is also used as the proxy variable. The parameters of interest in LP models are estimated in the GMM framework, and one-period lag terms are used as the instruments for labor, feed, and intermediate inputs. The last column of Table 1.5 reports the means and standard errors of the differences of the estimated input coefficients in Columns 2 and 4.

Comparing the results in Column 1 with those of the other columns, we find that the OLS overestimates the coefficients of the flexible inputs of labor and herd size represented by cow number because of the endogeneity or the positive relation between the inputs and unobserved productivity. This is consistent with the findings in the literature (e.g., Jang and Du 2019).[17] Including breeding investment in the law of motion of productivity has no statistically significant effects on the estimated factor shares. Coefficients do change, but the differences between Column 2 and 4 are not statistically significant. Including a non-linear interaction term does not appear to affect estimation given that the Column 3 and Column 4 estimates are essentially the same. While including breeding investments may affect factor shares, our sample size is too small to determine whether these changes are statistically significant.

Panel B of Table 1.5 presents the coefficients for the law of motion of productivity. As expected, breeding expenditure that occurred three years ago has a positive and significant effect on current productivity. While there was some risk of attenuation bias using breeding expenditure, we still see a positive and significant impact. The probability of exiting is negatively related to productivity, which implies that firms more likely to exit have lower productivity. This fits with what we expect from an industry that is consolidating: less productive firms are exiting and leaving only the most productive firms in the industry

[17]We do not find a downward bias of the OLS estimate on the capital input as suggested in the literature probably because of relatively small size and number of farms in the sample. We thank a reviewer for pointing this out.

Table 1.5: Estimates of the Production Function and Law of Motion

	OLS	Standard LP Model	De Loecker Model		Coefficient Difference, (2) - (4)
			Panel A: Production Function		
	(1)	(2)	(3)	(4)	
Labor	0.077***	0.067***	0.059	0.059***	0.008
	(0.012)	(0.012)	(0.047)	(0.010)	(0.006)
Capital	0.022***	0.016**	0.016**	0.017***	-0.001
	(0.008)	(0.007)	(0.007)	(0.005)	(0.004)
Herd Size	0.809***	0.786***	0.795***	0.804***	-0.018
	(0.026)	(0.052)	(0.048)	(0.038)	(0.036)
Feed	0.110***	0.111*	0.116**	0.105***	0.005
	(0.021)	(0.062)	(0.048)	(0.032)	(0.039)
Material	0.026	0.079*	0.041	0.041***	0.038
	(0.020)	(0.044)	(0.073)	(0.029)	(0.027)
Constant	8.233***	8.062***	8.053***	7.994***	0.068
	(0.143)	(0.045)	(0.034)	(0.038)	(0.056)
Observations	366	366	366	366	
Interaction	-	-	No	Yes	-
			Panel B: Law of Motion		
Productivity, $t-1$	-	-	0.523***	0.614	-
			(0.071)	(0.383)	
Breeding Expend, $t-3$	-	-	0.014***	0.117	-
			(0.003)	(0.316)	
Productivity $\times$ Breeding Expend			-	-0.012	
				(0.038)	
Exit Probability $P_t(k_{t-1}, b_{t-1})$	-	-	-0.060***	-0.062***	-
			(0.021)	(0.022)	
Observations	-	-	283	283	-
Year FE	-	-	Yes	Yes	-
R-squared	-	-	0.726	0.723	-

Note: Standard errors are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

(Olley and Pakes 1996). When we include the interaction term, we see a negative sign on the interaction but only the probability of exit remains statistically significant. This pattern suggests that there are diminishing returns to investments in genetics but the size of the standard errors makes it impossible to confirm whether this is the case. Still, since we do not find the interaction term to be positive, this is evidence against breeding attempts being a major component of breeding expenditure (see discussion in Section 3).

Table 1.6 examines the same questions but compares using breeding expenditures to genetic indices, a less noisy measure of investment in genetic improvement. Panel A of Table 1.6 compares the factor shares for the models that use breeding investment, genetic indices, or both in the law of motion respectively. The coefficients change very little between these models, except the materials coefficients that are not statistically different from zero in any model. Panel B of Table 1.6 shows how the genetic indices factor in the law of motion compare to that of breeding expenditure. Production and health indices from bulls chosen three years ago correlate positively to current productivity, though only the production index is statistically different than zero at the 95% level. The interaction term for the production index is negative just as in breeding expenditure but is statistically significant. The interaction term is an estimate of the parameter θ_2 from Equation 1.6 which measures how the new cohort's improved productivity enhances the productivity of the current cohort. We find evidence of diminishing returns to genetics, meaning already productive farms will benefit less from genetic investments than farms that have less productive cows. This makes intuitive sense, since cows are likely to have biological constraints that prevent genetic improvement from indefinitely increasing productivity. For example, cows that have higher milk production are more likely to develop certain health problems (Oltenacu et al. 2010). Farms with lower productivity have more to gain from genetic improvement than farms that are already productive and may be dealing with this trade-off between health and production.

As a robustness check, we examine the relationship between realized quality and

Table 1.6: Production Function, CDP-DHIAs Sample

	Breeding Expenditure	Genetic Indices
	Panel A: Production Function	
	(1)	(2)
Labor	0.057***	0.055
	(0.019)	(0.107)
Capital	0.013	0.011
	(0.009)	(0.029)
Herd Size	0.819***	0.817***
	(0.054)	(0.243)
Feed	0.124*	0.147**
	(0.069)	(0.062)
Material	0.009	0.031
	(0.048)	(0.081)
Constant	8.025***	8.082***
	(0.049)	(0.162)
Observations	87	87
R-squared	0.609	0.491
	Panel B: Law of Motion	
Productivity, t-1	0.664	0.970***
	(1.421)	(0.340)
Breeding Expend, t-3	0.296	-
	(1.153)	
Production Index, t-3	-	3.895**
		(1.645)
Health Index, t-3	-	2.873
		(2.718)
Productivity, t-1 ×	-0.033	-
Breeding Expend, t-3	(0.135)	
Productivity, t-1 ×	-	-0.482**
Production Index, t-3		(0.203)
Productivity, t-1 ×	-	-0.351
Health Index, t-3		(0.335)
Exit Probability,	-0.185**	-0.186**
$P_t(k_{t-1}, b_{t-1})$	(0.070)	(0.082)
Observations	55	55
Year FE	Yes	Yes
R-squared	0.609	0.491

Note: Standard errors are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

genetic investments by comparing the coefficients on the law of motion with and without quality-adjusted output. Since many genetics investments involve quality (e.g., protein and butterfat output), it is important to understand how quality and genetics investments interact. Table 1.7 show the model parameters from Table 1.6 with and without the quality adjustment and the difference between them. As before, the production function coefficients differ but are not statistically different, which is likely due to small sample size. In the unadjusted model, more of the coefficients such as labor and feed are insignificant than in the quality-adjusted model. When the quality dimension is not taken into account, it appears that many inputs have no relationship to output. Similarly, investments in production traits have no impact on productivity in the law of motion when output is not quality adjusted.

There are two insights generated from this exercise. First, inputs may be used to generate quality (butterfat and protein) instead of volume. If this is the case, ignoring quality adjustment may lead us to think some inputs do not matter to production. The second insight is that farms investing in quality are likely also making breeding investments into quality. Without adjusting for quality, these investments will not appear to have any impact on productivity.

1.5 Conclusion

Our work examines the relationship between investments in genetics through breeding and productivity growth in the US dairy industry. Using a detailed dataset of Wisconsin dairy farms, we incorporate investment in breeding services and genetic indices into the productivity law of motion. We test whether or not omitting genetics investments impacts the estimation of factor shares in the dairy production function. We find that including genetics investments in the law of motion changes the point estimates for feed, labor and materials in the production function, though no differences are statistically different than

Table 1.7: Model Results With and Without Quality Adjustment

	Unadjusted Model	Quality-Adjusted Model	Difference between Two Models
	Panel A: Production Function		
Labor	0.056	0.055	0.001
	(0.059)	(0.107)	(0.073)
Capital	0.000	0.011	-0.011
	(0.017)	(0.029)	(0.025)
Herd Size	0.802***	0.817***	-0.016
	(0.149)	(0.243)	(0.265)
Feed	0.153	0.147**	0.006
	(0.093)	(0.062)	(0.148)
Material	0.050	0.031	0.019
	(0.138)	(0.081)	(0.100)
Constant	8.009***	8.082***	-0.074
	(0.091)	(0.162)	(0.138)
Observations	87	87	-
	Panel B: Law of Motion		
Productivity, t-1	0.726**	0.970***	0.244
	(0.326)	(0.340)	(0.471)
Production Index, t-3	1.195	3.895**	2.700
	(1.917)	(1.645)	(2.526)
Health Index, t-3	4.094	2.873	-1.220
	(2.892)	(2.718)	(3.968)
Productivity, t-1 $\times$ Production Index, t-3	-0.147	-0.482**	-0.336
	(0.235)	(0.203)	(0.310)
Productivity, t-1 $\times$ Health Index , t-3	-0.503	-0.351	0.152
	(0.355)	(0.335)	(0.488)
Exit Probability, $P_t(k_{t-1}, b_{t-1})$	-0.260***	-0.186**	0.073
	(0.095)	(0.082)	(0.125)
Observations	55	55	-
Year FE	Yes	Yes	-
R-squared	0.490	0.491	-

Note: Standard errors are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

zero. While breeding investments made three years ago positively impact productivity in the current period, the returns to investment are lowest for farms with high productivity. Put differently, we find that there are diminishing returns to investments in genetics on Wisconsin dairy farms.

Our work has several limitations that may be able to be addressed in future work. First, the sample size in this study is very small. The sorts of data needed to understand investments in genetics in the context of other inputs are hard to come by. While datasets like the Agricultural Resource Management Survey (ARMS) have detailed information about capital and labor inputs, they do not have information about breeding investments. This a critical data gap that still exists when studying dairy farm operations and leads us to our second limitation. In this study, we use spending on breeding as a proxy for genetic investments. This may be a crude proxy for what we are actually trying to measure: investments in genetic quality. Data on individual breeding decisions from DHIAs can help fill this gap, but the lack of cross-over in our sample between DHIAs and CDP farms makes estimation very imprecise. Also, since the breeding decisions are at the animal level, it is an open question what the best way to aggregate this information to the farm level is. Further data collection efforts should particularly focus on trying to understand how much dairy farmers pay for different kinds of genetics. This kind of data is vital to understanding the link between investments in genetics and productivity growth in a more precise way.

Strategic breeding has been one of the most important innovations in animal agriculture in the past century. The next century poses new challenges for animal industries such as dairy, especially addressing excessive greenhouse gas emissions from livestock industries. Part of these externalities may be addressed in the future by breeding away from production and towards sustainability of the operation (Wall et al. 2010). Our work is a crucial first step in determining just how important farm investments in genetic improvement have been in the on-farm dynamics of productivity growth, and thus a crucial fist step in better understanding the role of genetics in the future of the dairy industry.

Appendix

Principal Component Analysis

Dairy farms make investments into the genetics of their herd by purchasing bulls with certain traits. Farms can choose bulls with higher production traits, for example a higher fat or protein yield, in order to have replacement cows in the future that yield more fat or protein. Fat and protein yield are especially important to Wisconsin dairy farms because they are located in the Upper Midwest Milk Marketing Order (Federal Milk Marketing Order 30) and are paid based on the amount of protein and fat they produce in pounds. Given these incentives, most dairy bull selection indices do not include milk yield independent of fat and protein (that is increasing volume). Instead, fat and protein yield are the primary trait indices using in dairy bull selection.[18] Farms can also choose to invest in health by choosing bulls with a lower "somatic cell count" (a measure that correlates to the incidence of mastitis) or a higher "daughter pregnancy rate" (a measure that correlated to fertility).

Since production traits are highly correlated to other production traits and health traits to other health traits, we construct a production and health index by using Principal Component Analysis (PCA) and a large dataset of dairy bull evaluations. Dairy bulls are evaluated three times a year, and in each period their scores are updated. The scores are calculated by the CDCB using a statistical formula that calculates the contribution of a bull to the performance of its offspring. The offspring data is from DHIA, meaning as bulls are used more widely there is more and more data available to calculate the scores. These scores are used by genetics companies to price bulls and by farmers to decide which bulls to purchase.

For our purposes, we use five traits: milk yield, fat yield, protein yield, somatic cell

[18] See, for example, the weights for the Net Merit index used widely in the dairy industry. In this index, fat and protein are valued at $4 a pound whereas milk yield is valued at less than $.01 a pound. Milk yield is given a low weight because, independent of fat and protein yield, there is little market value to producing high volume.

count, and daughter pregnancy rate. The first three are production traits whereas the last two are related to health. The first two principal components explained about 85% of variation in this data and capture the fact the production traits tend to be correlated with production traits and health traits with health traits. Table A1 shows the loading scores for each component, coefficients which determine the hyperplane through the trait space. The larger these coefficients are the stronger the association between this component and the trait is.

Table A1: Principal Component Analysis Loading Scores

Loading Scores	Component 1	Component 2
Milk Yield	-0.567	-0.029
Fat Yield	-0.552	0.055
Protein Yield	-0.580	0.009
Somatic Cell Count[1]	-0.118	0.714
Daughter Pregnancy Rate	0.149	0.697
Percentage of Variation Explained	58.06	27.25
Eigenvalues	2.90	1.36
Observations	138,108	

Note: [1] We use the negative of the somatic cell count in order to maintain the interpretation that higher values mean higher health.

The first component has large and negative loading scores for the three production traits whereas the second component has large and positive loading scores for the health components. This suggests that the first component can be a proxy for production traits and the second component can be a proxy for health traits. Since the production traits have negative load scores for the first component, we use the negative of the first component as our production index so that higher values are interpreted as more production (like the health index).

Since breeding values are estimates that are frequently updated, there is potentially measurement error in these values that would have implications for using the PCA components as covariates. However, Hellton and Thoresen (2014) specifically investigates the impact of measurement error on PCA and finds that measurement error does not typically impact PCA in a substantive way when PCA components are used as covariates

in a regression. They do find evidence of an attenuation effect on the parameters when the loading scores are small enough. This means that the parameters on the production and health components in the law of motion may be biased toward zero. Since our loading scores are fairly large, more than .5 for the production component and about .7 for the health component, we do not anticipate measurement error in breeding values to have a large impact on our results. Still, the impact of the indices may appear less significant in the law of motion than they are in reality.

Table A2: Breeding Expenditure and Genetic Quality Measures (Averaged)

	Dependent Variable:			
	Breeding Expenditure, Level		Breeding Expenditure, Logarithm	
	(1)	(2)	(3)	(4)
PCA Production	−52.515		0.019	
	(1,344.789)		(0.083)	
PCA Health	2,777.029*		0.191	
	(1,657.157)		(0.150)	
Herd PTA Milk		−6,078.876		0.051
		(3,713.791)		(0.224)
Herd PTA Fat		7,400.035**		0.333*
		(3,154.222)		(0.174)
Herd PTA Protein		−485.460		−0.380
		(3,109.226)		(0.274)
Herd PTA SCC[1]		−2,388.237**		−0.117*
		(978.182)		(0.069)
Herd PTA DPR		−1,608.360		0.018
		(1,367.826)		(0.148)
Herd Size	82.391***	81.258***	0.003***	0.003***
	(10.517)	(9.662)	(0.001)	(0.001)
Observations	214	214	214	214
Adjusted R^2	0.765	0.787	0.603	0.613

Note: *** p<0.01, ** p<0.05, * p<0.1. [1] We use the negative of the somatic cell count in order to maintain the interpretation that higher values mean higher health.

To understand the relationship between annual breeding expenditure at the farm level

Table A3: Breeding Expenditure and Genetic Quality Measures (Summed)

	Dependent Variable:			
	Breeding Expenditure, Level		Breeding Expenditure, Logarithm	
	(1)	(2)	(3)	(4)
PCA Production	−1.234		−0.0003***	
	(1.298)		(0.0001)	
PCA Health	6.084*		0.0003**	
	(3.443)		(0.0001)	
Herd PTA Milk		−98,776.720***		−4.037***
		(17,644.290)		(1.072)
Herd PTA Fat		53,371.880***		1.486
		(19,718.950)		(1.124)
Herd PTA Protein		33,528.420		1.185
		(22,020.540)		(1.751)
Herd PTA SCC[1]		12,305.090**		1.049***
		(4,806.982)		(0.335)
Herd PTA DPR		−1,028.596		0.005
		(1,569.777)		(0.108)
Herd Size	84.094***	86.404***	0.005***	0.005***
	(8.067)	(8.120)	(0.001)	(0.001)
Observations	214	214	214	214
Adjusted R^2	0.770	0.820	0.632	0.652

Note: *** p<0.01, ** p<0.05, * p<0.1. [1] We use the negative of the somatic cell count in order to maintain the interpretation that higher values mean higher health.

and these cow-level measures of genetic quality, Tables A2 and A3 present regressions of the average and sum of the indices of bulls that are purchased on breeding expenditure. Of the PCA components, only the health component correlates positively to breeding expenditure. Using the traits individually, PTA milk has a consistently negative coefficient due to the correlation between milk production and fat and protein production. Cows with the same fat and protein production but more milk production are worth less since they have higher feed requirements but the same fat and protein production. Alternatively, cows with the same milk production but higher fat production cost more according to

these results. This is in line with other hedonic analyses of dairy bull prices which find fat and protein production to be the strongest determinant of price (Richards and Jeffrey 1996; Schroeder et al. 1992). Measuring genetic quality using the average across cows results in a better SCC score (higher since we take the negative of it) correlating to lower expenditure, in contrast to previous hedonic analyses. If genetic quality is measured using the sum of the SCC trait then it correlates positively to expenditure, a results more in line with the literature.

Our results here illustrate a downside to using breeding expenditure as a proxy for genetic investment. It is tricky to compare the genetic indices of the bulls being purchases to the sum total of breeding expenditure for that year since one is at the cow level and one is at the herd level. Breeding expenditure can also be noisy, as it may involve purchases that are not related to genetic quality. In contrast, genetic indices are relatively straightforward to interpret and more clearly measure the genetic investments farms made that year.

Exit Probabilities

Olley and Pakes (1996) pioneered controlling for firm exit in the estimation of production functions by estimating the probability a firm would exit the sample using capital, firm age, and investment. Firm exit is especially a problem when studying the dairy sector because of the long-run trends towards consolidation. Figure A1 shows the number of farms in each year of our sample. In the span of ten years, the number of farms in our sample decreases by about 50%.

Our method adapts their approach but makes a significant contribution to the literature on production function estimation by using a random forest algorithm instead of OLS with polynomials, probit, or kernel estimation. Random forest has already been used in econometrics for estimation of causal treatment effects, notably the "causal tree model" of Wager and Athey (2018), but much less for the probability estimation. Random forest is a relatively lightweight superivesd machine learning algorithm that can be trained

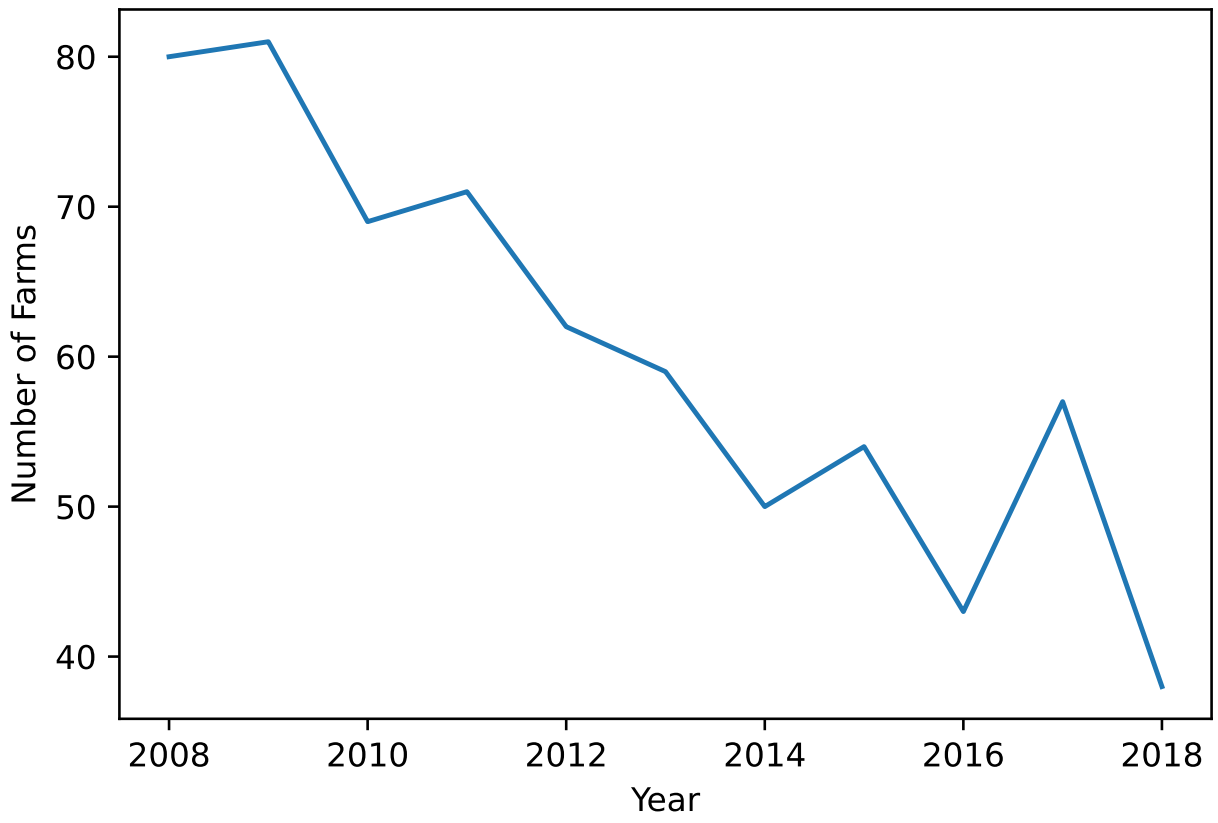
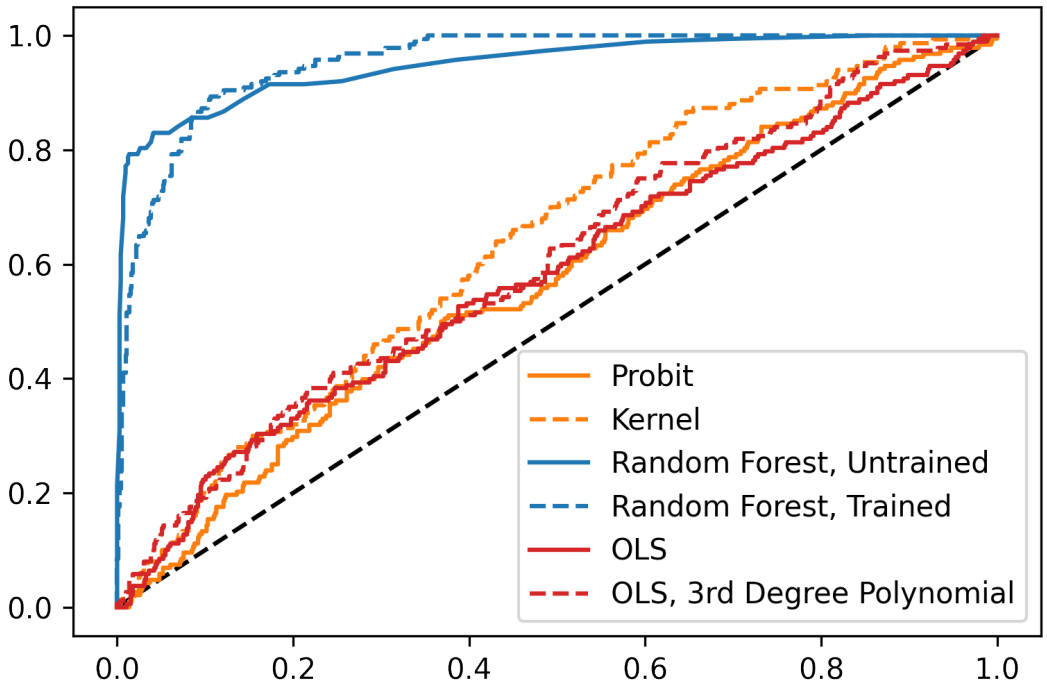


Figure A1: Number of Farms in Each Year

effectively to estimate probability distributions (Dankowski and Ziegler 2016; Niculescu-Mizil and Caruana 2005). We demonstrate a perfect use case for the random forest algorithm in economics, which is estimating probabilities which ultimately end up as corrective covariates in models.

In our case, we estimate probabilities using capital, the number of cows, and investment proxied by breeding expenditure. In Table A2 we benchmark six models: trained random forest, untrained random forest, OLS, OLS with third degree polynomials, probit, and kernel estimation. (Olley and Pakes 1996) uses kernel estimation and probit and benchmarked the methods using the correlation coefficients between the binary survival variable and their estimates. In our benchmarking, we include the correlation coefficient as well as two standard metrics used in supervised machine learning: Area Under the Curve (AUC) scores and Root Mean Squared Error (RMSE).

Figure A2: Exit Probability Estimators



	Random Forest Trained	Random Forest Untrained	OLS	OLS 3rd Degree Poly	Probit	Kernel
AUC Scores	0.955	0.950	0.581	0.604	0.575	0.636
RMSE	0.331	0.246	0.402	0.399	0.553	0.557
Correlation	0.719	0.819	0.094	0.162	0.178	0.096

Note: AUC measures the area under the AUC-ROC curve, which are plotted in the graph. The area under the curve measures the algorithm's ability to distinguish between classes (0s and 1s in this case), with higher scores denoting better peformance. RMSE is the square root of the average of the squared errors, with lower scores denoting better performance. The untrained random forest uses 50 trees with default parameters of a minimum of two samples for splitting and one sample per leaf. The trained random forest choose parameters using 5-fold cross validation and a randomized grid search over a range of parameters.

Even with relatively little training, the random forest significantly outperforms OLS, Probit, and Kernel estimation. In Olley and Pakes (1996), no model had a higher correlation coefficient than 0.6 (p. 1278). In our relatively small sample, both random forest probabilities have a correlation coefficient larger than 0.7 and significantly outperform the other algorithms in terms of AUC score.

Our results demonstrate some advantages the random forest in estimating probabilities from these sorts of firm data. First, the random forest is significantly less computationally-intensive than other supervised machine learning algorithms. Second, as demonstrated here, it needs relatively little training to take into account non-linearities. By nature, the random forest takes into account non-linearities in the relationship between the variables and the predicted outcome. This frees the researcher from having to make decisions about how many degrees of interaction to include. Also, including too many interactions run the risk of overfitting, a risk that can be avoided by doing out-of-sample cross validation.

In our analysis, we use the trained random forest probability predictions in all of our models. Supervised machine learning algorithms are not ideal for attempting to understand the structural relationships between variables because the underlying parameters are not inherently meaningful. However, supervised machine learning algorithms like random forest are absolutely ideal for cases where probabilities have to be estimated for selection indices and prediction accuracy is the only important performance metric. Future work could make a great contribution to the production function literature by understanding to what extent a better probability prediction using machine learning improves production function estimation.

2 TAXING FOR HEALTH: THE ENDURING BENEFITS OF IN UTERO CIGARETTE TAX EXPOSURE ON ADULT HEALTH

2.1 Introduction

Maternal smoking and exposure to second-hand smoke during pregnancy have long been known to be harmful to unborn babies. Tobacco smoke is a deadly mix of more than 7,000 chemicals and can damage the growing brain, lungs, arteries, and other delicate tissues (United States Department of Health and Human Services 2010). It is also the largest preventable cause of low birth weight, which is one of the best predictors of health in later life (Black et al. 2007; Case et al. 2005; Currie 2009; McEvoy and Spindel 2017). Governments have adopted a series of policies and regulations such as cigarette taxes, smoking bans in public places, youth access restrictions, warning labels on tobacco packaging, and bans on advertising, to fight against smoking and improve public health. Among them, cigarette taxes are viewed as the single most effective policy (World Health Organization 2015).

Researchers have found causal evidence that cigarette taxes reduce maternal smoking during pregnancy (Adams et al. 2012; Bradford 2003; Colman et al. 2003; Evans and Lien 2005) and that in utero exposure to cigarette taxes improves infant and childhood health (Evans and Lien 2005; Evans and Ringel 1999; Patrick et al. 2016; Simon 2016; Tominey 2007). Yet, very little is known about the effect of in utero exposure to cigarette taxes on long-term human capital accumulation. Because smoking is a behavior commonly linked to low socioeconomic status, and because cigarette smoke could be one channel of the intergenerational transmission of poor health and human capital outcomes, the long-term effect of in utero exposure to cigarette tax has profound implications for policymakers and public health interventions.

This paper explores the impact of in utero exposure to cigarette taxes on adult health. I employ a general difference-in-difference approach that allows me to control for unobserved

heterogeneity at both the year and state level. In addition, I apply an event study on a specific period to show the discrete impact of in utero exposure to cigarette tax hikes. Considering the recent criticism that a two-way fixed effects estimator could be biased in the case of heterogeneous treatment effects (Callaway et al. 2021; De Chaisemartin and d'Haultfoeuille 2020), I adopt a staggered DID estimator that is valid in the presence of such heterogeneity (De Chaisemartin and d'Haultfoeuille 2020). This allows me to estimate the effect on adult health of exposure to different discrete in utero cigarette tax levels.

I make use of the Panel Study of Income Dynamics (PSID) data spanning from 1968 to 2019. This dataset offers vital information on family backgrounds at the year of birth, childhood health and ability assessment, smoking behavior, and various other adult outcomes. I investigate the mechanisms underlying long-term health effects, such as the impact of taxes on parental smoking behavior during pregnancy, childhood cognitive and noncognitive ability, smoking behavior in adolescence and adulthood, educational attainment, and the age of first childbirth among individuals who were treated in utero. To investigate the underlying mechanisms, I also make use of the PSID-Child Development Supplements (PSID-CDS) data and the publicly available Vital Statistics Natality files.

I find that exposure to higher cigarette taxes while in utero significantly improves health in adulthood. A 10-cent higher tax that is in effect at the time of birth reduces the probability that individuals aged 25-35 have any of several health conditions (asthma, lung disease, heart disease or heart attack) by around two percentage points. This effect corresponds to eight percent of the mean, indicating a substantial reduction in the population who suffer from any of these conditions. Furthermore, the results suggest that the size of the beneficial health effect is increasing as people age. The results of the event study show that even exposure to a cigarette tax hike happened in the third trimester improves long-term health significantly. The results from the staggered DID estimator also support my findings. The results are stable across subgroups along key dimensions of demographic characteristics, family background, and different cohorts. The findings are also robust to a number of

specification tests.

I then proceed to explore the underlying mechanisms behind the long-term health effects. Using the PSID, I find that higher cigarette taxes reduce the probability of parental smoking, especially for parents who are about to have a baby, a result I then confirm using the Natality data. Moreover, higher cigarette taxes during the in utero period improve birth outcomes measured using average birth weight, average gestational age, average Apgar score, low Apgar score rate, very low birth weight rate, and preterm delivery rate. In addition, higher cigarette taxes during the in utero period are associated with better physical and neurological, psychiatric, and behavioral health in childhood (between ages 6 and 12), higher cognitive ability, a higher likelihood of obtaining a college or higher degree, a lower probability of smoking, and reduced smoking intensity for smokers in both adolescence and adulthood. Lastly, I offer evidence that higher cigarette taxes during the in utero period are associated with postponed age of first childbirth for individuals who were treated in utero, which is positively correlated with better health (Lee and Park 2020; Shadyab et al. 2017).

To the best of my knowledge, this paper is the first to investigate the long-run impact of early-life exposure to cigarette taxes on health. Most previous studies in the field focus on contemporaneous impacts of cigarette taxes, such as the association between cigarette taxes and pregnant women's smoking behavior (Adams et al. 2012; Bradford 2003; Colman et al. 2003; Evans and Lien 2005), the association between cigarette taxes in early life and infants' and children's health (Evans and Lien 2005; Evans and Ringel 1999; Patrick et al. 2016; Simon 2016), and the association between cigarette taxes in early life and educational attainment of adolescents (Settele and Van Ewijk 2018). In the study most closely related to mine, Hoehn-Velasco et al. (2023) find that, when girls who were exposed to cigarette taxes in early life grow up, they have lower rates of pre-pregnancy and prenatal smoking and are less likely to be overweight or obese during pregnancy. These are the only adult health outcomes or behaviors of which I am aware that have been studied in terms of the

effects of early-life exposure to cigarette taxes.

This paper also contributes to the literature on the long-run impacts of early life environment.[1] These studies provide evidence that early life environments are critical for the development of human capital. Earlier, much of this literature exploited variation from natural disasters and disease (Almond 2006; Barreca 2010; Lindeboom et al. 2010). More recently, studies have looked at policy experiments such as the Earned Income Tax Credit (EITC), food stamps, the supplemental nutrition program for Women, Infant and Children (WIC), and alcohol availability (Bastian and Michelmore 2018a; Hoynes et al. 2016b; Hwang 2019; Nilsson 2017). I extend the literature by examining the impact of a policy that is inexpensive to implement and generates new revenues. Moreover, most policy experiments usually happen within a short period of time, but people are exposed to cigarette taxes for a much longer duration, which allows us to compare the effect of exposure to cigarette taxes at different periods of the lifecycle. My findings reveal that in utero exposure to cigarette taxes has a greater impact on adult health than exposure to cigarette taxes in childhood or adulthood.

Finally, this study has important policy implications. First, this research underscores the enduring impact of early-life policy interventions, suggesting that, when evaluating the benefits of various interventions, policymakers should consider not just immediate outcomes but also longer-term effects. Second, given that in utero cigarette taxes are found to have significant long-term health benefits, governments could consider increasing these taxes. Third, the link between higher cigarette taxes while in utero and improved physical and neurological, psychiatric, and behavioral health, enhanced cognitive ability, higher education, and reduced smoking in both adolescence and adulthood suggests that such tax policies should be a critical component of broader public health strategies. Lastly, my findings suggest that early-life environments play a crucial role in the development of human capital. Policymakers therefore might investigate other potential early-life inter-

[1] See, e.g., Currie (2009) and Currie and Almond (2011) for reviews of this literature.

ventions that could yield similar long-term benefits. For example, there could be potential in studying the long-term effects of taxes on other harmful substances, such as alcohol or sugary beverages, and their association with health and developmental outcomes.

The remainder of the paper is structured as follows. Section I summarizes the medical literature on the effects of exposure to tobacco smoke during pregnancy and the economic literature on the effects of cigarette taxes. Section II presents the theoretical framework. Section III describes the data and Section IV presents the empirical model. Section V shows the results. I conclude in Section VI.

2.2 Background and Previous Research

Maternal smoking during pregnancy is harmful to unborn babies. According to the Surgeon General's reports, "the nicotine in cigarettes may cause constrictions in the blood vessels of the umbilical cord and uterus, thereby decreasing the amount of oxygen available to the fetus. Nicotine also may reduce the amount of blood in the fetal cardiovascular system" (United States Department of Health and Human Services 2004, p.564). Moreover, smoking during pregnancy can cause tissue damage in the unborn baby, particularly in the lung and brain (United States Department of Health and Human Services 2010). Maternal smoking during pregnancy leads to worse birth outcomes. Infants born to mothers who smoke during pregnancy are more likely to be small for gestational age and have a lower average birth weight than infants born to women who do not smoke during pregnancy; low birth weight, in turn, is associated with increased risk for neonatal, perinatal, and infant morbidity and mortality. Environmental tobacco smoke (ETS) is also harmful to the fetus. Compared to infants born to women who are not exposed to ETS, infants born to women who are exposed to ETS during pregnancy may have a small decrease in birth weight and a slightly increased risk for intrauterine growth retardation. The timing of exposure is important. Smoking in the third trimester is particularly detrimental, while infants of

mothers who stop smoking by the first trimester have birth weights and body measurements comparable with those of mothers who do not smoke (United States Department of Health and Human Services 2001).

Some studies further suggest that maternal smoking during pregnancy is associated with health and behavior problems in later life. Children whose mothers smoked during pregnancy are at a higher risk for a wide range of problems such as overweight, asthma, lung disease, attention deficit hyperactivity disorder, and behavioral problems (Gilliland et al. 2000, 2001; Milberger et al. 1996; Oken et al. 2008). Associations also have been found between maternal smoking during pregnancy and substance abuse and criminal outcomes in adulthood (Brennan et al. 1999).

Moreover, maternal smoking during pregnancy, after controlling for postnatal smoking, increases the risk that children will smoke and develop nicotine dependence when they grow up (Biederman et al. 2017; Kandel et al. 1994; Lieb et al. 2003). Smoking leads to health problems and harms nearly every organ of the body (Lushniak et al. 2014). Young people who smoke are at risk of addiction to nicotine, reduced lung growth and lung function, and early cardiovascular damage (Centers for Disease Control and Prevention 2012). The risk increases when smoking continues for many years. Smoking can cause more severe health conditions such as lung disease, heart disease, stroke, cancer, diabetes, and chronic obstructive pulmonary disease, while it increases risk for tuberculosis, certain eye diseases, and problems of the immune system (Lushniak et al. 2014).

However, maternal smoking during pregnancy is preventable. Prevention policies such as cigarette taxes, smoking bans in public places, youth access restrictions, and warnings about the dangers of tobacco have been adopted by governments. Among them, raising taxes on tobacco is the single most effective way to reduce smoking. There are several reasons for this. Higher tobacco taxes and prices reduce consumption and promote quitting; a tax increase is inexpensive to implement; taxation is especially effective in reducing tobacco use by young people, who are very price sensitive; and a tax increase generates new

revenues, which can support tobacco control and other health initiatives (World Health Organization 2015).

Economic literature has provided causal evidence that cigarette taxes improve children's health at birth. Using data from the 1989-1992 Natality files, Evans and Ringel (1999) find that a one-cent increase in real cigarette tax during the conception month (in 1982-4 dollars) leads to a reduction of around 0.08 percentage points in the smoking rate for pregnant women and an increase of around 0.21 grams in birth weight, a finding largely supported by the literature (Adams et al. 2012; Bradford 2003; Colman et al. 2003; Evans and Lien 2005; Patrick et al. 2016).

Moreover, economists have found that cigarette tax during pregnancy has impacts on children in later life. For the medium-term effect, using data from the National Health Interview Survey (NHIS), Simon (2016) finds that a one-dollar higher real tax during the first month of the third trimester leads to a 10 percent decrease in sick days from school and around a 5 percent decrease in the probability of having two or more doctor visits per year, for children born from 1988 to 2009. Settele and Van Ewijk (2018) find that higher cigarette taxes during pregnancy improve educational attainment for children born between 1988 and 1998 with mothers who have less than high school education. These studies suggest that the effects of cigarette tax during pregnancy on health in childhood and educational attainment in adolescence can be mediators for long-term health effects. For the long-run effect, Hoehn-Velasco et al. (2023) find that a one-percent higher cigarette tax during the in utero period reduces the probability of any smoking prior to conception for pregnant women by 0.21% and reduces the probability of prenatal smoking by 0.24%. They also find that higher cigarette taxes are associated with a lower pre-pregnancy BMI and a lower likelihood of developing diabetes before or during pregnancy.

My work makes important contributions to this literature by providing the first empirical evidence on the long-term health effects of exposure to higher in utero cigarette taxes.

2.3 Theoretical Framework

Cunha and Heckman (2007) provide a theoretical framework for the production of an individual's human capital. Their model provides theoretical evidence on how cigarette taxes in the prenatal period could have long-term health impacts. Human capital refers to cognitive ability, noncognitive ability, and health condition. Denote an individual's human capital at period t as $\theta_t \equiv (\theta_t^C, \theta_t^N, \theta_t^H)$, where θ^C refers to cognitive ability, θ^N refers to noncognitive ability, and θ^H refers to health condition. Based on Cunha and Heckman (2007), I model human capital as being produced by parental characteristics, investment, and environment (I add the environment to the original Cunha-Heckman model in order to understand the impact of cigarette tax). Each period's human capital depends on the capital in the previous stage, investment in human capital, environmental quality in the previous stage, and parental characteristics. Let h denote parental characteristics, I_t denote the investment at period t, and E_t denote the environmental quality at period t. The human capital at period $t+1$ is presented by

$$\theta_{t+1} = f_t(h, \theta_t, I_t, E_t). \tag{2.1}$$

The human capital θ_{t+1} increases with investment and environment in the last period – that is, $\frac{\partial f_t}{\partial I_t} > 0$ and $\frac{\partial f_t}{\partial E_t} > 0$. Repeatedly substituting Equation 2.1 for θ_{t-1}, θ_{t-2}, ..., the human capital at period $t+1$ can be written as a function of parental characteristics, initial human capital, environmental quality, and investments in all the past periods from the in utero period to period t.

$$\theta_{t+1} = m(h, \theta_1, E_1, \ldots, E_t, I_1, \ldots, I_t).$$

This framework captures three features: the "sensitive and critical period," "self-productivity," and "dynamic complementarity" in the development of children's human

capital (Cunha and Heckman 2007).

The "sensitive period" refers to periods that are more important than others in producing certain kinds of human capital. The "critical period" refers to the single period that is most effective in producing a particular kind of human capital. For example, the prenatal period is a sensitive period for the development of the brain and lungs and other organs. This implies that the in utero environment has a larger effect than the later-life environment in human capital development, i.e., $\frac{\partial f_t}{\partial E_1} > \frac{\partial f_t}{\partial E_s}$ where $s > 1$.

"Self-productivity" suggests that individuals with higher human capital are more productive and accumulate more capabilities. For example, emotionally stable children are more focused on learning activities and accumulate more human capital, i.e., $\frac{\partial f_t}{\partial \theta_t} > 0$. This characteristic indicates that the difference in human capital expands over time if all other factors stay the same.

Another feature of capability development captured by the model is "dynamic complementarity." That is, individuals with higher human capital gain a higher return from investment and environmental improvement. For example, healthy children can be more productive in learning activities compared to children with attention deficit/hyperactivity disorder (ADHD), i.e., $\frac{\partial^2 f_t}{\partial \theta_t \partial I} > 0$. This feature indicates that the difference in human capital expands faster with investment or environmental improvement.

Cigarette taxes can lead to two distinct outcomes. On the one hand, they tend to decrease smoking behaviors. On the other hand, for individuals heavily addicted to cigarettes, who may have a reduced response to cigarette taxes, higher taxes may result in increased expenditures on cigarettes. Consequently, whether exposure to higher cigarette taxes during pregnancy is beneficial is a question that requires empirical investigation. Previous studies generally indicate that the first effect, which is the reduction in smoking, prevails. Research has demonstrated that higher cigarette taxes reduce smoking behavior for pregnant women, thus leading to an improved uterine environment during pregnancy. Denote the in utero period to be period 1, that is $\frac{\partial E_1}{\partial \text{CigTax}_1} > 0$. Because the uterine environment has a strong

effect on the development of critical organs such as the brain and lungs, as well as the general health of the fetus, cigarette taxes during pregnancy directly affect human capital in early life, i.e. $\frac{\partial \theta_2}{\partial \text{CigTax}_1} = \frac{\partial f_1}{\partial E_1} \cdot \frac{\partial E_1}{\partial \text{CigTax}_1} > 0$. The effect lasts over time because individuals with higher early-life human capital are more productive in accumulating later-period capability according to the "self-productivity" theory. The corresponding inequality is $\frac{\partial \theta_t}{\partial \text{CigTax}_1} = \frac{\partial f_{t-1}(\theta_{t-1}, I_{t-1}, E_{t-1}, G)}{\partial \text{CigTax}_1} = \frac{\partial f_{t-1}}{\partial \theta_{t-1}} \cdot \frac{\partial \theta_{t-1}}{\partial \text{CigTax}_1} = \frac{\partial f_{t-1}}{\partial \theta_{t-1}} \cdot \frac{\partial f_{t-2}}{\partial \theta_{t-2}} \cdots \frac{\partial f_1}{\partial \theta_1} \cdot \frac{\partial \theta_1}{\partial \text{CigTax}_1} > 0$. The effects could even be strengthened as individuals with higher early-life human capital gain higher returns from investment and environmental improvement according to the "dynamic complementarity" theory, i.e. $\frac{\partial^2 \theta_t}{\partial \text{CigTax}_1 \partial I_s} = \frac{\partial f_{t-1}}{\partial \theta_{t-1}} \cdot \frac{\partial f_{t-2}}{\partial \theta_{t-2}} \cdots \frac{\partial f_s}{\partial I_s} \cdot \frac{\partial f_s}{\partial \theta_s} \cdots \frac{\partial f_1}{\partial \theta_1} \cdot \frac{\partial \theta_1}{\partial \text{CigTax}_1} > 0$. Moreover, in utero cigarette taxes may indirectly affect investment in later periods, if poor initial health requires increased expenditures of money and time on health care, which reduce the budget and time available to invest in human capital development (I). The theoretical model implies that increasing cigarette taxes in early life affects individuals' initial human capital; the effect can last long into adulthood and can get stronger with age.

Cigarette taxes in childhood, adolescence, and early adulthood may also have effects on health in adulthood. However, the effect should be smaller than the effect of in utero cigarette tax, according to the "sensitive period" theory, i.e. $\frac{\partial f_t}{\partial \text{CigTax}_1} = \frac{\partial f_t}{\partial E_1} \cdot \frac{\partial E_1}{\partial \text{CigTax}_1} > \frac{\partial f_t}{\partial \text{Cigtax}_s} = \frac{\partial f_t}{\partial E_s} \cdot \frac{\partial E_s}{\partial \text{CigTax}_s}$, assuming that the effect of cigarette taxes on the environment does not change over time, i.e. $\frac{\partial E_1}{\partial \text{CigTax}_1} = \frac{\partial E_s}{\partial \text{CigTax}_s}$.

I borrowed Cunha and Heckman (2007)'s theoretical framework to explore how cigarette taxes might affect long-term human capital accumulation. The framework yields three primary forecasts. Firstly, exposure to in utero cigarette taxes directly improves the environment and enhances human capital in early life. Moreover, the early life effect persists into adulthood and may expand over time due to the properties of self-productivity and dynamic complementarity. Lastly, because the in utero period is a sensitive period for physiological development, exposure to cigarette taxes in this period exhibits a larger impact on human capital accumulation than cigarette taxes in later life.

2.4 Data

Cigarette Taxes

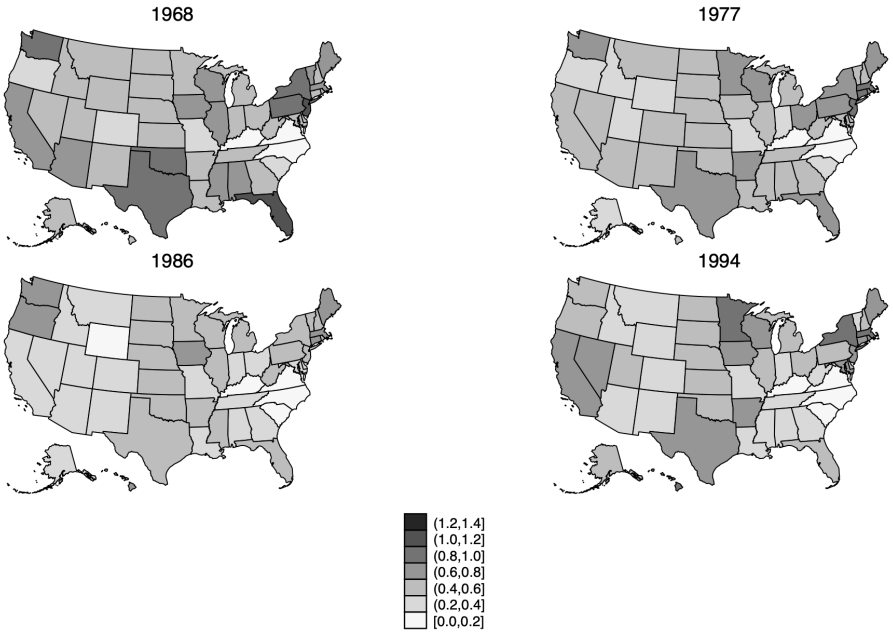
Cigarette taxes can be implemented by federal, state, and municipal governments. Because federal cigarette tax rarely changes and municipal cigarette taxes are not common, I focus on state excise taxes. Figure 2.1 shows the variation of state cigarette taxes between 1968 and 1994. States in the tobacco belt, such as North Carolina, Virginia, Kentucky, and South Carolina, have the lowest state cigarette taxes. Conversely, Connecticut, New Jersey, Minnesota, Florida, and Massachusetts tend to impose relatively high state cigarette taxes. I trim the states with very high or low average state cigarette taxes to test the robustness of the results. During the sample period, the average nominal state cigarette tax increased from \$0.08 in 1968 to \$0.27 per pack in 1994. The smallest increment was 1 cent, and the largest increment (in Washington D.C. in 1993) was 33 cents. The average real state cigarette tax in this period in 2020 dollars[2] varies between \$0.37 and \$0.71. There were 186 increases in real state cigarette tax during the period. Among them, 63% were equal to or greater than 10 cents. The largest change was an increase from 31 cents in 1992 to 90 cents in 1993 in Washington D.C. The average cigarette tax during 1968 and 1994 was 50 cents, while the average price for a pack of cigarettes before taxes was \$2.5.

Panel Study of Income Dynamics (PSID)

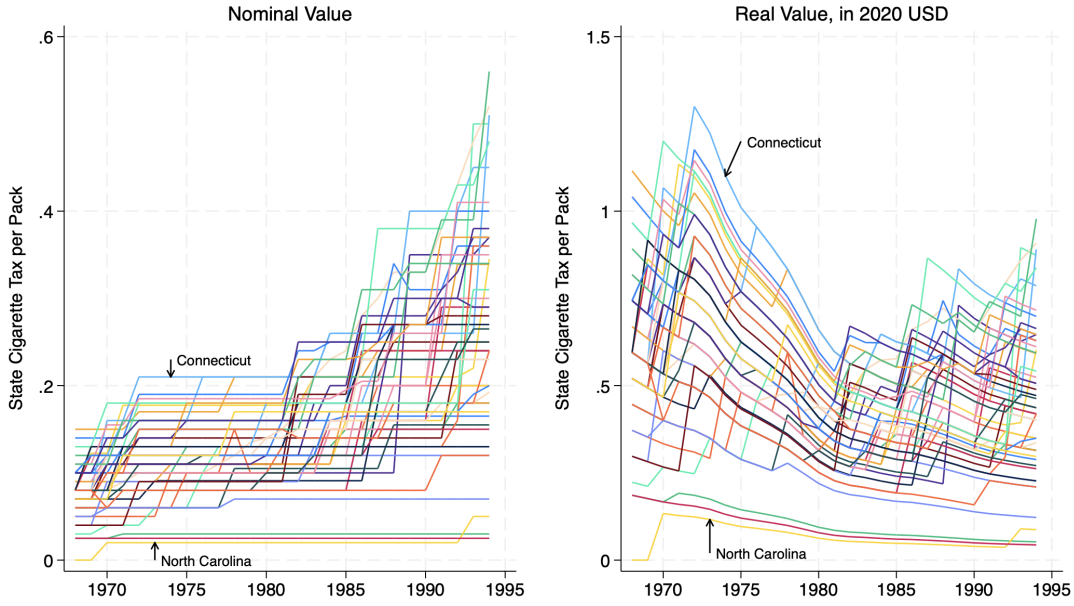
PSID is a longitudinal household survey that started in 1968 with a nationally representative sample of around 5,000 families in the United States. It surveyed household heads and spouses and their offspring annually from 1968 to 1997 and then biennially from 1997 on.

Three unique features of the PSID make it the perfect dataset for this study. Firstly, for respondents in PSID, I have detailed family background information, including the educational level of the mother, whether the mother was older than 35 when she gave birth,

[2]All the values below are 2020 dollars without further notation.



(a) Real (in 2020 Dollar) Cigarette Tax per Pack in Selected Years from 1968 to 1994



(b) Nominal and Real (in 2020 Dollar) Cigarette Tax per Pack from 1968 to 1994

Figure 2.1: Variation of State Cigarette Taxes

Source: Author's calculation. The data for cigarette taxes is obtained from The Tax Burden on Tobacco, and CPI data is sourced from the U.S. Bureau of Labor Statistics.

Note: Figures show the state cigarette tax per pack for all 50 states and the District of Columbia.

gender and marital status of the household head, number of children in the family, and the family income-Census needs standard ratio[3] in the year of birth. Moreover, in 1999, the PSID started collecting health information and therefore can provide detailed information on health status and health behavior for the household heads and spouses. In addition to health in adulthood, I have other adult performance information, such as education and fertility, for the individuals I study.

PSID-Child Development Supplement Data

I utilize PSID's Child Development Supplement (CDS) data for the analysis of the effect of cigarette taxes on health and cognitive ability in childhood, as well as smoking behavior in adolescence. CDS data is available for 1997, 2001, 2007, 2014, and 2019, providing valuable information on child development for children aged 0 to 17. I can therefore use the CDS data to explore the effect of early-life exposure to cigarette tax on childhood development as one potential underlying mechanism. The CDS started in 1997, and information was gathered on children between ages 0 and 12. For each PSID family, data was gathered for CDS on one or two eligible children. These children were followed in the surveys in 2001 and 2007 until they turned 18. In the CDS ongoing waves in 2013 and 2019, data was gathered on all the children aged 0 to 17 in the PSID families. CDS collects information on chronic physical conditions, including anemia, allergies, asthma, diabetes, ear disease, hearing difficulty, eye disease, and obesity,[4] and chronic neurological, psychiatric, or behavioral conditions, including seizure disorders, autism, speech problems, learning disabilities, behavioral disabilities, and hyperactivity for children between ages 0 and 18. I generate four health indices based on these health conditions: 1) whether the child has any chronic physical condition; 2) whether the child has any chronic neurological or psychiatric condition; 3) how many chronic physical conditions the child has; and 4) how many chronic neurological

[3]According to the PSID codebook, the Census needs standard is a poverty threshold by size of family, the number of persons in the family under 18, and the age of the householder; it is taken from the Census Bureau's website. The ratio can be seen as an adjusted measure of family income.

[4]The question about obesity was not asked in 1997.

or psychiatric conditions the child has. CDS also collects cognitive ability information, including scores on Woodcock-Johnson tests taken by children between ages 6 and 18, and collects information on smoking behavior for adolescents between ages 12 and 18.

Vital Statistics Natality Files [5]

I also use the US Natality birth data maintained by the National Center for Health Statistics (NCHS) from 1969 to 1994 to study the effect of cigarette taxes during pregnancy on the maternal smoking rate[6] and birth outcomes. Birth outcomes include average birth weight, very low birth weight rate (birth weight $<$ 1500 gram), average gestational age, preterm delivery rate (gestational age $<$ 28 weeks), average Apgar score, and low Apgar score rate (Apgar score $<$ 7). I do not include data from 1968 because limited information was recorded in 1968. Natality birth data record information collected from state birth certificates, including birth year, birth month, birth state, birth order, and the age, race, marital status, and educational level of the mother. I generate cell-level outcome variables following Baughman and Dickert-Conlin (2009).

Sample Selection

The main sample consists of household heads and spouses aged 25-35 who were born to PSID families between 1968 and 1994.[7] This age range was selected based on several considerations. One reason is that most respondents assumed the roles of household heads or spouses after the age of 25. Moreover, individuals born in 1968 were first posed health-related questions at the age of 31 in 1999 (when the PSID started collecting health information, and when household heads and their spouses were first queried about whether a medical professional had ever diagnosed them with asthma, lung disease, heart disease,

[5]The individual level Natality birth data from 1969-1994 can be downloaded from the website: https://www.nber.org/research/data/vital-statistics-natality-birth-data.

[6]The data on smoking behavior during pregnancy is not available in the Natality Files until 1989.

[7]Other members of the household are not included because the PSID only collects health information from the household heads and spouses.

or heart attack). Therefore, any upper age limit below 31 would exclude the 1968 cohort. By setting the upper limit at 35, I ensure that this 1968 cohort had three opportunities to participate in the survey between the ages of 25 and 35. This flexibility is crucial given that some became household heads or spouses after the age of 31 and may not have consistently participated in the PSID survey. This relatively narrow age range is chosen so that I can focus on an age group where health conditions exhibit limited variability. However, I have conducted robustness checks with different age ranges and the results are robust.

Only individuals born to PSID families after 1968 (the first year of the PSID data) are included in the sample because I need information on birth state and family background at birth. Additionally, because the latest released PSID survey at the time of writing this paper is the 2019 wave, individuals born after 1994 (and therefore younger than 25 in 2019) are excluded from the main sample.

In the process of refining the sample, I adhered to a systematic series of steps to ensure that the most relevant observations were included. Initially, I retained observations of household heads and spouses born to families in the 1968 sample between 1968 and 1994, who were between the ages of 25 and 35. This resulted in a sample size comprising 5,791 unique IDs. Further refinement involved removing those observations that were not part of at least one survey conducted by PSID during the ages of 1 to 18, narrowing the sample to 5,645 unique IDs. Next, any observation lacking a complete record of state of residence between the ages of 1 to 18 was excluded. The primary rationale was the inability to ascertain the cigarette tax during the birth year, even if I assumed that they did not relocate before turning 18. This step led to a slight reduction in the sample size to 5,644 unique IDs. Essential demographic information was also crucial for the analysis. Hence, observations were excluded if data were unavailable on race, the age of the mother at birth, or the mother's educational background. This brought the sample down to 5,609 unique IDs. Because health conditions are integral to my study, observations lacking information on specific health conditions (asthma, heart attack, heart disease, and lung conditions) were

removed, resulting in 5,418 unique IDs. Finally, to ensure the accuracy and relevance of the data, observations without clear information on the state of residence between the ages of 25 and 35 were dropped. Consequently, the final sample encompassed 5,402 unique IDs originating from 1,494 families surveyed in 1968.

The CDS sample consists of children born between 1989 and 2013. I exclude those born after 2013, because they were under age six during the most recent CDS survey conducted in 2019. Children born prior to 1989 were not considered for this study either, primarily because the question concerning obesity was introduced only in the 2001 survey. By that time, those born before 1989 were already older than 12. My analysis specifically targets the health conditions observed between ages 6 and 12, aiming to focus on a period when health conditions are relatively consistent across the age group.

Sample Statistics

Table 2.1 offers a comprehensive view of the weighted summary statistics for the estimation sample. Each observation in the sample is a unique individual. Upon analyzing the data, it becomes evident that approximately 24% of the sampled individuals had experienced at least one of the health conditions – asthma (18%), lung disease (7%), heart disease (2%), or heart attack (1%) – when they were surveyed between the ages of 25 and 35.

Demographically, the sample paints a diverse picture. Nearly half of the sample are male (48%). White individuals account for 81% of the sample, followed by Black individuals at 17%. The average age at which they were last surveyed during the 25 to 35 age bracket is 32 years. Eighteen percent were born to mothers aged over 35. Maternal education also provides interesting insights: 21% of the sampled individuals were born to mothers who hadn't completed high school. In contrast, 60% had mothers with educational attainments spanning from high school to some college, and 19% had mothers with a college degree or a more advanced educational background.

Diving deeper into familial structures, 14% hailed from female-headed households,

whereas 84% were born into two-parent families. The order of birth also plays a role in the analysis: 59% represent the eldest child in their family, 24% hold the position of second child, and the remaining 18% are either the third child or born later in the birth order. The average family income at the time of birth, measured as a ratio of the Census needs standard budget, stood at 3.56 (i.e., nearly four times the poverty level).

Table 2.1: Summary Statistics

Variable	Mean	Std. Dev.	Min	Max
Health Condition between Ages 25-35				
Any Health Condition = 1[1]	0.24	0.43	0	1
# of Health Conditions[2]	0.28	0.53	0	3
Asthma = 1	0.19	0.39	0	1
Lung Disease = 1	0.07	0.25	0	1
Heart Disease = 1	0.02	0.14	0	1
Heart Attack = 1	0.00	0.07	0	1
Demographics				
Birth Year	1981.20	7.20	1968	1994
Male	0.48	0.5	0	1
White	0.81	0.39	0	1
Black	0.17	0.37	0	1
Other Race	0.03	0.16	0	1
Age	32.43	2.97	25	35
Family Background at Birth Year				
Mother's Age >35	0.18	0.39	0	1
Mother Less than High School Education	0.21	0.41	0	1
Mother High School or Some College Education	0.60	0.49	0	1
Mother College or Above Education	0.19	0.39	0	1
Female-Headed Household	0.14	0.35	0	1
Married Parents	0.84	0.37	0	1
First Kid	0.59	0.49	0	1

Variable	Mean	Std. Dev.	Min	Max
Second Kid	0.24	0.43	0	1
Second+ Kid	0.18	0.38	0	1
Income to Needs Ratio	3.56	2.66	0	25.05
Cigarette Taxes and Regulations				
In Utero Cigarette Taxes (2020$)	0.50	0.23	0	1.31
Cigarette Tax at Age 25 (2020$)	1.21	0.98	0.04	5.00
Minimum Legal Age for Purchasing Tobacco Products	17.89	0.86	15	24
Substitutes				
State-Level Beer Tax Per 31 Gallon (2020$)	53.28	28.71	20.28	231.37
State-Level Wine Tax Per Gallon (2020$)	2.51	3.44	0.34	47.21
State-Level Spirit Tax Per Gallon (2020$)	31.20	28.71	0	107.83
Economic, Welfare and Medical Policies/Conditions				
Minimum Wage (2020$)	5.75	3.49	0	13.42
Maximum Marginal Income Tax Rate	5.63	4.10	0	21.8
Per Capita GDP (1,000, 2020$)	38.83	7.65	21.00	148.47
EITC Benefit (2020$)	1,402.32	912.53	0	6,621.93
CDCTC Benefit (2020$)	2,530.95	1,575.97	0	7,483.37
AFDC/TANF Benefit (2020$)	907.03	380.11	171.11	2158.94
Number of Hospital Beds (1,000)	48.56	37.47	1.30	206.4
Expenditures on Medicaid and Similar Programs (1,000, 2020$)	2807.07	3467.70	0	24999.96

Observations	5,402

Note: Authors' tabulations based on 1968-2019 PSID data. The main sample consists of individuals born into 1968 PSID families between 1968-1994 who became household heads or spouses between ages 25-35 during later PSID surveys. State cigarette taxes are obtained from The Tax Burden on Tobacco. The minimum legal age for purchasing tobacco products is collected through Nexis Uni database. Alcohol taxes are from the Alcohol Policy Information System (Ponicki 2004). The minimum wage is from the U.S. Department of Labor. The maximum marginal income tax rate is from the Council of State Governments. The per capita GDP is calculated using state GDP from the U.S. Bureau of Economic Analysis and state population from the U.S. Census Bureau and Fred Economic Data. The EITC benefit is from the National Bureau of Economic Research. The CDCTC benefit is collected through the Nexis Uni database and Hein Online database. The AFDC/TANF benefit is from legislative history references from U.S. Government Publishing Office. The number of hospital beds and welfare vendor payment for medical are from the U.S. Census Bureau. [1]1(Any health condition) = 1 if the individual has any of the following health conditions: asthma, lung disease, heart disease, and heart attack. [2]# of health conditions = the number of health conditions the individual has out of asthma, lung disease, heart disease, and heart attack.

Shifting the focus to policy and economic indicators, several data points emerge. The average in utero cigarette tax, which is determined either at the close of the third trimester or at the birth month, is calculated at $0.50. By the time these individuals reached 25, the average cigarette tax had increased to $1.21. Concurrently, the average state minimum legal age for tobacco product purchases was 17.89.

Economic indicators during the year of birth reveal that the state beer tax per 31 gallons was $53.28, the state wine tax per gallon was $2.51, and the state spirit tax per gallon was $31.20. The state minimum wage, on average, equaled $5.75. Other financial parameters include an average marginal income tax rate of 5.63%, a per capita GDP of $38,830, and an array of benefits: the average maximum Earned Income Tax Credit (EITC) was approximately $1,402 per year, the Child and Dependent Care Tax Credit (CDCTC) hovered around $2,530, and the Aid to Families with Dependent Children/Temporary Aid to Needy Families (AFDC/TANF) benefit was roughly $907 per month. The health infrastructure, as represented by the total number of hospital beds, was 48.56 thousand on average. Lastly, the average state expenditure for Medicaid and similar programs stood at about $2,807 million.

2.5 Empirical Strategy

Difference-in-difference

We utilize a generalized difference-in-difference approach with continuous treatment. The main specification for the baseline model is as follows:

$$y_{isym} = \alpha + \delta_1 CigTax_{sym} + \delta_2 CigTax_{(s,age25)} + X_{isym}\beta_1 + Z_{sy}\beta_2 + \lambda_y + \eta_s + \epsilon_{isym}. \quad (2.2)$$

The term y_{isym} refers to the health outcomes, measured during adulthood, for individual i born in state s in year y and month m. The main explanatory variable is $CigTax_{sym}$, which captures the real state cigarette tax in state s at birth. Birth time refers to the birth year-month; as noted above, cigarette smoking has the strongest effect on birth outcomes during the last trimester of pregnancy (United States Department of Health and Human Services 2001). $CigTax_{(s,age25)}$ captures the real cigarette tax in individual i's state of residence when s/he is 25.

The term X_{isym} represents a vector of individual-level characteristics, including gender, race (white and nonwhite), age squared at survey year, and family background at birth year, including the educational level of the mother (dropout, high school or some college, college and beyond), whether the mother was over 35 when she gave birth to the individual, gender of the household head, marital status of the household head, number of children in the family, and family income-Census needs standard ratio.

The term Z_{sy} represents a vector of state-level covariates at birth year, including minimum legal age for purchasing tobacco products, alcohol taxes (including tax rates for beer, spirits, and wine), social welfare benefits (including EITC benefit, CDCTC benefit, and AFDC/TANF benefits), economic status and policies (including the minimum wage, the maximum marginal income tax rate, and per capita GDP), and health care investment (including state expenditure for Medicaid and similar programs and the number of hos-

pital beds). These state-level covariates also affect children's early-life environment and long-term health outcomes.

The terms λ_y and η_s are birth year and birth state fixed effects, respectively. The birth year fixed effect controls for factors that change each year, such as the size of the cohort and public awareness about the harm of cigarette smoking. The state fixed effect controls for relatively time-invariant state characteristics, such as culture and climate.

I use the average PSID longitudinal weights during ages 1 to 18 in the estimation and cluster the standard errors by state of birth to account for unobserved correlation of the error terms within states.

The analysis relies on two sources of variation: cross-state variation in cigarette taxes in a particular year and within-state changes in cigarette taxes over time. We use state-level cigarette taxes as the treatment variable for several reasons. Firstly, the state-level cigarette tax affects the cost of consuming cigarettes in a given state; this could, in turn, influence the smoking behavior or exposure to secondhand smoke of pregnant women. Thus, there is a plausible causal relationship between state-level cigarette taxes and in utero exposure to tobacco smoke. Secondly, the variation in cigarette taxes at the state level is exogenous to the specific individual within states. Finally, a Two-Way Fixed Effects (TWFE) approach enables us to control for unobserved heterogeneity at both the year and state level. This helps address potential sources of bias, such as omitted variable bias, which could arise if we only examined the relationship between parental smoking behavior (or tobacco smoke in the environment during pregnancy) and adult health for the grown children at the individual level. However, it is still possible that the cigarette taxes are endogenous and could bias the results. We address these issues in the next section.

Threat to Identification

The identification depends on the exogeneity of cigarette taxes, conditional on the individual-level and state-level controls. The literature has explored what drives an increase in cigarette

taxes. Originally, legislatures enacted tobacco taxes primarily to boost state revenue. As the public awareness of the harm of smoking and second-hand smoke grew, states have also employed taxes to reduce cigarette consumption (Gruber 2001). However, considering that legislation usually takes years to happen, it is unlikely that a cigarette tax increase is correlated with a sudden shift of public awareness (Gruber and Köszegi 2001).

Another potential concern is whether changes in state cigarette taxes are influenced by state demographics, economic policies, or economic conditions that also impact health conditions. For example, if states with more highly educated people tend to adopt higher cigarette taxes, and if cigarette taxes tend to have a larger impact on better-educated people, then estimates of the effect of cigarette tax on health would be biased upward. If states with higher per capita GDP tend to adopt higher cigarette taxes, and if cigarette taxes tend to have a smaller impact on people with higher income, then estimates of the effect of cigarette tax on health would be biased downward. If states raise cigarette taxes to finance public health care or public welfare programs which improve public health, then estimates of the effect of cigarette tax on health would be biased upward.

To address these concerns, I conduct formal tests to examine the correlation between state cigarette taxes and state demographics, economic policies, and economic conditions. Column 1 in Table 2.2 presents the results of regressing the cigarette tax on the state-by-year characteristics (including percent male, percent white, percent married, percent high school or some college, percent with college or higher education, average age, state per capita GDP, state minimum wage, state marginal income tax rate, maximum AFDC/TANF benefit, maximum EITC benefit, maximum CDCTC benefit, number of hospital beds, state expenditures for Medicaid and similar programs, state beer/wine/spirits excise tax, and minimum legal age for purchasing tobacco products). In column 2, state fixed effect is included as a control, and in column 3, year fixed effect is added as an additional control. The difference between column 3 and the last column is that column 4 use cigarette tax rates at period $t + 1$ instead of period t as the outcome variable. Overall, the model can explain

more than 80% of the variation in state cigarette tax rates after controlling for state fixed effect and year fixed effect. The results suggest that a state's cigarette excise tax is correlated with its beer excise tax at the 10% level. However, the cigarette tax is not correlated with other state covariates, and these state-level variables cannot predict state cigarette taxes.[8] In addition, considering that all these factors may be correlated with public health, I control for them in the models to mitigate any potential biases.

Another threat to identification is sample selection bias – the potential bias due to the sample being endogenously selected by cigarette tax. If a cigarette tax affects fertility or the probability of entering the sample, the health effect could be endogenous. That is, the health effect could be a result of the fertility effect or the sample I use. Table 2.3 shows that the cigarette tax is not statistically significantly correlated with the probability that a woman aged 20 to 39 gave birth to a baby in the same year or the following year, regardless of the gender of the baby. Table 2.4 further shows that, for all the individuals born to PSID families between 1968 and 1994, in utero cigarette tax is not statistically significantly correlated with the probability of being included in the sample. These results suggest that selection bias is unlikely to be an issue for this study.

In addition, the effect of in utero cigarette tax could be biased upward if cigarette tax levels at different times are correlated; that is, in utero cigarette taxes could be correlated with the cigarette taxes to which people are exposed in later years. To mitigate this concern, I control for cigarette taxes at age 25 in the baseline model. I further conduct various specification tests, including controlling for cigarette taxes from other periods in later life. The results are robust.

[8] I also regressed each state covariate in time t+1 on cigarette tax at time t while controlling the full set of other state-level covariates to see whether the cigarette tax affects future state-year characteristics. The results show that state cigarette tax does not have a significant effect on state-year characteristics in the next year. Please see detailed results in Table B1.

Table 2.2: Correlation between State Cigarette Taxes and State-Level Demographics, Economic Policies, and Economic Conditions

	Cigarette Tax at Time t (1)	Cigarette Tax at Time t (2)	Cigarette Tax at Time t (3)	Cigarette Tax at Time t + 1 (4)
State Characteristics at Time t				
Male Percent	-0.147	0.013	-0.009	-0.025
	(0.106)	(0.071)	(0.067)	(0.061)
White Percent	0.158	0.025	-0.025	0.001
	(0.103)	(0.088)	(0.086)	(0.080)
Married Percent	-0.005	0.044	0.049	0.037
	(0.065)	(0.034)	(0.034)	(0.035)
High School or Some College Percent	-0.021	0.056	0.005	-0.051
	(0.097)	(0.076)	(0.080)	(0.080)
College or Higher Education Percent	0.145	0.034	-0.014	-0.069
	(0.121)	(0.084)	(0.086)	(0.101)
Average Age	0.004	-0.000	-0.000	-0.001
	(0.003)	(0.001)	(0.001)	(0.001)
State Per Capita GDP	-0.001	0.002	0.000	0.001
	(0.002)	(0.002)	(0.002)	(0.002)
State Min. Wage	0.004	0.004	0.000	-0.000
	(0.005)	(0.004)	(0.003)	(0.003)
State Income MTR	-0.002	-0.003	-0.003	-0.004
	(0.004)	(0.004)	(0.004)	(0.005)
State Max. AFDC/TANF Benefit	0.155***	0.027	0.030	0.057
	(0.058)	(0.051)	(0.055)	(0.056)
State Max. EITC	-0.007	0.003	-0.007	0.007
	(0.011)	(0.007)	(0.028)	(0.038)
State Max. CDCTC	-0.014	-0.014**	0.016	0.019
	(0.009)	(0.006)	(0.019)	(0.018)
State Hospital Beds	0.001	0.001	0.001	0.000
	(0.001)	(0.002)	(0.002)	(0.002)
State Expenditure on Medicaid and Similar Programs	-0.001	-0.002	-0.004	-0.001
	(0.006)	(0.010)	(0.011)	(0.011)
State Beer Excise Tax	0.001	0.003***	0.002*	0.002*
	(0.001)	(0.001)	(0.001)	(0.001)
State Wine Excise Tax	0.001	0.001	0.001	0.001
	(0.002)	(0.001)	(0.001)	(0.001)
State Spirit Excise Tax	0.002**	0.002	0.001	0.001
	(0.001)	(0.001)	(0.001)	(0.001)
Minimum Legal Age for Purchasing Tobacco Products	0.026	0.001	0.007	0.008
	(0.025)	(0.010)	(0.008)	(0.009)
R-squared	0.363	0.779	0.813	0.812
State FE	N	Y	Y	Y
Year FE	N	N	Y	Y
Observations		1,281		

Note: Coefficients in each column are from a separate regression. All the covariates are aggregated to the state-year level. The data is from 1968-1994. Standard errors, clustered by state, are in parentheses. Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Table 2.3: Endogeneity Check I: Impact of In Utero Exposure to Cigarette Taxes on Fertility

	1(Have a Newborn in Year $t/t+1$) (1)	1(Have a Boy in Year $t/t+1$) (2)	1(Have a Girl in Year $t/t+1$) (3)
Cigarette Taxes, t (10 Cents)	0.027	0.020	0.006
	(0.022)	(0.020)	(0.015)
Observations	68,259	68,259	68,259
R-squared	0.107	0.062	0.069
Y-mean	0.254	0.150	0.149
Std. Dev. of Y	(0.435)	(0.357)	(0.356)
Birth Year FE	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes
Interview Year FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
State Covariates at Time t	Yes	Yes	Yes

Note: Coefficients in each column are from a separate regression. We use PSID data from 1968-1994. The sample consists of females at ages 20-39. Estimates are weighted using PSID family longitudinal weights. Cigarette tax at survey year is CPI-adjusted to 2020 dollars. The unit is 10 cents. Other specifications follow the baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Table 2.4: Endogeneity Check II: Survival Bias

	1(Be a Household Head/Spouse between Ages 25-35)
In Utero Cigarette Taxes (10 Cents)	-0.003
	(0.006)
Observations	11,148
R-squared	0.089
Y-mean	0.586
Std. Dev. of Y	(0.493)
Cigarette Taxes at Age 25	Yes
Birth Year FE	Yes
Birth State FE	Yes
Individual Controls	Yes
Family Background at Birth	Yes
State Controls	Yes

Note: The sample consists of individuals born to PSID families between 1968-1994. Estimates are weighted using the average PSID longitudinal weights for ages 1-18. Outcome variable is a dummy variable indicating whether the individual became a household head or spouse between ages 25-35, in other word, whether the individual is eligible to be contained in our main sample. Other specification follows baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Event Study

I used an event study to determine the impact of early life exposure to a tax hike on adult health. The equation corresponding to the event study is:

$$y_{isym} = \alpha + \delta_1 \sum_{j=-J}^{J} e_{sj} + \delta_2 CigTax_{(s,age25)} + X_{isy}\beta_1 + Z_{sy}\beta_2 + \lambda_y + \eta_s + \epsilon_{isym}, \quad (2.3)$$

where e_{sj} equals one when an observation is j periods away from the event. The other variables are the same as in equation 2.2. Event time is defined in quarters. Period 0 presents the year-quarter in which the event happened.

Because the majority of the states in my sample experienced multiple tax hikes during 1968-1994, it is difficult to analyze each tax hike separately. Furthermore, event studies tend to be most effective at identifying divergent trends when many events take place within a shared time frame. Therefore, I defined a discrete tax event as any tax hike exceeding the fiftieth percentile (17 cents) of all hikes and selected the "high-frequency" tax period of 1968-1976, following Simon (2016). During this period, there happened 89 tax hike events and the average increase in tax is around 30 cents for these tax hikes. Under this criterion, almost all the states have only one discrete event in the time frame. In the event study sample, only states that experience an event are included. I also balance the event study such that an event is included only if it is the only event in the state for three years before the event and three years after the event.

Staggered Difference-in-Difference Estimator

There is a potential for bias in the presence of heterogeneous treatment effects for the difference-in-difference with continuous treatment with two-way fixed effects estimator (Callaway et al. 2021). Hence, I have chosen to employ the staggered Differences-in-Differences estimator for a non-binary ordered treatment, known as DID_M, as proposed by De Chaisemartin and d'Haultfoeuille (2020). This estimator maintains its validity even

when treatment effects vary across time or different groups. However, it does come with certain limitations.

Firstly, there is a bias-variance trade-off between DID_M and the TWFE estimator as DID_M often has a larger variance than that of TWFE estimator. Secondly, the DID_M estimator requires that, for each pair of consecutive dates, there exist groups whose treatment remains constant to act as control groups. Consequently, it cannot accommodate treatments such as the real cigarette tax that fluctuates from year to year. To address this, I have opted for a discrete treatment approach, specifically using discrete cigarette tax levels rather than real cigarette tax values to establish control groups that have stable treatment. It's important to strike a balance in the number of discrete levels; too many might put a restrict on the number of observations for control groups, while too few might result in limited number of switchers between these levels. I create seven distinct tax tiers for cigarettes by rounding the cigarette tax by 20 cents. That is, the first tier ranges from 0 to 10 cents, while the subsequent tiers go from 10 to 30 cents, then from 30 to 50, and so on, with the highest tier spanning 110 to 130 cents. Lastly, there is a constraint on the number of control variables that can be included in the model. Excessive control variables can inflate the standard error, which may affect the model's precision.

While the DID_M estimator is resilient against heterogeneous treatment effects, it's important to use caution because of the bias-variance trade-off, the requirement for groups with constant treatment within consecutive dates, and other constraints. By including the DID_M estimator as a robustness check, I aim to ensure the reliability of my findings and to assess the consistency of results between the TWFE and the staggered DiD approaches. This dual approach allows for a comprehensive evaluation of treatment effects while acknowledging the strengths and limitations of each method.

2.6 Results

I start by presenting the results on the instantaneous effect of cigarette tax on smoking behavior. I then present the main results on the effects of changes in cigarette taxes during childhood on health as adults, followed by heterogeneity of the effect and robustness tests. I further investigate the potential mechanisms/mediators and, lastly, I discuss the size of the effect.

Impact of Cigarette Tax on Smoking

Because smoking behavior was surveyed in 1986 and from 1999 on in the PSID, I use data from these years to estimate the effects of cigarette tax on smoking rates for household heads and spouses in the reproductive ages from 20 to 39. The results are shown in Table 2.5. Note that observations in this sample are not necessarily parents of individuals in the main sample. Column 1 of Table 2.5 shows that a 10-cent higher cigarette tax reduces the probability of smoking by 2 percentage points, which is significant at the 10% level and around 8% of the mean.

The sample for column 2 of Table 2.5 is a subsample of that for column 1 and consists of household heads and spouses who were aged 20-39 and had a newborn within two years after the year when the question on smoking behavior was asked. Column 2 shows that a 10-cent increase in cigarette tax reduces the probability of smoking by 2.9 percentage points, which is significant at the 5% level and corresponds to 15% of the mean. This result indicates that the tax effect could be even stronger for household heads and spouses who had a baby within that two-year period.

The sample for column 3 is a subsample of column 1 and only includes parents of individuals in the main sample. The result shows that a 10-cent increase in cigarette tax reduces the probability of smoking by 10 percentage points, which corresponds to 35% of the mean. The relatively large magnitude (compared to columns 1 and 2 in Table 2.5)

could be because around one-fifth of the sample are from 1986, when both the price of cigarettes and cigarette taxes were relatively low, and people may have been more sensitive to the increase of cigarette tax at that time.

Estimates with Natality data support this finding. Column 4 of Table 2.5 shows that a 10-cent higher cigarette tax during the in utero period is associated with a decrease of 1.6 percentage points in the smoking rate of the mother during pregnancy, which corresponds to 9% of the mean.

Main Results

Table 6 presents the effect of exposure to in utero cigarette tax on health in adulthood. I use two health outcome indices as the main measures of long-term health in adulthood. Health outcome index A is a binary variable that equals one if the individual has any of the following conditions: asthma, lung disease, heart disease, or heart attack. I choose these four health conditions because respiratory and cardiovascular conditions are the main problems caused by exposure to cigarette smoke (United States Department of Health and Human Services 2001, 2004).[9] Health outcome index B is the number of these health conditions that individuals have. Columns in Table 2.6 progressively add controls, including birth year and birth state fixed effects, demographics, family background at birth, and different state-level covariates. The results are stable, with the coefficients ranging from -1.3 to -1.8 percentage points for health outcome index A and -0.018 to -0.021 for health outcome index B. With the full set of controls, a 10-cent higher in utero cigarette tax reduces the probability of ever having any of those conditions between ages 25 and 35 by around 1.8 percentage points and reduces the number of conditions the individual has between age 25 and 35 by around 0.02. Effects on both indices correspond to 7-8% of their means, respectively, indicating a substantial reduction in the probability of suffering from

[9]Physical health conditions available in PSID include high blood pressure, diabetes, heart attack, heart disease, stroke, arthritis, cancer, psychosis, obesity, lung disease, asthma, learning disorder, and other conditions.

Table 2.5: Contemporary Effect: Impact of Exposure to Cigarette Taxes on Smoking Behavior

	PSID: 1(Smoke Now)			Natality: Smoke Rate of Mothers
	Individuals Aged 20-39	Individuals Aged 20-39 and with Newborns in Year t/t + 1	Individuals Aged 20-39 Whose Children in Main Sample	Pregnant Women Aged 20-39
	(1)	(2)	(3)	(4)
Cigarette Taxes, t (10 Cents)	-0.020*	-0.029**	-0.095***	-0.016*
	(0.011)	(0.012)	(0.023)	(0.008)
Observations	37,036	6,155	4,140	34,328
R-squared	0.136	0.184	0.174	0.975
Y-mean	0.246	0.199	0.270	0.170
Std. Dev. of Y	(0.431)	(0.399)	(0.444)	(0.132)
Year FE	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	N/A
Individual Controls	Yes	Yes	Yes	N/A
Family Background at Birth Year	Yes	Yes	Yes	N/A
State Covariates at Time t	Yes	Yes	Yes	Yes
Smoke Bans at Time t	Yes	Yes	Yes	Yes
Educational Level	Yes	Yes	Yes	Yes
Group FE	N/A	N/A	N/A	Yes

Note: Each column is from a separate regression. The first three columns are estimated with PSID data. The sample of first column consists of household heads and spouses aged 20-39 in PSID survey in 1986 and post-1999 (smoking behavior is only surveyed in these years). The sample of the second column consists of household heads and spouses who had newborn at year t/t+1 and aged 20-39 in PSID survey in 1986 and post-1999. The sample of the third column contains household heads and spouses aged 20-39 whose children contained in our main sample in PSID survey in 1986 and post-1999. Estimates are weighted using the PSID longitudinal weight. Outcome variables for the first three columns are individuals' smoking behaviors. The last column is estimated with Natality birth data from years 1969 to 1994. We do not include data from 1968 because the educational attainment of mother and marital status of mother are not recorded in 1968. The sample of the last column contains pregnant women aged 20-39. The outcome variable is smoke rate of pregnant women for cells defined by state, year, marital status, age, race, educational attainment of the mother, and the birth order of the newborn. Estimates are weighted by the number of birth in the cells. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

the four health conditions which are most related to exposure to smoking. The preferred model is the model corresponding to the last column.

Once I control for the cigarette tax during the in utero period, cigarette tax at age 25 has a much smaller effect on adult health. For example, a 10-cent higher cigarette tax at age 25 decreases the probability of ever having any of those four health conditions between age 25 and 35 by around 0.2 of a percentage point, which is only about 1% of the mean. The effect on the number of health conditions is statistically significant, but only at the 10% level.

Table 2.7 shows the regression results for the individual components of the two indices and the effect on adult health status in different age ranges. The magnitude of the health effect increases as individuals age. For health status during ages 35 to 45, a 10-cent higher cigarette tax while in utero reduces the probability of ever having any of those four health conditions by 2.8 percentage points and reduces the number of health conditions by 0.04; both estimates are larger than their counterparts for ages 25 to 35. For ages 25 to 35, the main driver is the effect on asthma, while for ages 35 to 45, the main drivers are the effects on asthma and lung disease. Figure 2.2 and Table B2 further shows that the magnitude of the beneficial effect of in utero cigarette tax on health status increases with age when I examine age ranges from 25 to 30, 30 to 35, 35 to 40, and to 40 to 45.

Event Study and Staggered $\mathrm{DID_M}$ Estimator

Figure 2.3 shows the event study for long-term health condition at the fiftieth percentile cut-off. As a reference group, I use the cohort born in the quarter just before the quarter in which the tax hike was implemented. For example, take a case in which there is only one tax hike in the sample. In Colorado in the third quarter of 1973, the reference cohort would be the children born in the second quarter of 1973, who were exposed to the tax hike in the quarter after the quarter of birth (the third quarter of 1973). This means the effect on long-term health for this cohort could be due to the tax hike reducing secondhand smoke exposure after birth. The coefficient for pre-period j compares the effect of exposure to

Table 2.6: The Long-Term Effects of In Utero Cigarette Tax Exposure on Adult Health

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Outcome A:1(Any Health Condition)								
In Utero Cigarette Taxes (10 Cents)	-0.013*	-0.014*	-0.014*	-0.014**	-0.013**	-0.014**	-0.015**	-0.018***
	(0.007)	(0.007)	(0.007)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
Cig. Taxes at Age 25 (10 Cents)	-0.002**	-0.002**	-0.002**	-0.002**	-0.002**	-0.002**	-0.002*	-0.002*
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	5,402	5,402	5,402	5,402	5,402	5,402	5,402	5,402
R-squared	0.035	0.041	0.046	0.047	0.047	0.047	0.047	0.049
Y-mean				0.240				
Std. Dev. of Y				(0.427)				
Outcome B: # of Health Conditions								
In Utero Cigarette Taxes (10 Cents)	-0.018**	-0.018**	-0.018**	-0.018**	-0.017**	-0.018**	-0.018**	-0.021***
	(0.007)	(0.008)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)
Cig. Taxes at Age 25 (10 Cents)	-0.002*	-0.002*	-0.002	-0.002	-0.002	-0.002	-0.002	-0.002
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	5,402	5,402	5,402	5,402	5,402	5,402	5,402	5,402
R-squared	0.035	0.043	0.048	0.049	0.05	0.05	0.05	0.051
Y-mean				0.279				
Std. Dev. of Y				(0.530)				
Birth Year FE, Birth State FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Gender, Race, Age Square		Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family Background at Birth Year			Yes	Yes	Yes	Yes	Yes	Yes
Tax Rate for Beer, Wine & Spirit				Yes	Yes	Yes	Yes	Yes
Minimum Legal Purchasing Age for Tobacco					Yes	Yes	Yes	Yes
AFDC, EITC, CDCTC Benefit						Yes	Yes	Yes
Per Capita Hospital Beds and State Expenditure on Medicaid and Similar Programs							Yes	Yes
Minimum Wage, Max income MTR, and Per Capita GDP								Yes

Note: Each column in each panel is from a separate regression. The sample consists of individuals who were born to PSID families between 1968-1994 and have became household head or spouse during ages 25-35. Estimates are weighted using the average PSID longitudinal weights for ages 1-18. Outcome variables are individuals' health condition measured during ages 25-35. 1(Any health condition) = 1 if ever had any disease out of asthma, lung disease, heart disease and heart attack. # of health condition = the number of aforementioned diseases the individual ever had. In utero cigarette taxes refer to the state cigarette tax at the year-month of birth in the birth state. The model also accounts for the cigarette tax in the individual's residence state at age 25. Both taxes are CPI-adjusted to 2020 dollars. The unit is 10 cents. Standard errors clustered by state of birth are in parentheses. Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Table 2.7: Decompose Long-Term Health Effects: Investigating Effects on Specific Health Conditions

	1(Any Health Condition) (1)	# of Health Condition (2)	Asthma (3)	Lung Disease (4)	Heart Attack (5)	Heart Disease (6)
Panel A. Baseline, Effects on Health Condition for Individuals Aged 25-35						
In Utero Cigarette Taxes (10 Cents)	-0.018***	-0.021***	-0.017***	-0.004	-0.001	0.001
	(0.006)	(0.008)	(0.006)	(0.003)	(0.001)	(0.001)
Cigarette Taxes at Age 25 (10 Cents)	-0.002*	-0.002	-0.001	0.000	0.000	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)
Observations	5,402	5,402	5,402	5,402	5,402	5,402
R-squared	0.049	0.051	0.047	0.045	0.036	0.035
Y-mean	0.240	0.279	0.185	0.070	0.005	0.019
Std. Dev. of Y	(0.427)	(0.530)	(0.388)	(0.254)	(0.071)	(0.137)
Panel B. Effects on Health Condition for Individuals Aged 35-45						
In Utero Cigarette Taxes (10 Cents)	-0.028***	-0.047***	-0.022**	-0.014**	-0.005	-0.007
	(0.010)	(0.015)	(0.009)	(0.006)	(0.003)	(0.005)
Cigarette Taxes at Age 35 (10 Cents)	0.000	0.001	0.000	0.001	0.000	0.000
	(0.001)	(0.002)	(0.001)	(0.001)	(0.000)	(0.000)
Observations	2,983	2,983	2,983	2,983	2,983	2,983
R-squared	0.06	0.064	0.052	0.061	0.043	0.05
Y-mean	0.223	0.283	0.170	0.064	0.017	0.032
Std. Dev. of Y	(0.416)	(0.587)	(0.376)	(0.245)	(0.129)	(0.176)
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes

Note: Each column in each panel is from a separate regression. For the baseline model in panel A, the sample consists of individuals who were born to PSID families between 1968-1994 and became household heads or spouses during age 25-35. The sample for Panel B consists of individuals who were born to PSID families between 1968-1994 and became household heads or spouses during age 35-45. Other specifications follows the baseline model. Standard errors clustered by state of birth are in parentheses. Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

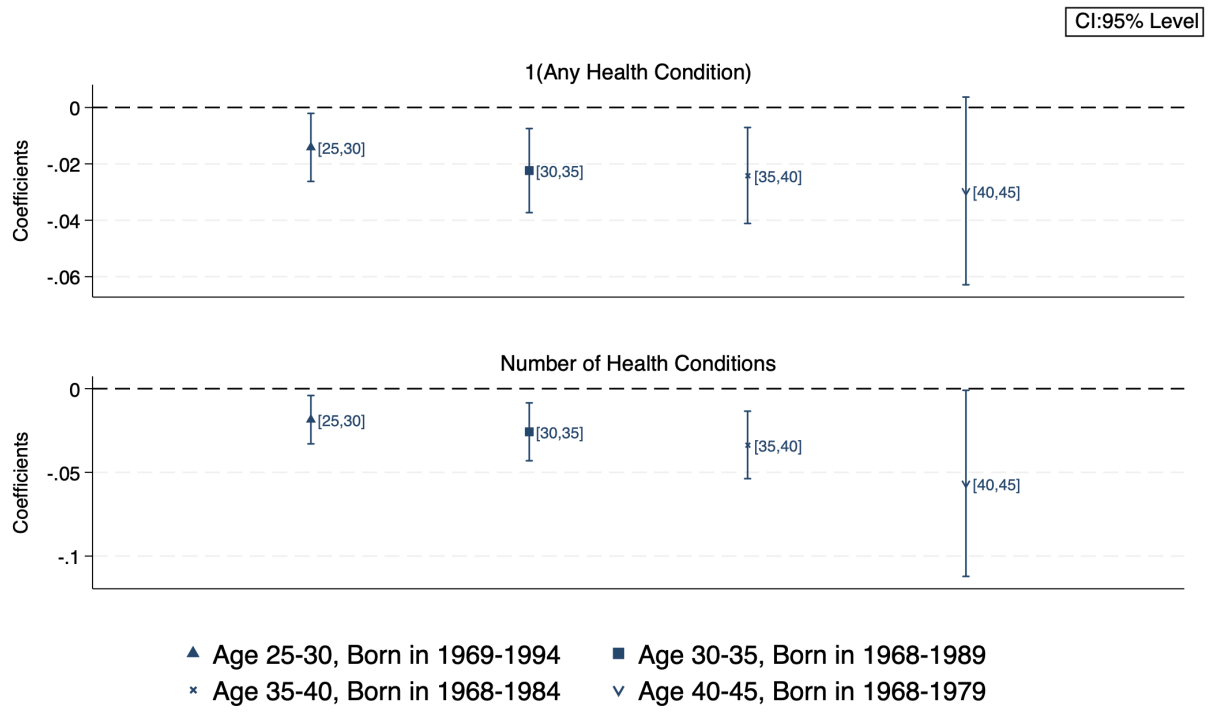
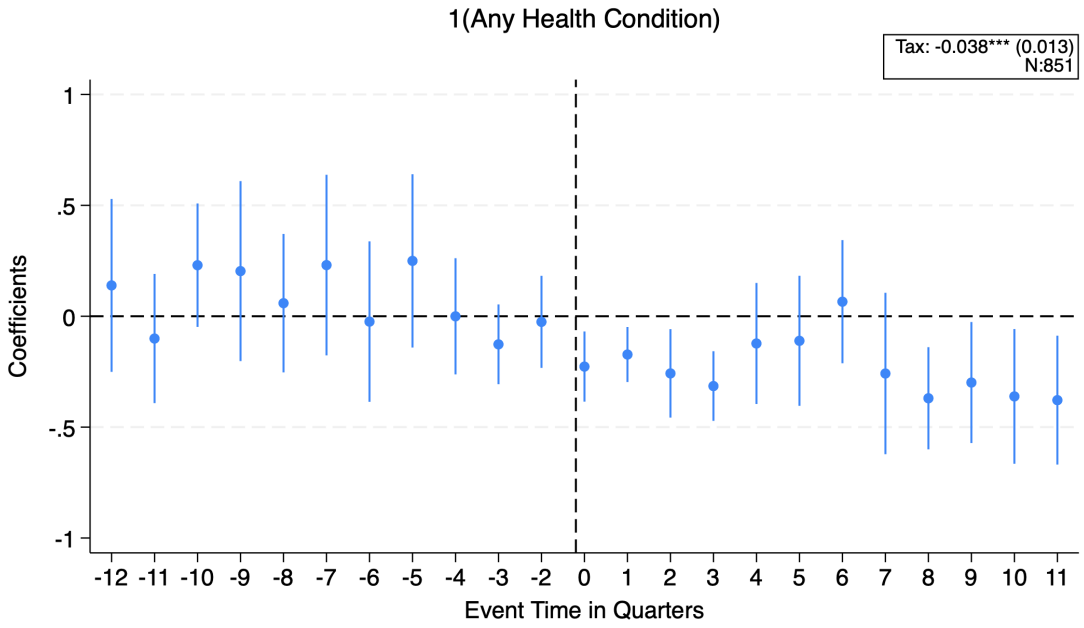


Figure 2.2: Effects of In Utero Cigarette Tax on Health Conditions in Adulthood Across Age Groups

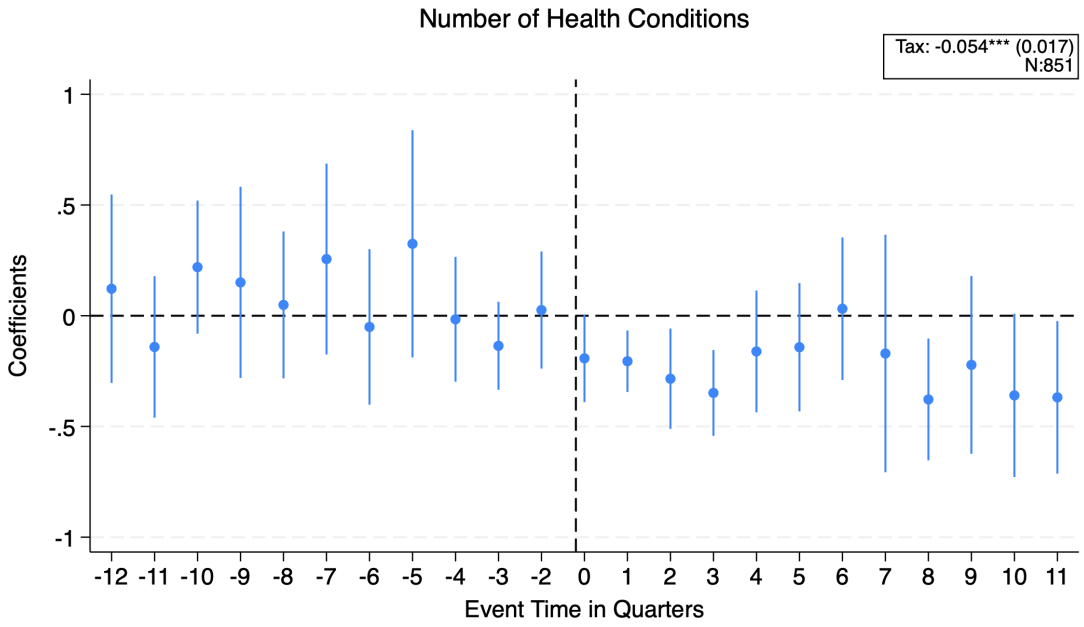
Note: Each estimated coefficient is from a separate regression. Estimates are weighted using the average PSID longitudinal weights for ages 1-18. Outcome variables are adult health conditions across different age ranges. In utero cigarette tax refers to the state cigarette tax at the year-month of birth in the birth state. The model also accounts for the cigarette taxes in the individuals' residence state at the lower bound of the age range. Taxes are CPI-adjusted to 2020 dollars. The unit of cigarette taxes are 10 cents. Other specification as baseline model. Standard errors clustered by state of birth are in brackets. Confidence intervals are at the 95%.

the tax hike starting in the jth quarter after birth to a tax hike starting in the first quarter after birth. The post-period coefficients compare the effect of a tax hike starting in the jth quarter before birth to exposure to a tax hike starting in the first quarter after birth. Figure 2.3 shows that there is no significant pre-trend effect but there are significant post-trend effects. Exposure to a tax hike in the third trimester in pregnancy or earlier improves long-term health outcomes significantly compared to being exposed to a tax hike after birth. I conducted a robustness check using the seventy-fifth percentile, and the result yielded a similar pattern (Figure B1).

Table 2.8 shows the results from the staggered $\mathrm{DID}_{\mathrm{M}}$ Estimator. A one-level higher cigarette tax during the in utero period (a 20 cent increase on average) causes a 3.9 percent-



(a) Any Health Condition = 1



(b) Number of Health Conditions

Figure 2.3: Event Study of In Utero Exposure to Tax Hikes on Health Condition in Adulthood

Note: Each figure represents a separate estimation. Events are defined as tax hikes exceeding the median increase during 1968-1994. Event time is denoted in quarters. The coefficient at period j reflects the event effect for the cohort born in that period. The sample comprises individuals born to PSID families during the high frequency tax period of 1968-1976, who became household heads or spouses at ages 25-35. Only individuals born in states where only one event happened in the period are included. Estimates are weighted using average PSID longitudinal weights from ages 1-18. Outcome variables are individuals' health conditions aged 25-35, with other settings as the baseline model. Confidence intervals are at 95%.

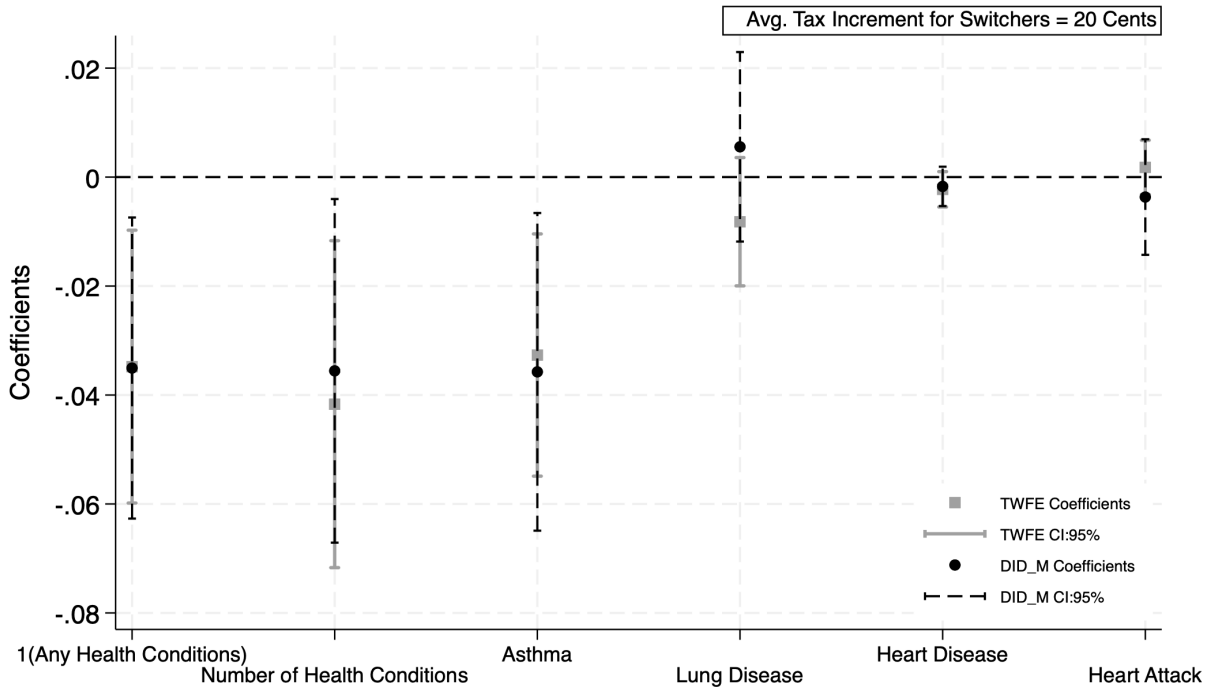


Figure 2.4: Comparing Two-Way Fixed Effects and Staggered Difference-in-Differences Estimations

Note: Each estimate is from a separate estimation. Staggered DID estimation, as proposed by De Chaisemartin and d'Haultfoeuille (2020), is applied to ordered treatments. For ease of comparison, the unit of the cigarette tax is adjusted to 20 cents, and confidence intervals are calculated at the 95% level.

age point reduction in the probability of ever having any of the four health conditions. This is close to the value from the TWFE estimation, where a 10 (20) cent increase in cigarette tax while in utero is associated with a 1.8 (3.6) percentage points reduction in the probability of ever having any of the four health conditions. Figure 2.4 shows the comparison between the TWFE estimation and the DID estimator after adjusting the unit of cigarette tax to 20 cents per pack for the TWFE estimation.

Heterogeneity by Subgroups

Socioeconomic status (Ekblad et al. 2014; Rumrich et al. 2019) and educational attainment (Gould et al. 2017) are primary predictors for smoking during pregnancy. In addition, the effect of early life environment may be different across genders (Nilsson 2017). For these

Table 2.8: Estimation Results Using Staggered DID Estimator

	1(Any Health Condition) (1)	# of Health Condition (2)	Asthma (3)	Lung Disease (4)	Heart Attack (5)	Heart Disease (6)
Panel A. Effects on Health Condition for Individuals Aged 25-35						
Discrete In Utero Cigarette Taxes Level	-0.039***	-0.049**	-0.032***	-0.013	-0.004	0.000
	(0.015)	(0.021)	(0.012)	(0.014)	(0.002)	(0.006)
Observations	3,669	3,669	3,670	3,670	3,670	3,669
Number of Switchers	1,040	1,040	1,040	1,040	1,040	1,040
Panel B. Effects on Health Condition for Individuals Aged 35-45						
Discrete In Utero Cigarette Taxes Level	-0.064***	-0.072***	-0.055***	-0.009	-0.005	-0.003
	(0.020)	(0.023)	(0.016)	(0.015)	(0.008)	(0.009)
Observations	1,936	1,936	1,936	1,936	1,936	1,936
Number of Switchers	627	627	627	627	627	627

Note: Results from the staggered DID estimator proposed by De Chaisemartin and d'Haultfoeuille (2020). Each column in each panel is from a separate regression. The sample for panel A consists of individuals who were born to PSID families between 1968-1994 and became household heads or spouses during age 25-35 . The sample for Panel B consists of individuals who were born to PSID families between 1968-1994 and became household heads or spouses during age 35-45 . Estimates are weighted using the average PSID longitudinal weights for ages 1-18. Outcome variables are individuals' health condition at the corresponding ages. In utero cigarette taxes refer to the state cigarette taxes at the year-month of birth in the birth state. We divided in utero cigarette tax into seven distinct tiers by rounding it by 20 cents. The first tier ranges from 0 to 10 cents, while the subsequent tiers go from 11 to 30 cents, then from 31 to 50, and so on, with the highest tier cigarette tax spanning from 110 to 130 cents. Switchers refer to switchers from one cigarette tax level to a higher cigarette tax level. Standard errors clustered by state of birth are in parentheses. Significance levels: * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

reasons, I explore the heterogeneity of cigarette tax effects by the respondents' race, gender, mother's education, family economic status, and parents' marital status at the time of birth. Figure 2.5 show the heterogeneous effects of cigarette taxes. The result is largely robust across subgroups. The effect is relatively larger for males, which is consistent with literature showing that males suffer more from an adverse environment in early life (Nilsson 2017). There is an opposing effect observed within the subgroup born to single-parent families, where a higher in utero cigarette tax is associated with an insignificant increased likelihood of experiencing health conditions in later life. This phenomenon may be attributed to persistent nicotine addiction for single parents that they do not reduce smoking in respond to cigarette tax that cigarette taxes become pure economic burden for them. Notably, the coefficient for the subgroup born to mothers with an educational level of college or above has a wide confidence interval, likely because of the very small size of this group.[10]

Robustness Checks

Columns 2 and 6 in Table 2.9 show the robustness of the health effect of in utero cigarette taxes while controlling for cigarette tax in later life. Friedson et al. (2023) finds that increasing the average cigarette tax during adolescence (between ages 14 and 17) reduces the probability of adult smoking, which could improve health status in adulthood. Building on this finding, I control for average cigarette taxes during adolescence. The result shows that controlling for average cigarette tax in adolescence does not change the baseline results on the health effects of exposure to cigarette tax while in utero. The average cigarette tax in adolescence does improve health status in adulthood, as shown by Friedson et al. (2023), although the effect is insignificant in my empirical setting. In columns 3 and 7, I control for both average cigarette tax between ages 1 and 13 and average cigarette tax between ages 14 and 17. The effect of cigarette taxes during the in utero period is the same as in the baseline model. The average cigarette taxes in the two childhood periods improve health status in

[10]The sample size for the subgroup born to mothers with a college degree or above is 754.

Table 2.9: Robustness Checks

	1(Any Health Condition)				# of Health Condition			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
In Utero Cigarette Taxes (10 Cents)	-0.018***	-0.018***	-0.016**		-0.021***	-0.021***	-0.020**	
	(0.006)	(0.006)	(0.007)		(0.008)	(0.007)	(0.007)	
Avg. Cig. Taxes at Ages 1-13			-0.04				-0.034	
			(0.092)				(0.099)	
Avg. Cig. Taxes at Ages 14-17		-0.043	-0.035			-0.036	-0.03	
		(0.034)	(0.037)			(0.039)	(0.043)	
Cig. Taxes at Age 25	-0.002*	-0.002*	-0.002**		-0.002	-0.002	-0.002	
	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)	(0.001)	
In Utero Cigarette Taxes (10 Cents)*Born in 1968-1980				-0.017***				-0.021***
				(0.006)				(0.008)
Difference in Cig. Tax Effect: 1968-1980 Cohorts vs. 1981-1994 Cohorts				-0.002				0.001
				(0.007)				(0.010)
Observations	5,402	5,402	5,402	5,402	5,402	5,402	5,402	5,402
R-squared	0.049	0.049	0.049	0.049	0.051	0.052	0.052	0.051
Y-mean			0.240				0.279	
Std. Dev. of Y			(0.427)				(0.530)	
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Note: Each column is from a separate regression. Specifications follow the baseline model except for the additional terms displayed in the table. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

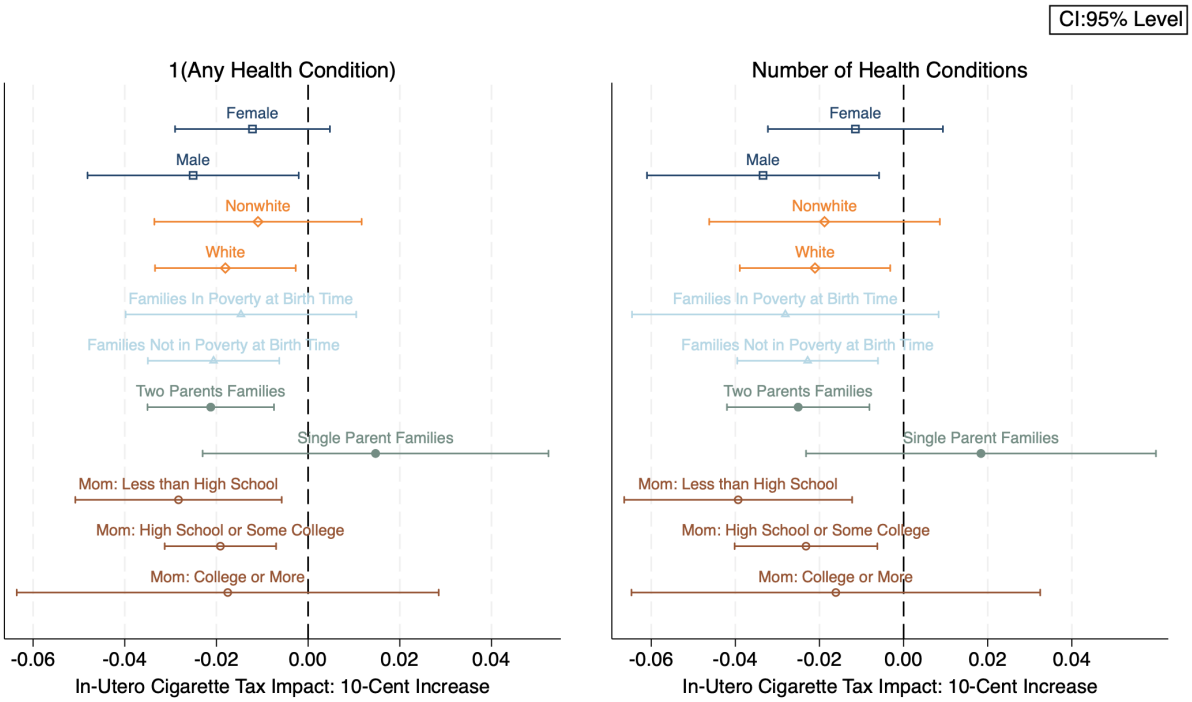


Figure 2.5: Subgroup Analysis

Note: Each estimated coefficient is from a separate regression. Mother's educational level is categorized as less than high school (if highest grade < 12), high school/some college (if highest grade 12-15), and college or above (if highest grade $\geqslant 16$). For subgroups by economic status, "in poverty" families are defined as families with income less than or equal to 1.5 times the Census need standard. Outcome variables are individuals' health conditions during ages 25-35. In utero cigarette tax refers to the state cigarette tax at the year-month of birth in the birth state. The model also accounts for the cigarette taxes in the individual's residence state at age 25. Both taxes are CPI-adjusted to 2020 dollars. The unit is 10 cents. Other specifications follow the baseline model. Confidence intervals are at 95%.

adulthood, although the effects are insignificant.

Because the timeframe in the study lasts for more than two decades, I check whether the effect is consistent over time by dividing the sample into two subsamples by median birth cohort: people born between 1968 and 1980 and people born between 1981 and 1994. The results in columns 4 and 8 show that there is no significant difference in the health effect of cigarette taxes during the in utero period for people born in these different decades.

As noted above, the effect of exposure to cigarette smoking is strongest in the third trimester (United States Department of Health and Human Services 2001). Figure 2.6 shows that the results are robust if I change the explanatory variable from the cigarette tax at the end of the third trimester to the cigarette tax at the beginning of the third trimester

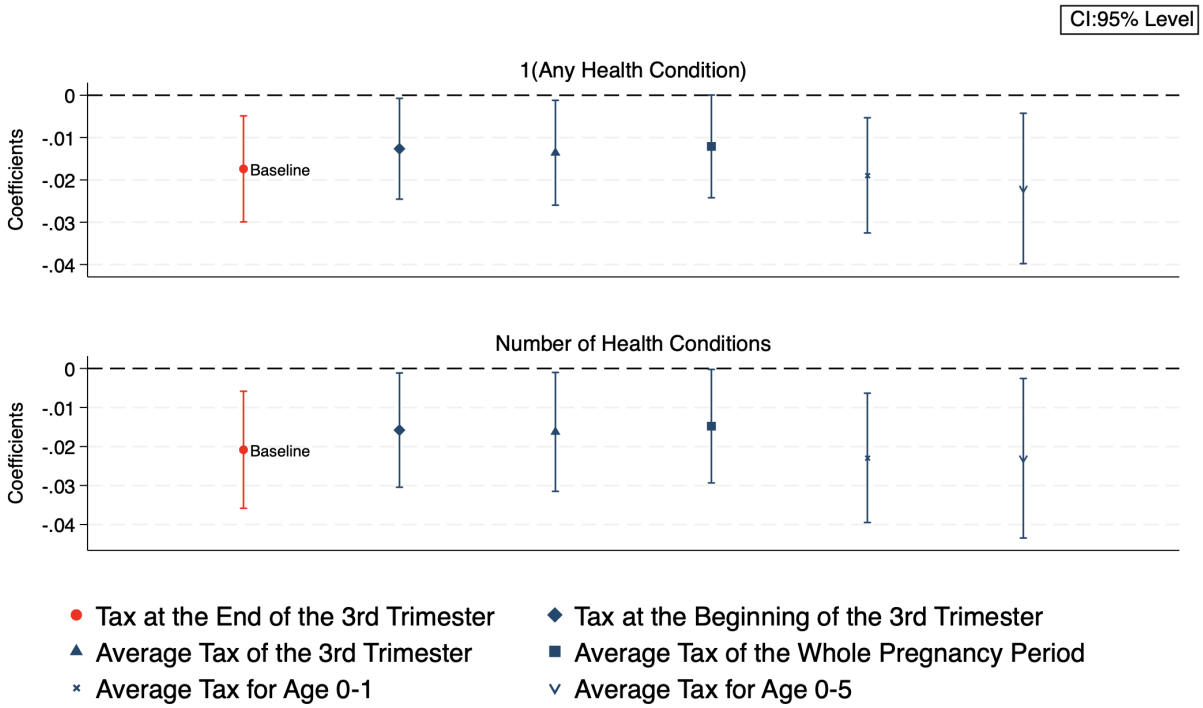


Figure 2.6: Robustness Check, Alternative Measures of Exposure to Cigarette Taxes
Note: Each estimated coefficient is from a separate regression. Specification follows the baseline model except for the measure of early life exposure to cigarette taxes. Standard errors are clustered by state of birth. Confidence intervals are at the 95%.

(as in Simon (2016)), the average cigarette tax during the third trimester, or the average cigarette tax during the whole period. In Figure 2.6, I also compare the effects of in utero cigarette tax at the end of the third trimester, the average cigarette tax in the first two years of life, and the average cigarette tax in the first six years of life. Compared to the cigarette tax during the in utero period, increasing the average cigarette tax in the first two years of life or in the first six years of life has about the same effect on health in adulthood. This indicates that the effect of the average cigarette taxes in the first several years of life could be mainly driven by the effect of the cigarette tax during the in utero period. However, I am cautious in interpreting this result because an increase in cigarette tax in birth year usually means the cigarette tax remains high in the following years.

One concern regarding our specification is the potential influence of outlier states, specifically those with exceptionally high or low levels of cigarette taxes within our sample.

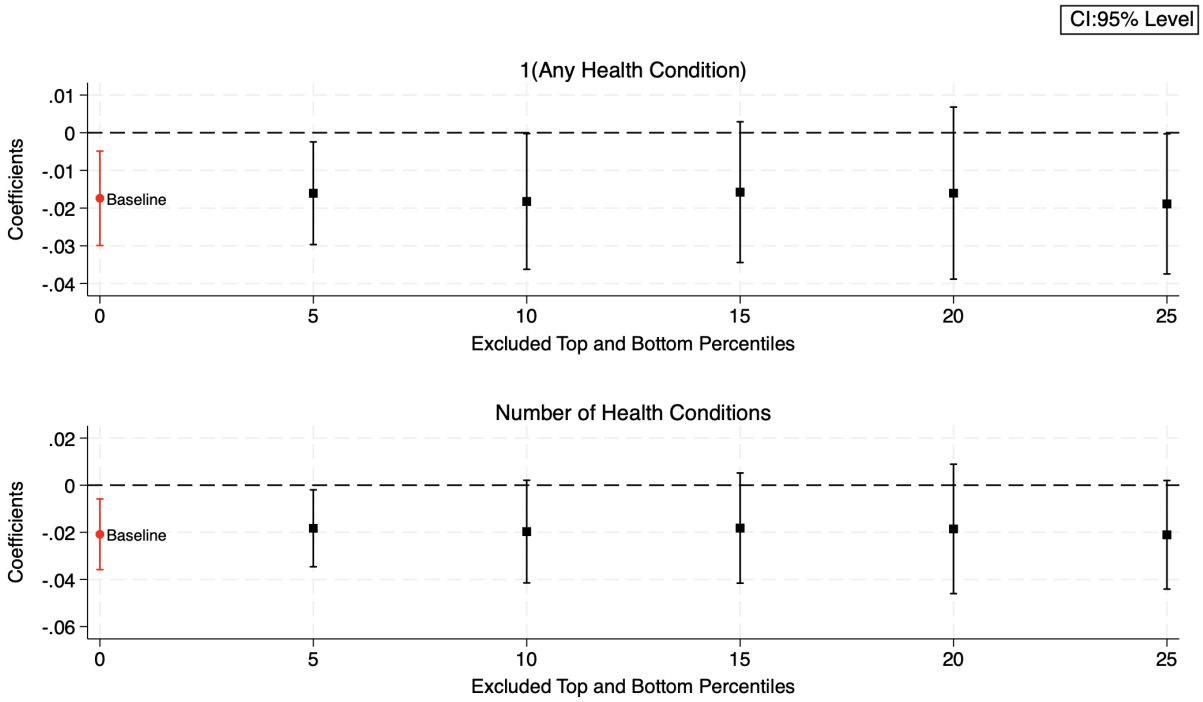


Figure 2.7: Robustness Check, Trimming States with High or Low Cigarette Taxes
Note: Coefficients were estimated by sequentially trimming states with average cigarette taxes in the bottom and top percentiles during 1968-1994. Each estimated coefficient is from a separate regression. Specification follows baseline model. Standard errors are clustered by state of birth. Confidence intervals are at the 95%.

We assess the robustness of our coefficient estimates concerning this aspect by progressively excluding states with average cigarette taxes at both the upper and lower extremes at the 5^{th}, 10^{th}, 15^{th}, 20^{th}, and 25^{th} percentiles. Consequently, this process retains the middle 90 percentiles, 80 percentiles, 70 percentiles, 60 percentiles and 50 percentiles when trimming the top and bottom percentiles. In Figure 2.7, the first coefficient estimate represents our baseline specification. The coefficient remains relatively stable in the trimming process. However, the confidence interval expands because this exercise mechanically constrains the variation in causality that would be required to estimate the coefficient of interest. Nevertheless, even with this limitation, the coefficient estimate remains statistically significant when we exclude the top and bottom 25^{th} percentiles.

Table B3 shows that the results are robust across different sampling weights. I further test the robustness of the baseline model to include state-specific linear time trends. In this

way, the identifying variation in cigarette tax comes from tax changes within states over time that deviate from the linear trend. Table B4 shows that the results are robust when controlling the state-specific linear time trends.

Table B5 compares the estimated coefficients and the corresponding odds ratio from OLS regression and logit/ordered logit regression. The odds ratio from the two regressions is similar and further supports the robustness of the results.[11] The results from logistic regression show that the odds ratio of ever having any health condition over having no health condition, associated with a 10-cent increase in cigarette tax, is 0.9 times the corresponding odds ratio without the 10-cent increase in cigarette tax. In other words, people have 10% lower odds (0.9-1=0.1) of ever having one of the health conditions in response to a 10-cent higher cigarette tax while in utero.

Effect of Discrete Levels of Cigarette Taxes

I use cigarette tax bins instead of continuous cigarette tax to explore the effect of discrete levels of cigarette taxes. The estimation model is

$$y_{isym} = \alpha + \sum_{j} \delta_j \text{Cigtax bin}_{sym,j} + \gamma \text{Cigtax}_{s,age25} + X_{isy}\beta_1 + Z_{sy}\beta_2 + \lambda_y + \eta_s + \epsilon_{isym} \quad (2.4)$$

where $\text{Cigtax bin}_{sb,j}$ is equal to 1 if the cigarette tax during an in utero month falls in the jth bin. The bins range from \$0 to \$1.5 with an increment of \$0.30 (cigarette taxes range from \$0 to \$1.31). Other variables are the same as in Equation 2.2.

Figure 2.8 shows the effects of discrete levels of cigarette taxes during the in utero period. It shows that the magnitude of the in utero cigarette tax effect on the two health indices is increasing with the cigarette tax levels. This indicates that the health effect increases in the cigarette tax, at least within the range of \$0 to \$1.31.

[11] The convergence is not achieved for heart attack.

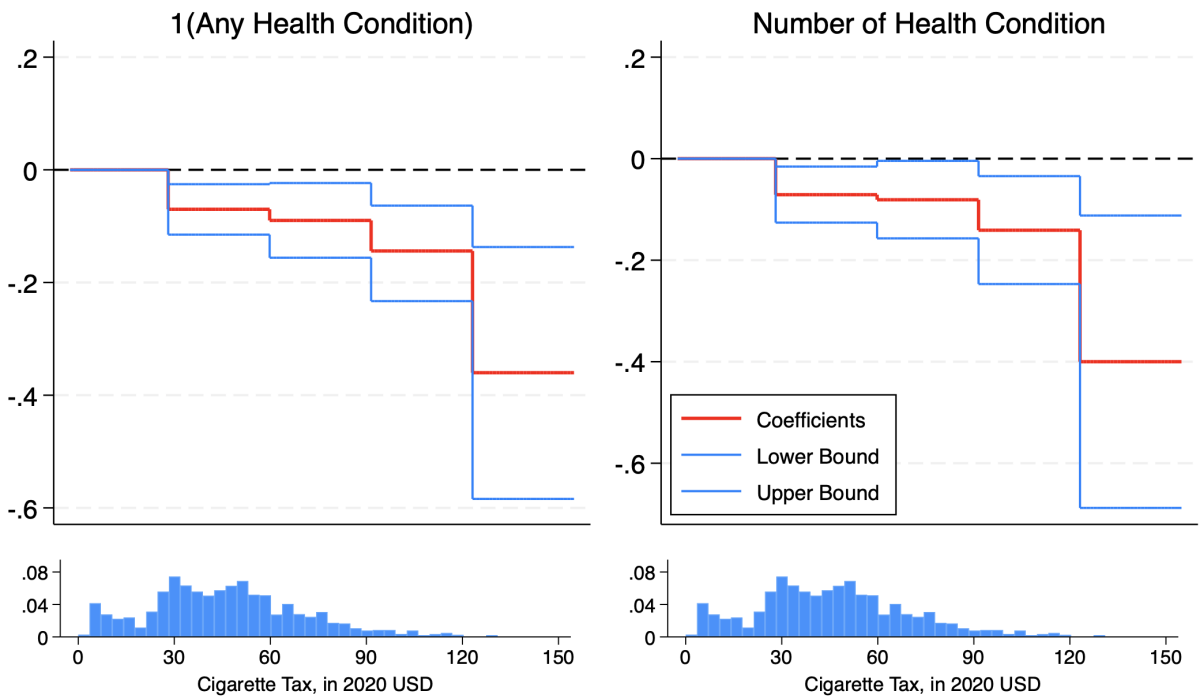


Figure 2.8: Effects of Discrete Levels of Cigarette Taxes
Note: The top two figures represent results from distinct regression analyses. The explanatory variables are five discrete levels of in utero cigarette taxes (ranging from 0-30, 30-60, 60-90, 90-120, and 120-150 cents), with the reference level being 0-30 cents. Cigarette taxes are cpi-adjusted and reported in 2020 dollars. Other specifications follow the baseline model. Confidence interval at 95%.

Mechanism

Effect on Birth Outcomes

Birth weight is one of the best proxies for infant health (Conley et al. 2006). It is also one of the best predictors for later life health (Black et al. 2007; Case et al. 2005; Currie 2009), educational and labor market outcomes (Behrman and Rosenzweig 2004; Currie and Hyson 1999; Johnson and Schoeni 2007), and life expectancy (Oreopoulos et al. 2008; Van den Berg et al. 2006). Public health professionals have identified maternal smoking during pregnancy as the largest risk factor for low birth weight that can be modified by behavior (Almond et al. 2005; Kramer 1987; Shiono and Behrman 1995) and cigarette tax can effectively reduce the likelihood that infants are born with low birth weight (Almond et al. 2005; Evans and Lien 2005; Evans and Ringel 1999).

Because information on birth weight is not available for every individual in the PSID, I use the Natality birth data from 1969 to 1994 to estimate the effect of in utero cigarette tax on birth outcomes.[12] Natality birth data not only provide birth weight, but also information on other measures of birth outcomes, such as gestational age, Apgar score, and maternal smoking behavior during pregnancy. Following Baughman and Dickert-Conlin (2009), I divide birth records into cells by state, year, the birth order of the child, and the age range, race, educational level, and marital status of the mother.[13] The birth outcomes I measure are average birth weight, average gestational age, average Apgar score, rate of very low birth weight (< 1500g), rate of preterm delivery (gestational weeks < 28), and rate of low Apgar score (< 7). The results in Table 2.10 show that a higher cigarette tax in the year of birth improves all these birth outcomes, although the effects on average birth weight and gestational age are statistically insignificant.

Health in Childhood

Table 2.11 provides suggestive evidence that exposure to cigarette tax while in utero affects health outcomes in adulthood by influencing health status in childhood. The sample consists of 4,260 children born between 1989 and 2013 and surveyed by the CDS. The results show that a 10-cent higher cigarette tax while in utero reduces the probability of ever having any physical health condition (anemia, allergies, asthma, diabetes, ear disease, hearing difficulty, eye disease, or obesity) by 0.4 percentage points; reduces the number of those physical health conditions by 0.010; reduces the probability of ever having any neurological, psychiatric, or behavioral condition (seizure disorder, autism, speech problem, learning disability, behavioral disability, or ADHD) by 0.3 percentage points (although statistically insignificant); and reduces the number of neurological, psychiatric, or behavioral conditions by 0.008 for children between ages 6 and 12. These effects account

[12] We do not use data from 1968 because it does not have information on marital status and education.

[13] Baughman and Dickert-Conlin (2009) do not use the marital status of mothers to generate the cells, possibly because they study the effects of the EITC on fertility, and EITC likely influences marital decisions. She does, however, conduct subgroup analysis by marital status.

Table 2.10: The Impact of In Utero Exposure to Cigarette Tax on Birth Outcomes and Maternal Smoking Behavior Using the Natality Data

	Average Birth Weight	Very Low Birth Weight Rate (<1500g)	Average Gestational Age	Preterm Delivery Rate (Weeks<28)	Average Apgar Score	Low Apgar Score Rate (<7)
	(1)	(2)	(3)	(4)	(5)	(6)
Cigarette Taxes, t (10 Cents)	9.038	-0.001**	0.028	-0.000*	0.064*	-0.002***
	(8.861)	0.000	(0.032)	0.000	(0.033)	(0.001)
Observations	106,025	106,025	106,025	106,025	83,918	83,918
R-squared	0.937	0.551	0.893	0.469	0.895	0.5
Y-mean	3,365	0.010	39.33	0.005	9.028	0.015
Std. Dev. of Y	128.9	0.008	0.431	0.006	0.163	0.009
Group FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes

Note: Each column is from a separate regression estimated with Natality birth data from years 1969 to 1994. We do not include data from 1968 because the educational attainment of mother and marital status of mother are not recorded in 1968. The outcome variables are average birth weight, very low birth weight rate, average gestational age, preterm delivery rate, average Apgar score, and low Apgar score rate for cells defined by state, year, marital status, age, race, educational attainment of the mother, and the birth order of the newborn. Estimates are weighted by the number of birth in the cells. Cigarette taxes are the state cigarette tax in the birth year in the child's birth state. Taxes are CPI-adjusted to 2020 dollars. The unit is 10 cents. Baseline specification includes group fixed effect and birth state fixed effect. Group is defined by state, marital status, age, race, educational attainment of the mother, and the birth order of the newborn. State covariates in the baseline model are under control. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

for no more than 3 percent of the mean, smaller than those for adult health status, indicating that the positive health effect of in utero exposure to a higher cigarette tax increases with age. However, I am cautious in interpreting these results because the sample used for the analysis on health effects in childhood is different from that used in the main analysis on health effects in adulthood and the outcome measures are not exactly the same.[14]

Smoking Behavior in Later Life

Maternal smoking during pregnancy increases the risk that the older and adult child will become a smoker and dependent on nicotine Biederman et al. (2017); Kandel et al. (1994); Lieb et al. (2003). My findings are consistent with this literature and show that higher cigarette taxes while in utero indeed reduce smoking behavior in later life. Columns 1 and 2 in Table 2.12, using the CDS data, show that a 10-cent higher cigarette tax while in utero reduces the probability of smoking by 0.5 percentage points for adolescents aged 12 to 18 and reduces the frequency of smoking in the month before the interview by around four days for those adolescents who smoke. These account for 6% and 23% of the means, respectively.

The results also show that a higher cigarette tax during the in utero period reduces smoking behavior in adulthood (Table 2.12, columns 3 and 4, based on the main PSID data). A 10-cent higher in utero cigarette tax reduces the probability of ever smoking by 1.6 percentage points and reduces the number of cigarettes smoked per day by 0.66 for smokers at age 35, both coefficients corresponding to around 5% of their means.[15]

However, reducing smoking behavior is not the only channel through which in utero cigarette tax affects health in adulthood. Table 2.13 shows that higher in utero cigarette taxes reduce health problems in adulthood even for people who never smoke. This is the

[14]Only 629 individuals are in both samples.

[15]When the information at age 35 is not available, we use data from the age closest to 35. Figure B2 shows the heterogeneous effects on smoking behavior in adulthood. Similar to the heterogeneous effects on the health condition indices, the effect on smoking behavior in adulthood is also relatively larger for individuals who are white, male, from families that were not in poverty at the time of their birth, and who had married parents at the time of their birth, compared to their counterparts.

Table 2.11: The Impact of In Utero Cigarette Tax Exposure on Childhood Health

	1(Any Physical Health Condition)	# of Physical Health Condition	1(Any Neurological, Psychiatric, or Behavioral Health Condition)	# of Neurological, Psychiatric, or Behavioral Health Condition
	(1)	(2)	(3)	(4)
In Utero Cigarette Taxes	-0.004**	-0.010*	-0.003	-0.008***
(10 Cents)	(0.002)	(0.006)	(0.002)	(0.002)
Observations	4,260	4,260	5,204	5,204
R-squared	0.06	0.077	0.082	0.084
Y-mean	0.568	0.973	0.215	0.308
(Std. Dev. of Y)	(0.495)	(1.105)	(0.411)	(0.701)
Cigarette Tax in Childhood	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes
Smoke Bans at Birth Year	Yes	Yes	Yes	Yes

Note: Each column is from a separate regression. The sample consists of children who were surveyed in Child Development Supplement in 1997, 2001, 2007, 2014 and 2019. These children born between 1989 and 2013. Estimates are weighted using the PSID longitudinal weights. Outcome variables are children's health condition at ages 6-12. The outcome variable in the first column, 1(Any physical health condition) = 1 if children have any physical problem out of anemia, allergies, asthma, diabetes, ear disease, hear difficulty, eye disease, obesity, or development delay. The outcome variable in the second column, # of physical health condition include obesity is the number if physical problem the children have out of anemia, allergies, asthma, diabetes, ear disease, hear difficulty, eye disease, obesity, or development delay. 1(Any Neurological, Psychiatric, or Behavioral Health Condition) = 1 if children have any neurological, psychiatric, or behavioral health condition out of convulsion, autism, speech problem, retardation, emotion disturbance, or hyperactivity. # of Neurological, Psychiatric, or Behavioral Health Conditions is the number of problem the children have out of convulsion, autism, speech problem, retardation, emotion disturbance, or hyperactivity. Cigarette tax at age 6 is controlled for health effect. Cigarette taxes are the state cigarette tax in the child's birth state. Taxes are CPI-adjusted to 2020 dollars. The unit is 10 cents. Other specification as baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Table 2.12: The Impact of In Utero Cigarette Tax Exposure on Smoking Behavior in Later Life

	In Adolescent (Data from PSID-CDS)		In Adulthood (Data from PSID)	
	Smoke	Smoke Days in Last Month	Ever Smoked	# Cigarettes/Day (Smokers only)
	(1)	(2)	(3)	(4)
In Utero Cigarette Taxes (10 Cents)	-0.005**	-3.981***	-0.016**	-0.660***
	(0.002)	(1.301)	(0.007)	(0.246)
Cigarette Taxes at Age 12	-0.002	-0.134	-	-
	(0.001)	(0.407)	-	-
Cigarette Taxes at Age 25	-	-	-0.001	0.004
	-	-	(0.001)	(0.039)
Observations	2,994	191	4,564	1,156
R-squared	0.199	0.571	0.085	0.242
Y-mean	0.079	17.850	0.388	12.010
Std. Dev. of Y	(0.269)	(11.44)	(0.487)	(7.855)
Birth Year FE	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes

Note: Each column is from a separate regression. For the first two columns, outcome variables are children's smoking behavior during age 12-18. The sample consists of children who were surveyed in Child Development Supplement in 1997, 2001, 2007, 2014 and 2019. Estimates are weighted using the PSID longitudinal weights. Cigarette tax in the birth state at age 12 is controlled. For the last two columns, outcome variables are individuals' smoke behavior during age 25-35. The sample consists of individuals who were born to PSID families between 1968-1994 and became household heads or spouses during age 25-35. Estimates are weighted using the average PSID longitudinal weights for ages 1-18. In utero cigarette taxes refer to the state cigarette taxes at the year-month of birth in the birth state. The model also accounts for the cigarette taxes in the individual's residence state at age 25. Both taxes are CPI-adjusted to 2020 dollars. The unit is 10 cents. Other specification as baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

case whether I look at the probability of ever having any of the listed health conditions, the number of those health conditions, or the probabilities of ever having each of those health conditions. However, the magnitudes of these effects are smaller than the effect for the whole sample, which includes both smokers and nonsmokers. This is not a rigorous test since the nonsmoker sample is an endogenous sample that is less likely to be affected by in utero cigarette tax exposure. The significant effects for this subgroup underscore the important role of in utero cigarette tax exposure on adult health.

Cognitive Ability and Educational Attainment

Cognitive ability and educational attainment are other channels through which cigarette taxes while in utero can affect health outcomes in adulthood. First, education shapes access to a wide array of material and non-material resources, including income, secure neighborhoods, and healthier lifestyles (Link and Phelan 1995). These factors can either safeguard or enhance one's health. Secondly, education enhances individuals' knowledge, skills, reasoning abilities, effectiveness, and a diverse range of other aptitudes that can be harnessed to produce health (Mirowsky 2017).

Table 2.14 provides suggestive evidence that a higher cigarette tax while in utero improves cognitive ability in childhood. Cognitive ability is measured by scores of Woodcock-Johnson tests taken by children between ages 6 and 18. The scores are comparable between different ages. The first three columns show that a 10-cent higher in utero cigarette tax statistically significantly reduces the probability of getting a low score in reading by 0.7 percentage points and improves the probability of getting a medium score by 0.8 percentage points.[16] The last three columns show that a 10-cent higher in utero cigarette tax statistically significantly reduces the probability of getting a low score in math by 0.7 percentage points and improves the probability of getting a high score by 0.7 percentage points.

[16]For both math and reading, I classify scores below percentile 33.3 as low, scores between percentile 33.3 and percentile 66.7 as medium, and scores higher than 66.7 as high.

Table 2.13: The Long-Term Effects of In Utero Exposure to Cigarette Tax on Adult Health for Never Smokers

	1(Any Health Condition) (1)	# of Health Condition (2)	Asthma (3)	Lung Disease (4)	Heart Attack (5)	Heart Disease (6)
In Utero Cigarette Taxes (10 Cents)	-0.010*	-0.013**	-0.013**	0.000	-0.002	0.002
	(0.006)	(0.006)	(0.005)	(0.003)	(0.001)	(0.002)
Cigarette Taxes at Age 25 (10 Cents)	0.000	0.001	0.000	0.000	0.000	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)
Observations	2,980	2,980	2,980	2,980	2,980	2,980
R-squared	0.064	0.062	0.069	0.065	0.044	0.052
Y-mean	0.200	0.218	0.161	0.039	0.002	0.016
Std. Dev. of Y	(0.400)	(0.457)	(0.368)	(0.194)	(0.049)	(0.124)
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes

Note: Each column is from a separate regression. The sample consists of individuals who were born to PSID families between 1968-1994 and became household heads or spouses during age 25-35 and never smoked. Other specification as baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Table 2.14: The Impact of In Utero Exposure to Cigarette Tax on Academic Performance in Childhood

	Reading Score			Math Score		
	Low Score	Medium Score	High Score	Low Score	Medium Score	High Score
	(1)	(2)	(3)	(4)	(5)	(6)
In Utero Cigarette Taxes (10 Cents)	-0.007***	0.008**	-0.001	-0.007**	0.000	0.007**
	(0.002)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
Observations	5,100	5,100	5,100	5,080	5,080	5,080
R-squared	0.224	0.059	0.197	0.205	0.051	0.213
Y-mean	0.245	0.356	0.399	0.230	0.330	0.440
Std. Dev. of Y	(0.430)	(0.479)	(0.490)	(0.420)	(0.470)	(0.496)
Cigarette Taxes in Childhood	Yes	Yes	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes

Note: Each column is from a separate regression. The sample consists of children who were surveyed in Child Development Supplement in 1997, 2001, 2007, 2014 and 2019. Estimates are weighted using the PSID longitudinal weights. Outcome variables are children's Woodcock-Johnson test standard scores. The test is provided to children older than 6 and the scores are comparable for different age. In utero cigarette taxes refer to the state cigarette taxes at the year-month of birth in the birth state. The model also accounts for the cigarette taxes in the individual's residence state at age 6. Both taxes are CPI-adjusted to 2020 dollars. The unit is 10 cents. Other specifications follow the baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Table 2.15: The Impact of In Utero Exposure to Cigarette Tax on Educational Attainment

	Highest Grade (1)	1(College or Above) (2)
In Utero Cigarette Taxes (10 Cents)	0.041	0.015**
	(0.035)	(0.007)
Observations	5,583	5,583
R-squared	0.312	0.275
Y-mean	14.16	0.39
Std. Dev. of Y	(2.128)	(0.488)
Cigarette Taxes at Age 25	Yes	Yes
Birth Year FE	Yes	Yes
Birth State FE	Yes	Yes
Individual Controls	Yes	Yes
Family Background at Birth Year	Yes	Yes
State Covariates at Birth Year	Yes	Yes

Note: Each column is from a separate regression. The outcome variables are derived from educational attainment information for individuals aged 25-35. Other specification as baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Columns 1 and 2 of Table 2.15 report estimates for the highest grade completed and the probability of having a college degree or above. A 10-cent higher in utero cigarette tax increases the highest grade completed by 0.04 and increases the probability of having college or above degree by around 1.4 percentage points, although only the latter is statistically significant.

Age at First Childbirth

Table 2.16 shows that a 10-cent higher cigarette tax while in utero delays the age at first childbirth by 0.17 years. There is a positive correlation between postponement of the first childbirth and health (Einiö et al. 2019; Lee and Park 2020; Shadyab et al. 2017). The finding that higher cigarette taxes while in utero delay the age at first childbirth could explain some of the adult health results reported above. However, there are no significant effects for the male subsample and female subsample.

In Figure 2.9, I have collected evidence for several mechanisms in which the outcome

Table 2.16: The Impact of In Utero Exposure to Cigarette Tax on Age at First Childbirth

	Age at First Childbirth		
	Whole Sample (1)	Male Sample (2)	Female Sample (3)
In Utero Cigarette Taxes	0.173*	0.15	0.188
(10 Cents)	(0.092)	(0.151)	(0.115)
Observations	3,658	1,584	2,074
R-squared	0.321	0.317	0.34
Y-mean	25.33	26.41	24.45
Std. Dev. of Y	(5.448)	(5.417)	(5.314)
Cigarette Taxes at Age 25	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes

Note: Each column is from a separate regression. The sample of the first column consists of individuals who were born to PSID families between 1968-1994. The sample of the second column and the third column are subgroups of male and female respectively. Estimates are weighted using the average PSID longitudinal weights for ages 1-18. Outcome variable is the age at first childbirth. In utero cigarette taxes refer to the state cigarette taxes at the year-month of birth in the birth state. The model also accounts for the cigarette taxes in the individual's residence state at age 25. Both taxes are CPI-adjusted to 2020 dollars. The unit is 10 cents. Other specification as baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

variables are binary, aiming to illustrate the mechanism underlying the long-term effect more clearly.

Discussion of the Effect Size

A 10-cent increase in cigarette tax during the in utero period is approximately 20% of the mean. This increase is associated with a reduction in the probability of adults aged 25-35 ever having asthma, lung disease, heart disease, or heart attack by 1.8 percentage points, corresponding to an effect size of 8% of the mean probability. Specifically, the reduction in the probability of ever having asthma is 1.7 percentage points (9% of the mean), and for lung disease, it is 0.7 percentage points (6% of the mean).

These findings are in line with previous literature. Table 2.17 presents a detailed

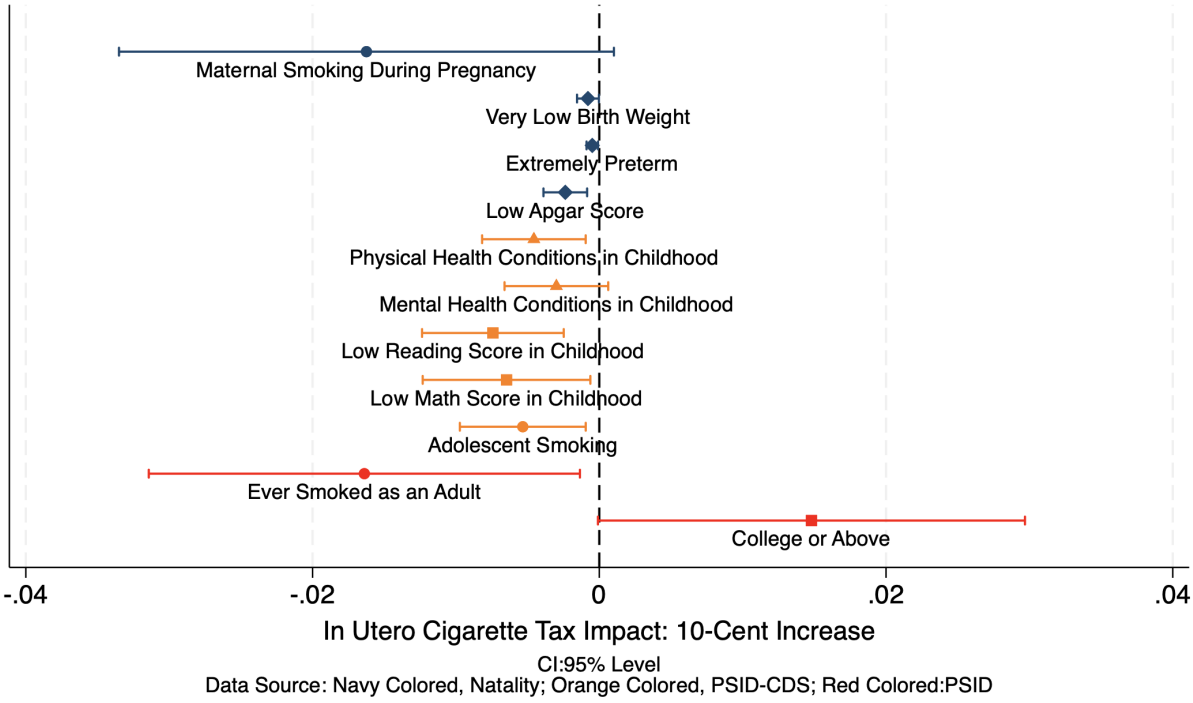


Figure 2.9: Mechanism Analysis

Note: Each coefficient is from a separate regression. All the outcome variables are dummy variables. In utero cigarette tax refers to the state cigarette tax in the birth state at the year-month of birth. Taxes are cpi-adjusted and reported in 2020 dollars. The unit is 10 cents. The navy colored results are estimated with data from Natality Files. The orange colored results are estimated with PSID-CDS data. The red colored results are estimated with PSID data. Confidence interval at 95%.

comparison of the estimated effect sizes with scaled effect sizes in previous literature that are comparable to the results. Hoehn-Velasco et al. (2023) is the only study that investigated the long-term effect of cigarette taxes during the in utero period. That study shows that a 10-cent in utero cigarette tax significantly reduces the probability that a woman will be overweight or obese during pregnancy by 0.4 percentage points, which corresponds to an effect of 1% of the mean. Simon (2016) estimated the effect of in utero cigarette tax on health conditions in childhood, and found that a 10-cent higher in utero cigarette tax significantly reduces the probability of children ages 2 to 17 having asthma by 0.8 percentage points, which corresponds to 14% of the mean. The effect sizes are not exactly the same because the outcome variables are not the same or are measured at different ages, and there are differences in sample characteristics and average cigarette tax. However, it is safe to say

that my findings are consistent with previous literature.

I want to emphasize that the estimates are based on relatively modest cigarette tax rates between 1968 and 1994, with an average tax of 0.50. Therefore, I cannot straightforwardly extend the estimates to predict the impact of cigarette taxes outside of this tax rate range.

I also compare the estimated long-term effects of cigarette taxes while in utero with the long-term effects of changes in other aspects of the in utero environment in Table 2.18. Hoynes et al. (2016b) find that access to the food stamps program in childhood (from no exposure to full exposure starting in utero and continuing until age five) reduces metabolic syndrome by 0.3 of a standard deviation, while my study finds that a 10-cent higher cigarette tax during the in utero period reduces the probability of ever having asthma, lung disease, heart disease or heart attack by 0.04 of a standard deviation. Hwang (2019) finds that a one-year exposure to the WIC program in early life reduces the probability of having asthma in adulthood by 3.7 percentage points, while my study finds that a 10-cent higher cigarette tax while in utero reduces the probability of ever having asthma by 1.8 percentage points. With a linear extrapolation, a 10-cent increase in cigarette tax while in utero is equivalent to about a half-year exposure to the food stamps program or WIC program.

2.7 Conclusion and Discussion

In this study, I investigate the effects of the level of the cigarette tax during the in utero period on long-term health outcomes in adulthood. I find that exposure to higher cigarette taxes while in utero leads to better health in adulthood and that the effects strengthen with age. My findings resonate with a robust body of literature that underscores the effects of early-life exposure on a spectrum of health parameters in the long run.

By delving into the multifaceted mechanisms at play, I have established a compelling case for the role of birth outcomes, health in childhood, smoking behavior in later life, cognitive ability, educational attainment, and age at first childbirth as pathways through

Table 2.17: Comparison of Effect Sizes of In Utero Cigarette Tax Exposure on Health Condition

	Adult Health				Mechanism: Childhood Health			
	Our Analysis			Hoehn-Velasco et al. (2023)	Our Analysis	Simon (2016)		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Data Source	PSID			Natality Files	PSID-CDS	NHIS		
Birth Cohorts	1968-1994			1965-2000	1989-2013	1988-2009		
Average Cig. Taxes	0.50			0.84	0.99	1.24		
Outcome (Y)	Probability of ever having any of the following health conditions: asthma, lung disease, heart disease, heart attack.	# health conditions ever have among asthma, lung disease, heart disease, heart attack	Probability of ever having asthma	Probability of being overweight or obese during pregnancy	Probability of ever having any physical health conditions	Probability of having asthma	Probability of two or more doctor visits per year	Sick days from school
Surveyed Age	25-35	25-35	25-35	First-delivery pregnant women aged 18-49	6-12	2-17	2-17	5-17
Coefficient Corresponding to a 10 Cents Tax Increase	-1.8 pp	-0.021	-1.7 pp	-0.4 pp	-0.4 pp	-0.8 pp	-0.2 pp	-0.03
Mean of Y	24%	0.279	18.5%	48.2%	56.8%	5.8%	61.9%	3.4%
Effect Compared to the Mean	8%	8%	9%	1%	1%	14%	0.4%	1%

Note: This table compares sample characteristics and effect sizes in Hoehn-Velasco et al. (2023) and Simon (2016). All effect sizes estimated by Hoehn-Velasco et al. (2023) and Simon (2016) are scaled to be comparable to our estimated effects, i.e., they reflect the impact of 10-cent tax increases in terms of real 2020 dollars.

Table 2.18: Comparison of Effect Sizes of Early-Life Exposure to Different Polies on Health Condition

	Our Analysis (1)		Hoynes et al. (2016) (2)	Hwang (2019) (3)
Policy Exposure	Cigarette Taxes		Food Stamp Program	Special Supplemental Nutrition Program for Women, Infants, and Children
Exposed Period	In Utero		In Utero to Age 5	In Utero to Age 5
Data Source	PSID		PSID	PSID
Birth Cohort	1968-1994		1956-1981	1970-1985
Sample	No limitation on parental education		Children whose parents had less than high school education	Children whose parents are at most high school graduates
Outcome (Y)	Probability of ever having any of the following health conditions: asthma, lung disease, heart disease, heart attack.	# Health conditions ever had among asthma, lung disease, heart disease, heart attack	Metabolic Health Index in Adulthood	Asthma
Surveyed Age	25-35	25-35	18-53	18-45
Main Results	A 10-cent (2020$) increasing in in utero cigarette taxes reduces the probability of ever developing asthma, lung disease, heart disease, or heart attack by 1.8 percentage points	A 10-cent (2020$) increasing in in utero cigarette taxes reduces the number of health conditions among asthma, lung disease, heart disease, or heart attack by 0.021	Change from no access to food stamp to full exposure to food stamp in utero to age five reduces metabolic syndrome by 0.3 standard deviations	Change from no access to full exposure to the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) in utero to age five reduces the probability of having asthma in adulthood by 14.7 percentage points
Mean of Y	24%	0.279	0.01	0.132
Effect Compared to the Mean	8%	8%	N/A	111%

Note: This table compares our research to Hoynes et al. (2016b) and Hwang (2019).

which cigarette taxes while in utero affect health in adulthood.

The significance of my study lies in its comprehensive approach to unraveling the intricate web of effects stemming from early-life exposure to higher cigarette taxes. The identification of various mechanisms and their interconnectedness showcases the complexity of the relationship between exposure at birth and adult health, offering valuable insights for both researchers and policymakers.

My research is not without limitations. The main sample spans birth cohorts from 1968 to 1994, a period marked by relatively modest year-to-year fluctuations in state cigarette tax rates. As such, the estimates may not fully encapsulate the repercussions of more recent and substantial tax hikes. This presents an avenue for future exploration – to scrutinize the long-term ramifications of significant tax increases in the modern context and over a larger range of values, in order to enhance our understanding of the dynamic interplay between tax policies and health outcomes.

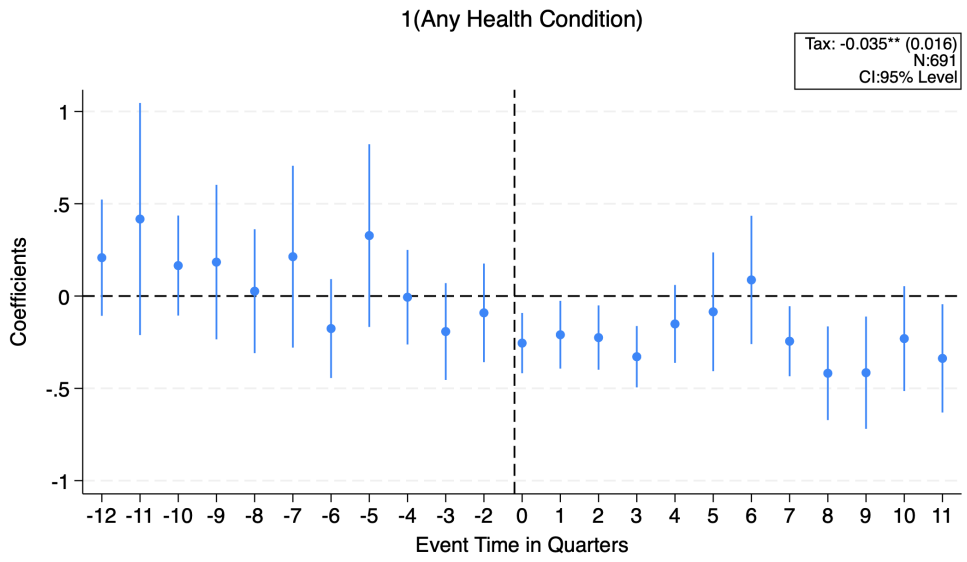
In conclusion, my study provides a comprehensive and illuminating assessment of the profound and enduring consequences of higher cigarette taxes during the in utero period for health outcomes in adulthood. By further uncovering the underlying mechanisms, my research contributes to the evolving discourse on early-life policy interventions and their far-reaching implications for public health and well-being.

Appendix

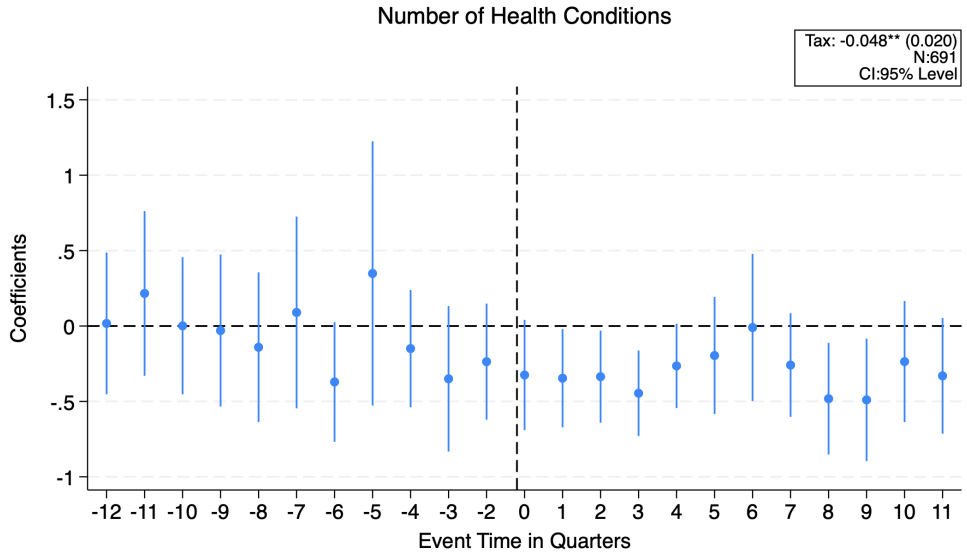
Table B1: The Effects of State Cigarette Taxes on State-Level Demographic, Economic Policies, and Economic Conditions

	Cigarette Taxes at Time t	
State Covariates at Time t + 1		
Male Percent	0.003	(0.025)
White Percent	-0.018	(0.030)
Married Percent	0.007	(0.050)
Less than HS percent	0.007	(0.030)
High School or Some College Percent	-0.001	(0.034)
College or Higher Education Percent	-0.006	(0.015)
Average Age	0.176	(1.843)
State Maximum CDCTC	0.187	(0.308)
State Maximum EITC	-0.039	(0.039)
State Maximum AFDC/TANF Benefit	0.009	(0.076)
State Hospital Beds	1.204	(4.713)
State Expenditure on Medicaid and Similar Programs	-0.308	(0.813)
State Minimum Wage	0.264	(0.828)
State Income MTR	-0.46	(1.244)
State Per Capita GDP	0.292	(3.288)
State FE	Yes	
Year FE	Yes	
N	1,230	

Note: Each row is from a separate model with the full set of covariates using samples aggregated to the state-year level. Data are from 1968-1994. Outcome variables are state-level demographics, economic policies, or economic conditions in year t+1. Cigarette taxes are from year t. State and year fixed effects are included in the model. Standard errors clustered by state are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.



(a) 1(Any Health Condition)



(b) Number of Health Condition

Figure B1: Robustness of Event Study Results to the Alternative Cutoff at the 75th Percentile
Note: Each figure represents a separate estimation. Events are defined as tax hikes exceeding the median increase during 1968-1994. Event time is denoted in quarters. The coefficient at period j reflects the event effect for the cohort born in that period. The sample comprises individuals born to PSID families during the "high frequency" tax period of 1968-1976, who became household heads or spouses at ages 25-35. Only individuals born in states where only one event happened in the period are included. Estimates are weighted using average PSID longitudinal weights from ages 1-18. Outcome variables are individuals' health conditions aged 25-35, with other settings as the baseline model. Confidence intervals are at 95%.

Table B2: The Effects of In Utero Exposure to Cigarette Tax on Health Conditions in Adulthood across Different Age Ranges

	1(Any Health Condition) (1)	# of Health Conditions (2)
Panel A. Outcome Variable = Health Condition at Ages 25-30		
In Utero Cigarette Taxes (10 Cents)	-0.014**	-0.019**
	(0.006)	(0.007)
Cig. Taxes at Age 25 (10 Cents)	-0.002	-0.001
	(0.001)	(0.001)
N	5,025	5,025
R-squared	0.049	0.05
Mean of Y	0.216	0.243
Std. Dev. of Y	(0.411)	(0.490)
Panel B. Outcome Variable = Health Condition at Ages 30-35		
In Utero Cigarette Taxes (10 Cents)	-0.023***	-0.026***
	(0.007)	(0.008)
Cig. Taxes at Age 30 (10 Cents)	-0.001	-0.001
	(0.001)	(0.001)
N	4,227	4,227
R-squared	0.05	0.055
Mean of Y	0.194	0.224
Std. Dev. of Y	(0.395)	(0.486)
Panel C. Outcome Variable = Health Condition at Ages 35-40		
In Utero Cigarette Taxes (10 Cents)	-0.025***	-0.034***
	(0.008)	(0.010)
Cig. Taxes at Age 35 (10 Cents)	0	0
	(0.001)	(0.001)
N	2,958	2,958
R-squared	0.057	0.056
Mean of Y	0.202	0.244
Std. Dev. of Y	(0.402)	(0.529)
Panel D. Outcome Variable = Health Condition at Ages 40-45		
In Utero Cigarette Taxes (10 Cents)	-0.031*	-0.058**
	(0.017)	(0.029)
Cig. Taxes at Age 40 (10 Cents)	-0.021	-0.027*
	(0.015)	(0.015)
N	1,707	1,707
R-squared	0.094	0.09
Mean of Y	0.209	0.27
Std. Dev. of Y	(0.406)	(0.588)
Birth Year FE	Yes	Yes
Birth State FE	Yes	Yes
Individual Controls	Yes	Yes
Family Background at Birth Year	Yes	Yes
State Covariates at Birth Year	Yes	Yes

Note: Each column of each panel is from a separate regression. Outcome variables are measured at different age range in each panel. Other specifications follow the baseline model. Standard errors clustered by state of birth are in parentheses.
Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

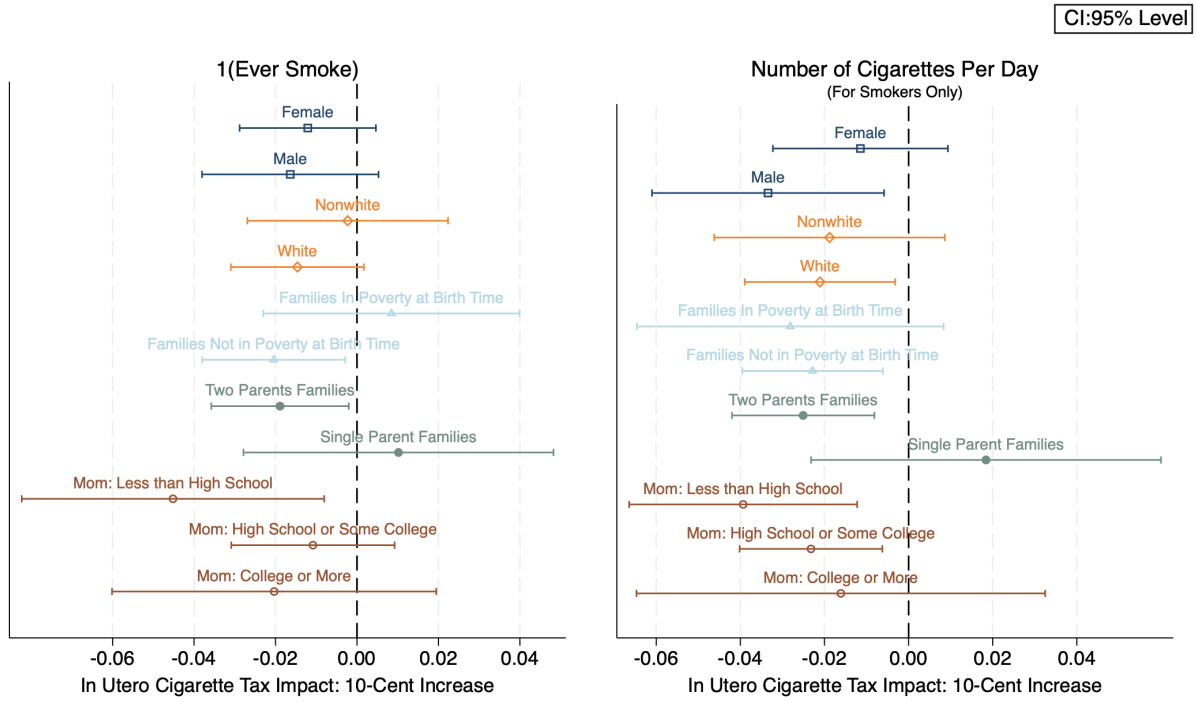


Figure B2: Subgroup Analysis for the Impact of In Utero Exposure to Cigarette Tax on Smoking Behaviors in Adulthood

Note: The sample size for the subgroup of individuals whose mothers have college degree or higher is very small (< 100). Each estimated coefficient is from a separate regression. The sample consists of individuals who were born to PSID families between 1968-1994 and became household heads or spouses during ages 25-35. Estimates are weighted using the average PSID longitudinal weights for ages 1-18. Mother's educational level is categorized as less than high school (if highest grade < 12), high school/some college (if highest grade 12-15), and college or above (if highest grade $\geqslant 16$). For the economic status subgroups, "in poverty" families are defined as families with income less than or equal to 1.5 times Census need standard. Outcome variables are smoking behaviors during age 25-35. Other specifications follow the baseline model. Standard errors are clustered by state of birth. Confidence intervals are at the 95%.

Table B3: Robustness Checks on Sampling Weight

	1(Any Health Condition) (1)	# of Health Conditions (2)
Panel A. Estimates Weighted Using PSID Longitudinal Weight at Birth Year		
In Utero Cigarette Taxes (10 Cents)	-0.017***	-0.021***
	(0.006)	(0.008)
Cigarette Taxes at Age 25 (10 Cents)	-0.002*	-0.002
	(0.001)	(0.001)
Observations	5,402	5,402
R-squared	0.049	0.051
Mean of Y	0.240	0.279
Std. Dev. of Y	(0.427)	(0.530)
Panel B. Estimates Weighted Using PSID Longitudinal Weight at Age 18		
In Utero Cigarette Taxes (10 Cents)	-0.016**	-0.021***
	(0.006)	(0.008)
Cigarette Taxes at Age 25 (10 Cents)	-0.002*	-0.002
	(0.001)	(0.001)
Observations	5,402	5,402
R-squared	0.048	0.051
Mean of Y	0.240	0.279
Std. Dev. of Y	(0.427)	(0.530)
Birth Year FE	Yes	Yes
Birth State FE	Yes	Yes
Individual Controls	Yes	Yes
Family Background at Birth Year	Yes	Yes
State Covariates at Birth Year	Yes	Yes

Note: Each column of each panel is from a separate regression. Specifications follow the baseline model except for the estimation weight. Standard errors clustered by state of birth are in parentheses. Significance levels: * p<0.1, ** p<0.05, *** p<0.01.

Table B4: Robustness Check: Control State-Specific Linear Time Trend

	1(Any Health Condition) (1)	# of Health Conditions (2)	Asthma (3)	Lung Disease (4)	Heart Attack (5)	Heart Disease (6)
In Utero Cigarette Taxes (10 Cents)	-0.020***	-0.022**	-0.022***	0.003	-0.001	-0.002
	(0.007)	(0.009)	(0.008)	(0.004)	(0.001)	(0.002)
Cigarette Taxes at Age 25 (10 Cents)	-0.001	-0.001	-0.001	0.000	0.000	-0.000*
	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)
Observations	5,402	5,402	5,402	5,402	5,402	5,402
R-squared	0.063	0.063	0.064	0.058	0.045	0.049
Mean of Y	0.240	0.279	0.185	0.070	0.005	0.019
Std. Dev. of Y	(0.427)	(0.530)	(0.388)	(0.254)	(0.071)	(0.137)
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes
State-Specific Linear Time Trend	Yes	Yes	Yes	Yes	Yes	Yes

Note: Each column in each panel is from a separate regression. Specifications follow the baseline model except for controlling state-specific linear trends. Standard errors clustered by state of birth are in parentheses. State-specific linear time trend is also controlled.
Significance levels: * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table B5: OLS Regression and Logistic Regression

	1(Any Health Condition) (1)	# of Health Condition (2)	Asthma (3)	Lung Disease (4)	Heart Attack (5)	Heart Disease (6)
Panel A. OLS Regression						
In Utero Cigarette Taxes (10 Cents)	-0.018***	-0.021***	-0.017***	-0.004	-0.001	0.001
	(0.006)	(0.008)	(0.006)	(0.003)	(0.001)	(0.001)
Odds Ratio	0.947	0.952	0.927	0.947	0.801	1.052
	(0.019)	(0.021)	(0.026)	(0.040)	(0.199)	(0.052)
Panel B. Logistic Regression						
In Utero Cigarette Taxes (10 Cents)	-0.105***	-0.107***	-0.122***	-0.080	-	-0.146
	(0.040)	(0.040)	(0.045)	(0.072)	-	(0.134)
Odds Ratio	0.900	0.900	0.885	0.923	-	0.864
	(0.036)	(0.036)	(0.039)	(0.066)	-	(0.115)
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth State FE	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Family Background at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes
State Covariates at Birth Year	Yes	Yes	Yes	Yes	Yes	Yes

Note: The convergence for heart attack for logistic regression is not achieved .
Results in Panel B-column 1,3,4,6 is from logit regression and results in Panel B-column 2 is from an ordered logit regression. Odds is the probability that the individual has any of the four smoking-related conditions rather than s/he does not have any of those conditions. For column 2, odds is the probability that the outcome variable is less than or equal to j rather than greater than j. Odds ratio is the odds at the 10-cent higher cigarette tax over the odds at the current cigarette tax. Specification follows the baseline model. Standard errors clustered by state of birth are in parentheses. Significance levels: * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

3 LONG-TERM HEALTH AND BEHAVIORAL EFFECTS OF EARLY CHILDHOOD EXPOSURE TO THE CHILD AND DEPENDENT CARE TAX CREDIT: A LONGITUDINAL ANALYSIS FROM AGES 6-18

3.1 Introduction

The Child and Dependent Care Tax Credit (CDCTC) is a childcare subsidy enacted by the federal government in 1976. The objective of the CDCTC is to encourage parents, particularly mothers, to join the labor force or stay in it. As the largest tax program in the U.S. specifically targeting childcare costs, it facilitated over 5 million households to claim $2.8 billion in expenses during fiscal year 2020.[1]

To be eligible for the CDCTC, both parents must be employed if they are married. Likewise, single parents must be employed to qualify for the tax credit, and for divorced couples, the child's main caregiver must be employed. Parents are responsible for covering their child's care costs. Thus, typical expenditures are those incurred for daycare centers, after-school care, and summer camps. Parents can claim the credit until their child is 12 years old. There is considerable evidence that the CDCTC increases labor supply for mothers, particularly married mothers (Averett et al. 1997; Averett and Wang 2023; Jiang 2020; Pepin 2020). To benefit from the credit, children must be in childcare while their parents work. Participation in non-parental childcare arrangements is now the norm for preschool age children in the U.S. but there is disagreement as to its effects on children (Herbst 2023). This paper asks whether early childhood exposure to the CDCTC, the central childcare subsidy for non-welfare recipients in the US, affects children's health in later years, specifically for children ages 6-18.

There are two potential channels by which the CDCTC can affect children's health. The first is that increased income from the mothers' work can positively affect a child's health

[1] See https://www.taxpolicycenter.org/statistics/credit-type-and-amount (accessed 6/12/2023).

as it increases the monetary resources available to the family. This may enable the family to afford higher-quality health care. The family may also be able to afford better housing and more nutritious food and live in a better neighborhood. All these factors have been shown to affect children's health positively. In addition, children in child care may develop better social skills, such as learning to cooperate and play with others, and follow rules. They may also receive developmentally appropriate care, which better prepares them for formal schooling. We can consider these benefits as income effects.

On the other hand, non-parental childcare may have negative implications for children's health. Mothers may be better positioned to provide children with individualized care to thrive. Thus, their children may suffer when mothers allocate more time to the labor market and substitute parental care with non-parental care. For example, children exposed to non-maternal care may struggle to form secure attachments with their mothers, potentially leading to behavioral issues and compromised mental health. Additionally, mothers typically excel as caregivers, whereas childcare workers may need to attend to the needs of multiple children simultaneously. Moreover, in childcare settings, children may encounter sick peers, increasing their susceptibility to illness. Furthermore, they may have access to less nutritious food and limited opportunities for physical activity, resulting in compromised physical health. This can be thought of as the substitution effect. However, there is an emerging body of evidence indicating that high-quality childcare can be beneficial for children from disadvantaged families. For these children, non-parental care may offer more age-appropriate and stimulating environments, potentially leading to improvements in both physical and mental health (Herbst 2023; Herbst and Tekin 2016). Because the income and substitution effects may work in opposite directions, the effect of the CDCTC on child health and health-related outcomes is an empirical question.

Childcare exposure may be endogenous to children's health. Therefore, we investigate how early-life exposure to the CDCTC subsidy affects children's health in later childhood. Specifically, we leverage the variation in maximum CDCTC benefits to which children born

and lived in different states and years were exposed to identify the effect of early childhood exposure to the CDCTC on health in later childhood. We use all available waves of the Child Development Supplement (CDS) of the Panel Survey of Income Dynamics (PSID) and the two-way fixed-effects (TWFE) model. We find that early childhood exposure to the CDCTC has differential effects on health in later childhood for children of mothers with different education levels. Children with mothers who have more than a high school education experience adverse physical and mental health impacts; those with mothers of high school education do not experience any difference in their physical and mental health, while those with mothers who did not finish high school benefit physically and mentally from exposure to the CDCTC. We further conduct several robustness checks.

This study makes significant contributions to the existing literature by being the first to examine the effect of early childhood exposure to the CDCTC on later childhood health using longitudinal data and variation in CDCTC benefits over two decades. While previous research on the CDCTC has predominantly focused on its effect on women's labor supply, with two exceptions, one which studies the fertility effects of the CDCTC (Averett and Wang 2023) and the other that studies the impact of the CDCTC on education (Jiang 2020), we are the first, to our knowledge, to investigate its potential effects on child health and behaviors comprehensively.

Our work has important policy implications. Our findings indicate that subsidizing childcare through tax credits may have consequences for children's health, but these impacts vary by the level of a mothers' education. These findings thus provide evidence to federal and state governments on the vital yet overlooked health effects of the CDCTC, which is crucial in a comprehensive evaluation of this important policy.

The rest of this paper is organized as follows. We start by providing a brief review of related literature, followed by an overview of the CDCTC. Then, we detail our data, empirical strategy, and results. We conclude with key insights from our research.

3.2 Previous Literature

Our work contributes to two strands of literature. The first is the effect of childcare on children's outcomes. The second is the effect of tax policies (mainly the Earned Income Tax Credit) on children's outcomes.

There is a fairly sizable literature in economics that aims to address the issue of the effect of childcare and childcare subsidies on children's outcomes (Baker et al. 2008; Black et al. 2014; Herbst 2017; Herbst and Tekin 2010, 2012, 2016). Herbst (2023) provides a recent summary of some of this work. This work focuses on a variety of outcomes including mental health, child development, behavioral outcomes, and physical health. Much of this work revolves around examining the effect of the Child Care and Development Fund (CCDF) which is a program aimed at subsidizing childcare for the working poor. In contrast, we know almost nothing about the effects of the CDCTC on any of these outcomes despite the fact that other than the CCDF, it is the only other mechanism for government subsidization of childcare in the US.

Herbst and Tekin (2012, 2016) estimate the short- and medium-run impact of the CCDF, a childcare subsidy program restricted to the working poor, as noted above. One of their studies suggests that this childcare subsidy may have short-term negative effects that disappear as children age (Herbst and Tekin 2016). They report that the behavioral effects of subsidized childcare are larger for children with more educated mothers. On the other hand, Herbst and Tekin (2012) find that subsidized childcare leads to sizeable increases in the prevalence of overweight and obesity among low-income children. When examining summer childcare, Herbst (2013) finds that the more negative impacts of childcare participation are limited to the children of relatively well-educated mothers, but he does not find that more disadvantaged children benefit from childcare participation, contrary to the findings of others.

A closely related literature examines the effect of another tax subsidy, the Earned Income Tax Credit (EITC), on children's outcomes. While the EITC is not aimed specifically at

childcare, it does create an income effect for eligible low-income families. However, because it also incentivizes the mothers' labor supply, it too may also have a time-allocation effect. This literature tends to find a positive effect of the EITC on a variety of child outcomes (Averett and Wang 2018; Bastian and Michelmore 2018b; Dahl and Lochner 2012).

We are aware of only one previous paper that specifically examines exposure to the CDCTC in early life on later outcomes (Jiang 2020). He finds that exposure to the CDCTC in early life is associated with fewer years of education as an adult and that this is particularly the case for mothers with higher levels of education. In this paper, we take an empirical approach similar to that of Jiang (2020).

3.3 Description of the CDCTC

The CDCTC is a tax credit designed to cover a portion of an individual's child and dependent care expenses related to employment. Although the non-refundable feature of the federal CDCTC benefits makes it difficult for low-income families to claim the tax credit, the primary goal of the federal CDCTC upon introduction in 1976 was to assist working mothers, particularly single mothers, in covering childcare costs while pursuing employment. Hence, the credit is contingent upon using childcare to support work (or schooling). Currently, eligible children must be under the age of 13.

Families can claim expenses up to $3,000 with one eligible child in care and $6,000 for families with two or more eligible children. The amount claimed cannot exceed the earned income of the taxpayer or their spouse with the lower income. The federal CDCTC is not refundable. Thus, a family cannot receive more benefits than they owe in federal income taxes. Finally, the credit is graduated on a sliding scale. Families with an Adjusted Gross Income (AGI) of $15,000 or less are eligible for a credit equal to 35 percent of eligible expenses. The rate decreases as AGI increases above $15,000 until it reaches 20 percent for families with AGIs above $43,000 (see Figure 3.1). Thus, in theory, families with an AGI of

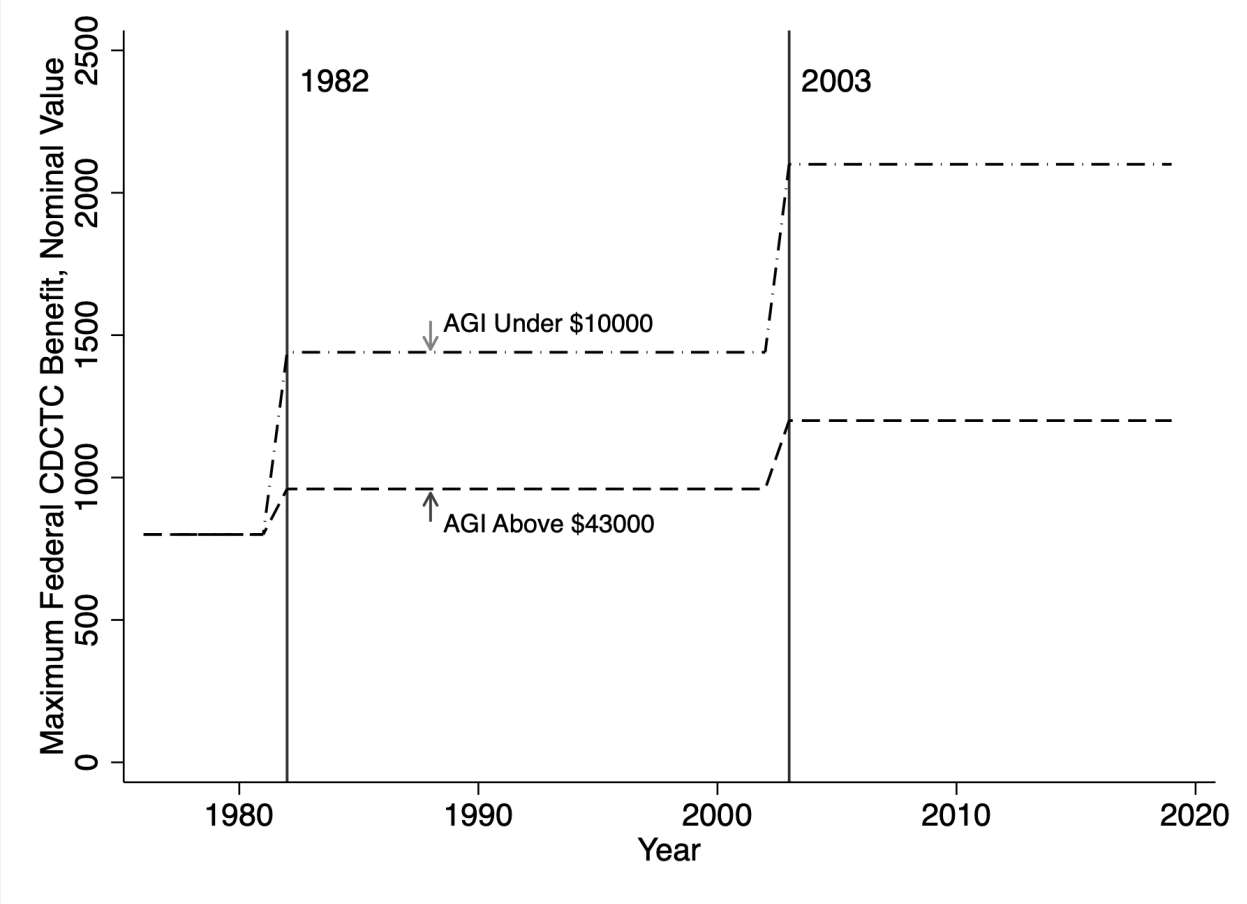


Figure 3.1: Structure of the Federal CDCTC over Time

$15,000 or less are eligible for a maximum credit of $1,050 per eligible child. In contrast, families with incomes above $43,000 are eligible for a maximum credit of $600 per eligible child. According to the Congressional Research Service, the average credit claimed in 2020 ranged between $500 and $600.

Figure 3.2 illustrates the distribution of federal CDCTC recipients by income. For comparison purposes, we also include the same statistics for the Earned Income Tax Credit (EITC)[2], another federal program aimed to incentivize employment among low- and medium-income families that may also affect children's health as described earlier. It is not surprising that there is limited overlap between those who claim the CDCTC and those who claim the EITC, given that the EITC primarily targets lower-income families

[2]Because the 2012 data are missing, we smoothed across adjacent years.

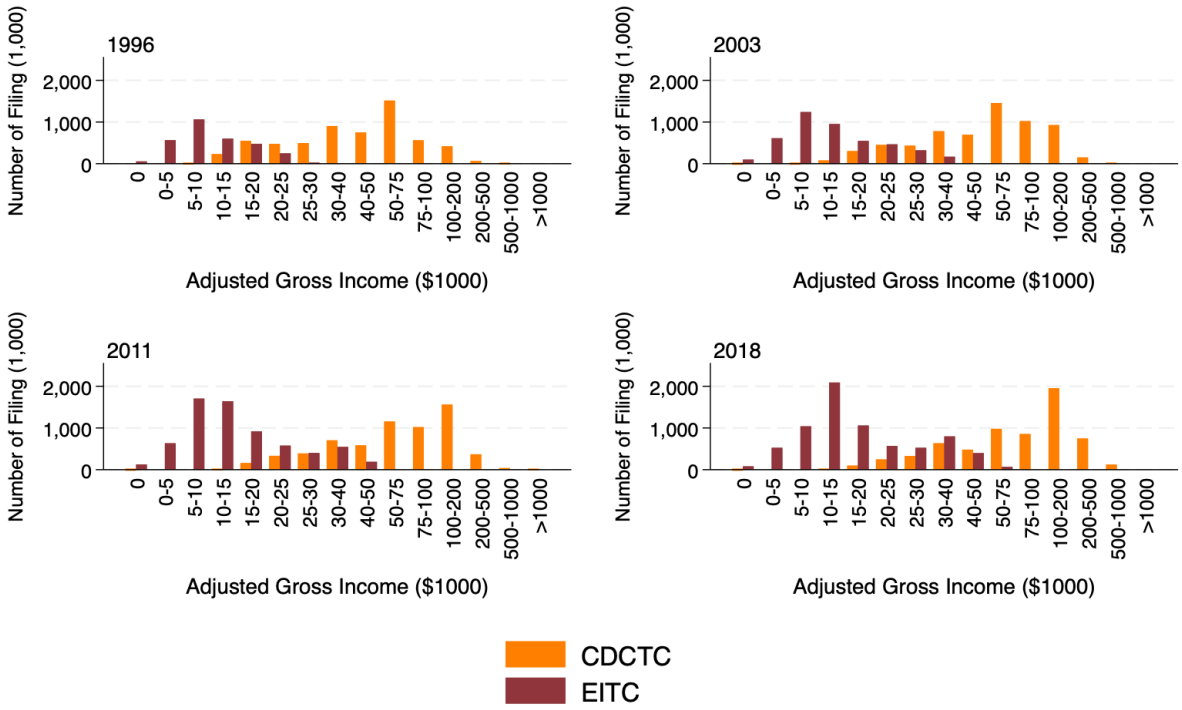


Figure 3.2: Distribution of Recipients of CDCTC Compared to EITC Recipients

than the CDCTC and phases out completely once the AGI reaches a certain level while the federal CDCTC is not refundable which means it may provide fewer benefits to low-income families. The figure indicates that middle- and upper-income families are more likely to use the CDCTC, while lower-income families are more likely to benefit from the EITC. Appendix Figure A1 displays the eligibility rate for the CDCTC by AGI for various years, while Appendix Figure A2 presents the take-up rates for the CDCTC by AGI for different years. These figures further suggest that the CDCTC is most often used by middle- and higher-income families.

Shortly after the federal CDCTC was enacted, states began implementing their CDCTCs. The initial group of states introduced their state CDCTCs between 1976 and 1977, almost immediately after the federal CDCTC was enacted. As shown in Figure 3.3, by 2019, 25 states and Washington D.C. had implemented their CDCTC. Many of these state CDCTCs are structured as a percentage of the federal CDCTC. For example, in our sample period, Iowa had the lowest percentage of 5 percent, while Oregon offered a generous credit of 188

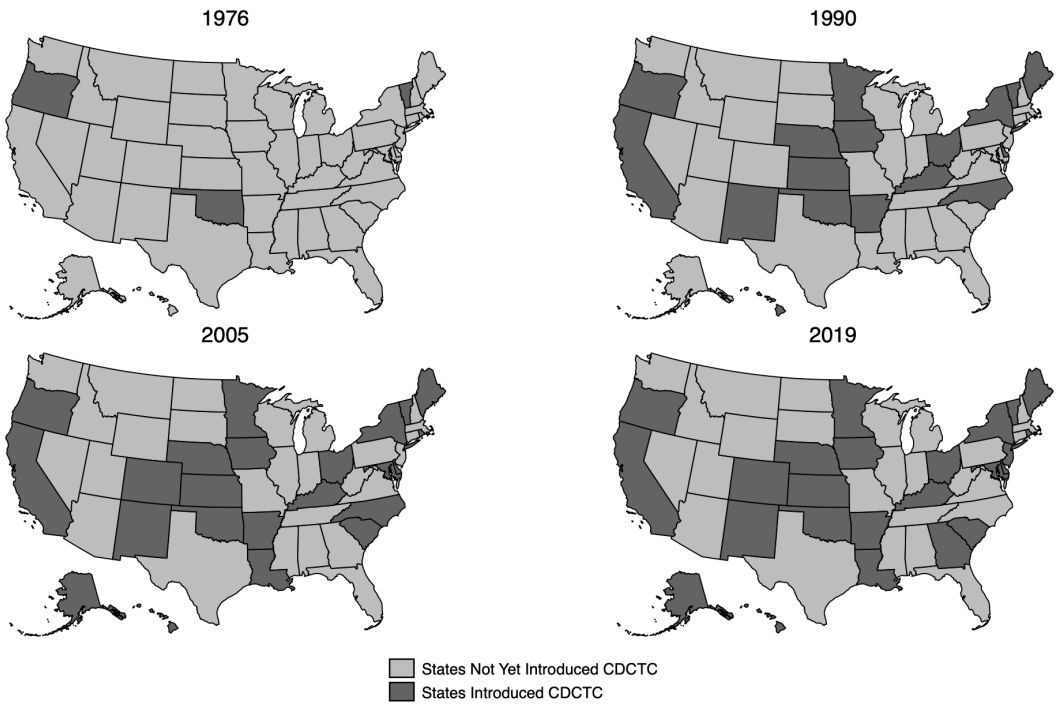


Figure 3.3: State-Level Introduction of the CDCTC over Time

percent of the federal credit. Although the CDCTC is not refundable at the federal level, 12 states had refundable credits in 2019. In these cases, taxpayers can be refunded if the state CDCTC exceeds the state tax liability. Figure 3.4 depicts the generosity of state CDCTCs using a heat map, with darker colors indicating states and years with the most generous CDCTC. Over time, CDCTC generosity has generally diminished because the CDCTC is not indexed to inflation. Yet, more families have been exposed to the CDCTC over time due to many states enacting refundable CDCTCs. Figure 3.5 depicts the variation in the generosity of State CDCTC benefits over time using a line graph.

Parents do not receive the tax credit until they file their taxes, which means they must pay for childcare upfront and wait up to a year to be reimbursed. Moreover, only certain expenses are eligible for the credit, which tend to be more formal childcare arrangements. Therefore, lower-income families may be less likely to use the CDCTC, as they are more inclined to rely on family members, such as grandparents, for care (Laughlin 2010). Conversely, those with higher incomes are more likely to benefit from the credit due to higher

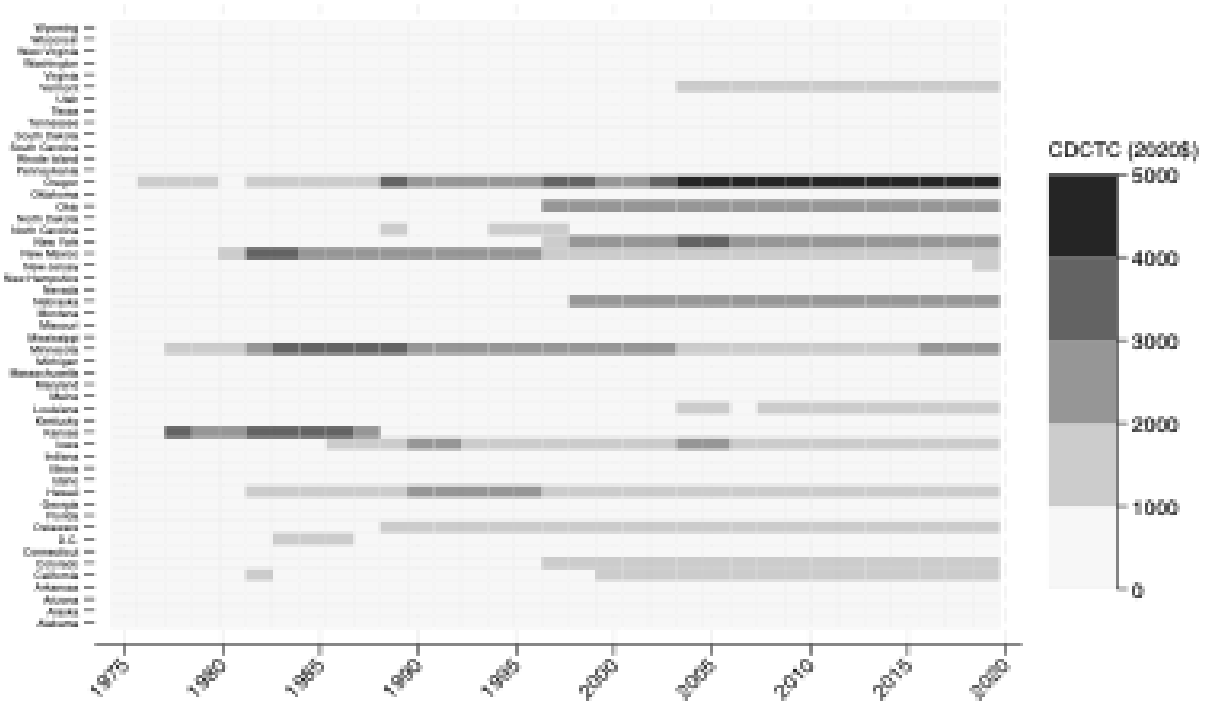


Figure 3.4: Heat Map of CDCTC Generosity by State

childcare expenses and a greater likelihood of paying for childcare. Those at the lower end of the income distribution are less likely to incur a tax liability and, hence, less likely to be eligible for the tax credit. Early studies on the CDCTC have documented that making the CDCTC refundable would significantly increase the amount families spent on market-provided childcare (Michalopoulos et al. 1992). Newer studies suggest that making the credit refundable would enhance the likelihood of utilization among single parents (Pepin 2021). Finally, evidence indicates that a ten-percentage point increase in the CDCTC leads to a 5-percentage point rise in the use of paid childcare (Pepin 2020).

3.4 Data

We use data from the Panel Study of Income Dynamics (PSID) and its Child Development Supplement (CDS). The PSID is the longest-running longitudinal survey in the world, starting in 1968 with a nationally representative sample of around 5,000 families in the

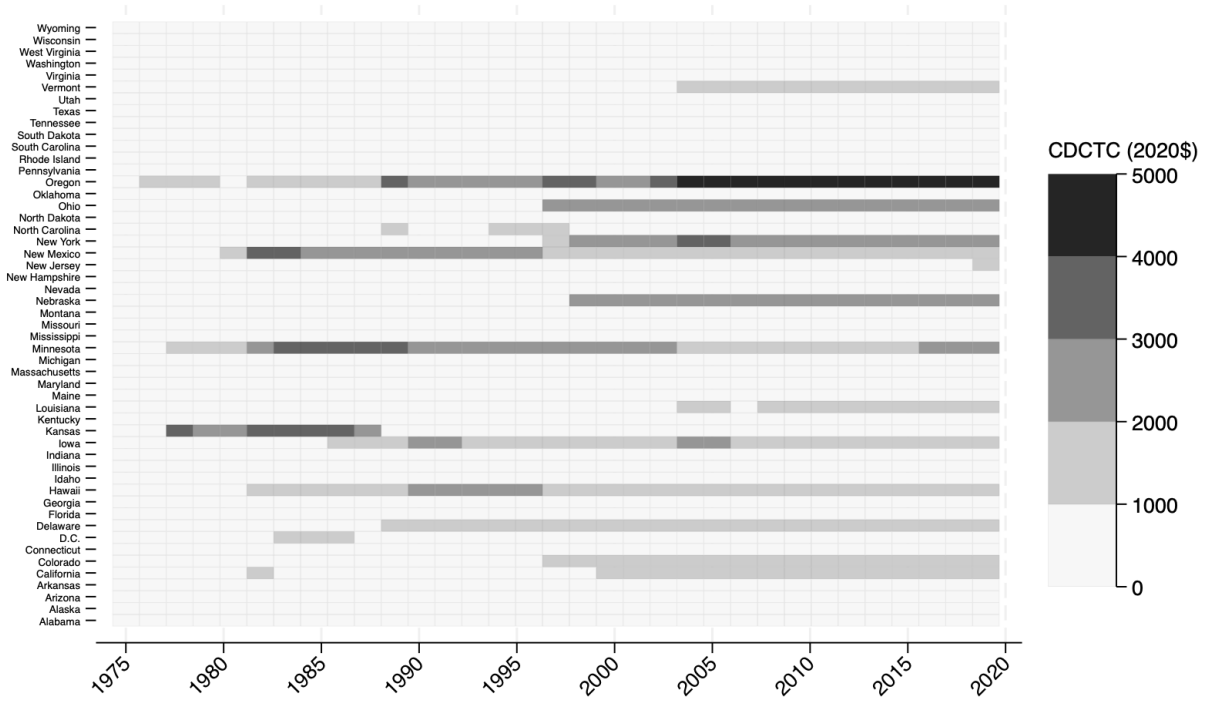


Figure 3.5: The Variation of Maximum State CDCTC Benefit from 1975-2019
Note: The following states never introduced a state CDCTC, and therefore, the state CDCTC benefit is zero: Alabama, Arizona, Connecticut, Florida, Idaho, Illinois, Indiana, Massachusetts, Michigan, Mississippi, Missouri, Montana, Nevada, New Hampshire, North Dakota, Pennsylvania, South Dakota, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin, and Wyoming.

United States. It surveys all family members of the respondents and their descendants to this day. In 1997, the PSID expanded its primary dataset to include comprehensive details about 3,563 children ranging from infancy to twelve years old, as well as their guardians, specifically for the CDS. These children were selected randomly from the PSID's pool of contributing households, with each family providing information on no more than two of their offspring. Post-1997, these children were tracked through subsequent surveys in 2002 and 2007, remaining within the CDS framework until they reached adulthood at age 18. During the CDS's subsequent iterations in 2013 and 2019, the study broadened its scope to encompass all children between the ages of 0 and 17 within the surveyed households.

PSID-CDS has two unique features that make it suitable for this study. First, the panel design provides detailed family background information at the year of birth, including the educational level of the mother, whether the mother was older than 35 when she

gave birth, gender and marital status of the household head, number of children in the family, and the family income. Moreover, PSID-CDS collects health status measures in childhood covering doctor-diagnosed chronic conditions such as asthma, diabetes, anemia, orthopedic impairment, allergies, vision problems, hearing problems, convulsion, speech impairment, emotional disturbance, hyperactivity, and other conditions, in addition to subjective measures of health such as the behavior problem index, primary caregiver (PCG) rated health status, and limitations on activities.

Following the methodology of Kling et al. (2007), we construct two physical and mental health indices with doctor-diagnosed chronic conditions, which are simple averages across standardized z-score measures of each component with converted signs. The first physical health index is constructed with doctor-diagnosed chronic conditions, including asthma, diabetes, anemia, orthopedic impairment, allergies, vision problems, hearing problems, and other conditions. The second physical health index is similar to the first one but without other conditions. The first mental health index is constructed with doctor-diagnosed chronic conditions, including convulsion, speech impairment, emotional disturbance, hyperactivity, and other conditions. The second mental health index is similar to the first but without other conditions. The classification of chronic health conditions is drawn from Van Cleave et al. (2010) and Vaughn (2023). While all the components are "bads" (i.e., indicate worse health, behavioral conditions) since the indices use converted signs of these components, an increase in these health indices indicates a better outcome (i.e., improved health or behavior).

We develop a health quality index using subjective health measures. Our approach mirrors the composite measure used by Johnson and Schoeni (2007), Halliday et al. (2021), Vaughn (2023). This measure combines the PCG-rated health of the child with reports of any limitations in typical childhood activities. It draws upon the composite measure devised by Erickson (1995), which transforms rated health and activity limitations into a continuous health measure similar to healthy life years.

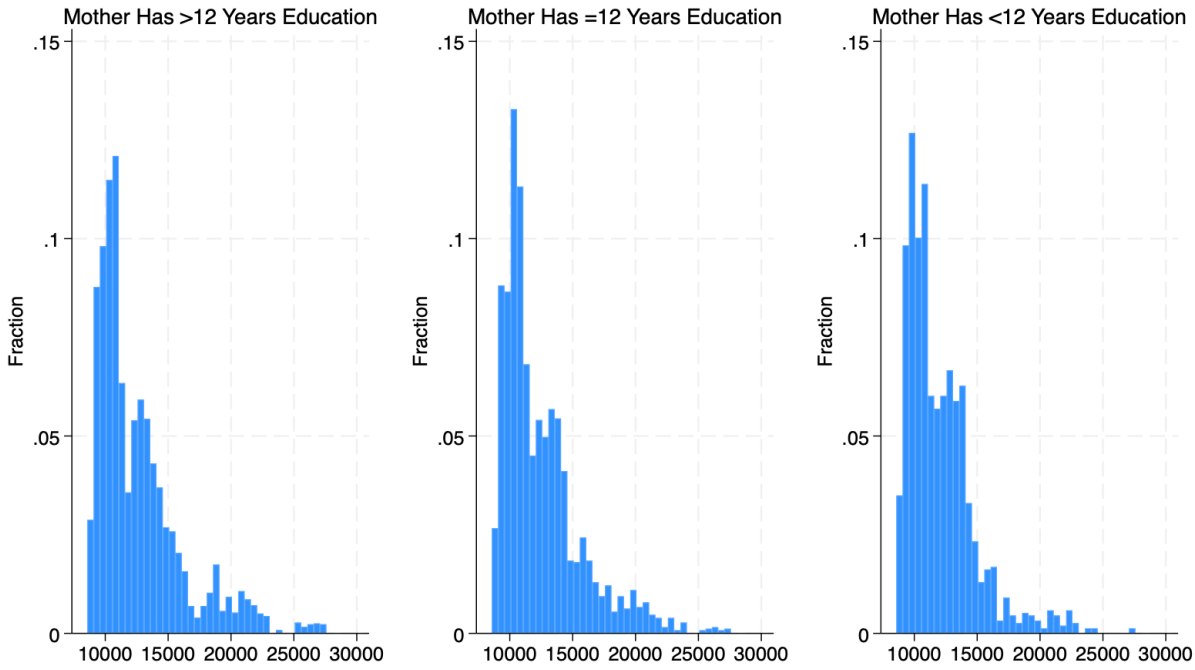


Figure 3.6: Statistic Distribution of Maximum CDCTC Exposure between Ages 0-3 in Main Sample

We also use the 30-item Behavior Problems Index (BPI) developed by James Peterson and Nicholas Zill to measure a survey of the incidence and severity of child behavior problems. The BPI was administered to PCGs of children. PCGs indicated whether the behavior or trait described in each item in the series was "often," "sometimes," or "never" true of the child. Overall scale scores and subscale scores for internalizing and externalizing behavior are included in the data.

3.5 Method

We exploit the accumulated federal and state maximum CDCTC benefit in the first four years of life for children born between 1983 and 2013 to empirically estimate a generalized difference-in-differences model with continuous treatment variables. This measure of exposure, rather than actual CDCTC eligibility based on reported family income, is our

preferred measure due to concerns of endogeneity of own CDCTC eligibility with child health outcomes. Figure 3.6 illustrates the distribution of the accumulated federal and state maximum CDCTC benefit in the first four years of life for children in our sample. The distribution is categorized by mothers' education level. This approach is similar to that of Hoynes et al. (2016a), Bastian and Michelmore (2018b), Jiang (2020), and Vaughn (2023). The model enables a comparison of the outcomes for children exposed to different CDCTC generosity over the first four years of their lives. The model takes the following form:

$$Y_{isby} = \beta_0 + \beta_1 \text{CDCTCexposure}_{(s,\text{age0-3})} + +\beta_2 X_{ib} + \beta_3 Z_{sb} + \gamma_s + \eta_b + \theta_y + \epsilon_{isb} \quad (3.1)$$

Where i denotes the individual, s the state of birth, b the year of birth, and y the interview year.

Y_{isby} measures health outcomes or intermediate outcomes mentioned in the data section for children aged 6-18. $\text{CDCTCexposure}_{(s,\text{age0-3})}$ refers to accumulated CDCTC exposure in the first four years of life.

X_{ib} represents a vector of individual-level characteristics, including gender, race, age squared at the survey year, and family background at birth year, such as the educational level of the mother, whether the mother was over 35 when she gave birth to the individual, gender of the household head, marital status of the household head, number of children in the family, and family income-Census needs standard ratio at the year of birth.

The term Z_{sb} represents a vector of state-level covariates at the birth year, including state maximum EITC benefit, state maximum AFDC/TANF benefit, economic status and policies (including the minimum wage, the maximum marginal income tax rate, and per capita GDP), and health care investment (including state expenditure for Medicaid and similar programs and the number of hospital beds). These state-level covariates also affect children's health status.

We allow for unrestricted state effects γ_s, unrestricted cohort effects at the national level

η_b, and unrestricted interview year fixed effect θ_y.

The parameter of interest is β_1, the effect of exposure to CDCTC benefit in the first four years of life, which is identified from variation within states across birth cohorts. All models are estimated using the PSID sample weights, and we cluster standard errors by the state of birth.

Threat to Identification

One challenge to identification lies in the potential presence of confounding factors at the state level that could simultaneously influence both CDCTC generosity and health outcomes. To address this concern, we investigated the relationship between CDCTC benefits and various state-level variables, as outlined in Table 3.1. In the first column of Table 3.1, we regressed CDCTC benefits against a range of state variables, including other state welfare programs (EITC, TANF/AFDC), state economic policies (minimum wage, maximum marginal income tax rate), state economic indicators (per capita GDP, tax revenue), and state medical resources (number of hospital beds, expenditure on Medicaid and similar programs). Subsequently, we introduced state fixed effect in the second column and included both state fixed effect and year fixed effect in the third column. Our findings indicate that there is no significant correlation between CDCTC benefits and other state variables when accounting for both state fixed effect and year fixed effect.

Additionally, we explored whether CDCTC influenced families' relocation in Table 3.2. In the first column of Table 3.2, we solely controlled for state fixed effect and year fixed effect. In subsequent columns, we progressively incorporated family characteristics and state covariates. However, we did not find evidence suggesting that people relocate due to CDCTC benefits.

Furthermore, we observed differential effects of CDCTC exposure across maternal education levels, as depicted in the results section. We do not think there are omitted variables that would have effects of a similar pattern across different levels of maternal

Table 3.1: Correlation between CDCTC Benefits and Other State Welfare Programs, Economic Policies, and Economic Conditions

	Maximum CDCTC Benefit		
	(1)	(2)	(3)
EITC Benefit	0.046*	-0.061**	0.036
	(0.027)	(0.030)	(0.067)
TANF/AFDC	-0.947***	-2.782***	0.189
	(0.202)	(0.294)	(0.201)
Minimum Wage	47.022**	-1.462	19.433
	(19.795)	(24.568)	(18.025)
Maximum Marginal Income Tax Rate	77.311***	32.857	8.711
	(20.084)	(35.550)	(23.766)
Per Capita GDP	6.442	1.405	4.452
	(5.857)	(7.188)	(4.157)
Tax Revenue	0.018**	0.020*	-0.012
	(0.007)	(0.011)	(0.010)
Number of Hospital Beds	-7.177***	-9.406	-6.114
	(2.080)	(10.530)	(5.515)
Expenditure on Medicaid and Similar Programs	-0.021	-0.029**	0.019
	(0.024)	(0.014)	(0.014)
State FE	N	Y	Y
Year FE	N	N	Y
Observations	2,634	2,634	2,634
R-squared	0.207	0.402	0.898

Note: Standard errors are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

education.

3.6 Results

Because child health is at least partly produced in the home, there can be heterogeneity by education regarding mothers' abilities to produce child health. Indeed, Grossman (2006, 2017) theorizes that more educated mothers are more capable of "producing" better child health with a given set of health inputs (i.e., productive efficiency), and they are also able to allocate health inputs more efficiently compared to women who are less educated (i.e., allocative efficiency). Furthermore, variation in the generosity of the CDCTC over two decades and panel data on individuals from several birth cohorts allows for estimating

Table 3.2: Correlation between CDCTC Benefits and Relocation

	Relocation		
	(1)	(2)	(3)
Maximum CDCTC Benefit	0.002	0.002	-0.003
	(0.005)	(0.005)	(0.004)
Observations	115,695	115,695	115,695
R-squared	0.021	0.035	0.035
Mean Y	0.083	0.083	0.083
State FE, Year FE	Y	Y	Y
Family Characteristics	N	Y	Y
State Covariates	N	N	Y

Note: Standard errors are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

heterogeneous treatment effects by mothers' education. Thus, we disaggregate all our analyses by mothers' education. We focus on three groups: mothers with less than a high school diploma, mothers with a high school education, and mothers with more than a high school education.[3]

Table 3.3 presents the summary statistics for outcome variables concerning child health outcomes, including physical and mental health indices, general health quality, and behavioral issues, disaggregated by the mothers' educational attainment.

The table illustrates subtle gradients in Physical Health Indices 1 and 2, with children of mothers with more than a high school education exhibiting better health scores than those with less than a high school education. Notably, the incidence of asthma, diabetes, and vision problems increases as maternal education decreases. Conversely, allergies are most prevalent among children of the most educated mothers, possibly reflecting greater diagnostic rates or reporting biases in higher socio-economic groups.

When considering Mental Health Indices, a clear trend emerges, showing a deterioration in mental health scores moving from children of mothers with more than a high school education to those with less. This pattern is also mirrored in the prevalence rates of specific conditions like epilepsy, speech impairment, and hyperactivity, with the rates generally

[3]Herbst and Tekin (2016) also conduct heterogeneity analysis by education of the mother. Their focus is on childcare subsidies to low-income families; thus, their results are not directly comparable to ours.

Table 3.3: Statistics Summary for Outcome Variables

Variable	Mother Has More than High School Education		Mother Has Just High School Education		Mother Has Less than High School Education	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Physical Health Indice and Components						
Physical Health Index 1[1]	0.005	0.406	0.012	0.383	-0.034	0.448
Physical Health Index 2[2]	0.008	0.414	0.011	0.403	-0.043	0.478
Asthma=1	0.139	0.346	0.154	0.361	0.186	0.390
Diabetes=1	0.002	0.044	0.004	0.063	0.010	0.099
Anemia=1	0.031	0.174	0.025	0.157	0.046	0.209
Orthopedic Impairment=1	0.037	0.190	0.034	0.181	0.016	0.127
Allergies=1	0.257	0.437	0.240	0.427	0.222	0.416
Vision Problem=1	0.026	0.161	0.035	0.183	0.050	0.218
Hearing Problem=1	0.023	0.151	0.016	0.125	0.035	0.185
Other Conditions=1	0.075	0.264	0.067	0.250	0.065	0.246
N	4762		2553		1546	
Mental Health Indice and Components						
Mental Health Index 1[3]	0.007	0.525	-0.002	0.544	-0.037	0.600
Mental Health Index 2[4]	0.013	0.574	-0.007	0.610	-0.053	0.673
Epileptic=1	0.013	0.113	0.020	0.141	0.029	0.168
Speech Impairment=1	0.104	0.305	0.089	0.284	0.094	0.292
Emotional Disturbance=1	0.048	0.214	0.053	0.223	0.068	0.252
Hyperactive=1	0.090	0.286	0.106	0.309	0.114	0.318
Other Conditions=1	0.075	0.264	0.067	0.250	0.065	0.246
N	4762		2553		1546	
Other Health Indice						
Health Quality[5]	0.951	0.081	0.932	0.092	0.912	0.105
N	4758		2551		1544	
Behavior Problem Index-External Problem	0.086	0.949	-0.090	1.047	-0.229	1.082
N	4603		2474		1499	
Behavior Problem Index-Internal Problem	0.053	0.952	-0.043	1.021	-0.177	1.154
N	4604		2468		1503	
Behavior Problem Index-Total[6]	0.080	0.939	-0.078	1.051	-0.223	1.125
N	4587		2457		1496	

Note: For all the health indexes (except for the components), a higher value means better health.
[1] Physical health index 1 equals the mean of the following standardized physical health conditions: asthma, diabetes, anemia, orthopedic impairment, allergies, vision problems, hearing problems, and other conditions. [2] Physical Health Index 2 is similar to Physical Health Index 1, except it excludes "Other Conditions". [3] Mental health index 1 equals the mean of the following standardized mental health conditions: epileptic, speech impairment, emotional disturbance, hyperactive, and other conditions. [4] Mental Health Index 2 is similar to Mental Health Index 1, except it excludes "Other Conditions". [5] Health quality is a measure generated with primary caregiver-rated child health and whether the primary caregiver reports that the child faces limitations in usual childhood activities. [6] The three behavior problem indexes are standardized values from PSID-CDS.

increasing as maternal education level decreases.

Health Quality ratings further confirm the observed socio-economic gradient in child health, with the highest ratings among children of mothers with more than a high school education and the lowest among those whose mothers have less than a high school education. This variable, reflecting both caregiver-reported general health and activity limitations, encapsulates the broader quality of life implications of child health status.

The Behavior Problem Indices—including external, internal, and total problem scores—reveal a stark contrast in behavioral issues across maternal education levels. Children from the least educated maternal background exhibit significantly more behavioral problems, evidenced by negative mean scores, suggesting a higher prevalence and severity of these issues in lower socio-economic groups.

Moving to Table 3.4, in terms of CDCTC exposure, the accumulated maximum exposure in the first four years, measured in 2020 USD, demonstrates a clear correlation with maternal education levels. Children of mothers with more than a high school education have a higher mean exposure ($12890.22) compared to those whose mothers have just a high school education ($12770.84) or less than a high school education ($12209.08).

Examining other variables, the data reveal more variation by maternal education. For instance, the mean family income-census needs ratio and household head's total earnings in the four years following the birth of a child illustrate economic disparities that are consistent with maternal educational attainment. Specifically, families where the mother has more than a high school education report higher mean values in both family income relative to needs (5.396) and household head's earnings ($276.708 in thousands) compared to families with lower levels of maternal education. Additionally, demographic variables such as the number of children under 18 and the prevalence of unmarried heads of households also vary by maternal education.

The clear patterns of maternal education across health outcomes, CDCTC exposure, and other critical variables shown in Table 3.3 and Table 3.4 highlight the potential influence of

Table 3.4: Statistics Summary for Control Variables

Variable	Mother Has More than High School Education		Mother Has Just High School Education		Mother Has Less than High School Education	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Accumulated Maximum CDCTC Exposure in the First 4 Years ($2020)	12890.22	3657.258	12770.84	3435.376	12209.08	2937.141
Individual and Family Characteristics						
Birth Year	1997.662	8.555	1996.287	8.281	1995.662	8.206
Male	0.516	0.500	0.493	0.500	0.494	0.500
Age	11.320	3.293	11.493	3.287	11.531	3.146
White Head	0.856	0.352	0.736	0.441	0.508	0.500
Mothers' Age >35	0.213	0.410	0.209	0.407	0.347	0.476
Female Head	0.070	0.255	0.185	0.388	0.385	0.487
Unmarried Head	0.090	0.286	0.217	0.412	0.445	0.497
Number of Children under 18 = 1	0.586	0.493	0.557	0.497	0.426	0.495
Number of Children under 18 = 2	0.263	0.441	0.258	0.438	0.291	0.454
Number of Children under 18 >2	0.150	0.358	0.185	0.388	0.283	0.451
Family Income-Census Needs Ratio	5.396	4.048	3.041	2.087	1.806	1.570
Head's Total Earning in 4 Years after the Birth of Child (1,000)	276.708	233.804	177.117	482.948	89.680	86.401
State Controls						
CDCTC Refundability	0.149	0.356	0.132	0.339	0.086	0.280
Maximum EITC benefit ($2020)	4326.704	2285.350	3995.710	2199.815	3822.619	2204.647
EITC Refundability	0.143	0.350	0.104	0.305	0.089	0.284
Maximum TANF/AFDC Benefit ($2020)	684.667	286.195	660.830	282.180	591.067	266.087
Number of Hospital Beds (1,000)	34.191	27.616	34.085	26.499	35.033	25.764
State Expenditure on Medicaid and Similar Programs ($2020) (1,000)	7691.533	8345.050	6728.834	7574.394	6752.142	7726.952
State Minimum Wage ($2020)	6.443	2.623	6.113	2.761	5.576	2.972
Maximum State Marginal Income Tax Rate	5.685	3.296	5.362	3.289	4.834	3.169
Per Capita GDP ($2020) (1000)	49.673	9.942	47.323	10.029	46.161	11.976
Paid Family Leave	0.024	0.154	0.017	0.131	0.007	0.085
N	4,762		2,553		1,546	

Note: Author's tabulation. Sample individuals born between 1983 and 2013. The CDCTC benefit is collected through the Nexis Uni database and Hein Online database. The EITC benefit is from the National Bureau of Economic Research. The AFDC/TANF benefit is from legislative history references from U.S. Government Publishing Office. The number of hospital beds and welfare vendor payment for medical are from the U.S. Census Bureau. The minimum wage is from the U.S. Department of Labor. The maximum marginal income tax rate is from the Council of State Governments. The per capita GDP is calculated with state GDP from the U.S. Bureau of Economic Analysis and state population from the U.S. Census Bureau and Fred Economic Data. The features of state paid family leave programs are from Bipartisan Policy Center.

maternal education on child health outcomes and the utilization of tax credits and other socio-economic factors. This motivates our empirical decision to analyze the effects of early childhood exposure to the CDCTC on later childhood health by maternal education.

Table 3.5 presents the main findings from our study on the impact of early childhood exposure to the CDCTC on various health outcomes and behavioral indices, employing data from the PSID-CDS spanning the years 1997, 2001, 2007, 2013, and 2019. As noted earlier, this analysis is stratified by the mothers' educational attainment, offering insights into how the effects of CDCTC exposure differ across socio-economic status.

In Panel A, focusing on children of mothers with more than a high school education, we observe statistically significant negative associations between state CDCTC exposure in the first four years (measured in $1,000, 2020 USD) and both Physical Health Indices 1 and 2, as well as Mental Health Indices 1 and 2, with coefficients ranging from -0.012 to -0.023 standard deviations, indicating an adverse effect of early CDCTC exposure on these health outcomes. This indicates that the substitution effect is larger than the income effect for this group.

Panel B reveals a different pattern for children of mothers with only a high school education. The effects of state CDCTC exposure are generally less pronounced and not statistically significant for most health and behavioral outcomes.

Panel C, representing children of mothers with less than a high school education, starkly contrasts with the other panels. Here, early CDCTC exposure exhibits positive impacts on Physical Health Indices and Mental Health Indices, with significant coefficients suggesting that CDCTC exposure may play a beneficial role in these groups; i.e., that the income effect prevails for this group.

Table 3.6 delves into the components of the Physical Health Index, offering a granular view of how early exposure to the CDCTC correlates with specific health conditions across different maternal education backgrounds.

Panel A, focusing on children whose mothers have more than a high school education,

Table 3.5: Main Results

	Physical Health Index 1	Physical Health Index 2	Mental Health Index 1	Mental Health Index 2	Health Quality	Behavior Problem Index-External Problem	Behavior Problem Index-Internal Problem	Behavior Problem Index-Total
Panel A: Children Born to Mother Has More than High School Education								
CDCTC Exposure at Ages 0-3	-0.014***	-0.015***	-0.018***	-0.021***	-0.001	-0.029	-0.032	-0.033
	(0.005)	(0.005)	(0.005)	(0.006)	(0.001)	(0.023)	(0.021)	(0.023)
Y-mean	0.005	0.008	0.008	0.013	0.951	0.086	0.053	0.080
Observations	4,762	4,762	4,762	4,762	4,758	4,603	4,604	4,587
R-squared	0.065	0.07	0.091	0.094	0.077	0.069	0.083	0.082
Panel B: Children Born to Mother Has Just High School Education								
CDCTC Exposure at Ages 0-3	-0.009	-0.004	-0.006	0.003	0.001	0.029	0.007	0.02
	(0.012)	(0.012)	(0.007)	(0.007)	(0.002)	(0.019)	(0.017)	(0.019)
Y-mean	0.012	0.012	-0.002	-0.007	0.932	-0.090	-0.043	-0.078
Observations	2,553	2,553	2,553	2,553	2,551	2,474	2,468	2,457
R-squared	0.096	0.095	0.13	0.125	0.112	0.136	0.141	0.155
Panel C: Children Born to Mother has Less than High School Education								
CDCTC Exposure at Ages 0-3	0.033**	0.039**	0.047**	0.062**	0.005	0.055	0.046	0.048
	(0.014)	(0.015)	(0.022)	(0.024)	(0.004)	(0.033)	(0.032)	(0.034)
Y-mean	-0.034	-0.043	-0.037	-0.053	0.912	-0.229	-0.177	-0.223
Observations	1,546	1,546	1,546	1,546	1,544	1,499	1,503	1,496
R-squared	0.193	0.176	0.245	0.23	0.158	0.153	0.223	0.183

Note: Each column is from a separate regression. We use PSID-CDS data from 1997, 2001,2007,2013, 2019. The sample consists of children at ages 6-18. Estimates are weighted using PSID family longitudinal weights. Outcome variables include health indexes which are the mean of standardized physical/mental health conditions, measure of health quality generated with primary caregiver rated general health condition and limit on activities, binary variable indicating if height is less than 5 percentile, and standardized behavior problem total score, external behavior problem score, internal behavior problem score. The main explanatory variable is the accumulated maximum CDCTC exposure in the first 4 years of life, and CPI-adjusted to 2020 dollars. The unit is $1,000. Baseline fixed effects include brith year fixed effect, birth state fixed effect, and survey year fixed effect. Individual demographic controls include gender, race, age and age cubic. Family background at birth includes the educational level and age of mother, gender of head, marital status of head, number of children in the family, family income-Census needs standard ratio at birth year, the total earning of household head. State covariates include the refundability of CDCTC and EITC, the maximum AFDC/TANF benefits, maximum EITC benefits, the minimum wage, the maximum income marginal tax rate, per capita GDP, the number of hospital beds, welfare vendor payment for medical, and paid family leave at the year of birth. Standard errors clustered by state of birth are in parentheses. Significance levels: * p<0.1, ** p<0.05, ***p<0.01.

reveals a statistically significant positive relationship between early CDCTC exposure and the incidence of anemia, vision problems, and hearing problems, with coefficients of 0.004 and 0.005 respectively, all indicating an increase in these conditions with increased CDCTC exposure. This suggests a potential adverse effect of financial support through CDCTC in early childhood against these specific health issues.

Panel B reports on children born to mothers with just a high school education, where the effects of CDCTC exposure are generally less statistically significant. However, a notable exception is the positive association between early CDCTC exposure and "Other Conditions" (0.010), suggesting a potential area where financial intervention may have an adverse effect on health issues not captured by the more common conditions listed.

Panel C presents outcomes for children of mothers with less than high school education, where the analysis uncovers a significant negative effect of early CDCTC exposure on hearing (-0.019). This finding indicates that CDCTC exposure can have protective effects on certain health outcomes.

Table 3.7 offers a detailed analysis of the components of the Mental Health Index, separated by maternal education level. This table provides insight into how early exposure to the CDCTC influences various facets of children's mental health, including convulsions, speech impairments, emotional disturbances, hyperactivity, and other conditions.

Panel A, which examines children born to mothers with more than a high school education, indicates a significant adverse effect of early CDCTC exposure on speech impairment, emotional disturbance, and hyperactivity, with coefficients of 0.014, 0.006, and 0.007, respectively. These findings suggest that increased CDCTC exposure in the first four years is associated with increased instances of these mental health issues among children in this group.

Panel B shifts the focus to children of mothers with just a high school education. Here, the effects of early CDCTC exposure are less uniform, with a notable positive correlation with "Other Conditions" (0.010).

Table 3.6: Childhood Physical Health Index Components

	Asthma	Diabetes	Anemia	Orthopedic Impairment	Allergies	Vision Problem	Hearing Problem	Other Conditions
Panel A: Children Born to Mother Has More than High School Education								
CDCTC Exposure	0.002	0	0.005***	0.001	0.002	0.005***	0.004**	0.002
at Ages 0-3	(0.004)	0.000	(0.001)	(0.003)	(0.005)	(0.002)	(0.002)	(0.002)
Y-mean	0.139	0.002	0.031	0.037	0.257	0.027	0.023	0.075
Observations	4,762	4,762	4,762	4,762	4,762	4,762	4,762	4,762
R-squared	0.075	0.033	0.046	0.043	0.097	0.056	0.037	0.06
Panel B: Children Born to Mother Has Just High School Education								
CDCTC Exposure	-0.001	-0.002	0.002	-0.004	0.013	0.004	0.003	0.011**
at Ages 0-3	(0.009)	(0.002)	(0.002)	(0.004)	(0.008)	(0.004)	(0.002)	(0.005)
Y-mean	0.154	0.004	0.025	0.034	0.240	0.035	0.016	0.067
Observations	2,553	2,553	2,553	2,553	2,553	2,553	2,553	2,553
R-squared	0.091	0.108	0.074	0.1	0.14	0.071	0.061	0.096
Panel C: Children Born to Mother Has Less than High School Education								
CDCTC Exposure	-0.023**	-0.004	0.002	0.003	-0.022*	-0.001	-0.019***	0.003
at Ages 0-3	(0.011)	(0.003)	(0.006)	(0.003)	(0.013)	(0.010)	(0.006)	(0.009)
Y-mean	0.186	0.010	0.046	0.016	0.222	0.050	0.036	0.065
Observations	1,546	1,546	1,546	1,546	1,546	1,546	1,546	1,546
R-squared	0.233	0.086	0.126	0.319	0.256	0.169	0.21	0.183

Note: Each coefficient is from a separate regression. The outcome variables are binary, equaling one if the individual has ever had the corresponding health condition. The main explanatory variable is the accumulated maximum CDCTC exposure in the first 4 years of life, and CPI-adjusted to 2020 dollars. The unit is $1,000. These models contain the full set of covariates as the main model.
Significance levels: * p<0.1, ** p<0.05, ***p<0.01.

Panel C addresses children whose mothers have less than a high school education. This panel reveals a significant negative effect of early CDCTC exposure on the occurrence of emotional disturbance (-0.017), which shows the potential protective impact of financial support on the mental health of children from the most socio-economically disadvantaged backgrounds.

This exploration into the physical and mental health components affected by CDCTC exposure underscores the complexity of its impacts. The findings reveal both protective and potentially adverse associations, dependent on the specific physical and mental health outcome and the family's socio-economic background (indicated by maternal education).

Tables 3.8, 3.9, and 3.10 present the results of a series of robustness checks to the main findings reported in Table 3.5, on the impact of the CDCTC exposure on various health and behavior outcomes in children.

Table 3.8 focuses on children of higher maternal education. First, compared to the baseline model (Panel A, which is the same as Table 3.5, Panel A), the extended exposure models (Panels B and C) show a consistent negative impact of CDCTC exposure during the first 5 and 6 (instead of 4) years on Physical and Mental Health Indices, with slight variations in magnitude, indicating the robustness of the main findings (Table 3.5, Panel A). Panels D and E explore the impact using different surveys of the CDS, which also largely confirm the main results, though with some variations in effect sizes, suggesting contextual or temporal factors may influence the outcomes. Controlling for family fixed effect (Panel F) or using an alternative one-individual-one-observation sample (Panel G) introduces additional controls or data restrictions, further demonstrating the persistence of the negative impacts on children's health and behavior, albeit with some changes in significance levels and effect sizes, emphasizing the robustness of the analysis against various methodological adjustments.

Tables 3.9 and 3.10 focus on children with mothers who are high school graduates and those with mothers who did not finish high school, respectively. The results are very

Table 3.7: Childhood Mental Health Index Components

	Convulsion	Speech Impairment	Emotion Disturbance	Hyperactive	Other Conditions
Panel A: Children Born to Mother Has More than High School Education					
CDCTC Exposure at Ages 0-3	-0.001	0.011***	0.006*	0.009*	0.002
	(0.001)	(0.004)	(0.003)	(0.005)	(0.002)
Y-mean	0.013	0.104	0.048	0.090	0.075
Observations	4,762	4,762	4,762	4,762	4,762
R-squared	0.042	0.074	0.065	0.081	0.06
Panel B: Children Born to Mother Has Just High School Education					
CDCTC Exposure at Ages 0-3	0.002	0.004	-0.004	-0.009	0.011**
	(0.002)	(0.004)	(0.003)	(0.005)	(0.005)
Y-mean	0.020	0.089	0.053	0.106	0.067
Observations	2,553	2,553	2,553	2,553	2,553
R-squared	0.093	0.102	0.099	0.156	0.096
Panel C: Children Born to Mother Has Less than High School Education					
CDCTC Exposure at Ages 0-3	-0.001	-0.016	-0.019***	-0.030*	0.003
	(0.006)	(0.011)	(0.006)	(0.018)	(0.009)
Y-mean	0.029	0.094	0.068	0.114	0.065
Observations	1,546	1,546	1,546	1,546	1,546
R-squared	0.239	0.231	0.19	0.176	0.183

Note: Each coefficient is from a separate regression. The outcome variables are binary, equaling one if the individual has ever had the corresponding health condition. The main explanatory variable is the accumulated maximum CDCTC exposure in the first 4 years of life, and CPI-adjusted to 2020 dollars. The unit is $1,000. These models contain the full set of covariates as the main model.
Significance levels: * p<0.1, ** p<0.05, ***p<0.01.

Table 3.8: Robustness Checks – For Children Born to Mother Has More than High School Education

	Physical Health Index 1	Physical Health Index 2	Mental Health Index 1	Mental Health Index 2	Health Quality	Behavior Problem Index-External Problem	Behavior Problem Index-Internal Problem	Behavior Problem Index-Total
Panel A: Baseline Model, Main X = Accumulated Maximum CDCTC Benefit in the First 4 Years								
CDCTC Exposure	-0.014***	-0.015***	-0.018***	-0.021***	-0.001	-0.029	-0.032	-0.033
	(0.005)	(0.005)	(0.005)	(0.006)	(0.001)	(0.023)	(0.021)	(0.023)
Y-mean	0.005	0.008	0.008	0.013	0.951	0.086	0.053	0.080
Observations	4,762	4,762	4,762	4,762	4,758	4,603	4,604	4,587
R-squared	0.065	0.07	0.091	0.094	0.077	0.069	0.083	0.082
Panel B: Main X = Accumulated Maximum CDCTC Benefit in the First 5 Years								
CDCTC Exposure	-0.011***	-0.012***	-0.015***	-0.018***	-0.001	-0.024	-0.026	-0.026
	(0.004)	(0.004)	(0.005)	(0.006)	(0.001)	(0.019)	(0.016)	(0.018)
Y-mean	0.005	0.008	0.008	0.013	0.951	0.086	0.053	0.080
Observations	4,762	4,762	4,762	4,762	4,758	4,603	4,604	4,587
R-squared	0.065	0.069	0.091	0.095	0.077	0.069	0.083	0.082
Panel C: Main X = Accumulated Maximum CDCTC Benefit in the First 6 Years								
CDCTC Exposure	-0.009***	-0.010***	-0.013***	-0.015***	-0.001	-0.021	-0.023	-0.023
	(0.003)	(0.004)	(0.005)	(0.005)	(0.001)	(0.016)	(0.014)	(0.016)
Y-mean	0.005	0.008	0.008	0.013	0.951	0.086	0.053	0.080
Observations	4,762	4,762	4,762	4,762	4,758	4,603	4,604	4,587
R-squared	0.065	0.069	0.091	0.094	0.077	0.069	0.083	0.082
Panel D: Main X = Accumulated Maximum CDCTC Benefit at Ages 1-4								
CDCTC Exposure	-0.013**	-0.014**	-0.016**	-0.020***	-0.001	-0.032	-0.033*	-0.034
	(0.005)	(0.005)	(0.006)	(0.007)	(0.001)	(0.023)	(0.019)	(0.022)
Y-mean	0.005	0.008	0.008	0.013	0.951	0.086	0.053	0.080
Observations	4,762	4,762	4,762	4,762	4,758	4,603	4,604	4,587
R-squared	0.065	0.069	0.09	0.094	0.077	0.07	0.083	0.082
Panel E: Control for Family Fixed Effect								
CDCTC Exposure	-0.037	-0.037	-0.036*	-0.037*	-0.002	-0.049***	-0.034**	-0.046***
	(0.026)	(0.026)	(0.020)	(0.021)	(0.003)	(0.012)	(0.013)	(0.012)
Y-mean	0.00691	0.00981	0.00965	0.0154	0.951	0.09	0.054	0.0826
Observations	4,588	4,588	4,588	4,588	4,584	4,425	4,428	4,410
R-squared	0.358	0.365	0.4	0.407	0.405	0.407	0.388	0.421
Panel F: One-Individual-One-Observation								
CDCTC Exposure	-0.070*	-0.068**	-0.050**	-0.049**	0	-0.022	-0.027	-0.027
	(0.038)	(0.033)	(0.022)	(0.020)	(0.001)	(0.023)	(0.019)	(0.022)
Y-mean	0.0351	0.0526	0.0186	0.0361	0.949	0.0815	0.0519	0.0765
Observations	3,192	3,192	3,192	3,192	3,191	3,093	3,087	3,081
R-squared	0.068	0.074	0.09	0.091	0.089	0.078	0.088	0.09

Note: Standard errors are in parentheses.
Significance levels: * p<0.1, ** p<0.05, ***p<0.01.

Table 3.9: Robustness Checks – For Children Born to Mother Has Just High School Education

	Physical Health Index 1	Physical Health Index 2	Mental Health Index 1	Mental Health Index 2	Health Quality	Behavior Problem Index-External Problem	Behavior Problem Index-Internal Problem	Behavior Problem Index-Total
Panel A: Baseline Model, Main X = Accumulated Maximum CDCTC Benefit in the First 4 Years								
CDCTC Exposure	-0.009	-0.004	-0.006	0.003	0.001	0.029	0.007	0.02
	(0.012)	(0.012)	(0.007)	(0.007)	(0.002)	(0.019)	(0.017)	(0.019)
Y-mean	0.012	0.012	-0.002	-0.007	0.932	-0.090	-0.043	-0.078
Observations	2,553	2,553	2,553	2,553	2,551	2,474	2,468	2,457
R-squared	0.096	0.095	0.13	0.125	0.112	0.136	0.141	0.155
Panel B: Main X = Accumulated Maximum CDCTC Benefit in the First 5 Years								
CDCTC Exposure	-0.009	-0.006	-0.004	0.004	0.001	0.029*	0.01	0.021
	(0.009)	(0.010)	(0.006)	(0.006)	(0.001)	(0.017)	(0.014)	(0.016)
Y-mean	0.012	0.012	-0.002	-0.007	0.932	-0.090	-0.043	-0.078
Observations	2,553	2,553	2,553	2,553	2,551	2,474	2,468	2,457
R-squared	0.096	0.095	0.13	0.125	0.112	0.137	0.141	0.155
Panel C: Main X = Accumulated Maximum CDCTC Benefit in the First 6 Years								
CDCTC Exposure	-0.009	-0.006	-0.003	0.004	0	0.028*	0.011	0.022
	(0.008)	(0.008)	(0.005)	(0.005)	(0.001)	(0.016)	(0.012)	(0.015)
Y-mean	0.012	0.012	-0.002	-0.007	0.932	-0.090	-0.043	-0.078
Observations	2,553	2,553	2,553	2,553	2,551	2,474	2,468	2,457
R-squared	0.096	0.095	0.13	0.125	0.112	0.137	0.141	0.155
Panel D: Main X = Accumulated Maximum CDCTC Benefit at Ages 1-4								
CDCTC Exposure	-0.012	-0.007	-0.004	0.007	0.001	0.042*	0.012	0.03
	(0.012)	(0.012)	(0.008)	(0.008)	(0.001)	(0.022)	(0.019)	(0.021)
Y-mean	0.012	0.012	-0.002	-0.007	0.932	-0.090	-0.043	-0.078
Observations	2,553	2,553	2,553	2,553	2,551	2,474	2,468	2,457
R-squared	0.096	0.095	0.13	0.125	0.112	0.137	0.141	0.155
Panel E: Control for Family Fixed Effect								
CDCTC Exposure	-0.019	-0.007	-0.036*	-0.019	0	0.014	-0.043	-0.016
	(0.015)	(0.017)	(0.021)	(0.026)	(0.003)	(0.025)	(0.043)	(0.032)
Y-mean	0.0181	0.0181	-0.00382	-0.00928	0.932	-0.0857	-0.0385	-0.0737
Observations	2,426	2,426	2,426	2,426	2,423	2,350	2,340	2,329
R-squared	0.436	0.433	0.437	0.417	0.408	0.489	0.488	0.511
Panel F: One-Individual-One-Observation								
	(0.122)	(0.115)	(0.039)	(0.029)	(0.002)	(0.021)	(0.023)	(0.022)
Y-mean	0.0667	0.0435	-0.0132	-0.0364	0.932	-0.0725	-0.029	-0.0614
Observations	1,703	1,703	1,703	1,703	1,702	1,649	1,646	1,640
R-squared	0.108	0.106	0.151	0.147	0.111	0.136	0.147	0.156

Note: Standard errors are in parentheses.
Significance levels: * p<0.1, ** p<0.05, ***p<0.01.

Table 3.10: Robustness Checks – For Children Born to Mother Has Less than High School Education

	Physical Health Index 1	Physical Health Index 2	Mental Health Index 1	Mental Health Index 2	Health Quality	Behavior Problem Index-External Problem	Behavior Problem Index-Internal Problem	Behavior Problem Index-Total
Panel A: Baseline Model, Main X = Accumulated Maximum CDCTC Benefit in the First 4 Years								
CDCTC Exposure	0.033**	0.039**	0.047**	0.062**	0.005	0.055	0.046	0.048
	(0.014)	(0.015)	(0.022)	(0.024)	(0.004)	(0.033)	(0.032)	(0.034)
Y-mean	-0.034	-0.043	-0.037	-0.053	0.912	-0.229	-0.177	-0.223
Observations	1,546	1,546	1,546	1,546	1,544	1,499	1,503	1,496
R-squared	0.193	0.176	0.245	0.23	0.158	0.153	0.223	0.183
Panel B: Main X = Accumulated Maximum CDCTC Benefit in the First 5 Years								
CDCTC Exposure	0.027**	0.031**	0.038*	0.050**	0.005	0.034	0.029	0.028
	(0.011)	(0.012)	(0.019)	(0.021)	(0.003)	(0.028)	(0.027)	(0.029)
Y-mean	-0.034	-0.043	-0.037	-0.053	0.912	-0.229	-0.177	-0.223
Observations	1,546	1,546	1,546	1,546	1,544	1,499	1,503	1,496
R-squared	0.192	0.175	0.245	0.23	0.158	0.152	0.222	0.182
Panel C: Main X = Accumulated Maximum CDCTC Benefit in the First 6 Years								
CDCTC Exposure	0.022**	0.026**	0.032*	0.041**	0.004	0.025	0.024	0.021
	(0.010)	(0.010)	(0.017)	(0.019)	(0.003)	(0.025)	(0.025)	(0.026)
Y-mean	-0.034	-0.043	-0.037	-0.053	0.912	-0.229	-0.177	-0.223
Observations	1,546	1,546	1,546	1,546	1,544	1,499	1,503	1,496
R-squared	0.192	0.175	0.244	0.229	0.158	0.152	0.222	0.182
Panel D: Main X = Accumulated Maximum CDCTC Benefit at Ages 1-4								
CDCTC Exposure	0.032**	0.038**	0.046*	0.059*	0.005	0.026	0.025	0.02
	(0.014)	(0.014)	(0.026)	(0.029)	(0.005)	(0.034)	(0.036)	(0.037)
Y-mean	-0.034	-0.043	-0.037	-0.053	0.912	-0.229	-0.177	-0.223
Observations	1,546	1,546	1,546	1,546	1,544	1,499	1,503	1,496
R-squared	0.192	0.175	0.244	0.229	0.158	0.152	0.222	0.181
Panel E: Control for Family Fixed Effect								
CDCTC Exposure	0.051*	0.061*	0.062**	0.081***	0.012*	0.054	0.027	0.06
	(0.027)	(0.031)	(0.024)	(0.024)	(0.007)	(0.099)	(0.111)	(0.112)
Y-mean	-0.0339	-0.0426	-0.0368	-0.0527	0.912	-0.229	-0.177	-0.223
Observations	1,546	1,546	1,546	1,546	1,544	1,499	1,503	1,496
R-squared	0.448	0.433	0.514	0.52	0.483	0.492	0.544	0.519
Panel F: One-Individual-One-Observation								
CDCTC Exposure	0.265**	0.297**	0.226**	0.258***	0.007*	0.055	0.062	0.056
	(0.102)	(0.117)	(0.108)	(0.089)	(0.004)	(0.038)	(0.040)	(0.041)
Y-mean	-0.219	-0.218	-0.174	-0.173	0.912	-0.242	-0.19	-0.234
Observations	1,079	1,079	1,079	1,079	1,078	1,041	1,043	1,039
R-squared	0.217	0.208	0.26	0.239	0.216	0.191	0.239	0.21

Note: Standard errors are in parentheses.
Significance levels: * p<0.1, ** p<0.05, ***p<0.01.

consistent with those shown in Panels B and C of Table 3.5, similar to what we have just observed from Table 3.8.

Across these three tables, the robustness checks affirm the general trends observed in the main results (Table 3.5) but also show the complexities underlying the relationship between CDCTC exposure and child health and behavior outcomes. The variations observed across different maternal education levels, model specifications, and data structure underscore the importance of considering socio-economic factors, policy design, and implementation features when evaluating the impacts of tax credit policies on child welfare.

3.7 Conclusion and Discussion

Our investigation into early childhood exposure to CDCTC on later childhood health and behavioral outcomes yields important findings that contribute significantly to both the economic literature and policy discussions. This study shows the complex interplay between childcare subsidies and child health and behavioral outcomes by leveraging the variation in CDCTC benefits across states and years and employing longitudinal data from the Child Development Supplement of the Panel Survey of Income Dynamics.

We find that the CDCTC's impact varies markedly across different maternal education levels. For children of mothers with more than a high school education, early exposure to CDCTC is correlated with adverse physical and mental health outcomes. In contrast, children of mothers with a high school education show no significant difference in health outcomes attributable to CDCTC exposure. Remarkably, children of mothers with less than a high school education benefit from CDCTC exposure, exhibiting positive physical and mental health effects. These differential impacts underscore the importance of considering socio-economic and educational contexts in assessing the effectiveness of childcare subsidies. Several robustness checks, including variations in the period of CDCTC benefit accumulation and analyses using different data structures, reinforce the reliability of our

findings.

This study makes critical contributions by being among the first to systematically examine the long-term health and behavioral effects of early exposure to the CDCTC. Our findings add depth to the existing literature that has primarily focused on the labor supply effects of the CDCTC, expanding the discourse to consider the policy's broader implications for child welfare.

Our results have important policy implications, especially in light of ongoing debates about the design and scope of childcare subsidies in the U.S. The differential health impacts we identify suggest that the CDCTC, while beneficial in facilitating labor market participation, may have unintended consequences for child health that vary across socio-economic groups. This highlights the need for policy designs that are sensitive to the diverse needs of families, potentially including provisions that ensure access to high-quality childcare or direct health interventions for children at risk of adverse outcomes. These results also contribute valuable insights to the ongoing discourse on tax credits as mechanisms for improving child health and well-being, with implications for policymakers and researchers alike in the quest to optimize the design and targeting of social safety net programs.

Moreover, the benefits observed among the children with the least educated mothers point to the potential of childcare subsidies in supporting vulnerable populations. Enhancing the accessibility and responsiveness of such programs could amplify these positive effects, suggesting a valuable area for policy innovation and expansion.

Future research should delve deeper into the mechanisms driving the observed health impacts, exploring how variations in childcare quality, parental stress, and child development practices contribute to these outcomes. Additionally, studies could examine the longitudinal effects of CDCTC exposure into adulthood, assessing its implications for educational attainment, employment outcomes, and long-term health.

In conclusion, our study highlights the complex and multifaceted effects of the CDCTC on child health outcomes, underscoring the need for comprehensive policy evaluations

that consider a range of child welfare dimensions. As the demand for childcare subsidies continues to grow, our findings offer valuable insights for policymakers aiming to balance support for working parents with the health and development needs of children. By carefully navigating these considerations, it is possible to design childcare subsidy programs that facilitate parental employment and contribute positively to the next generation's health and well-being.

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