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Essays on Sleep, Labor, and Child Development

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ABSTRACT OF THE DISSERTATION

Essays on Sleep, Labor, and Child Development

by

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Dr. Joseph Cummins, Chairperson

This dissertation investigates the relationships between economic conditions, employment, sleep patterns, health outcomes, and social factors. The research is organized into three chapters, each addressing a unique aspect of these dynamics.

Chapter 1 examines the impact of sleep on fatal vehicle crashes by leveraging variations in sunset times across different locations and seasons. Using sunset time as an instrumental variable, the study finds that a one-hour increase in monthly sleep leads to about a 2.4% reduction in fatalities. These findings highlight the potential benefits of aligning social schedules with natural sleep patterns to reduce accidents.

Chapter 2 explores how employment rates affect sleep patterns. The analysis reveals that weekday sleep is countercyclical, while weekend sleep is procyclical. Employed individuals compensate for reduced weekday sleep by sleeping more on weekends. The study also shows that industries with higher telework prevalence experience smaller decreases in sleep duration as employment rises. Demographic factors such as minority status, lower education levels, and single parenthood exacerbate sleep reductions during high employment periods.

Chapter 3 investigates caste differences in child height in India. The study documents differential growth patterns across caste groups, showing that lower caste children are born shorter and grow less quickly than their higher-caste counterparts. These differences are largely explained by observable covariates, particularly maternal characteristics and household wealth. The research reveals that the influence of these variables changes as children age, with health endowment related variables explaining birth length gaps and health investments becoming more significant over time. Lower caste children face persistent endowment disparities from birth and post-birth investment differentials, which together exacerbate height gaps as they develop.

Collectively, these chapters contribute to a deeper understanding of how economic and social factors influence sleep, health, and development, emphasizing the importance of policies that support adequate sleep and address social inequalities to improve well-being and productivity.

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Chapter 1

Sleep and Fatal Vehicle Crashes: Evidence from Sunset Time in the United States

1.1 Abstract

Adequate sleep is critical for overall healthy functioning. Insufficient sleep has been linked to a decline in attention and cognitive function, which poses a potential risk for vehicle crashes. This paper aims to study the impact of sleep on fatal vehicle crashes. For the short-term analysis, I explored the variation in sunset times throughout the year in a specific location. By using sunset time as an instrument, I found that a one-hour delay in sunset leads to a decrease of approximately 12 minutes in weekly sleep duration. Additionally, a one-hour increase in monthly sleep leads to about a 2.4% reduction in

fatalities. For the long-term analysis, I employed two different approaches. First, I utilized the geographical variation in sunset time across counties within a time zone. However, the results from this approach were not statistically significant. Second, I applied spatial regression discontinuity, focusing on the timing of sunset at a time-zone boundary. I found that there is no consistent and statistically significant effect of the later sunset side on the fatalities. This paper can help in creating a better policy solution regarding DST and clock changes, as well as designing social schedules that promote healthy sleep patterns, which are crucial for both health and productivity.

1.2 Introduction

Sleep is crucial for both human health and productivity, but its importance remains largely understudied in the fields of health and labor economics. Insufficient sleep is associated with fatigue-related accidents and injuries (Dinges, 1995; Lockley et al., 2007; Barnes and Wagner, 2009), attention, cognitive ability, coordination, motor skills, and processing speed (Dinges and Powell, 1985; Drummond et al., 2005; Banks and Dinges, 2007; Lim and Dinges, 2010), as well as productivity and psychological well-being (Bessone et al., 2021). However, identifying variations in sleep patterns that are both explainable and not strongly correlated with significant lifestyle choices poses a challenge. Measuring sleep outcomes is further complicated by the frequent delay, cumulative nature, and the challenge of quantification in large datasets. Therefore, by utilizing plausibly exogenous variations in sleep patterns, I aim to assess the potential impact of sleep-related cognitive outcomes on an immediate and measurable outcome: fatal vehicle crashes.

The timing of sunset and sunrise changes throughout the year in a specific location, as well as across different locations within a time zone. However, despite this natural variation, our work schedules and school start times often remain inflexible. These rigid school and work hours force individuals to wake up at the same early hour, preventing them from adjusting for this time difference by sleeping in later. This forced synchronization can negatively impact our circadian rhythms, ultimately affecting the duration and quality of our sleep. Consequently, this phenomenon produces both seasonal or short-term effects within a given year and long-term geographical effects across locations within a time zone.

I aim to address the following question: What are the short-run and long-run effects of sleep duration on fatal vehicle crashes in the United States? To answer this question, I utilize three different strategies to isolate the various factors that contribute to differences in sleep patterns caused by astronomical and time-keeping sources. These strategies consist of two instrumental variable (IV) approaches and a spatial regression discontinuity design (RDD).

For the instrumental variable (IV) approach, I exploit two different sources of identifying variation in sleep duration. Variation in sunset time throughout the year in one location isolates a short-term, seasonal variation in sleep duration, while geographic variation in sunset time across counties within the same time zone isolates long-term sleep differences across different areas. In the short term, there is variation in sunset time within a county throughout the year. For example, a later sunset in the summer could lead to a shorter sleep duration. In the long term, there are differences in sunset time among various counties in a time zone. For instance, the sunset is later for locations further west than for locations further east, and people in the western part of the time zone would sleep less.

The regression discontinuity design (RDD) strategy exploits the sharp discontinuity in sunset time across time zone borders. There is a distinct discontinuity in sunset time around the border, with sunset occurring approximately one hour later for counties situated on the right side of the time zone boundary compared to those on the left. For both strategies, I use sleep data from the American Time Use Survey (ATUS) and vehicle fatality data from the Fatality Analysis Reporting System (FARS).

Both the IV and RDD yield interesting first-stage results. The delay in sunset time can potentially disrupt the production of melatonin, consequently pushing sleep schedules to a later time. Using the seasonal, short-run IV method, I discovered that a one-hour delay in sunset results in a decrease of approximately 12 minutes in weekly sleep duration. According to related research, a one-hour delay in sunset time within a particular location is associated with a reduction in nighttime sleep by approximately 20 minutes per week (Gibson and Shrader, 2018). The results from the RDD analysis indicate that a one-hour delay in sunset leads to an average decrease of around 10 minutes in sleep duration. This finding aligns with the study conducted by Giuntella and Mazzonna (2019), which reported a decrease of 19 minutes in sleep duration due to a delayed sunset.

Previous research conducted by Giuntella and Mazzonna (2019) focused on the years 2003 to 2013 and found that employed individuals tend to sleep less when living on the side of the time zone border with later sunsets. When I replicate this analysis using the same dataset and time frame, my results closely mirror theirs. However, an interesting twist arises when I expand the analysis to include data from 2014 to 2019. In this later period, I observe a contrasting trend where employed individuals actually sleep more if they reside on the side of the time zone border with later sunsets.

Using the seasonal, short-term IV approach, I found that a one-hour increase in monthly sleep leads to a decrease of about 2.4% in fatalities in the short run. Related research has shown that the transition into Daylight Saving Time (DST) during the spring season leads to a significant 5.6% increase in fatal crashes, and this effect remains consistent for a period of six days following the transition (Smith, 2016). Based on the RDD methodology, my research findings suggest that the impact of residing on the late sunset side has no statistically significant or consistent effects on fatal crashes among employed individuals. One potential explanation for this phenomenon could be that people gradually adapt to the extended daylight hours and eventually adjust their sleeping patterns, thereby negating any significant influence on the occurrence of fatalities.

This paper contributes to three strands of literature in economics. First, it contributes to the lab studies of sleep in medical research by using observational data to study the causal impact of sleep on fatalities, providing understanding in real-world situations. There is a plethora of research on lab studies in sleep, which shows that sleep deprivation has a negative impact on attention, cognitive ability, coordination, motor skills, and processing speed (Dinges and Powell, 1985; Drummond et al., 2005; Banks and Dinges, 2007; Lim and Dinges, 2010). Second, this paper contributes to the recent literature focusing on the impact of sleep on productivity and health by examining both the short-run and long-run effects directly from sleep data on fatalities using the IV and RDD approaches. Previous research has found associations between sleep and various outcomes, including fatal vehicle crashes (Smith, 2016), wages (Gibson and Shrader, 2018), productivity and psychological well-being (Bessone et al., 2021), functioning of financial markets (Kamstra et al., 2000), hospital admissions (Jin and Ziebarth, 2020), cognitive skills and depression symptoms (Giuntella et al., 2017), and health outcomes (Giuntella and Mazzonna, 2019). Third, this paper contributes to the research that estimates the effects of school start times on academic achievement (Dills and Hernandez-Julian, 2008; Carrell et al., 2011; Edwards, 2012; Heissel et al., 2017; Avery et al., 2019) by providing additional causal evidence to assist policy makers in making decisions regarding school start times.

The rest of this paper proceeds as follows. Section 2 reviews the literature encompassing sleep studies in the medical fields and empirical evidence of sleep in Economics. Section 3 describes the data used in this paper. Section 4 illustrates the identification strategy and the empirical methods. Section 5 reports the main results, and Section 6 discusses the robustness checks. Section 7 concludes and discusses paths for future research.The following sections outline the structure of this paper. Section 2 provides a comprehensive review of the existing literature on sleep studies in both medical and economic fields. Additionally, it presents empirical evidence related to sleep in Economics. Section 3 provides a detailed description of the data utilized in this study. In Section 4, the identification strategy and empirical methods employed are explained. The main findings are reported in Section 5, while Section 6 discusses the robustness checks conducted. Finally, Section 7 concludes the paper and suggests potential avenues for future research.

1.3 Literature Review

1.3.1 Lab Studies of Sleep in Medical Research

There exists a plethora of research on lab studies in sleep, which shows that sleep deprivation has a negative impact on attention, memory, and mood. For example, Banks and Dinges (2007) reviewed recent experiments on chronic sleep restriction and found that restricting sleep can result in attention lapses, slowed working memory, reduced processing speed, depression, and preservative thinking. They also suggest that long-term sleep deprivation leads to unhealthy physiological results.

Besides chronic sleep restriction, Lim and Dinges (2010) reviewed studies on the impact of short-term sleep deprivation on cognition. They found that simple attention is strongly affected by short-term sleep deficit. The authors believe that sleep deprivation can pose significant safety risks, and implementing countermeasures targeting simple attention would be the most effective way to prevent accidents in industries.

One example of measuring simple attention is the laboratory study of the Psychomotor Vigilance Test (PVT) (Dinges and Powell, 1985). The PVT was initially invented in 1985 to measure sustained attention and has since become the most widely used test in studies of sleep and circadian rhythm research. Numerous studies have demonstrated that the PVT is a highly sensitive indicator of sleep deprivation.

A laboratory study conducted by Drummond et al. (2005) investigated the neural basis of PVT and found that optimal performance is dependent on the brain region responsible for these functions after a normal night of sleep. On the other hand, poor performance following sleep deprivation activates the brain's "default mode." This finding supports previous studies suggesting that sleep has an impact on attention.

This paper contributes to this literature by utilizing observational data to examine the causal impact of sleep on fatalities, providing insights into real-world situations.

1.3.2 Empirical Evidence on Sleep in Economics

Despite the extensive body of medical research highlighting the hazards of sleep deprivation, economists have only recently begun to explore the economic implications of insufficient sleep through empirical analysis. This paper aims to contribute to the emerging field of research on the consequences of sleep deprivation within the economic literature.

Productivity and Health

First, this paper is linked to the literature of estimating the impact of sleep on productivity and health (Kamstra et al., 2000). In a recent study, Smith (2016) uses regression discontinuity (RD) and day-of-year fixed effects (FE) model to study the short-run effects of Daylight Saving Time (DST) on fatal crashes and provides evidence of 5.6% increase in fatalities for six days after the spring transition of DST. He decomposes the aggregate effect of DST into an ambient light and sleep mechanism and finds that sleep deprivation is the channel that results in more fatal crashes while changing ambient light merely reallocates fatalities within a day. In addition, he discovers that losing an hour of sleep raises the risk of being in a drowsiness-related fatal crash by 46%.

I differentiate from Smith (2016), as rather than studying the short-run effects of DST on national fatalities using RD and FE models and analyzing sleep mechanism indirectly without using any sleep data or measurements, I examine both the short-run and long-run effect directly from sleep data on county-level fatal crashes using the IV and RDD approach.

The results would help us to form a better policy solution such as whether to keep

DST and end clock changes. The benefits of the DST include decreased crime (Doleac and Sanders, 2015) and cost of the DST would be related to sleep loss with transitions. A better solution would keep the benefits of DST while diminishing the costs of the transition. For example, on March 15, 2022, the U.S. Senate passed the Sunshine Protection Act of 2021, which would keep a permanent DST and end clock changes, but this Act has not made it to the U.S. House for discussion. In addition, the results could contribute to constructing social schedules such as work schedules and school start times in ways that promote sleeping, which is related to health and productivity.

This paper is also linked to Gibson and Shrader (2018), who use IV specification to study the impact of sunset variation within a location over time and sunset variation within a time-zone on wages and find that a one hour increase in weekly sleep results in 1.1% increases in wages in the short run and 5% in the long run. I employ a similar econometric approach to examine the effects of monthly sleep on fatal vehicle crashes at the county level, both in the short run and long run. Additionally, I incorporate the RDD method to estimate the long run effects.

A recent field experiment by Bessone et al. (2021) shows that a randomized three-week treatment to improve sleep in Chennai, India, increases sleep time by 27 minutes at night, which has no significant impact on cognition, productivity, or well-being. However, short naps in the afternoon help to improve the productivity, psychological well-being, and cognition. Instead of using field experiment, I am using non-experimental data to examine the impact of sleep.

Furthermore, Jin and Ziebarth (2020) study the hospital admissions impact of DST. Using an event study method, they find that the hospitalization rates decrease after the transition into standard time by adjusting the time back by one hour during fall and this effect continues for four days after the fall transition. My paper differs by using IV and RDD instead of event study method to estimate the short-run causal impact of sleep on traffic crashes.

In addition, Giuntella et al. (2017) uses IV method to analyze the causal impact of sleep deprivation on cognition and depression of older workers in urban China. They use sunset time as instrument and find that a later sunset time decreases sleep time and an increase in sleep duration could improve cognition and reduce depression. I am using the similar strategy of IV, but I am focusing on the short-run effects of sunset variation in the United States instead of the long-run impacts in urban China.

Another paper by Giuntella and Mazzonna (2019) uses spatial regression discontinuity design (RDD) to examine the health and income effects due to the discontinuity in sunset time at a time-zone boundary in the U.S. and find that an extra hour in sunset time leads to an average of 19 minutes decrease in sleep duration. In addition, they find the insufficient sleep is associated with negative health outcomes such as obesity, diabetes, cardiovascular diseases, and breast cancer. Rather than analyzing the long-term effects of exposure of light in the evening on health outcomes, I aim to measure both the short-run and long-run effects of sunset timing on fatalities. This paper confirms that sleep deprivation could affect the productivity and health of people through increasing the risk in fatal vehicle crashes.

Academic Achievement

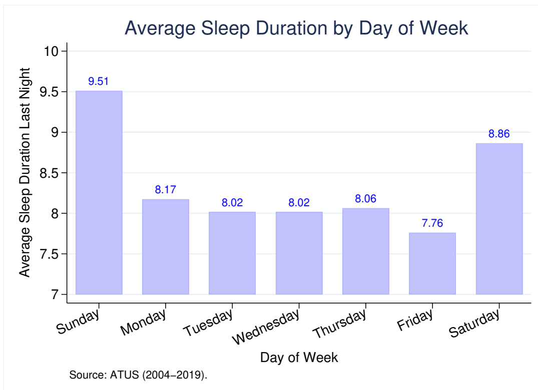
Second, this paper is related to the research that estimate the effects of school start times and sleep on academic achievements (Dills and Hernandez-Julian, 2008; Edwards, 2012). Researchers find that starting school later has a significant positive impact on academic scores for students and sleep is one of the mechanisms that could explain this impact. For example, Carrell et al. (2011) use the policy adjustments in the daily timetable at the US Air Force Academy as well as randomized allocation of freshman students to courses and conclude that starting school 50 minutes later has substantial constructive effect on test scores, corresponding to a one-standard-deviation increase in teacher quality.

In addition, a related work by Heissel et al. (2017) uses students moving across time zone border in Florida as instrument for hours of sunlight and finds that changing school start time one hour later relative to sunrise improves academic performance for adolescents in math and reading. The results are in line with sleep researchers' findings, which shows that later start times are beneficial for adolescent learning. However, it is not clear if sleep has a direct causal impact on the academic scores.

A field experiment by Avery et al. (2019) studies the effect of increased sleep on health and academic outcomes using commitment devices and monetary incentives. They find that the subjects in the treatment group are more likely to increase sleep duration and the treatment has positive but small impact on health and academic outcomes. This paper contributes to this literature by analyzing the direct causal impact of sleep on fatal vehicle crashes using non-experimental data to provide insights in the real-world scenarios.

1.4 Data

1.4.1 Individual Sleep Duration (ATUS)



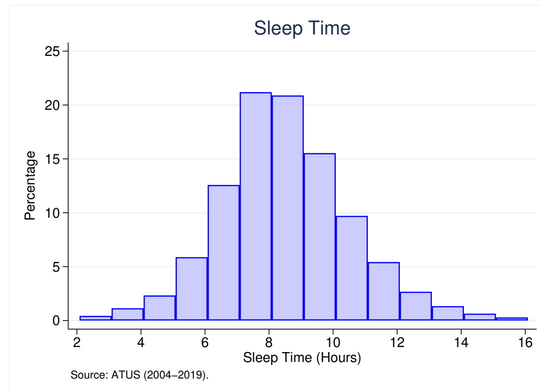


Figure 1.1: Sleep Duration

Notes: This graph shows the average sleep duration by day of week and distribution of sleep duration from ATUS (2004-2019).

The individual sleep duration data used in this study is derived from the American Time Use Survey (ATUS), which is sponsored by the U.S. Bureau of Labor Statistics (BLS) and conducted by the U.S. Census Bureau. The ATUS has been conducted since 2003 and is the first continuous survey on time use in the United States. The survey randomly selects individuals from households that have recently completed the eight-month interview for the Current Population Survey (CPS). The ATUS interviews are conducted between two and five months after the last CPS interview. The main objective of the ATUS is to gain insights into how people allocate their time.

To collect data on sleep duration, the ATUS utilizes a time diary method through computer-assisted telephone interviews. Respondents are asked to recall the time they spent on each activity from 4:00 am on the previous day to 4:00 am on the day of the interview. This approach ensures that the time diaries can be summed to a total of 24 hours, minimizing potential biases. For each activity, the ATUS collects either the ending time or the duration of the activity, and the interviewer records the responses verbatim, which are later coded (Hamermesh et al., 2005).

It is worth noting that the sleep duration measurements obtained from the ATUS tend to be higher than those obtained from other surveys that use stylized questions, such as the Behavioral Risk Factor Surveillance System (BRFSS), by approximately 1.7 hours. On average, the ATUS reports a sleep duration of around 8.7 hours per night, whereas the BRFSS reports an average sleep duration of about 7 hours (Kaplan et al., 2020). This disparity can be attributed to the fact that the ATUS diary measures encompass activities such as napping, dozing, falling asleep, and waking up (Basner et al., 2007).

I will use sleep data at the county level, which is only available from 2004 onwards, to examine the impact of sleep prior to the COVID-19 pandemic. For this analysis, I have included sleep data from the years 2004 to 2019, resulting in a total of 210,586 observations spanning from 2003 to 2020. To ensure the accuracy of the analysis, I have only included individuals who are part of the labor force, as indicated by the ATUS-CPS dataset from 2003 to 2020. It is important to note that the CPS dataset does not provide county information for individuals residing in counties with a population of less than 100,000. Consequently, I was only able to match 38.5% of the sample. As a result, the findings from the ATUS dataset are more representative of counties with a higher degree of urbanization.

To address potential confounding factors, I have narrowed down the analysis to individuals aged between 18 and 55, excluding retired individuals and high-school age workers. Additionally, I have limited the sample to individuals who sleep between 2 and 16 hours per night. It is worth noting that those who sleep less than 2 hours make up less than 1% of the total sample. After applying these restrictions, the final sample consists of 53,552 observations, with 49,671 individuals being employed. This employed group represents 92.8% of the total sample.

In this analysis, I have incorporated various socio-demographic variables, including age, race, sex, education, marital status, nativity status, and number of children. Additionally, I have taken into account geographic characteristics such as latitude and indicators for large counties and coastal counties.

Figure 1.1 illustrates the distribution of sleep duration, revealing that individuals tend to sleep more during weekends. To further support my findings, Table 1.1 presents the summary statistics for the analysis, combining data from both ATUS and FARS. Notably, the average sleep duration in my sample is 8.61 hours.

By utilizing the interview date and location coordinates (latitude and longitude), I was able to determine the daily sunset time for each individual in the sample from 2004 to 2019, as well as the average sunset time in the corresponding county in 2012. To perform these calculations, I utilized the R studio package called "suncalc." This package relies on the formulas provided by Astronomy Answers regarding the position of the sun and the planets. To ensure accuracy, I cross-verified the sunset time obtained through this method with the sunset time calculated using the Sunrise/Sunset and Solar Position Calculators provided by the National Oceanic and Atmospheric Administration (NOAA).

Table 1.1: Summary Statistics

	(1) All mean/sd	(2) Female mean/sd	(3) Male mean/sd	(4) Employed mean/sd	(5) Unemployed mean/sd
Crashes per 100,000 Population	0.74	0.74	0.73	0.74	0.72
	(0.46)	(0.47)	(0.46)	(0.47)	(0.45)
Sleep Duration (Hours)	8.61	8.69	8.52	8.56	9.25
	(2.05)	(2.04)	(2.06)	(2.04)	(2.13)
Age	38.75	38.61	38.90	39.01	35.28
	(9.71)	(9.74)	(9.67)	(9.54)	(11.13)
White	0.78	0.75	0.80	0.78	0.69
	(0.42)	(0.43)	(0.40)	(0.41)	(0.46)
Black	0.14	0.17	0.12	0.14	0.23
	(0.35)	(0.37)	(0.32)	(0.34)	(0.42)
High School	0.48	0.48	0.48	0.47	0.60
	(0.50)	(0.50)	(0.50)	(0.50)	(0.49)
College	0.44	0.45	0.43	0.45	0.21
	(0.50)	(0.50)	(0.49)	(0.50)	(0.41)
Married	0.54	0.50	0.57	0.55	0.39
	(0.50)	(0.50)	(0.49)	(0.50)	(0.49)
Nativity Status	0.77	0.79	0.76	0.77	0.75
	(0.42)	(0.41)	(0.43)	(0.42)	(0.43)
Number of Children	1.09	1.11	1.06	1.08	1.14
	(1.15)	(1.12)	(1.18)	(1.14)	(1.23)
Holiday	0.02	0.02	0.02	0.02	0.02
	(0.13)	(0.13)	(0.12)	(0.13)	(0.13)
Latitude	37.25	37.20	37.29	37.26	37.02
	(5.05)	(5.07)	(5.04)	(5.05)	(5.07)
Large County	0.68	0.68	0.68	0.68	0.72
	(0.47)	(0.47)	(0.46)	(0.47)	(0.45)
Weekend	0.51	0.51	0.50	0.51	0.50
	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)
N	36296	18569	17727	33811	2485

Notes: Data are from ATUS (2004-2019) and FARS (2004-2019). Latitude and longitude are from US Census Bureau. The sample is restricted to people who are in the labor force and aged between 18 and 55. The crashes data from FARS are matched to the ATUS data at county-year-month level.

1.4.2 Fatal Vehicle Crashes (FARS)

The data on fatal vehicle crashes is collected from the Fatality Analysis Reporting System (FARS), which was developed by the National Center of Statistics and Analysis (NCSA) of the National Highway Traffic and Safety Administration (NHTSA). FARS has been gathering information on fatal vehicle crashes from all 50 states in the United States since 1975. In order to be included in FARS, a crash must involve a motor vehicle traveling on a public trafficway and must result in the death of at least one motorist or non-motorist within 30 days of the crash.

FARS provides detailed information on the exact time and location of the accident, as well as the road type, light condition, and weather. For my analysis, I will be using the data from 2004 to 2019, which consists of 539,052 observations. On average, there are approximately 33,690 fatal crashes each year, resulting in about 92 fatal crashes per day across the entire United States. It is worth noting that fatal crashes are more likely to occur between 4pm and midnight, as well as on weekends.

Data on non-fatal crashes is not available for the entire nation, as many states do not maintain a standardized database for such incidents. While analyzing only the data on fatal vehicle crashes provides valuable insights, it does create a lower bound of the impact of sleep on all types of vehicle crashes.

1.5 Empirical Methods

1.5.1 Identification Strategy 1: Sunset Time as Instrument (IV)

This study aims to investigate the causal impact of sleep durations on fatal vehicle crashes. However, there are two main concerns that need to be addressed. The first concern is the possibility of omitted variables bias, which means that there may be other variables that are associated with both sleep duration and crashes. The second concern is reverse causality, which suggests that fatal vehicle crashes could actually affect sleep duration.

To overcome these challenges, I will employ an instrumental variable (IV) strategy to measure the causal relationship between sleep and crashes. This strategy has been used in previous studies, such as the one conducted by Gibson and Shrader (2018) to examine the causal influence of sleep duration on wages in the United States.

In order to estimate the short-term effects, I will use the variation in sunset times across a specific location throughout the year. This will allow me to capture the immediate impact of sleep duration on fatal vehicle crashes. For the long-term effects, I will consider the variation in sunset times across different locations in the United States to provide insights into the lasting effects of sleep duration on crashes.

I will provide background information on the relationship between sunset and sleep, then discuss the short-term and long-term specifications separately, explaining how each approach contributes to our understanding of the causal relationship between sleep and fatal vehicle crashes.

Relationship between Sunset and Sleep

The timing and duration of sleep are strongly associated with the rising and setting of sun. This biological relationship between sleep and daylight provides the reasoning for why selecting sunset as instrument for sleep. Roenneberg et al. (2007) show that light is the strongest signal from the environment for human biological clock and find that sun time, rather than social time, has the primary influence on the synchronization of human circadian rhythm. The circadian system is a strong force that synchronize with environmental stimuli. Nearly every living creature has an internal clock that is set to the Earth's 24-hour rotational timetable. This internal circadian rhythm helps the body to anticipate the external environment, such as when the sun will rise and set, as well as the optimal times to sleep, wake, eat, and exercise. Individuals who do not sleep at their ideal circadian timing or who are sleep deprived compared to intrinsic sleep need are facing more negative health outcomes (Ashbrook et al., 2020). Due to the circadian rhythm, the variation in daylight could affect sleep habits.

Location and seasonal variation in sunset time could all cause a change in sleep patterns. Researchers find that individuals living in a location with later sunset time tend to sleep later (Gibson and Shrader, 2018; Giuntella and Mazzonna, 2019). The sunlight changes across year also affect the sleep patterns (Hubert et al., 1998). Latitude and longitude could both influence the sunset and sunrise time. For example, Campante and Yanagizawa-Drott (2015) use the interaction of latitude and the rotation of lunar calendar to identify the causal relationship between the length of Ramadan fasting and the economic growth in Muslim countries. In addition, Brockmann et al. (2017) explore the associations between sleep duration and latitude in Chile and find that people sleep longer with increasing latitude. Furthermore, Friborg et al. (2012) analyze the associations between seasonal variations in day length and sleep comparing Ghana and Norway and find that lack of daylight was related to change of sleep patterns. The change in sleep pattern could affect the sleep duration due to work and school scheduling.

Rigid work and school schedule could disrupt human circadian rhythms and cause health and productivity issues. In the recent economic literature, the distribution of time among market work, home production work, leisure, and rest has been a major topic (Becker, 1965; Gronau, 1977; Aguiar and Hurst, 2007; Guryan et al., 2008; Aguiar et al., 2013; Carneiro et al., 2015; Bastian and Lochner, 2020). The allocation of time could depend on the working and school schedules, and the social times are usually synchronized for optimal welfare (Weiss, 1996; Hamermesh et al., 2008). If people could wake up late to compensate sleep late, then the sleep duration would be the same. However, workers and students have the forced synchronization of work and school scheduling, thus later sunset and bedtime would shorten sleep duration in the short and long term. A decrease in sleep duration could disrupt human circadian rhythms, which could post negative effects on health and productivity (Cappuccio et al., 2010).

Daily Sunset Time Variation for Short-Run Analysis

In the short run, I will use the daily sunset variation in one location across the year as the instrument. Figure 1.2 shows that the sunset time is like a cosine wave over a year. The latitude of the location determines the amplitude of the wave, and the longitude

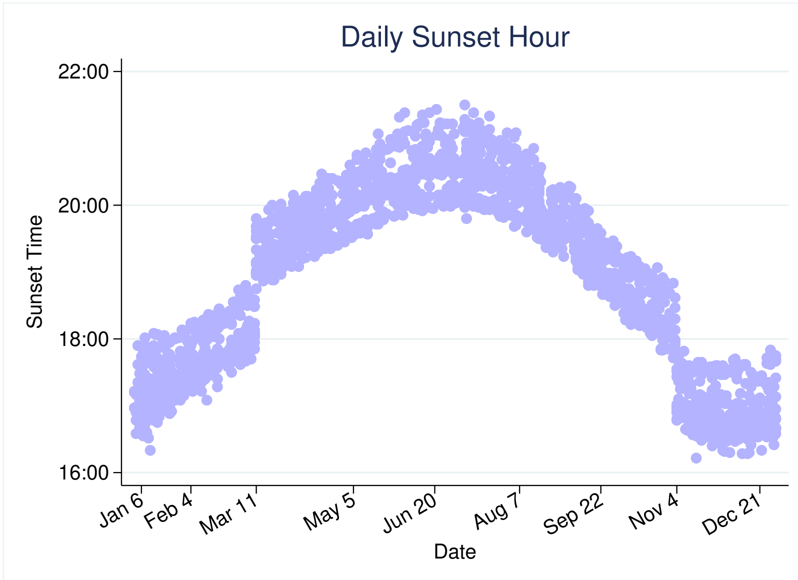


Figure 1.2: Daily Sunset Hour - Short Run Analysis

Notes: This graph depicts the daily sunset hours for counties sampled by ATUS in the continental United States in 2012. The y-axis shows the sunset hour in 24 hour time. For example, 16:00 is the same as 4:00pm. Mar 11 is when the DST starts and Nov 4 is when the DST ends in 2012. Jun 20 is the summer solstice and Dec 21 is the winter solstice. The setup of this graph is similar to Gibson and Shrader (2018).

variation within a time zone defines the average sunset time, which is used to estimate the long-run effects. The substantial spring and fall leaps generated by DST is another characteristic of the sunset time. There is a regular seasonal pattern, and the sunset time is generally late during summer and early during winter. The later sunset time in the summer could result in shorter sleep duration, which could impact the attention and disrupt circadian rhythm.

In terms of the instrument validity, the first requirement is the instrument of sleep must be strongly correlated with sleep. The F test for the first stage is 11.94 for unconditional model and 10.93 for conditional model, which are both greater than 10. This suggest that this instrument has a strong first stage. The second requirement is that the instrument of sleep cannot be correlated with the error term in the equation of interest. If the instrument meets this requirement, then this instrument satisfy the exclusion restriction. The exclusion restriction validity requires that other crashes determinants do not correlate with daily sunset time in a location. Since sunset time follows a predictable seasonal pattern, the major challenge to this assumption is seasonally varying crash determinants.

One potential concern of the identification strategy is that sunset time varies seasonally, so does sunrise time and daylight duration. Medical research show that the length of daylight has a positive effect on mood as the sunlight could help the body to produce vitamin D, which could affect mood and depression (Murase et al., 1995; Lambert et al., 2002; Kjærgaard et al., 2012; Friborg et al., 2012). Furthermore, exposure to more light in the evening could provide incentive to exercise more (Wolff and Makino, 2012). If daylight influences both crashes and sleep through mood or another channel, the short-run results could be misleading. To address the seasonality issue, I include the controls for seasonality, such as year-month fixed effects and I got the similar results. I assume that the crash determinants such as the mood due to seasonality do not correlate with the sunset time.

Other confounding factors may include icy road in winter and drinking behaviors etc. Those unobserved confounding factors could be correlated with sunset time as well as the crashes. Including the time fixed effects could alleviate the concern of different road conditions in various seasons. Adding the county fixed effects could address the issue that the north and south locations would have different road conditions during winters. I use sunset time instead of sunrise time because the rigid work and school schedules would affect the wake up time, and the sunset time may have a larger impact on the sleep duration for employed people.

Average Sunset Time Variation for Long-Run Analysis

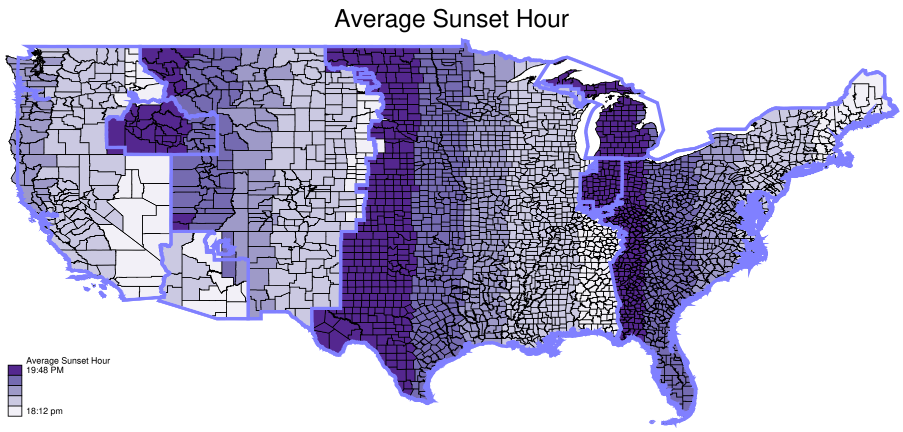


Figure 1.3: Average Sunset Hour - Long Run Analysis

Notes: This graph shows the average sunset hours for all counties in the continental United States in 2012. I used sunset time package "suncalc" from R studio to calculate the average sunset time. I separated counties into 5 quintile based on the average sunset time in 2012. Darker color implies later sunset. The time zone border lines are in blue. The setup of this graph is similar to Gibson and Shrader (2018) and Giuntella and Mazzonna (2019).

In the long run, I will use the average sunset variation across locations as instrument. Figure 1.3 depicts the average sunset time for the continental United States in 2012. The eastern part gets darker late in a time zone, which indicate that the people who live in eastern areas are more likely to go to bed later and sleep less. Within a U.S. time zone, the largest variance in sunset time is around 1 hour. The average sunset time is constant regardless of latitude. Since all counties in the continental U.S. have almost the same average annual daylight, this is not a confounding factor in the long-run study.

Time and scheduling were not consistent across the United States until the development of the railroad traffic after the Civil War. America's railroads started the first U.S. time zones on November 18, 1883, known as Standard Railroad Time. Later in 1918, the Standard Time Act established the current four continental U.S. time zones including

Eastern, Central, Mountain, and Pacific. Since then, the time zones have been in effect, with only minor adjustments at the margins. Currently, 12 of the 48 continental states are in more than one time zone (Bartky, 1989; Hamermesh et al., 2008).

The purpose of the invention of DST was to save energy during times of war. In 1918, the United States established a formal DST schedule, but it was overturned when World War I ended due to its inconvenient nature. In 1966, President Johnson signed the current U.S. DST scheme into law. Each state can surpass the law by enacting its own legislation. In 2007, the DST time was extended by four weeks. Except for Arizona and Hawaii, most states in the United States implement DST, and Indiana began to adopt DST in 2006 (Kamstra et al., 2000).

State and local government could require the Department of Transportation (DOT) to change time zones (Valpando, 2013). This alteration of time zone borders suggest that time zone is not set randomly. Counties have changed in both westward and eastward directions, and it is more common to switch to the east side, which has later sunset. Since the position of the border is not exogenous, comparing nearby counties on the opposite sides of the border could lead to biased results under regression discontinuity design. In addition, I could exclude counties that do not adopt DST to avoid possible endogeneity issue.

As for instrument validity, I first check if the average sunset instrument is strongly correlated with sleep. The F test for the first stage is 0.02 for unconditional model and 0.01 for conditional model and this suggest that this instrument is a weak instrument. Possible confounding factors include sorting and coastal distance, which could possibly correlates with the sunset instrument and the determinants that affect the crashes. Individuals could sort on the eastern of western side of a time zone border, which suggest that there is correlation between average sunset time and population density. In addition, the average sunset time could correlate with coastal distance since sunset time is related to longitude. Coastal distance could affect the risk of vehicle crashes because individuals report better overall health and mental health when they live close to the seaside (White et al., 2013).

1.5.2 Identification Strategy 2: Discontinuity in Sunset Time at Time-zone Border (RDD)

The RDD strategy exploits the sharp discontinuity in sunset time across time zone borders. Figure 1.4 shows there is a distinct discontinuity in sunset time around the border, with sunset occurring approximately one hour later for counties situated on the west side of the time zone boundary compared to those on the east side. Figure 1.5 illustrates the process of determining the distance of counties to the nearest time zone border within a 400-mile radius using QGIS. Initially, I isolated the time zone borders between each time zone by employing the "split features" function. Subsequently, I utilized the "shortest line" function between the centroids of each county and the time zone borders.

In the RDD framework, it is essential to assume that there are no disparities in observable or unobservable attributes that could introduce confounding effects into the outcomes. Unlike a conventional regression discontinuity design, it is not possible to directly compare individuals living on opposite sides of the time zone boundary because they would be residing at different latitudes. To enable the comparison of individuals residing in neighboring counties, this analysis includes a set of geographic reference variables and utilizes linear controls for latitude.

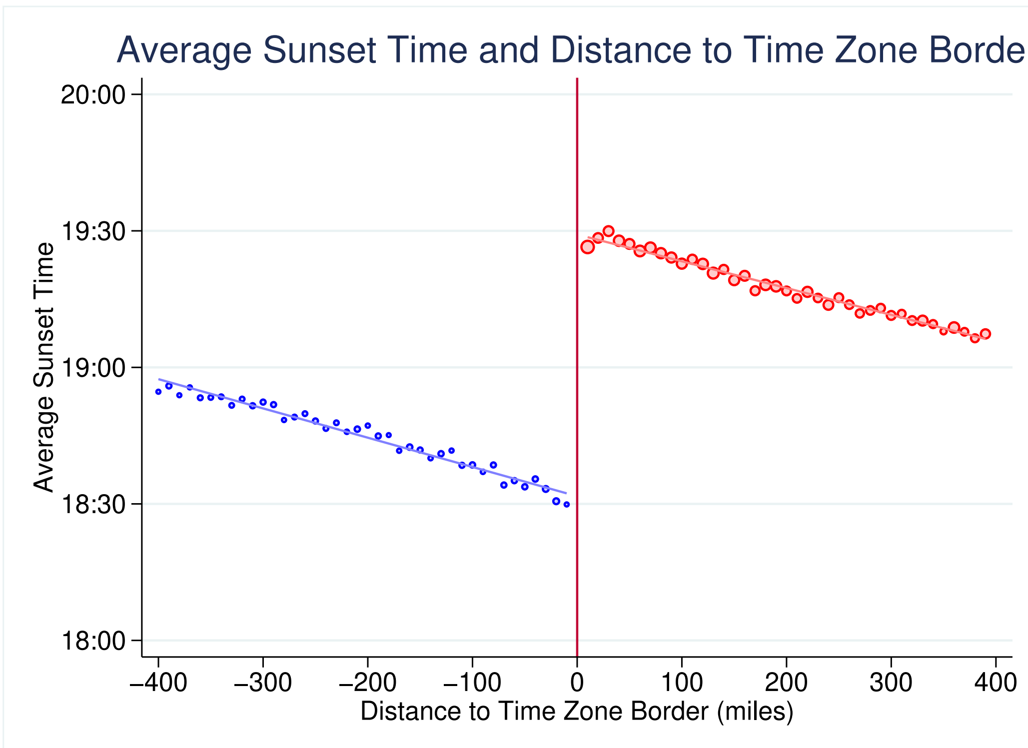


Figure 1.4: Sunset and Distance to Time Zone Border for Unemployed

Notes: This graph shows the discontinuity in sunset time over distance to time zone borders. The distance are calculated using QGIS. I used sunset time package "suncalc" from R studio to calculate the average sunset time. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

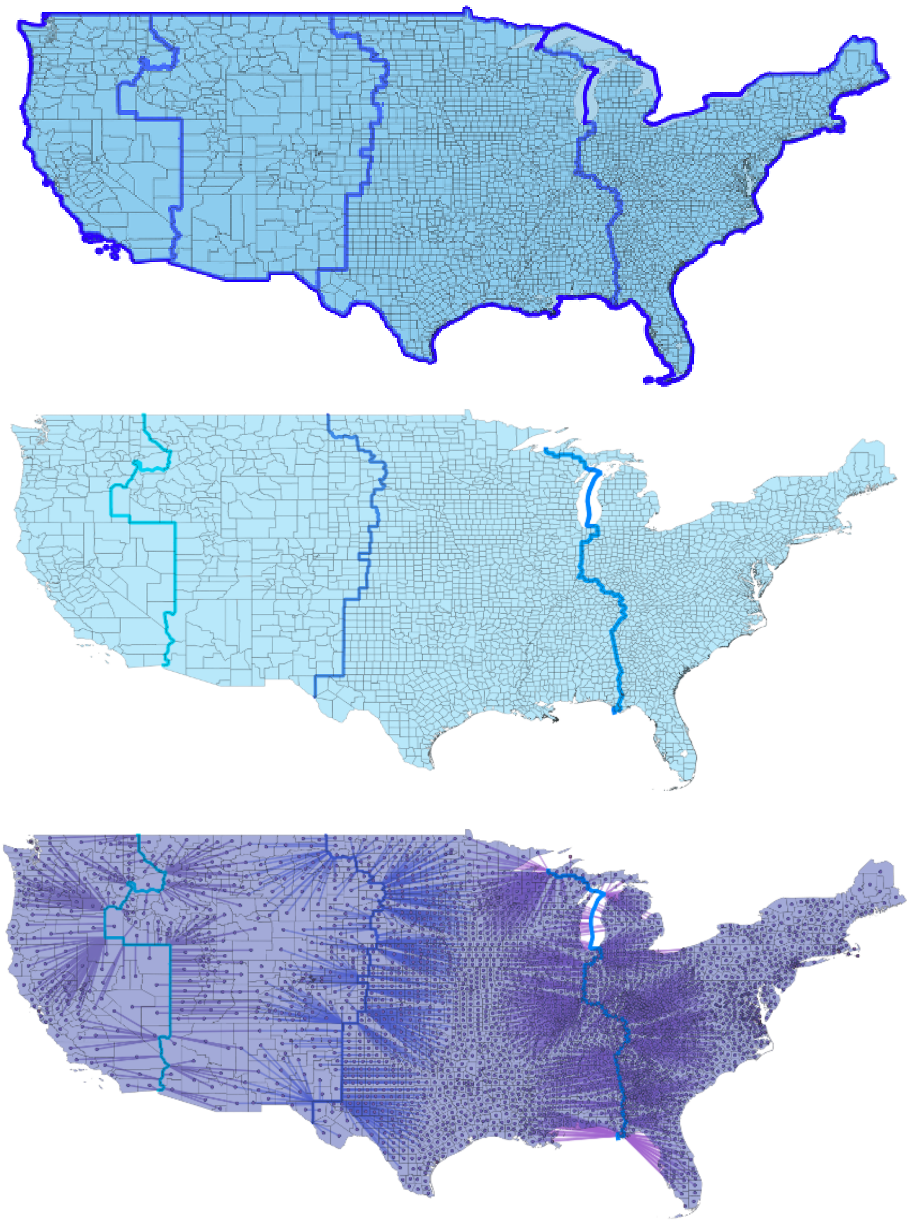


Figure 1.5: Distance of Counties to the Nearest Time Zone Border

Notes: This graph illustrates the process of determining the distance of counties to the nearest time zone border within a 400-mile radius using QGIS. Initially, I isolated the time zone borders between each time zone by employing the "split features" function. Subsequently, I utilized the "shortest line" function between the centroids of each county and the time zone borders.

1.5.3 Estimation Equations

1.5.4 IV Strategy

First, I use the instrumental variable method. To estimate the short-run effect of sleep, I will first use the monthly changes in sunset within a county as the first instrument. I estimate the following short-run first stage,

$$Sleep_{ijt} = \alpha_1 Sunset_{jt} + X'_{ijt}\delta_1 + \gamma_{1,j} + \eta_{1,ijt} \tag{1.1}$$

short-run second stage,

$$Crash_{jt} = \alpha_2 Sle\hat{e}p_{ijt} + X'_{it}\delta_2 + \gamma_{2,j} + \eta_{2,ijt} \tag{1.2}$$

and reduced form,

$$Crash_{jt} = \alpha_3 Sunset_{jt} + X'_{it}\delta_3 + \gamma_{3,j} + \eta_{3,ijt} \tag{1.3}$$

where $Sleep_{ijt}$ is the monthly sleep duration for individual i in county j for date t, $Sunset_{jt}$ is the sunset time on that date in that county, $\gamma_{1,j}$ includes county fixed effects, X_{it} is a vector controls including socio-demographics (age, race, sex, education, marital status, nativity status, and number of children), geographic characteristics (latitude, longitude, and indicator for large counties), and interview characteristics (indicators for holiday and weekend). $Crash_{jt}$ is the fatal crashes per 100 million VMT for the county j at county-year-month level. $\eta_{k,jm}$ is the error term for k $\in \{1, 2, 3\}$.

The second instrument is the annual average sunset, which captures the geographical differences in sunset time across counties in the United States. I estimate the following long-run first stage,

$$Sleep_j = \delta_1 Sunset_j + X_j' \beta_1 + \epsilon_{1,j} \tag{1.4}$$

short-run second stage,

$$Crash_j = \delta_2 \hat{Sleep}_j + X_j' \beta_2 + \epsilon_{2,j} \tag{1.5}$$

and reduced form,

$$Crash_j = \delta_3 Sunset_j + X_j' \beta_3 + \epsilon_{3,j} \tag{1.6}$$

where $Sleep_j$ is average monthly sleep duration in location j, $Sunset_j$ is the average sunset time in that location, X_j is a vector controls including county-level socio-demographics (age, race, sex, education, marital status, nativity status, and number of children), geographic characteristics (latitude and indicators for large counties and coastal counties), and interview characteristics (indicators for holiday and weekend). $Crash_j$ is the average fatal crashes per 100 million VMT at county-month level. $\epsilon_{k,j}$ is an error term for k $\in \{1, 2, 3\}$.

1.5.5 RDD Strategy

$$Sleep_{ijt} = \beta_0 + \beta_1 LS_j + \beta_2 f(D_j) + \beta_3 f(D_j) * LS_j + X'_{ijt}\beta_4 + u_{ijt} \tag{1.7}$$

where $Sleep_{ijt}$ is the daily sleep duration for individual i in county j for date t, LS_j is indicator for the county located on the late sunset side of a time zone border, D_j is the distance to the time zone border or the "running variable," X_{ijt} is a vector controls including individual socio-demographics characteristics (age, race, sex, education, marital status, nativity status, and number of children), geographic characteristics (latitude and indicators for large counties and coastal counties), and interview characteristics (indicators for holiday and weekend).

$$Crash_j = \beta_0 + \beta_1 LS_j + \beta_2 f(D_j) + \beta_3 f(D_j) * LS_j + X'_j\beta_4 + u_j \tag{1.8}$$

where $Crash_j$ is the average fatal crashes per 100 million VMT at county-month level., LS_j is indicator for the county located on the late sunset side of a time zone border, D_j is the distance to the time zone border or the "running variable," X_j is a vector controls including county-level socio-demographics (age, race, sex, education, marital status, nativity status, and number of children), geographic characteristics (latitude and indicators for large counties and coastal counties), and interview characteristics (indicators for holiday and weekend).

1.6 Results

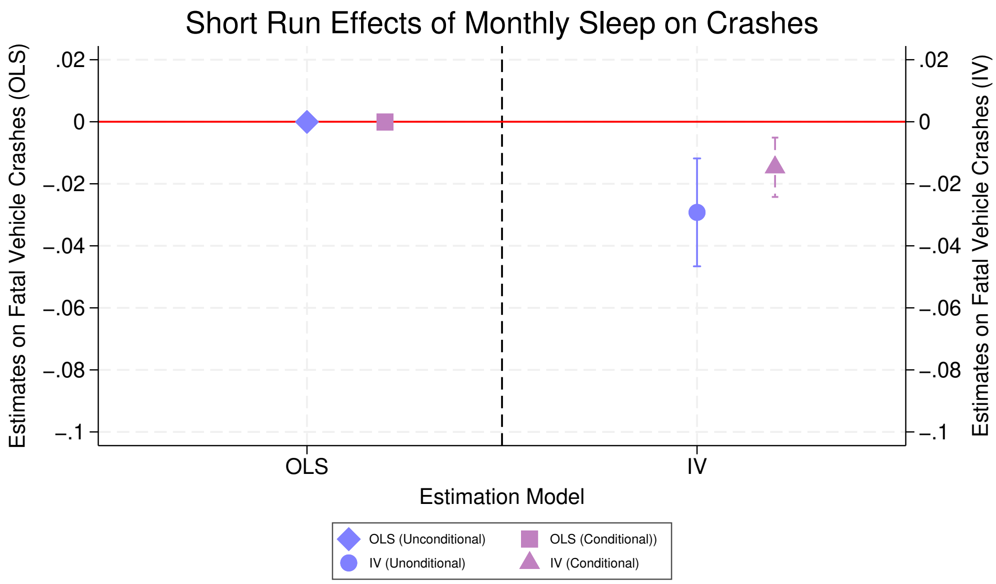
1.6.1 IV Strategy

Table 1.2 shows the results of the short run effects of sunset and sleep. The first and second column suggest that there is no major impact of average monthly sleep on crashes in terms of statistical significance and magnitude under ordinary linear regression (OLS) model. The third and fourth column show the results for Equation (1.1), which implies that one hour late in sunset will lead to about 12 minutes decrease in weekly sleep.

In column (6), the estimates suggest that increasing monthly sleep by one hour results in a decrease of 0.015 fatal crashes per 100,000 population at the county-year-month level. This reduction is equivalent to a 2.4% decrease in fatalities in the short term, as shown in 1.3 (column 6).

Figure 1.6 shows the same results for Table 1.2 and Table 1.3. In each panel, the left y-axis denotes the scale for OLS results and the right y-axis depicts the scale for IV estimates. The top panel indicates that one hour increase in monthly sleep causes 0.015 reduction in fatalities under IV (conditional model). The bottom panel suggests that additional one hour of sleep reduces fatalities by 2.4% under IV (conditional model). As a comparison, the OLS estimates are close to zero in both panels.

As for the long run results, Table 1.4 and Table 1.5 show that there is no significant impact of sleep on fatalities. One exception is that the sunset time has positive and significant impact on crashes in column (7) under the unconditional model, which is in line with the short-run results, suggesting that a later sunset time increases fatalities.



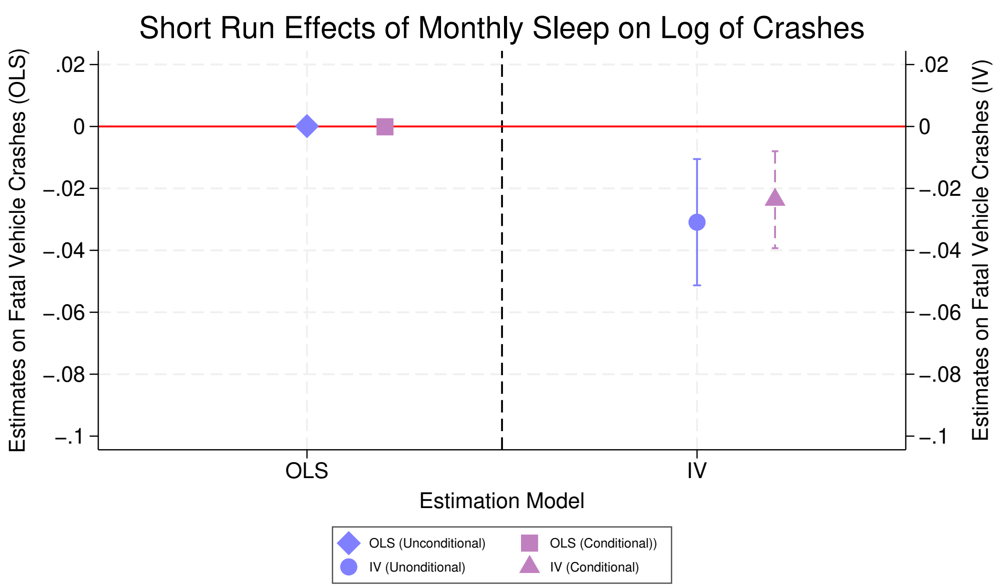


Figure 1.6: Short Run Effects of Monthly Sleep on Crashes

Notes: This graph shows estimates of the short run effects of monthly sleep on crashes using OLS and IV. The error bars are at 95% confidence intervals for the mean. Sleep denotes monthly average sleep hours. The dependent variable of crashes refers to fatal crashes per 100,000 population at county-year-month level. Controls include socio-demographics (age, race, sex, education, marital status, nativity status, and number of children) and geographic characteristics (latitude, longitude, and dummy for large counties).

Table 1.2: Short Run Effects of Sunset and Sleep

	OLS		IV(First-Stage)		IV(Second-Stage)		Reduced-Form	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Crashes	Crashes	Sleep	Sleep	Crashes	Crashes	Crashes	Crashes
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Average Monthly Sleep	-0.000	-0.000			-0.029***	-0.015***		
	(0.00)	(0.00)			(0.01)	(0.00)		
Sunset Hour			-0.815***	-0.778***			0.024***	0.011***
			(0.24)	(0.22)			(0.00)	(0.00)
Mean	0.81	0.81	261.28	261.28	0.81	0.81	0.81	0.81
Controls	No	Yes	No	Yes	No	Yes	No	Yes
County FEs	No	Yes	No	Yes	No	Yes	No	Yes
Observations	36296	36296	36296	36296	36296	36296	36296	36296
F test			11.99	12.55				

Notes: Sleep and sunset time are measured in hours by state-county level. The dependent variable of sleep is monthly average sleep hours. The dependent variable of crashes refers to fatal crashes per 100,000 population at county-year-month level. Controls include socio-demographics (age, race, sex, education, marital status, nativity status, and number of children), geographic characteristics (latitude, longitude, and indicator for large counties), and interview characteristics (indicators for holiday and weekend). The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). F test on the excluded instrument. Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.3: Short Run Effects of Sunset and Sleep (Log of Crashes)

	OLS		IV(First-Stage)		IV(Second-Stage)		Reduced-Form	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Crashes	Crashes	Sleep	Sleep	Crashes	Crashes	Crashes	Crashes
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Average Monthly Sleep	-0.000	-0.000			-0.044***	-0.024***		
	(0.00)	(0.00)			(0.01)	(0.01)		
Sunset Hour			-0.815***	-0.778***			0.036***	0.019***
			(0.24)	(0.22)			(0.01)	(0.00)
Mean	-0.41	-0.41	261.28	261.28	-0.41	-0.41	-0.41	-0.41
Controls	No	Yes	No	Yes	No	Yes	No	Yes
County FEs	No	Yes	No	Yes	No	Yes	No	Yes
Observations	36296	36296	36296	36296	36296	36296	36296	36296
F test			11.99	12.55				

Notes: Sleep and sunset time are measured in hours at county level. The dependent variable of crashes refers to fatal crashes per 100,000 population at county level. Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.4: Long Run Effect of Sunset on Sleep and Fatal Crashes

	OLS		IV(First-Stage)		IV(Second-Stage)		Reduced-Form	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Crashes	Crashes	Sleep	Sleep	Crashes	Crashes	Crashes	Crashes
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Average Monthly Sleep	-0.00	-0.00			-0.32	0.90		
	(0.00)	(0.00)			(2.32)	(99.51)		
Sunset Hour			-0.35	-0.02			0.11^{***}	-0.02
			(2.51)	(2.50)			(0.04)	(0.04)
Mean	0.97	0.97	261.39	261.39	0.97	0.97	0.97	0.97
Controls	No	Yes	No	Yes	No	Yes	No	Yes
County FEs	No	No	No	No	No	No	No	No
Observations	396	396	396	396	396	396	396	396
F test			0.02	0.00				

Notes: Sleep and sunset time are measured in hours by county level. The dependent variable of crashes refers to fatal crashes per 100,000 population at county level. Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.5: Long Run Effect of Sunset on Sleep and Log of Fatal Crashes

	OLS		IV(First-Stage)		IV(Second-Stage)		Reduced-Form	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Crashes	Crashes	Sleep	Sleep	Crashes	Crashes	Crashes	Crashes
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Average Monthly Sleep	-0.00	-0.00			-0.45	0.65		
	(0.00)	(0.00)			(3.21)	(72.16)		
Sunset Hour			-0.35	-0.02			0.16^{***}	-0.01
			(2.51)	(2.50)			(0.05)	(0.03)
Mean	-0.22	-0.22	261.39	261.39	-0.22	-0.22	-0.22	-0.22
Controls	No	Yes	No	Yes	No	Yes	No	Yes
County FEs	No	No	No	No	No	No	No	No
Observations	396	396	396	396	396	396	396	396
F test			0.02	0.00				

Notes: Sleep and sunset time are measured in hours by county level. The dependent variable of crashes refers to fatal crashes per 100,000 population at county level. Significance levels: * 0.10, ** 0.05, *** 0.01.

1.6.2 RDD Strategy

The first stage results of the RDD study reveal an important caveat. Previous research conducted by Giuntella and Mazzonna (2019) focused on the time frame from 2003 to 2013 and found that employed individuals tend to sleep less when living on the later sunset side of the time zone border. When I replicate this analysis using the same dataset and period from 2004 to 2013, my findings closely align with Giuntella and Mazzonna (2019). However, an intriguing twist emerges when I extend the analysis to include data collected from 2014 to 2019. During this later period, I observe a contrary trend, wherein employed individuals actually tend to sleep more if they reside on the later sunset side of the time zone border.

Figure 1.7 illustrates the discontinuity in sleep and crash rates in relation to the distance from the time zone border. The first row indicates that employed individuals located on the right side of the time zone border have similar sleep and fatality rates compared to those on the left side (2004-2019). The second row demonstrates that employed people living on the side of the time zone border with later sunsets experience less sleep and fewer crashes (2004-2014), which is consistent with results by Giuntella and Mazzonna (2019). Conversely, the third row shows that individuals on the later sunset side have more sleep and higher fatality rates (2014-2019).

The first column of Figure 1.7 shows the relationship between sleep and distance to the time zone border. The data indicates that employed individuals on the right side of the border have similar sleep compared to those on the left side from 2004 to 2019. From 2004 to 2014, people on the side with later sunsets have reduced sleep, supporting previous research by Giuntella and Mazzonna (2019). However, from 2014 to 2019, individuals on the later sunset side have increased sleep.

Similar results are observed in Table 1.6, Table 1.7, and Table 1.8. Specifically, the findings in column 3 and 6 of Table 1.6 indicate that the impact of the late sunset side on sleep is both statistically significant and of small magnitude. Most columns in Table 1.7 demonstrate that employed individuals sleep less on the late sunset side, with statistically significant results. Notably, column 6 reveals that residing in the late sunset side can lead to an average reduction in sleep duration by 21 minutes. However, while column 4 and 5 in Table 1.8 show positive and significant effects, column 6 does not reach statistical significance. This indicates that there may be other factors that could have affected the results in the later period from 2014 to 2019.

Moving on to the second stage, the results from Table 1.9, Table 1.10, and Table 1.11 indicate that there are almost no statistically significant and consistent effects of sunsets on sleep. However, it is important to note that the negative and significant effect observed in column 2 of Table 1.9 and Table 1.10 becomes smaller and loses significance after accounting for county fixed effects. Furthermore, the signs of the effects are not consistent when using the 250 mile bandwidth and the 100 mile bandwidth. In Table 1.11, column 6 shows a negative and significant effect, but the other columns exhibit inconsistent signs and lack statistical significance.

In summary, my RDD analysis reveals that employed individuals residing on the later sunset side of the time zone border experienced a decrease in sleep duration from 2004 to 2013, which aligns with the findings of Giuntella and Mazzonna (2019). However, from 2014 to 2019, there was an increase in sleep duration. Furthermore, the second-stage results indicate that the impact of late sunset on sleep is not consistently significant across various time periods and bandwidths.

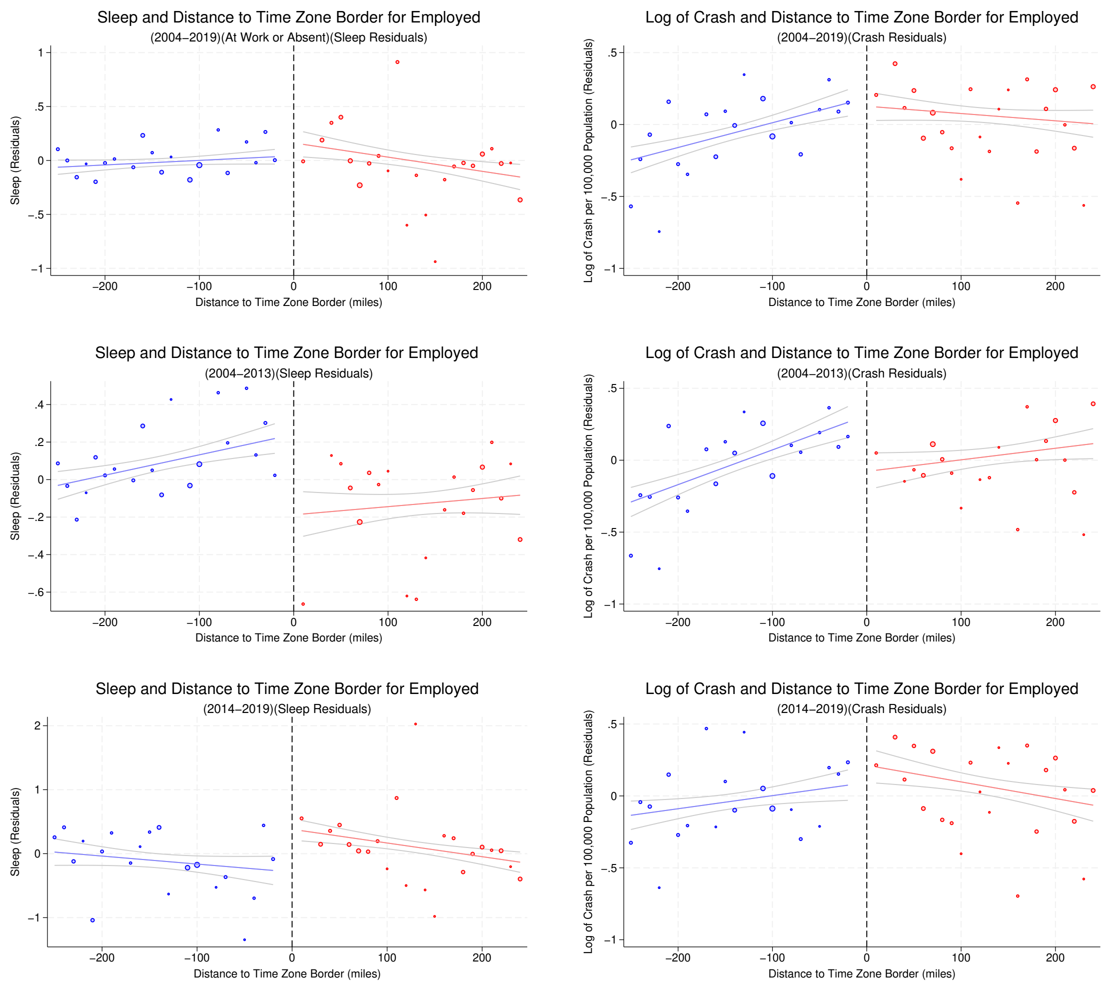


Figure 1.7: Sleep and Crash Discontinuity

The figure illustrates the discontinuity in sleep and crashes in relation to the distance from the time zone border. Data are from ATUS and FARS (2004-2019). Each point represents the the mean residuals (10 miles average) of sleep on a set of geographical controls (a linear control for latitude and dummy for large counties). The right figure shows the discontinuity in crash and distance to time zone border. Each point represents the mean residuals (10 miles average) of the crash per 100,000 population on a set of geographical controls (a linear control for latitude and dummy for large counties). The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

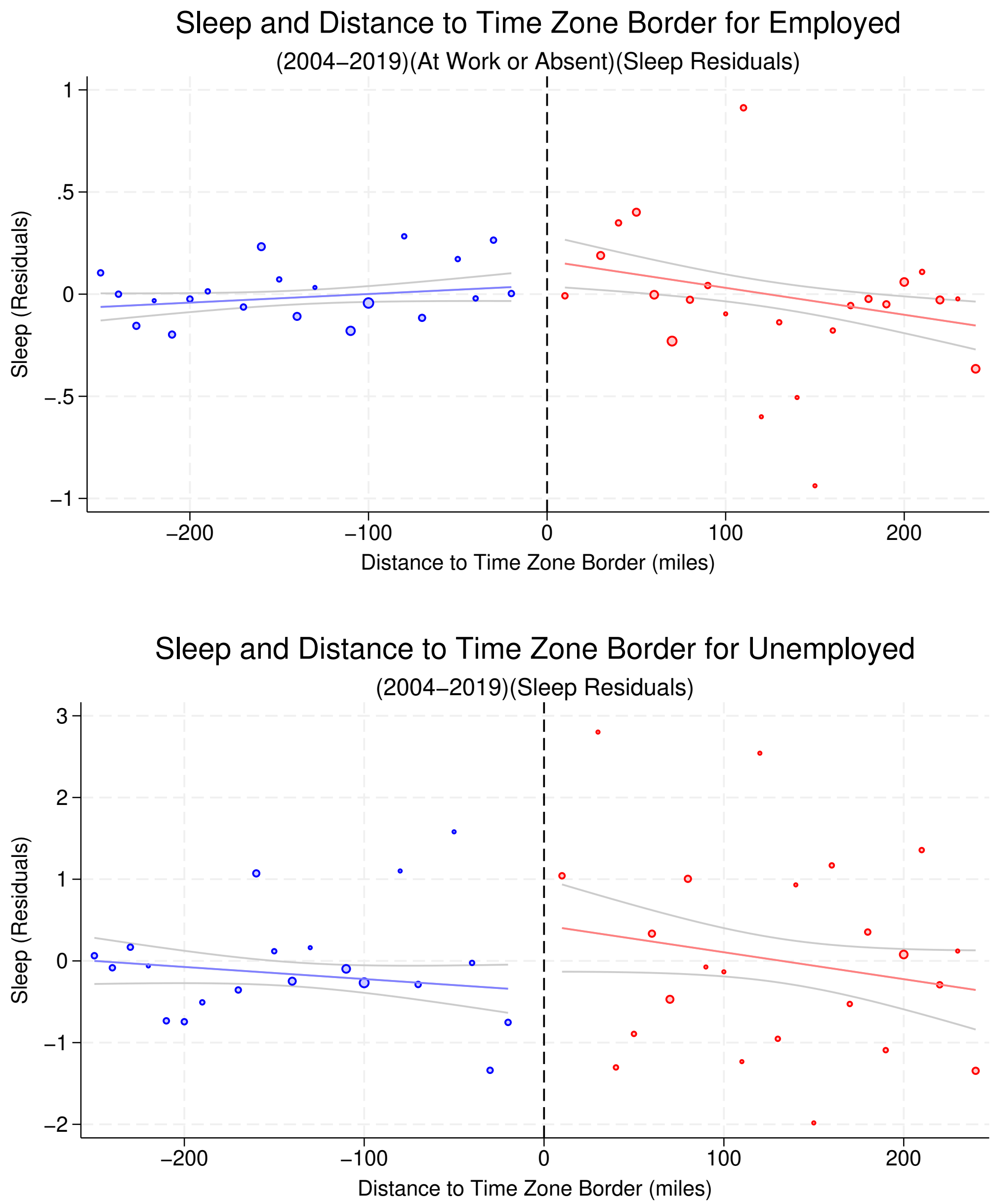


Figure 1.8: Sleep and Distance to Time Zone Border (2004-2019)

This figure show the discontinuity in sleep and distance to time zone border for employed and unemployed individuals. Data are from ATUS (2004-2019). Each point represents the the mean residuals (10 miles average) of sleep on a set of geographical controls (a linear control for latitude and dummy for large counties) on the right panel. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

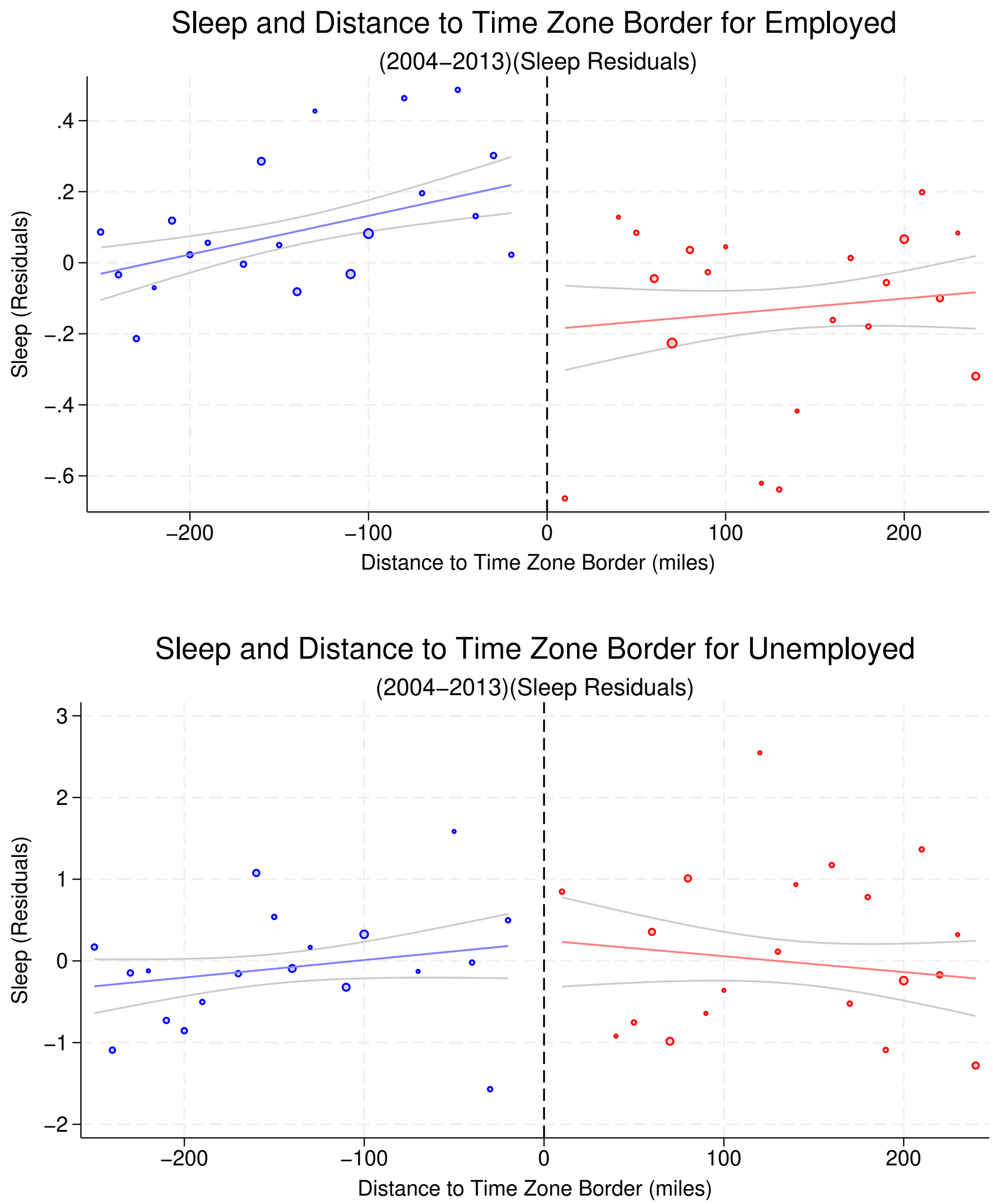


Figure 1.9: Sleep and Distance to Time Zone Border (2004-2013)

This figure show the discontinuity in sleep and distance to time zone border for employed and unemployed individuals. Data are from ATUS (2004-2013). Each point represents the the mean residuals (10 miles average) of sleep on a set of geographical controls (a linear control for latitude and dummy for large counties) on the right panel. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

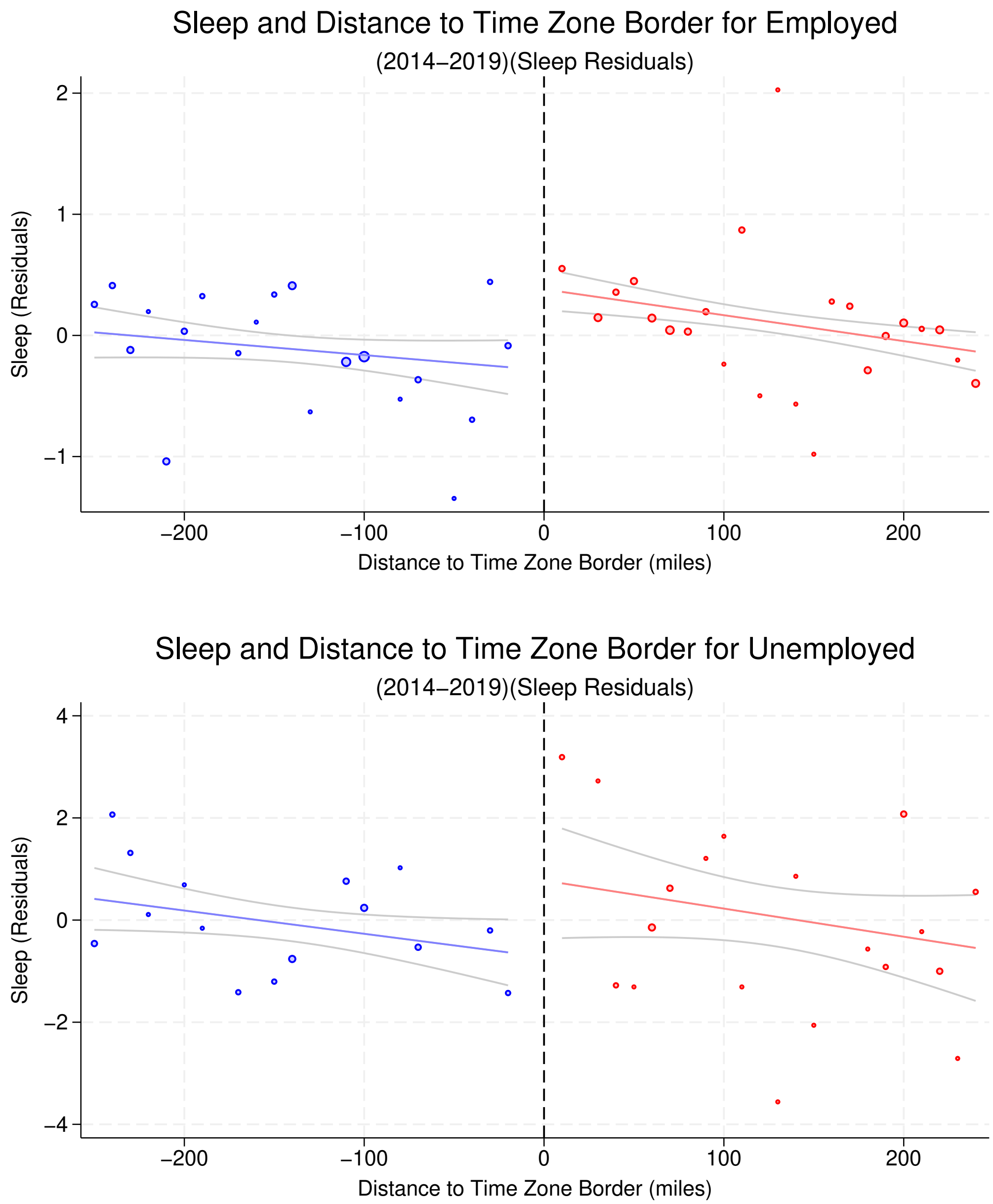


Figure 1.10: Sleep and Distance to Time Zone Border (2014-2019)

This figure show the discontinuity in sleep and distance to time zone border for employed and unemployed individuals. Data are from ATUS (2014-2019). Each point represents the the mean residuals (10 miles average) of sleep on a set of geographical controls (a linear control for latitude and dummy for large counties) on the right panel. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

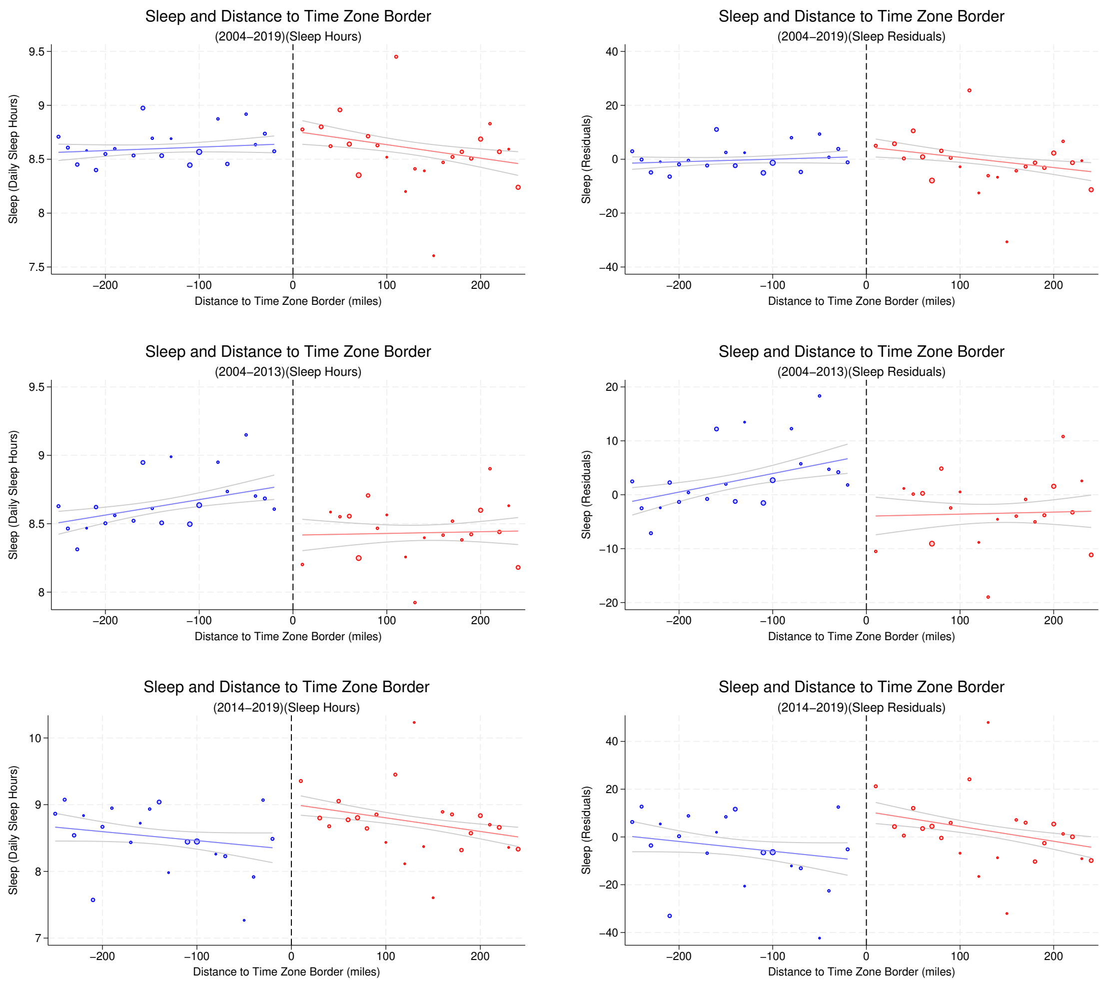


Figure 1.11: Sleep and Distance to Time Zone Border

This figure show the discontinuity in sleep and distance to time zone border. Data are from ATUS (2004-2019). Each point represents the mean daily sleep hour on the left panel and mean residuals (10 miles average) of sleep on a set of geographical controls (a linear control for latitude and dummy for large counties) on the right panel. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

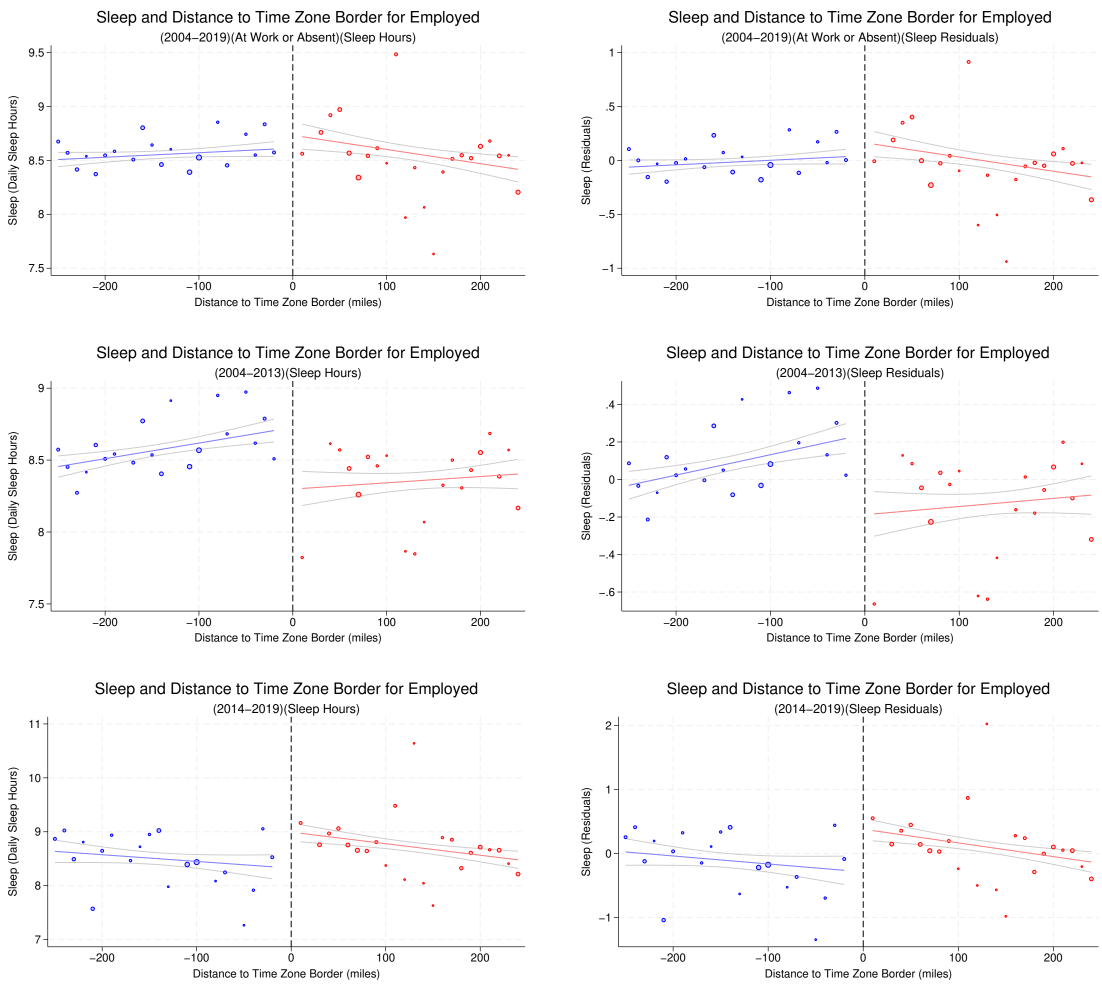


Figure 1.12: Sleep and Distance to Time Zone Border for Employed

This figure show the discontinuity in sleep and distance to time zone border for employed individuals. Data are from ATUS (2004-2019). Each point represents the mean daily sleep hour on the left panel and mean residuals (10 miles average) of sleep on a set of geographical controls (a linear control for latitude and dummy for large counties) on the right panel. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

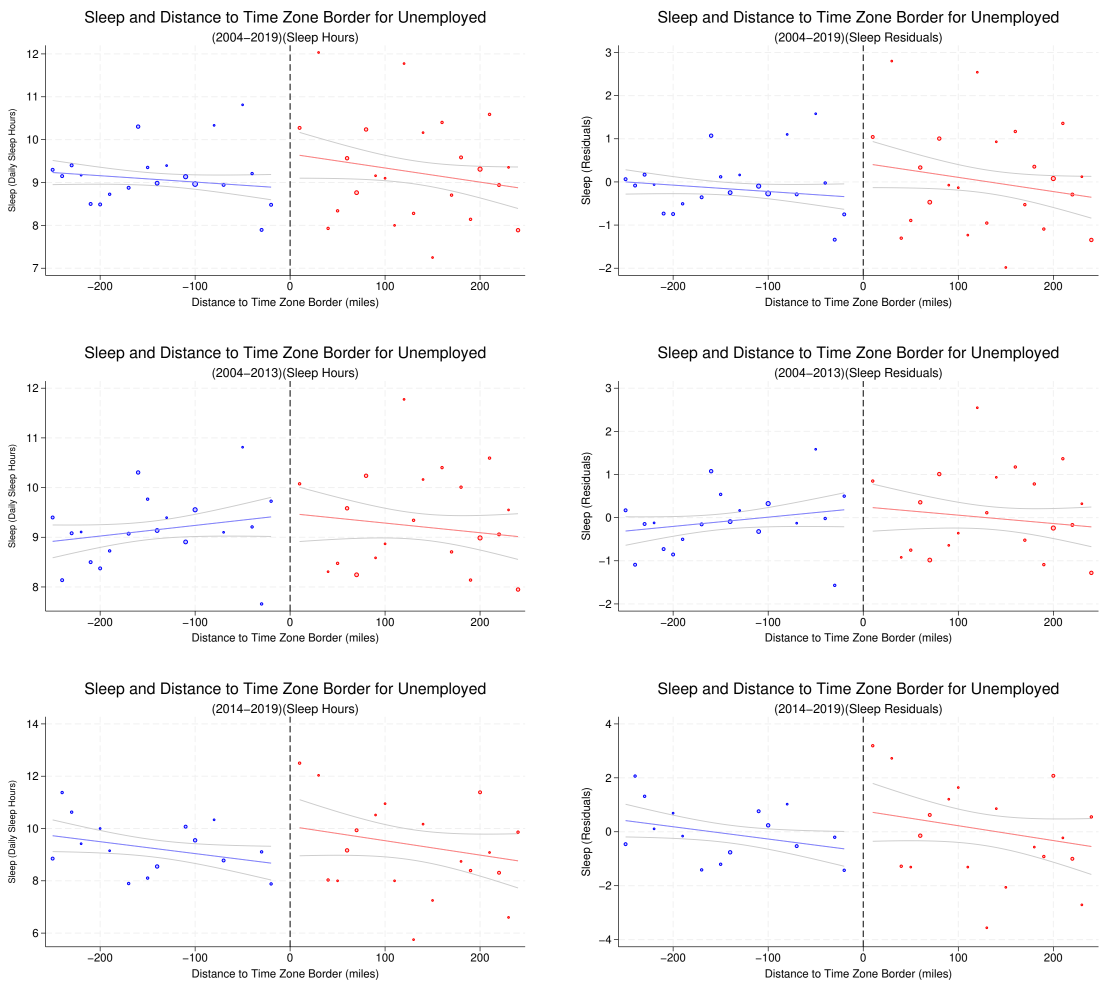


Figure 1.13: Sleep and Distance to Time Zone Border for Unemployed

This figure show the discontinuity in sleep and distance to time zone border for unemployed individuals. Data are from ATUS (2004-2019). Each point represents the mean daily sleep hour on the left panel and mean residuals (10 miles average) of sleep on a set of geographical controls (a linear control for latitude and dummy for large counties) on the right panel. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

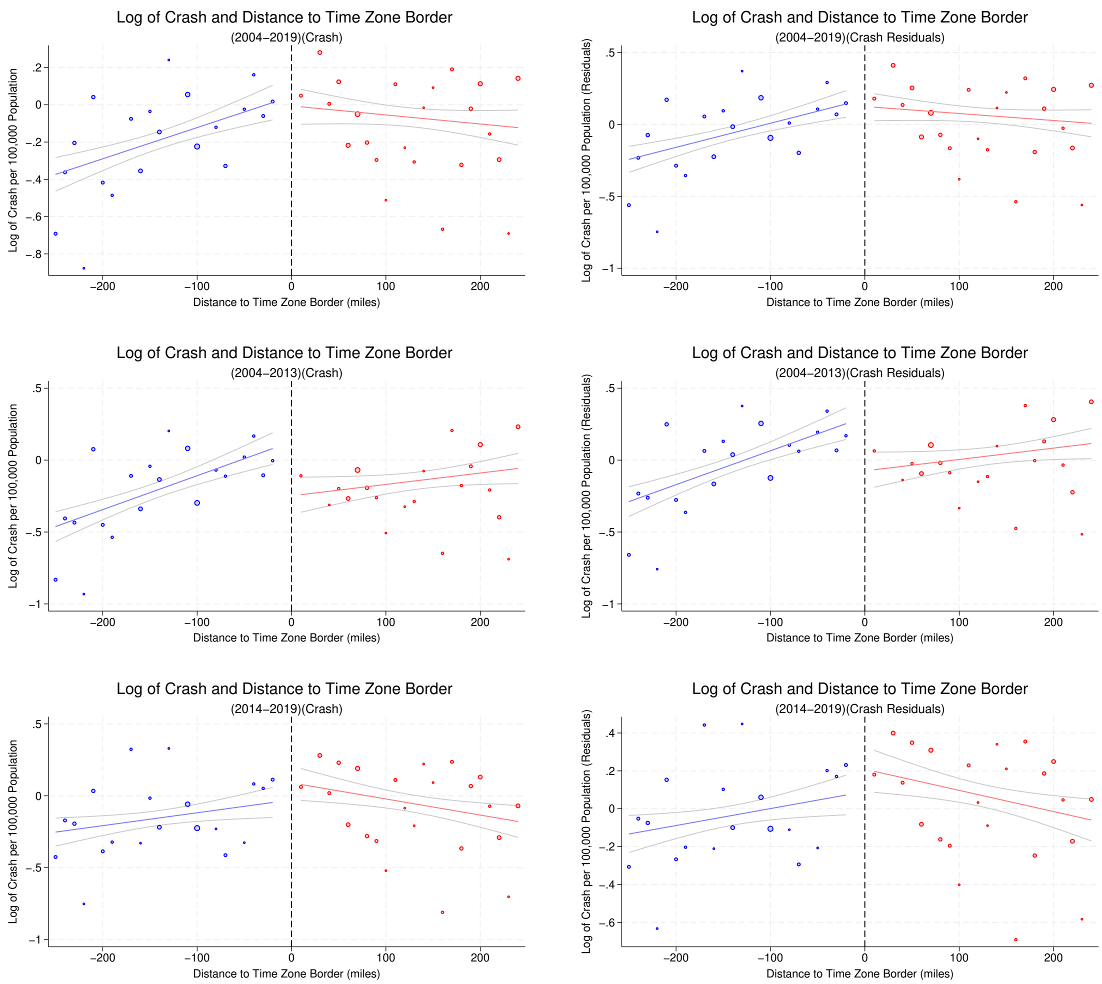


Figure 1.14: Crash and Distance to Time Zone Border

This figure show the discontinuity in crash and distance to time zone border. Data are from ATUS (2004-2019). Each point represents the mean crash per 100,000 population on left panel and mean residuals (10 miles average) of the crash per 100,000 population on a set of geographical controls (a linear control for latitude and dummy for large counties) on the right panel. The scatterplot is weighted by the number of observations in distance group. The distance group is calculated using the cut command in Stata.

Table 1.6: Effects of Late Sunset Side on Sleep for Employed (2004-2019)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Sleep	Sleep	Sleep	Sleep	Sleep	Sleep	Sleep $\geq 8hrs$
	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Late Sunset Side=1	-0.158	0.035	-0.241*	0.119	0.354**	-0.009	-0.009
	(0.12)	(0.11)	(0.14)	(0.17)	(0.16)	(0.18)	(0.03)
Mean	8.59	8.59	8.59	8.59	8.59	8.59	8.59
Controls	No	Yes	Yes	No	Yes	Yes	Yes
County FEs	No	No	Yes	No	No	Yes	No
Bandwidth (miles)	250	250	250	100	100	100	250
Observations	27542	27542	27542	7172	7172	7172	27542

Notes: Data are from ATUS (2004-2019). Estimates include the distance to the time-zone boundary and the interaction with the late sunset border, socio-demographics (age, race, sex, education, marital status, nativity status, and number of children), geographic characteristics (latitude, longitude, and indicator for large counties), and interview characteristics (indicators for holiday and weekend). The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.7: Effects of Late Sunset Side on Sleep for Employed (2004-2013)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Sleep	Sleep	Sleep	Sleep	Sleep	Sleep	Sleep $\geq 8hrs$
	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Late Sunset Side=1	-0.443***	-0.273***	-0.229	-0.363*	-0.066	-0.356**	-0.077**
	(0.10)	(0.10)	(0.15)	(0.19)	(0.10)	(0.17)	(0.03)
Mean	8.49	8.49	8.49	8.53	8.53	8.53	0.61
Controls	No	Yes	Yes	No	Yes	Yes	Yes
State FEs	No	No	Yes	No	No	Yes	No
Bandwidth (miles)	250	250	250	100	100	100	250
Observations	8305	8305	8305	2598	2598	2598	8305

Notes: Data are from ATUS (2004-2019). The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.8: Effects of Late Sunset Side on Sleep for Employed (2014-2019)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Sleep	Sleep	Sleep	Sleep	Sleep	Sleep	Sleep $\geq 8hrs$
	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Late Sunset Side=1	0.385**	0.657***	0.023	0.405*	0.597***	0.404	0.090**
	(0.19)	(0.14)	(0.19)	(0.21)	(0.19)	(0.26)	(0.04)
Mean	8.63	8.63	8.63	8.64	8.64	8.64	0.65
Controls	No	Yes	Yes	No	Yes	Yes	Yes
State FEs	No	No	Yes	No	No	Yes	No
Bandwidth (miles)	250	250	250	100	100	100	250
Observations	3605	3605	3605	1108	1108	1108	3605

Notes: Data are from ATUS (2004-2019). The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.9: Effects of Late Sunset Side on Log of Crashes for Employed (2004-2019)

	(1)	(2)	(3)	(4)	(5)	(6)
	Crashes	Crashes	Crashes	Crashes	Crashes	Crashes
	b/se	b/se	b/se	b/se	b/se	b/se
Late Sunset Side=1	-0.198	-0.347**	-0.260*	0.246	0.185	-0.033
	(0.18)	(0.14)	(0.16)	(0.21)	(0.14)	(0.26)
Mean	8.59	8.59	8.59	8.59	8.59	8.59
Controls	No	Yes	Yes	No	Yes	Yes
County FEs	No	No	Yes	No	No	Yes
Bandwidth (miles)	250	250	250	100	100	100
Observations	27542	27542	27542	7172	7172	7172

Notes: Data are from FARS and ATUS (2004-2019). The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.10: Effects of Late Sunset Side on Log of Crashes for Employed (2004-2013)

	(1)	(2)	(3)	(4)	(5)	(6)
	Crashes	Crashes	Crashes	Crashes	Crashes	Crashes
	b/se	b/se	b/se	b/se	b/se	b/se
Late Sunset Side=1	-0.259	-0.384**	-0.220	0.249	0.233	0.033
	(0.21)	(0.18)	(0.18)	(0.19)	(0.17)	(0.27)
Mean	8.55	8.55	8.55	8.55	8.55	8.55
Controls	No	Yes	Yes	No	Yes	Yes
County FEs	No	No	Yes	No	No	Yes
Bandwidth (miles)	250	250	250	100	100	100
Observations	20027	20027	20027	5160	5160	5160

Notes: Data are from FARS and ATUS (2004-2013). The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.11: Effects of Late Sunset Side on Log of Crashes for Employed (2014-2019)

	(1)	(2)	(3)	(4)	(5)	(6)
	Crashes	Crashes	Crashes	Crashes	Crashes	Crashes
	b/se	b/se	b/se	b/se	b/se	b/se
Late Sunset Side=1	-0.083	-0.270**	-0.319*	0.184	0.009	-0.556**
	(0.19)	(0.13)	(0.17)	(0.29)	(0.15)	(0.25)
Mean	8.69	8.69	8.69	8.69	8.69	8.69
Controls	No	Yes	Yes	No	Yes	Yes
County FEs	No	No	Yes	No	No	Yes
Bandwidth (miles)	250	250	250	100	100	100
Observations	7515	7515	7515	2012	2012	2012

Notes: Data are from FARS and ATUS (2014-2019). The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01.

1.7 Robustness Check

Confounding factors such as weather, road conditions, and ambient light may be correlated with both the sunset hour and crashes, so I control for seasonality by adding time fixed effects. For instance, the road could be icy in the northern regions during winters, which poses a higher risk of fatal vehicle crashes. Table 1.12 includes year, year-month, and county-month fixed effects, and the results all show similar estimates as Table 1.2. The impact of sleep on the log of crashes is similar to the original estimates after including controls for seasonality, which indicates that additional sleep has a short-run negative impact on fatalities. For example, columns (2) to (5) imply that one extra hour of monthly sleep causes a 2.4% reduction in the log of fatal crashes per 100,000 population.

Road type could affect fatal vehicle crashes since the speed is different on various roads. Table 1.13 illustrates the short-run effects of sleep on fatal vehicle crashes by types of roads, such as highways, county roads, and local streets. The road type is available in FARS since 1987, and I categorize the road as a highway if it is an interstate, U.S. highway, or state highway. In addition, I classify the road as a local street if it is a township, municipality, or frontage road. The results show that the impact of sleep on fatalities is mostly driven by the fatalities on highways. Specifically, column (2) indicates that one additional hour of monthly sleep results in a 1.9% reduction in fatalities on highways.

Light could play a crucial role in fatal vehicle crashes as more ambient light could create a safer driving environment. If the sunset is late by one hour, then additional light during the evening should reduce the risk of crashes. Similarly, decreasing light by one hour in the morning would increase the risk of fatalities. To test this hypothesis, I will decompose the crashes into morning crashes (two hours more or less from the local average sunrise time), evening crashes (two hours more or less from the local average sunset time), least light-impacted daytime crashes (the remaining hours), and nighttime crashes following a similar setup from Smith (2016).

Table 1.14 provides the short-run effect of sleep on fatalities by light condition. Columns (2) and (3) suggest that sleep has a statistically insignificant impact on crashes during the morning and evening. Column (4) suggests that increasing monthly sleep by one hour could lead to about a 2.1% reduction in fatal crashes per 100,000 population. The result indicates that the impact of sleep on fatalities is mostly driven by the impact on daytime crashes.

Table 1.12: Short Run Effects of Sleep on Log of Crashes (Seasonality)

	(1) Crashes b/se	(2) Crashes b/se	(3) Crashes b/se	(4) Crashes b/se	(5) Crashes b/se
Average Monthly Sleep	-0.044***	-0.024***	-0.025***	-0.023***	-0.024***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Mean	-0.41	-0.41	-0.41	-0.41	-0.41
Controls	No	Yes	Yes	Yes	Yes
County FEs	No	Yes	Yes	Yes	Yes
Year FE	No	No	Yes	No	No
Year-Month FE	No	No	No	Yes	No
County-Month FE	No	No	No	No	Yes
Observations	36296	36296	36296	36296	36296

Note: Sleep and sunset time are measured in hours at county-year-month level. The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01

Table 1.13: Short Run Effects of Sleep on Log of Crashes by Types of Roads

	(1) Crashes (All Roads)	(2) Crashes (Highway)	(3) Crashes (County Road)	(4) Crashes (Local Street)
	b/se	b/se	b/se	b/se
Average Monthly Sleep	-0.024***	-0.019**	-0.006	-0.002
	(0.01)	(0.01)	(0.00)	(0.01)
Mean	-0.41	-0.92	-1.33	-1.27
Controls	Yes	Yes	Yes	Yes
County FEs	Yes	Yes	Yes	Yes
Observations	36296	30623	16172	23986

Notes: Sleep is measured in hours at county-year-month level. The dependent variable of sleep is monthly average sleep hours. The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). Significance levels: * 0.10, ** 0.05, *** 0.01.

Table 1.14: Short Run Effects of Sleep on Log of Crashes by Light Condition

	(1) Crashes (All Hours)	(2) Crashes (Morning)	(3) Crashes (Evening)	(4) Crashes (Daytime)	(5) Crashes (Nighttime)
	b/se	b/se	b/se	b/se	b/se
Average Monthly Sleep	-0.024***	0.025	0.155	-0.021***	-0.000***
	(0.01)	(0.02)	(0.12)	(0.01)	(0.00)
Mean	-0.41	-1.71	-1.43	-1.28	-1.88
Controls	Yes	Yes	Yes	Yes	Yes
County FEs	Yes	Yes	Yes	Yes	Yes
Observations	36296	18075	23521	27022	28100

Notes: Sleep is measured in hours at county-year-month level. The dependent variable of sleep is monthly average sleep hours. The standard errors are robust to heteroscedasticity and clustered at state-county level (reported in parentheses). "Morning" is defined as +/- two hours from the average sunrise time in that county. "Evening" is defined as +/- two hours from the average sunset time in that county. Significance levels: * 0.10, ** 0.05, *** 0.01.

1.8 Conclusion

Sleep deprivation has negative effects on alertness and attention, increasing the risk of fatal vehicle crashes. While medical studies have linked sleep deprivation to adverse health outcomes, there is limited understanding of its impact in real-world situations. This paper investigates the causal impact of sleep on fatal vehicle crashes in the US using IV and RDD methods, and data from the ATUS and FARS.

By employing a seasonal, short-run IV approach, the study reveals that a one-hour delay in sunset results in a decrease of approximately 12 minutes in weekly sleep duration. Furthermore, a one-hour increase in monthly sleep is associated with a 2.4% reduction in fatalities.

The RDD analysis shows that employed individuals on the later sunset side of the time zone border slept less from 2004 to 2013, but slept more from 2014 to 2019. The second-stage results of the RDD found no consistent, statistically significant effects of sunsets on sleep and fatal vehicle crashes across different time periods and bandwidths.

This paper could inform policy solutions regarding Daylight Saving Time (DST) and clock changes. It also provides insights into designing schedules that prioritize healthy sleep patterns for overall well-being and productivity. The findings highlight the importance of adequate sleep for health and safety, and suggest potential policy implications for optimizing work and school schedules. Future research could explore sleep's impact on other health and productivity outcomes.

Chapter 2

Employment and Sleep Patterns

2.1 Abstract

This paper examines how employment rates affect sleep patterns, using data from the American Time Use Survey (ATUS) and Local Area Unemployment Statistics (LAUS) from 2003 to 2022. Research suggests that while weekday sleep is countercyclical, weekend sleep is procyclical, with employed individuals sleeping more on weekends to compensate for shorter weekday sleep. The results show that a 1 percentage point increase in the employment-to-population rate reduces average sleep duration by approximately 1 minute and weekday sleep by around 2 minutes. This effect is less pronounced when using the data from 2003-2022, likely due to the increase in telework following the COVID-19 pandemic. Additionally, industries with a higher concentration of telework experience smaller decreases in sleep with increasing employment. Heterogeneous analysis shows that minorities, less educated individuals, women, and single adults face greater reductions in sleep during weekdays.

2.2 Introduction

Sleep is essential for maintaining good health and productivity, and a lack of sleep could incur substantial health and economic costs. According to a report by the Centers for Disease Control and Prevention (CDC), sleeping fewer than 7 hours per night can increase the risk of developing conditions such as high blood pressure, heart disease, stroke, diabetes, obesity, and frequent mental distress (Liu, 2016).

Besides the health costs, insufficient sleep can lead to increased mortality and decreased productivity. Sleep deprivation is associated with accidents and injuries caused by fatigue (Dinges, 1995; Lockley et al., 2007; Barnes and Wagner, 2009). It affects attention, cognitive skills, coordination, motor functions, and processing speed (Dinges and Powell, 1985; Drummond et al., 2005; Banks and Dinges, 2007; Lim and Dinges, 2010). Additionally, it impacts productivity and psychological well-being (Bessone et al., 2021).

The business cycle has a significant impact on sleep patterns. According to a study by Colman and Dave (2013), sleep duration tends to be countercyclical. During the Great Recession, people spend more time sleeping and television watching (Aguiar et al., 2013). By analyzing data on sleep duration and unemployment rates, Figure 2.1 illustrates a positive correlation between unemployment rates and the amount of sleep. The unemployment data is seasonally adjusted, and sleep durations are smoothed using a 12-month moving average. Both sets of variables have been adjusted to remove any linear trends and normalized by subtracting the mean from the detrended data and then dividing by the standard deviation. Figure 2.2 indicates the countercyclical nature of sleep applies to both employed and not-employed individuals.

Individuals display different work hours and sleep patterns on weekdays versus weekends. According to Figure 2.3, people generally work about 5.6 hours more during the weekdays. Meanwhile, Figure 2.4 shows that employed individuals usually sleep an extra 1.25 hours on weekends than on weekdays. The relationship between sleep and economic conditions also varies by the day of the week. Niekamp (2019) found that while sleep duration on weekdays is countercyclical, it tends to be procyclical during weekends

This paper investigates the relationship between employment rates and sleep patterns by utilizing data from the American Time Use Survey (ATUS) and Local Area Unemployment Statistics (LAUS) covering the period from 2003 to 2022. In line with the research conducted by Niekamp (2019) on the economic impact, I replicate his analysis using data from 2003 to 2022. Moreover, I expand the analysis to encompass data up to 2022, allowing for an examination of the effects of the COVID-19 Recession, commonly referred to as the Great Lockdown. Additionally, this study incorporates an analysis of the telework component to provide a comprehensive understanding of the topic.

The analysis conducted using a standard linear regression model reveals interesting patterns in sleep behavior. Specifically, it shows that weekday sleep follows a countercyclical trend, while weekend sleep exhibits a procyclical pattern, although the latter is not statistically significant. The results indicate that a 1 percentage point increase in the employment-to-population rate leads to a reduction in average sleep duration by approximately 1 minute. Moreover, weekday sleep experiences a more substantial decrease of around 2 minutes. However, it is worth noting that the impact of employment on sleep duration appears to be smaller when considering data from 2003-2015. This could be attributed to the rise in teleworking practices following the COVID-19 pandemic. Industries with higher rates of telework also demonstrate smaller reductions in sleep as employment levels increase.

Furthermore, a heterogeneous analysis reveals that certain demographic groups experience more significant reductions in weekday sleep. Specifically, minorities, individuals with lower education levels, women, and single individuals are particularly affected.

The following sections of this paper will provide a detailed overview of the topic. Section 2 will review the existing literature on sleep and economic condition studies. In Section 3, the data used in this paper will be described. Section 4 will outline the empirical methods employed in the study. The main results will be presented in Section 5, while Section 6 will conclude the paper and discuss potential avenues for future research.

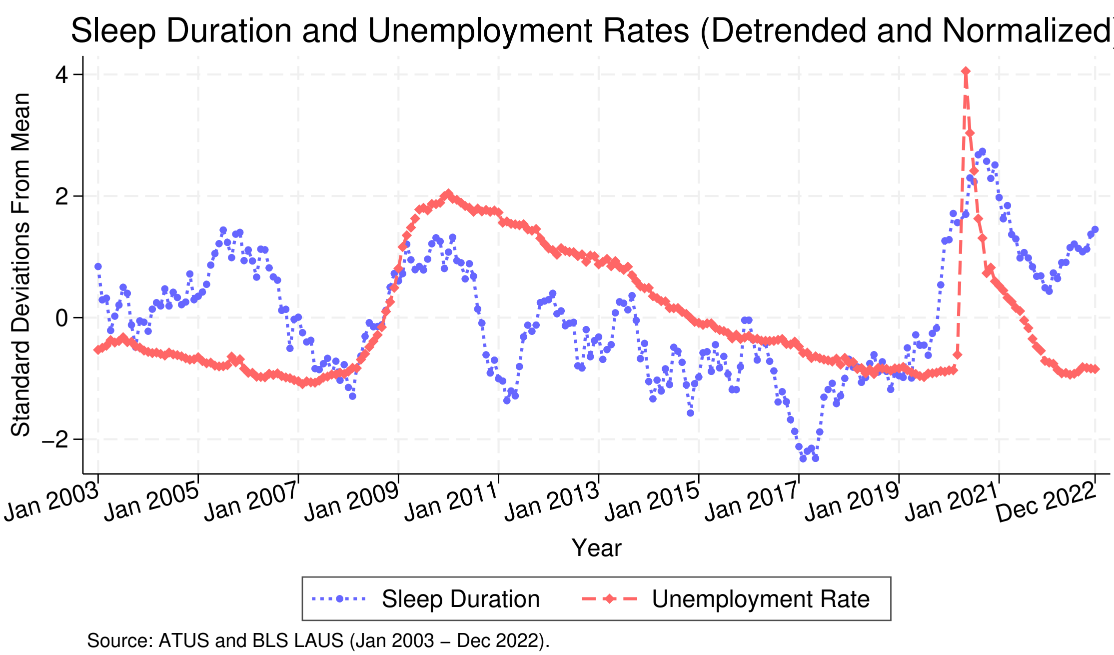


Figure 2.1: Sleep Duration and Unemployment Rates (Detrended and Normalized)

Notes: Unemployment rates are seasonally adjusted and sleep durations are smoothed by applying a moving average. The variables have been detrended by removing a linear trend and normalized by subtracting the mean of the detrended variables and dividing by their standard deviation. The figure shows a positive relationship between unemployment rates and sleep duration.

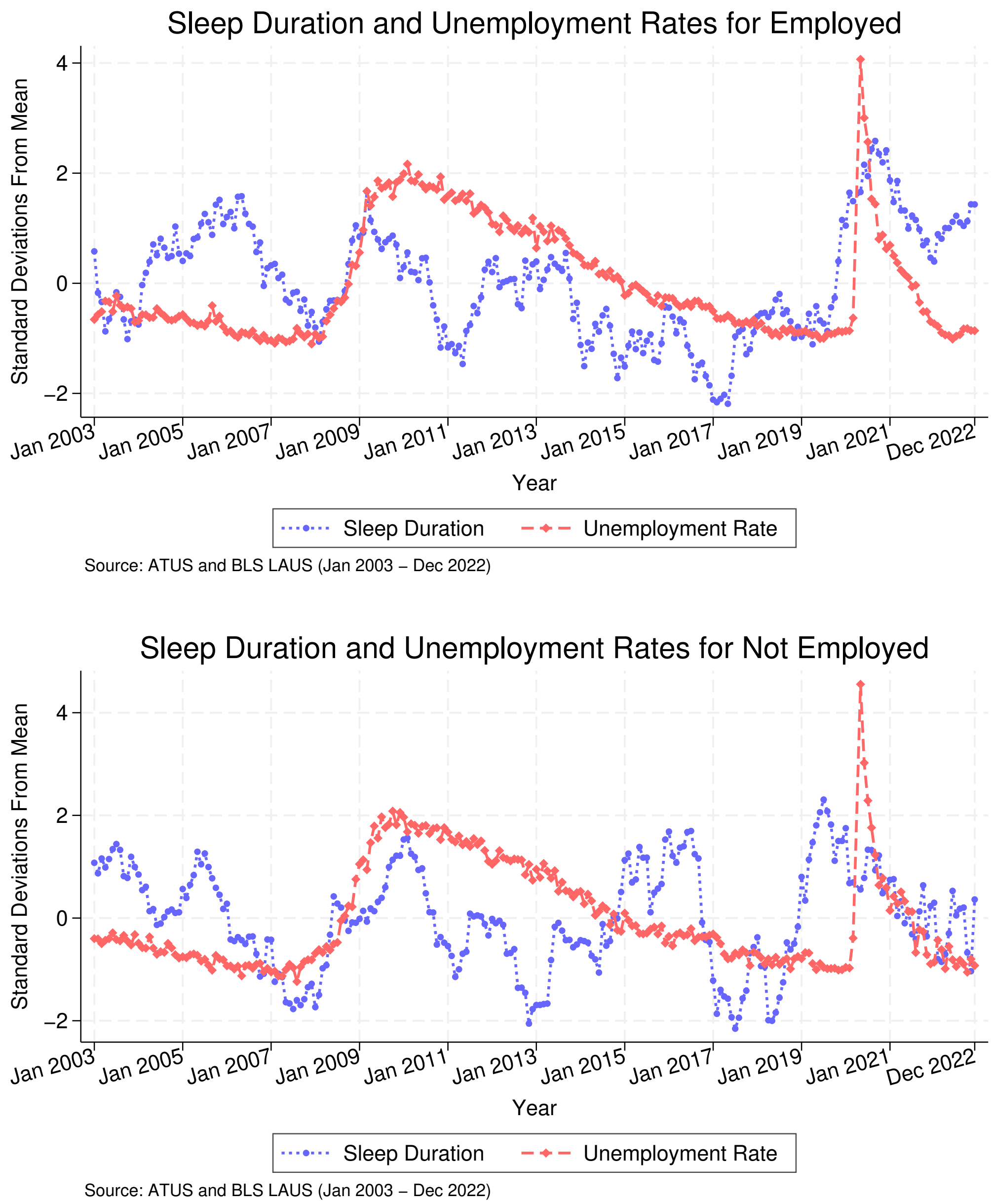


Figure 2.2: Sleep Duration and Unemployment Rates (Employed v.s. Not Employed)

Notes: Unemployment rates are seasonally adjusted and sleep durations are smoothed by applying a moving average. The variables have been detrended by removing a linear trend and normalized by subtracting the mean of the detrended variables and dividing by their standard deviation. The figure shows a positive relationship between unemployment rates and sleep duration. Not employed refers to those who are unemployed and not in the labor force.

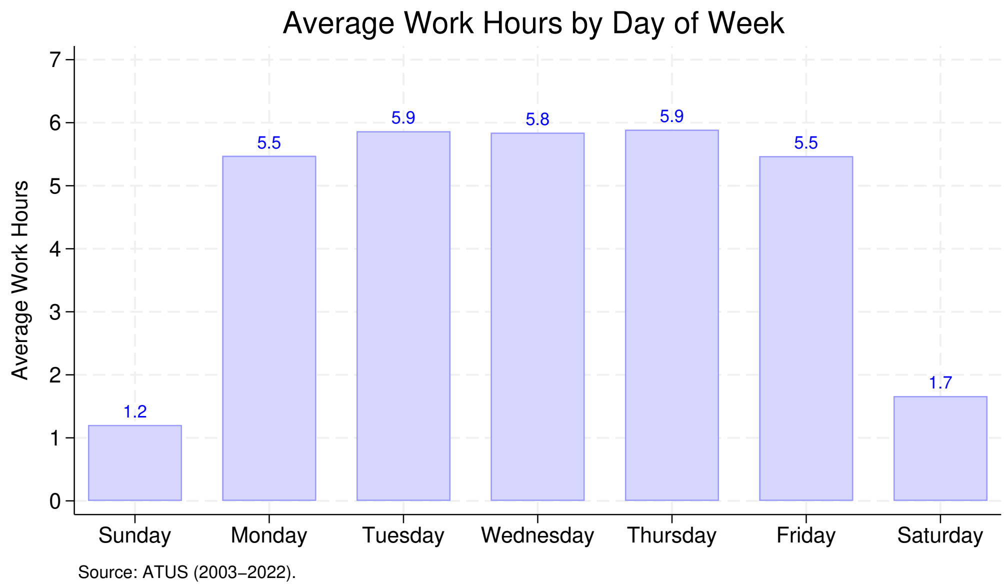


Figure 2.3: Average Work Hours by Day of Week

Notes: The sample averages only include data from individuals aged 25 to 55 who have reported at least 23 hours of time use.

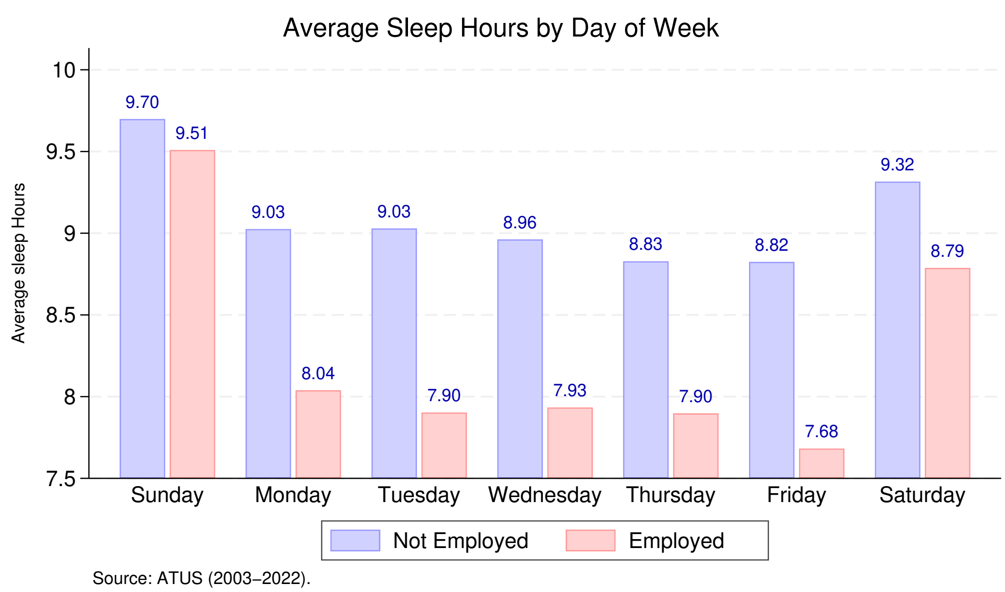


Figure 2.4: Average Sleep Hours by Day of Week

Notes: The sample averages only include data from individuals aged 25 to 55 who have reported at least 23 hours of time use. Not employed refers to those who are unemployed and not in the labor force.

2.3 Literature Review

Sleep deprivation can negatively impact both mental and physical health. Medical studies have demonstrated that lack of sleep negatively impacts attention, memory, and mood (Banks and Dinges, 2007). Additionally, the Centers for Disease Control and Prevention (CDC) report that sleeping less than 7 hours per night increases the risk of developing serious health issues such as high blood pressure, heart disease, stroke, diabetes, obesity, and frequent mental distress (Liu, 2016).

Medical research has long indicated the risks of sleep deprivation, but only recently have economists begun to empirically analyze its effects. Studies have shown varying impacts of sleep on different aspects of life (Kamstra et al., 2000). For instance, Smith (2016) found a 5.6% increase in fatal crashes following the spring transition to Daylight Saving Time (DST). Similarly, Gibson and Shrader (2018) demonstrated that an extra hour of weekly sleep could increase short-term wages by 1.1% and long-term wages by 5%.

Further research includes a field experiment in Chennai, India, by Bessone et al. (2021), which increased sleep duration by 27 minutes nightly but did not significantly affect cognition, productivity, or well-being—though afternoon naps did improve these factors. Jin and Ziebarth (2020) noted a decrease in hospitalization rates following the fall transition of DST, an effect lasting four days. Additionally, studies by Giuntella et al. (2017) and Giuntella and Mazzonna (2019) found that later sunset times reduced sleep duration and were linked to several negative health outcomes, emphasizing the profound impact of sleep on health and economic variables.

This paper contributes to the literature on how economic conditions influence health outcomes, aligning with established literature that typically views recessions as beneficial for health. For instance, Ruhm (2000) noted that mortality rates fluctuate procyclically, while his later work observed health-improving behaviors such as reduced smoking and increased physical activity during economic downturns (Ruhm, 2005). Similarly, Miller et al. (2009) found that higher unemployment rates correlate with lower state-level mortality rates, and Stevens et al. (2015) found that most additional deaths that occur when the economy is strong are among the elderly, particularly elderly women and those residing in nursing homes.

Additionally, Charles and DeCicca (2008) found that deteriorating labor market conditions result in weight gain and diminished mental health among African-American men, as well as reduced mental health among less-educated males. Similarly, Colman and Dave (2013) observed that during a recession, decreased work hours lead to increased recreational exercise, TV-watching, sleeping, childcare, and housework, with the most pronounced effects seen among low-educated men.

Building on these foundations, my research draws a close parallel with the findings of Niekamp (2019), who investigated the cyclical nature of sleep patterns across different days of the week, noting that sleep duration on weekdays is countercyclical but procyclical on weekends. My study replicates Niekamp's analysis using data spanning from 2003 to 2015 and extends it to include analysis up to 2022 to examine the impacts of the COVID-19 Recession. This extension provides a comprehensive view of how recent economic disruptions, coupled with the rise in teleworking, have altered daily routines and health behaviors.

This paper also explores the impact of the COVID-19 pandemic on the labor market, building on previous studies that have documented shifts in work habits. Before the pandemic, Pabilonia and Vernon (2020) documented a rising trend in remote work in the United States, observing that teleworkers spent less time on commuting and grooming while allocating more time to leisure, sleep, household production, and family activities on work-from-home days. The pandemic significantly accelerated this shift, as Bick et al. (2023) reported a persistent rise in work from home (WFH), increasing from 14.4 percent of workdays in February 2020 to 39.6 percent in May 2020. They predict a permanent change post-pandemic, with twice as many workers expected to WFH full-time.

Similarly, Barrero et al. (2021) found that 20 percent of full workdays will likely be conducted from home after the pandemic, suggesting a lasting transformation in the labor market dynamics. My research contributes to this literature by documenting how economic conditions differently affect sleep patterns across industries, with a particular focus on those that have a high prevalence of telework.

In summary, while existing research provides substantial insights into the effects of sleep deprivation and economic conditions on health and labor market dynamics, there remains a need for a nuanced analysis of how these elements interact across different industries, especially in the context of increased telework. This paper fills this gap by leveraging recent data to assess the impacts of economic disruptions on sleep patterns, particularly during the COVID-19 recession. By focusing on industries with high rates of telework, this study contributes to a more detailed understanding of the relationship between economic conditions and health outcomes in the modern labor market.

2.4 Data

The individual sleep duration comes from the American Time Use Survey (ATUS) sponsored by the U.S. Bureau of Labor Statistics (BLS) and conducted by the U.S. Census Bureau since 2003. ATUS is the first continuous survey on time use in the United States. Individuals are randomly selected from the households that just finished the eight-month interview for the Current Population Survey (CPS) and the interviews for ATUS are conducted between two and five months after the last CPS interview. The goal of ATUS is to understand how people allocate their time.

The time diary of the ATUS is conducted through computer-assisted telephone interviews. The respondent is asked to recall the time spend in each activity from 4:00 am on the previous day to 4:00am on the interview day. This method allows the time diaries to be summed to 24 hours to minimize possible biases. For each activity, the ATUS gathers either the ending time or the duration of the activity and the interviewer collects the answers verbatim, which are coded later (Hamermesh et al., 2005).

Following Niekamp (2019) and Colman and Dave (2013), I limit the analysis for individuals with age between 25 to 55 and also restrict the analysis to observations with at least 23 documented hours (92% of observations). The employment data (unemployment rates and employment to population ratio) is from the Local Area Unemployment Statistics (LAUS) monitored by BLS. I exploit the variation of economic condition at the month-state level.

Table 2.1 shows the summary statistics.

Table 2.1: Summary Statistics

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	All	Female	Male	Employed	Not Employed	$<$ Bachelor's	$\geq$ Bachelor's
	mean/sd	mean/sd	mean/sd	mean/sd	mean/sd	mean/sd	mean/sd
Sleep Time (Hours)	8.66	8.76	8.53	8.52	9.23	8.45	8.79
	(2.23)	(2.21)	(2.25)	(2.15)	(2.49)	(1.94)	(2.40)
Age	40.42	40.19	40.68	40.38	40.59	40.21	40.55
	(8.49)	(8.55)	(8.41)	(8.39)	(8.89)	(8.15)	(8.71)
Married	0.60	0.59	0.62	0.61	0.59	0.67	0.56
	(0.49)	(0.49)	(0.49)	(0.49)	(0.49)	(0.47)	(0.50)
Number of Children	1.17	1.24	1.08	1.13	1.33	1.16	1.18
	(1.19)	(1.19)	(1.19)	(1.15)	(1.32)	(1.14)	(1.23)
Children Under 3	0.16	0.16	0.15	0.15	0.20	0.18	0.14
	(0.36)	(0.37)	(0.36)	(0.35)	(0.40)	(0.38)	(0.35)
White	0.66	0.65	0.68	0.68	0.58	0.74	0.61
	(0.47)	(0.48)	(0.47)	(0.47)	(0.49)	(0.44)	(0.49)
Black	0.13	0.14	0.11	0.12	0.16	0.09	0.15
	(0.33)	(0.35)	(0.31)	(0.32)	(0.37)	(0.28)	(0.36)
Hispanic	0.15	0.15	0.15	0.14	0.19	0.08	0.20
	(0.36)	(0.36)	(0.36)	(0.35)	(0.40)	(0.27)	(0.40)
American Indian	0.01	0.01	0.01	0.01	0.01	0.00	0.01
	(0.09)	(0.09)	(0.09)	(0.08)	(0.11)	(0.06)	(0.10)
Holiday	0.02	0.02	0.02	0.02	0.02	0.02	0.02
	(0.13)	(0.13)	(0.13)	(0.13)	(0.13)	(0.13)	(0.13)
Incomplete Diary	0.08	0.09	0.07	0.08	0.08	0.09	0.07
	(0.27)	(0.28)	(0.25)	(0.27)	(0.27)	(0.29)	(0.26)
N	120743	65868	54875	97713	23030	48733	72010

Notes: Data are from ATUS and LAUS (2003-2022). The sample is restricted to respondents aged 25-55.

2.5 Methodology

To estimate the impact of employment on sleep duration, I employ a standard linear regression model:

$$Sleep_{isdmt} = \beta_0 + \beta_1 E_{smt} + X'_{ismt}\beta_2 + \gamma_s + \delta_m + \lambda_d + \theta_t + u_{isdmt} \quad (2.1)$$

where $Sleep_{isdmt}$ represents the daily sleep duration in minutes for individual i in state s on day of week d in month m in year t. The variable X_{ismt} is a vector of control variables that includes socio-demographic factors (age, gender, race, education, race interacted with education, marital status, number of children, an indicator for having a child under 3, and industry codes) and interview characteristics (indicators for holiday and incomplete diary). E_{smt} denotes the civilian employment-population ratio for state s in month m of year t.

The state fixed effects, γ_s, account for the time-invariant unobserved heterogeneity of state-specific factors. The month fixed effects are represented by δ_m, the day of the week fixed effects by λ_d, and the year fixed effects by θ_t. The error term, u_{isdmt}, has standard errors clustered at the state level. This empirical approach aligns with the extensive literature on economic conditions and health outcomes (Niekamp, 2019; Charles and DeCicca, 2008; Ruhm, 2000, 2005).

2.6 Results

The impact of the employment-to-population ratio on sleep duration from 2003 to 2022 are illustrated in Table 2.2. According to Column 2, a one percentage point increase in the employment rate reduces sleep by approximately one minute per night. The point estimate of -1.02 (using data from 2003-2022) aligns closely with the findings of -1.1 from Niekamp (2019) and -0.97 from Colman and Dave (2013). This consistency supports the conclusion of prior research that overall sleep is counter-cyclical. The effects vary between weekdays and weekends. Columns 4 and 6 report that a one percentage point increase in the employment rate decreases weekday sleep by 2.3 minutes per night, while it increases weekend sleep by 0.26 minutes per night.

The effects of employment can vary significantly across different demographics. Table 2.3 presents the results segmented by education, race, and gender. Columns 2 and 3 indicate that the effects of employment on sleep are greater and statistically significant for individuals without a Bachelor's degree. Columns 4 and 5 show that the impact is more pronounced among minorities, including Blacks, Hispanics, and American Indians. Columns 6 and 7 suggest that females experience a higher impact compared to males. Column 8 reveals that white males without a Bachelor's degree are particularly sensitive to changes in the employment rate, especially during weekdays. Table 2.4 replicates the analysis using data from 2003 to 2015, as done by Table Niekamp (2019). The results are similar, with Column 1 showing slightly higher estimates (using data from 2003-2015). The weekday effects are relatively greater, while the weekend effects are smaller and not statistically significant.

Marital status can also affect the estimates in different ways. Table 2.5 presents the effects by marital status. Columns 1 and 2 reveal that single individuals experience a greater impact on weekday sleep. Columns 3 and 4 indicate that the employment effects are more pronounced for single parents, who tend to be less educated and belong to minority groups, making them more strongly affected. Additionally, single parents sleep less during weekends when employment rates increase. Columns 5 and 6 show that the sleep or work of low-educated females is not sensitive to economic conditions. Table 2.6 shows the results for the period from 2003 to 2015. The estimates are similar to those found by Table Niekamp (2019), except that married parents experience a stronger impact.

Table 2.2: Effects of Employment Rate on Sleep (2003-2022)

	All		Weekday		Weekend	
	(1)	(2)	(3)	(4)	(5)	(6)
	Sleep	Sleep	Sleep	Sleep	Sleep	Sleep
	b/se	b/se	b/se	b/se	b/se	b/se
Employment to Population Rate	-1.375***	-1.019***	-1.461***	-2.308***	-1.333***	0.259
	(0.22)	(0.35)	(0.23)	(0.44)	(0.25)	(0.53)
Mean	519	519	519	519	519	519
Controls	No	Yes	No	Yes	No	Yes
State FEs	No	Yes	No	Yes	No	Yes
Observations	120743	120743	60250	60250	60493	60493

Notes: Data are from ATUS and BLS LAUS (2003-2022). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Controls include socio-demographics (age, gender, race, education, race interacted with education, marital status, number of children, indicator for having a child under 3, and industry codes) and interview characteristics (indicators for holiday and incomplete diary). The standard errors are robust to heteroscedasticity and clustered at state level (reported in parentheses).

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.3: Effects of Employment Rate on Sleep by Subgroups (2003-2022)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	All	< Bachelor's	≥ Bachelor's	White	BHAI	Female	Male	WM < Bachelor's
All	-1.019**	-1.155*	-0.965	-0.529	-1.777*	-1.183*	-0.822	-1.113
	(0.352)	(0.541)	(0.515)	(0.507)	(0.828)	(0.564)	(0.468)	(0.809)
Observations	120743	72010	48733	80057	33617	65868	54875	20985
R^2	0.11	0.10	0.13	0.11	0.10	0.11	0.12	0.10
Weekday	-2.308***	-2.900***	-1.699*	-1.496*	-4.455***	-2.753***	-1.824**	-2.785*
	(0.441)	(0.625)	(0.675)	(0.639)	(1.136)	(0.699)	(0.595)	(1.249)
Observations	60250	35690	24560	40287	16411	32706	27544	10625
R^2	0.07	0.07	0.05	0.06	0.07	0.07	0.07	0.07
Weekend	0.259	0.512	-0.201	0.461	0.658	0.353	0.128	0.471
	(0.532)	(0.825)	(0.628)	(0.715)	(1.153)	(0.712)	(0.766)	(1.516)
Observations	60493	36320	24173	39770	17206	33162	27331	10360
R^2	0.05	0.05	0.06	0.05	0.05	0.06	0.06	0.05

Notes: Data are from ATUS and BLS LAUS (2003-2022). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Controls include socio-demographics (age, gender, race, education, race interacted with education, marital status, number of children, indicator for having a child under 3, and industry codes) and interview characteristics (indicators for holiday and incomplete diary). The standard errors are robust to heteroscedasticity and clustered at state level (reported in parentheses). Column 5 refers to (BAHI) Black, Hispanic, or American Indian. Column 8 restricts to white males with education less than a Bachelor's degree.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.4: Effects of Employment Rate on Sleep by Subgroups (2003-2015)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	All	< Bachelor's	≥ Bachelor's	White	BHAI	Female	Male	WM < Bachelor's
All	-1.133*	-1.234*	-1.051	-0.710	-1.855*	-1.196	-1.058	-1.631
	(0.444)	(0.589)	(0.712)	(0.596)	(0.918)	(0.681)	(0.703)	(0.846)
Observations	91184	56659	34525	61415	24970	50314	40870	16524
R^2	0.11	0.10	0.13	0.11	0.10	0.11	0.11	0.10
Weekday	-2.875***	-3.644***	-1.709	-2.245**	-4.232***	-3.155***	-2.553**	-4.731**
	(0.512)	(0.703)	(1.063)	(0.782)	(1.203)	(0.870)	(0.932)	(1.626)
Observations	45346	28025	17321	30812	12140	24893	20453	8360
R^2	0.07	0.07	0.05	0.06	0.07	0.07	0.06	0.06
Weekend	0.549	1.042	-0.344	0.785	0.340	0.602	0.406	1.425
	(0.673)	(0.950)	(0.793)	(0.857)	(1.372)	(0.917)	(0.984)	(1.555)
Observations	45838	28634	17204	30603	12830	25421	20417	8164
R^2	0.05	0.05	0.06	0.05	0.05	0.05	0.06	0.05

Notes: Data are from ATUS and BLS LAUS (2003-2015). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Column 5 refers to (BHAI) Black, Hispanic, or American Indian. Column 8 restricts to white males with education less than a Bachelor's degree.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.5: Effects of Employment Rate on Sleep by Marital Status (2003-2022)

	(1)	(2)	(3)	(4)	(5)	(6)
	Single	Married	Single Parent	Married Parent	MF(sleep)	MF(work)
All	-0.791	-1.141*	-3.081**	-0.958	-1.795	2.102
	(0.643)	(0.504)	(0.974)	(0.524)	(0.972)	(1.109)
Observations	48062	72681	18122	64968	21066	21066
R^2	0.10	0.13	0.11	0.10	0.11	0.31
Weekday	-3.234***	-1.692*	-5.507***	-2.866***	-2.814*	2.080
	(0.909)	(0.681)	(1.261)	(0.759)	(1.395)	(1.854)
Observations	23969	36281	8959	32453	10325	10325
R^2	0.07	0.06	0.08	0.07	0.06	0.34
Weekend	1.447	-0.438	-0.765	0.848	-0.610	2.138
	(0.898)	(0.654)	(1.237)	(0.682)	(1.137)	(1.737)
Observations	24093	36400	9163	32515	10741	10741
R^2	0.05	0.06	0.05	0.05	0.06	0.09

Notes: Data are from ATUS and BLS LAUS (2003-2022). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Column 5 refers to married females without college degree for sleep in minutes. Column 6 refers married females without college degree for work in minutes.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.6: Effects of Employment Rate on Sleep by Marital Status (2003-2015)

	(1)	(2)	(3)	(4)	(5)	(6)
	Single	Married	Single Parent	Married Parent	MF(sleep)	MF(work)
All	-1.080	-1.220*	-3.278*	-1.295*	-1.829	1.226
	(0.824)	(0.586)	(1.408)	(0.603)	(1.167)	(1.219)
Observations	35733	55451	13837	48373	17124	17124
R^2	0.10	0.12	0.11	0.10	0.11	0.30
Weekday	-3.849**	-2.175**	-5.476**	-3.758***	-2.532	0.282
	(1.239)	(0.759)	(1.862)	(0.988)	(1.633)	(2.096)
Observations	17766	27580	6794	24113	8381	8381
R^2	0.07	0.06	0.08	0.07	0.06	0.33
Weekend	1.532	-0.155	-1.169	1.080	-1.002	2.657
	(1.097)	(0.918)	(1.949)	(0.840)	(1.652)	(2.088)
Observations	17967	27871	7043	24260	8743	8743
R^2	0.05	0.06	0.05	0.05	0.06	0.09

Notes: Data are from ATUS and BLS LAUS (2003-2015). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Column 5 refers to married females without college degree for sleep in minutes. Column 6 refers married females without college degree for work in minutes.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

There is heterogeneity across occupation industries due to variations in work time structures. Using the CPS major industry code, I use the variable similar to Niekamp (2019),

$$\text{Percentage of Weekday Work Time} = \frac{W_{\text{weekday}}}{W_{\text{weekday}} + W_{\text{weekend}}},$$

where W_{weekday} is the mean reported work time in minutes on weekdays, and W_{weekend} is the mean reported work time in minutes on weekends. Figure 2.5 shows the percentage ranges from 62% in Leisure and Hospitality to 88% in Financial Activities sector.

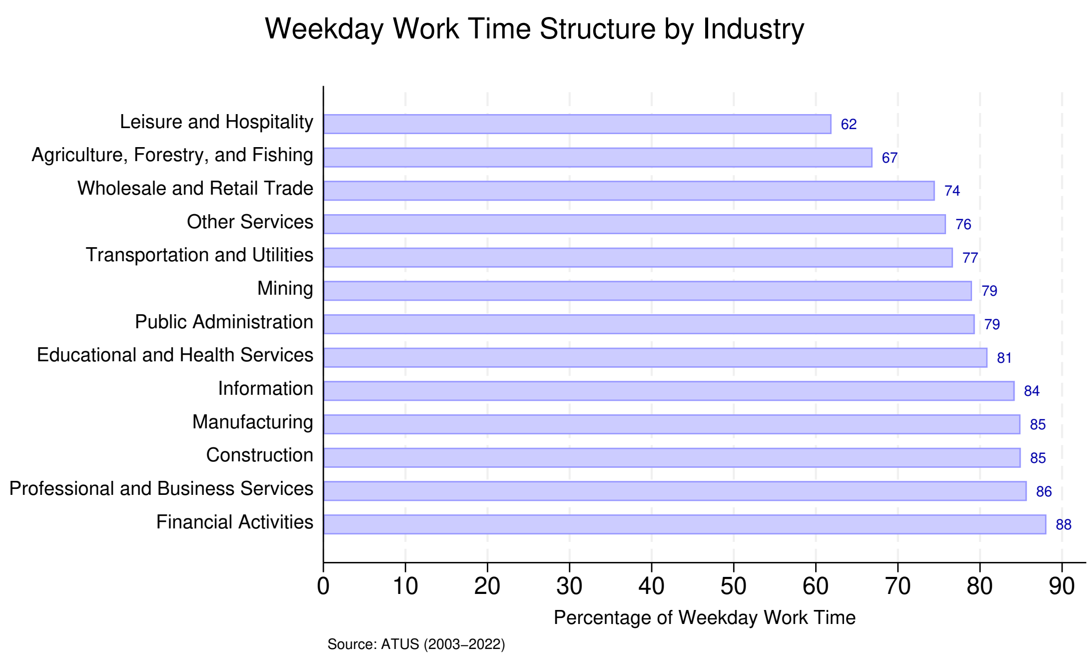


Figure 2.5: Weekday Work Time Structure by Industry

Notes: Data are from ATUS (2003-2022). W_{weekday} is the mean reported work time in minutes on weekdays, and W_{weekend} is the mean reported work time in minutes on weekends. The percentage of weekday work time is given by the formula:

$$\text{Percentage of Weekday Work Time} = \frac{W_{\text{weekday}}}{W_{\text{weekday}} + W_{\text{weekend}}}.$$

Table 2.7 presents the results by work time structure in different industries. Columns 1 and 2 indicate that the effects are primarily driven by those who are employed. Columns 3 and 4 show estimates for individuals working in industries with a percentage of weekday work time above and below the median. The impacts are comparable, albeit marginally greater for individuals below the median. Columns 5 and 6 divide the industries into blue-collar and white-collar workers, revealing a higher impact on blue-collar workers.

Table 2.8 shows the analysis from 2003 to 2015. The results are similar to those of Table Niekamp (2019), except that the not-employed individuals also experience a significant effect on weekday sleep. The results are mainly driven by those working in industries with a percentage of weekday work time below the median. The estimates for blue-collar workers are lower.

If we break the analysis into different periods as shown in Table 2.9, we observe that the impact of employment on sleep is smaller from 2003-2022 compared to 2003-2015 (Columns 1-2). Columns 3 to 9 show that the effects are generally larger and more significant for the periods from 2011-2015 (after the Great Recession) and 2020-2022 (after the pandemic). The effects are smaller after the pandemic, possibly due to the rise of work-from-home (WFH) or telework, which has been shown to increase sleep time according to previous literature (Pabilonia and Vernon, 2020).

To examine the impact across industries with varying levels of telework concentration, I utilize a new survey question introduced in July 2020 in ATUS. This question asks respondents, "At any time in the last 4 weeks, did you telework or work at home for pay?" Figure 2.6 displays the ranking of industries by their telework percentages, which range from 4% in Agriculture, Forestry, and Fishing to 43% in the Financial Activities sector.

Table 2.10 shows the effects of by telework concentrated industry. Column 2 shows that those who answered yes to the telework question exhibits positive impact of employment on sleep, although the effects are not statistically significant. Table 2.11 show that the impact is smaller for those who work in industries where the percentage of telework exceeds the mean (Column 2 and Column 4). This indicates that the impact of employment rate on sleep could be more driven by those industries that are not telework concentrated. The rise of the telework after the pandemic may alleviate the impact of employment on sleep, especially for the industries that are telework concentrated.

Table 2.7: Effects of Employment Rate on Sleep by Work Time Structure (2003-2022)

	(1)	(2)	(3)	(4)	(5)	(6)
	Employed	Not Employed	$>$ Med Weekday	$\leq$ Med Weekday	Blue-collar	White-collar
All	-1.140**	0.010	-1.010*	-1.178	-1.279	-0.351
	(0.392)	(0.900)	(0.381)	(0.624)	(1.110)	(0.661)
Observations	97713	23030	73864	29712	18907	20764
R^2	0.13	0.06	0.14	0.10	0.16	0.16
Weekday	-1.992***	-2.642	-1.979***	-2.120*	-1.580	-0.313
	(0.445)	(1.391)	(0.458)	(0.804)	(1.011)	(0.844)
Observations	49045	11205	37106	14715	12859	14405
R^2	0.04	0.06	0.04	0.05	0.15	0.15
Weekend	-0.267	2.415	-0.148	-0.169	-0.111	-0.501
	(0.605)	(1.363)	(0.682)	(1.007)	(1.447)	(0.899)
Observations	48668	11825	36758	14997	12788	14291
R^2	0.06	0.05	0.06	0.05	0.14	0.15

Notes: Data are from ATUS and BLS LAUS (2003-2022). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Column 1 limits the sample to employed population and column 2 limites to unemployed and population that are not in the labor force. Column 3 limits to below median weekday work time percentage. Column 5 refers to blue-collar workers: construction and manufaturing. Column 6 refers to white-collar workers: financial activities and professional and business services.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.8: Effects of Employment Rate on Sleep by Work Time Structure (2003-2015)

	(1)	(2)	(3)	(4)	(5)	(6)
	Employed	Not Employed	> Med Weekday	≤ Med Weekday	Blue-collar	White-collar
All	-1.335*	-0.107	-0.794	-2.108**	-1.191	0.322
	(0.587)	(0.975)	(0.673)	(0.700)	(1.486)	(0.766)
Observations	73216	17968	55255	22770	14558	15132
R^2	0.13	0.06	0.13	0.09	0.16	0.16
Weekday	-2.618***	-3.176*	-2.166**	-3.042**	-3.360*	-0.676
	(0.605)	(1.400)	(0.694)	(0.984)	(1.489)	(1.019)
Observations	36579	8767	27557	11286	7234	7662
R^2	0.04	0.06	0.04	0.05	0.05	0.06
Weekend	-0.048	2.507	0.422	-0.994	0.727	1.327
	(0.775)	(1.734)	(0.922)	(1.107)	(2.138)	(1.398)
Observations	36637	9201	27698	11484	7324	7470
R^2	0.06	0.06	0.06	0.05	0.08	0.07

Notes: Data are from ATUS and BLS LAUS (2003-2015). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Column 1 limits the sample to employed population and column 2 limites to unemployed and population that are not in the labor force. Column 3 limits to below median weekday work time percentage. Column 5 refers to blue-collar workers: construction and manufaturing. Column 6 refers to white-collar workers: financial activities and professional and business services.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.9: Effects of Employment Rate on Sleep by Periods

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	2003-2022	2003-2015	2016-2022	2003-2007	2008-2010	2011-2015	2016-2019	2020-2022
All	-1.019**	-1.133*	-0.935	-1.020	-2.399	-2.889*	2.383	-1.688*
	(0.352)	(0.444)	(0.593)	(0.921)	(1.604)	(1.196)	(2.162)	(0.705)
Observations	120743	91184	29559	40772	20772	29640	18404	11155
R^2	0.11	0.11	0.12	0.11	0.12	0.11	0.12	0.12
Weekday	-2.308***	-2.875***	-1.536	-2.075	-2.694	-5.420**	4.251	-3.335**
	(0.441)	(0.512)	(0.938)	(1.118)	(1.441)	(2.021)	(2.475)	(1.052)
Observations	60250	45346	14904	20278	10230	14838	9213	5691
R^2	0.07	0.07	0.07	0.06	0.06	0.08	0.08	0.07
Weekend	0.259	0.549	-0.452	-0.063	-1.790	-1.247	0.498	-0.379
	(0.532)	(0.673)	(1.045)	(1.318)	(2.573)	(1.558)	(3.096)	(1.277)
Observations	60493	45838	14655	20494	10542	14802	9191	5464
R^2	0.05	0.05	0.06	0.06	0.06	0.06	0.05	0.06

Notes: Data are from ATUS and BLS LAUS (2003-2022). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

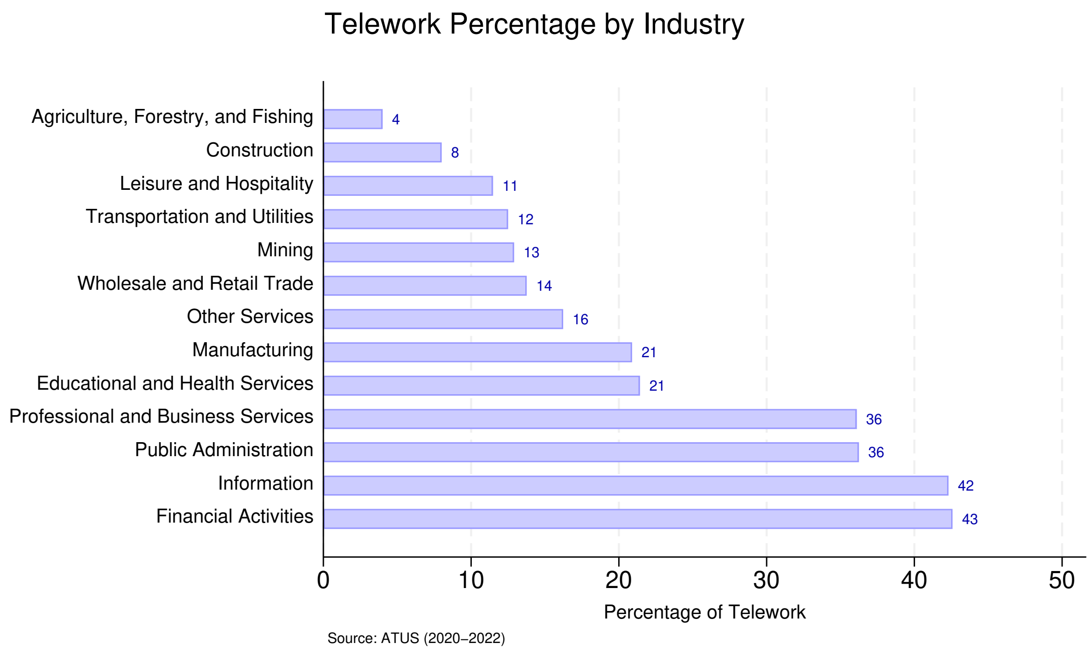


Figure 2.6: Telework Percentage by Industry

Notes: Data are from ATUS (2020-2022). Starting July 2020, a new survey question has been introduced asking respondents,“At any time in the last 4 weeks, did you telework or work at home for pay?” This graph depicts the percentage of telework by industry.

Table 2.10: Effects of Employment Rate on Sleep by Telework

	All(2003-2022)	Telework(2020-2022)	Industry(2003-2019)
	(1)	(2)	(3)
	Sleep	Sleep	Sleep
	b/se	b/se	b/se
Employment to Population Rate	-1.019***	-1.582	-0.967**
	(0.35)	(1.86)	(0.48)
Telework × Employment		0.953	
		(0.80)	
Above Mean Telework × Employment			-0.076
			(0.21)
N	120743	7445	93846
r2	0.11	0.14	0.12

Notes: Data are from ATUS and BLS LAUS (2003-2022). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Column 1 uses data from 2003-2019. Column 2 includes respondents who answered yes to the telework question beginning in July 2020 (2020-2022). Column 3 represents individuals employed in industries where the percentage of telework exceeds the mean (2020-2019).

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.11: Effects of Employment Rate on Sleep by Industry (Telework Percentage)

	(1) All 2003-2022	(2) > Mean Telework 2003-2022	(3) ≤ Mean Telework 2003-2022	(4) > Mean Telework 2003-2019	(5) ≤ Mean Telework 2003-2019
All	-1.019**	-0.964*	-1.201*	-0.754	-1.317*
	(0.352)	(0.465)	(0.533)	(0.702)	(0.630)
Observations	120743	54957	48590	49436	44410
R^2	0.11	0.13	0.12	0.12	0.12
Weekday	-2.308***	-1.502*	-2.583***	-1.461	-2.802***
	(0.441)	(0.590)	(0.626)	(0.765)	(0.711)
Observations	60250	27631	24178	24774	22057
R^2	0.07	0.04	0.05	0.04	0.05
Weekend	0.259	-0.515	0.162	-0.111	0.282
	(0.532)	(0.797)	(0.833)	(1.001)	(0.946)
Observations	60493	27326	24412	24662	22353
R^2	0.05	0.06	0.06	0.05	0.06

Notes: Data are from ATUS and BLS LAUS (2003-2022). The dependent variable is daily sleep in minutes for respondents aged 25-55. Each cell represents the estimates of employment to population rate on sleep. Column 1-3 uses data from 2003-2022. Column 4-5 uses data from 2003-2019. Column 2-5 represents individuals employed in industries where the percentage of telework exceeds the mean.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

2.7 Conclusion

This study investigates the impact of employment rates on sleep patterns using data from the American Time Use Survey (ATUS) and Local Area Unemployment Statistics (LAUS) spanning from 2003 to 2022. The analysis confirms that sleep duration is countercyclical, with higher employment rates leading to reduced sleep, particularly on weekdays.

Demographic factors significantly influence these effects, with greater impacts observed among individuals without a Bachelor's degree, minorities, females, and single parents. Industries with higher telework rates exhibit smaller sleep reductions as employment rises, suggesting that telework can mitigate some negative impacts on sleep, especially in the post-pandemic period.

These findings highlight the need for policies and employer strategies that support adequate sleep, particularly for vulnerable groups and those in less flexible work environments. Future research could explore the long-term effects of telework and economic cycles on sleep and overall well-being, as well as potential interventions to promote better sleep and work-life balance.

Chapter 3

Caste Differences in Child Growth: Disentangling Endowment and Investment Effects

3.1 Abstract

Using the fourth round of the Indian National Family Health Survey (NFHS-4), and subsequently replicating our results using the fifth round (NFHS-5), we document differential child physical growth patterns across caste groups in India, demonstrating that lower caste children are born shorter and grow less quickly than children from higher-caste households. We then show that, in line with work from previous rounds of the NFHS, these differences are largely explainable by observable covariates, particularly maternal characteristics and household wealth variables. Our research also reveals a previously undocumented

dynamic, that the influence of these variables changes as children develop, and suggests that caste-gaps are the result of multiple mechanisms impacting the child growth process at different stages of development. Using age-disaggregated decomposition methods, we demonstrate that health endowment related variables (e.g. maternal height) largely explain birth length gaps, and that variables related to health investments (e.g. household wealth, health care usage) become increasingly influential as children age. Children from lower caste households thus face two margins generating height gaps as they age: a persistent endowment disparity present from birth, and a post birth investment differential that exacerbates the initial deficit.

3.2 Introduction

We document large disparities in child height for age z-scores (HAZ) across Indian caste groups using data from the fourth round of the Indian National Family Health Survey (NFHS-4) and replicate the results using the fifth round (NFHS-5). Scheduled Caste (SC) and Scheduled Tribe (ST) children are, on average, around 0.4 and 0.5 standard deviations (sd) shorter than Upper Caste (UC) children in the first six-months of life, while children from Other Backwards Classes (OBC) are about 0.2 sd shorter. By age five, caste HAZ differentials have increased by an additional 0.1–0.3 sd in each group.

Caste differentials also largely disappear, or at least greatly attenuate in magnitude, after adjusting for a broad set of household and community variables in a regression model, a finding common to studies analyzing data from previous rounds of the NFHS (Van de Poel and Speybroeck, 2009; Coffey et al., 2019). Our results are fully consistent with this previous research on the topic. What separates our work from previous analyses of child HAZ gaps across caste groups is our focus on the biological process of human growth. While both Coffey et al. (2019) and Van de Poel and Speybroeck (2009) are concerned with the social processes generating caste HAZ disparities, we are interested in the inter-generational and contemporary disparities in health inputs that generate them. To accomplish this, we shift the analytic focus of our research from the static health measures used in previous work (HAZ, stunting), onto the dynamics of child growth as children age.

Figure 3.1 summarizes our main empirical focus and findings, graphing mean HAZ across child age in months for four broad caste groups: UC, OBC, SC and ST. The overall pattern is similar across all groups and mirrors the HAZ-age profile shape common across the developing world (Shrimpton et al., 2001; Victora et al., 2010). Indian children as a whole are, on average, born below the WHO reference population by between 0.2 sd and 0.5 sd. Over the first two years of life, Indian children then grow too slowly compared to the reference population, reflected in a decreasing mean HAZ over this period.

The caste differentials themselves display similar age-dynamics. SC, ST, and OBC children are, on average, born shorter than UC children. These gaps, in the standard deviation units of HAZ, then widen over the first two years of life, and subsequently remain relatively constant or slightly decrease between the ages of 2 and 5.

We structure our interpretation of these dynamics based on insights derived from a dynamic health capital accumulation model (Grossman, 1972). Health capital theory considers the realized health of a person at any age as the result of two distinct mechanisms: a health endowment provided to a child at birth, and a stream of subsequent health inputs

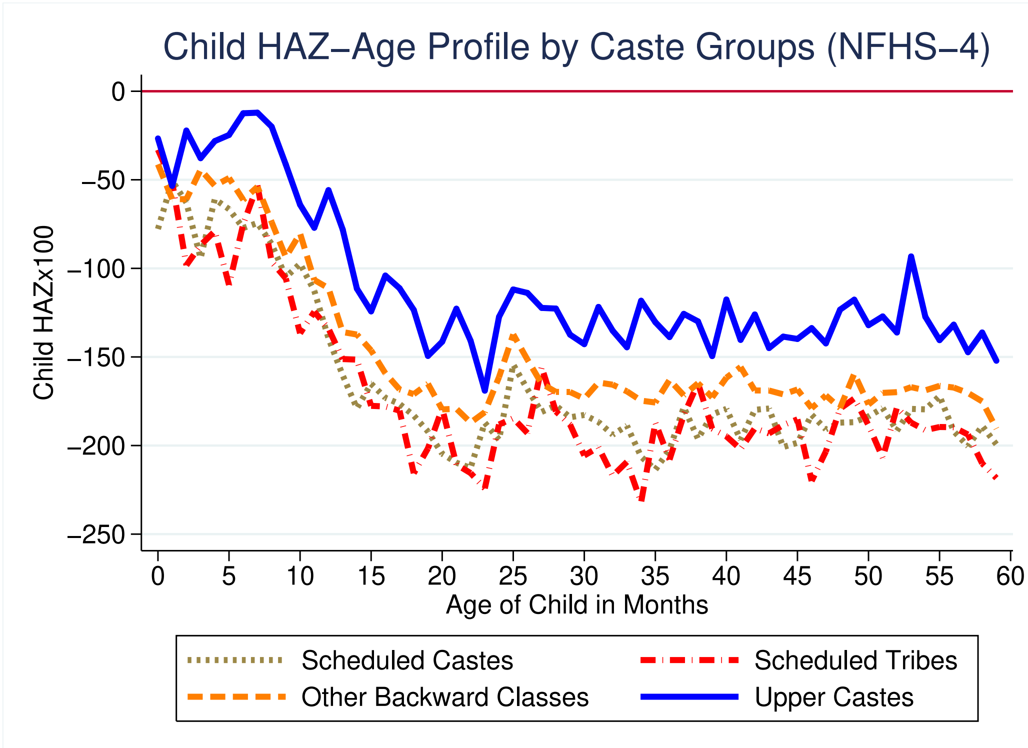


Figure 3.1: Child HAZ by Caste Groups

Notes: This figure graphs mean child HAZ score (x100) by caste groups for children from 0 to 5 years. The x-axis represents child age in months and the y-axis is mean weighted HAZ(x100). Median child HAZ for reference population children is 0. The mean child HAZ in this sample is -1.49. A child with HAZ between -2 and -3 indicates moderate chronic malnutrition (stunted), and HAZ below -3 indicates severe chronic malnutrition. The results are weighted by sample weights.

consumed by the child after birth. Following Aiyar and Cummins (2021), we interpret birth length as a measure of child health endowments, and the rate of child growth as a measure of the interaction between this health endowment and the subsequent stream of health inputs consumed by the child.

Such a model predicts caste HAZ disparities that change as children age. Lower caste women are smaller, sicker and have less access to maternity care than higher caste women, potentially generating an “endowment” effect in the caste-HAZ deficit (Dommaraju et al., 2008; Sanneving et al., 2013; Saikia et al., 2019; Blunch and Gupta, 2020; Hamal et al., 2020; Uddin et al., 2020). Similarly, lower caste households are significantly poorer than upper caste households, and many simply cannot afford health inputs like high-quality and sufficient food, or to live in areas with sanitation or clean air (Thorat and Sadana, 2009; Vart et al., 2015; LoPalo et al., 2019; Spears and Thorat, 2019; Biswas et al., 2023). Such disparities in post-birth private and public investments may exacerbate differences at birth and generate divergent growth trajectories (Dommaraju et al., 2008; Dewey and Begum, 2011; Mertens et al., 2023).

Moreover, such a model predicts that the determinants of the HAZ gaps change as children age as well. Initial birth length deficits are likely to be the result of long-term maternal health and nutrition and/or pre-natal health care differentials, but should not be explainable by determinants of post-birth health inputs. On the other hand, household wealth disparities across caste groups are likely to generate differential health consumption patterns post-birth (e.g. in terms of nutritional intake or health care demand) that are likely to affect the rate at which children grow. Similarly, caste locational sorting may generate differential access to public goods that provide non-rival and non-excludable benefits to everyone in some area (e.g. public sanitation, pollution exposure), potentially generating differential growth patterns across caste groups as children age. The health capital theory prediction is clear: endowment-related health inputs should predominantly explain birth length disparities; post-birth consumption of (private and public) health inputs should become increasingly influential as children grow.

Our decomposition results suggest precisely this: an initial HAZ deficit at birth that is statistically explained largely by differences in variables related to the health endowment; and household health investment variables that have little explanatory power over

HAZ gaps at birth, but which grow in explanatory power as children age. By the age of 5, we estimate that around half of the caste-HAZ deficit is due to disparities in average child health endowments, and around half due to deficits in post-birth child health inputs. Almost none of the caste gaps are ascribed by the model to disparities in public goods across caste groups. We conclude that initial endowment deficits across caste groups persist through early childhood, while private investment effects, driven by caste differentials in household wealth and expenditure, accumulate over the first few years of life.

While some of the dynamics we describe have been explored in the broader literature on growth faltering (Danaei et al., 2016; De Onis and Branca, 2016; Prendergast and Humphrey, 2014), we add new evidence documenting the age dynamics of childhood HAZ disparities across caste groups in India. We build on the economic demography literature examining the influence of health related variables in explaining caste HAZ disparities (Van de Poel and Speybroeck, 2009; Coffey et al., 2019) by studying the changing influence of household inputs over the life course of a child.

The goal of this work is to provide researchers and policy makers with economic insights into the biological (in contrast to social) processes behind the disparate and diverging child growth trajectories across castes. Our results cannot speak to questions of the economic and social discriminatory practices that have generated the disparities in child health endowments and investments that we document, nor to the effectiveness or efficiency of any particular policy aimed at remediating such disparities. We instead speak to the timing and subject of any policies or interventions aimed at reducing caste disparities in child health. An empirically equivalent definition of our "endowment" and "investment" related variables would be health inputs that could (in theory) be intervened upon either before or after a child is born. Our results thus reinforce an important overarching insight for both researchers and policy makers — understanding or ameliorating caste disparities in child HAZ will require addressing both contemporary disparities in early life child health inputs and nutritional intake, and disparities in the health and nutrition of the next generation of mothers.

3.3 Caste Disparities

Caste disparities in India exist across almost every meaningful human welfare measure including household earnings, educational attainment and life-course health outcomes (Deshpande, 2000, 2001; Borooah, 2005; Kijima, 2006; Munshi and Rosenzweig, 2006; Subramanian et al., 2006; Deshpande, 2007; Deshpande and Newman, 2007; Van de Poel and Speybroeck, 2009; Perkins et al., 2011; Zacharias and Vakulabharanam, 2011; Kumar, 2013; Deshpande and Sharma, 2016; Maity, 2017; Deshpande and Ramachandran, 2019; LoPalo et al., 2019; Munshi, 2019; Blunch and Gupta, 2020; Vyas et al., 2022; Goraya, 2023). Health outcome disparities across caste groups are well documented at every age: lower caste children are more likely to experience stunted growth; adult lower caste men and women are shorter and less healthy; and people from lower castes die younger (Kijima, 2006; Subramanian et al., 2006; Van de Poel and Speybroeck, 2009; Perkins et al., 2011; Maity, 2017; Saikia et al., 2019; Raushan et al., 2022; Vyas et al., 2022). The literature on these caste disparities in child health outcomes has largely focused on either quantifying the extent of current discrimination against lower caste members, or tracing out the mechanisms through which the long history of caste discrimination has translated religious social hierarchies into political and economic hierarchies that generate large health disparities (Coffey et al., 2019; LoPalo et al., 2019; Blunch and Gupta, 2020).

Both sets of discriminatory practices – contemporaneous and historical – are potential explanations of the child HAZ disparities across castes that we investigate. Caste-based occupational sorting that arises due to closely-knit caste networks leads to exclusion of SC and ST communities from lucrative livelihood opportunities (Deshpande, 2000, 2001; Ito, 2009; Siddique, 2011; Munshi, 2019), generating large income and wealth disparities across caste groups. These disparities in spending power then translate into disparities in health investment levels by the household, particularly disparities in food consumption and nutritional intake (Thorat and Sadana, 2009; Mahadevan and Suardi, 2013; Parappurathu et al., 2015; Choudhury et al., 2021; Biswas et al., 2023). Furthermore, direct discriminatory practices lower access to health care for SC, ST, and OBC children both directly (Coffey et al., 2019; LoPalo et al., 2019; Spears and Thorat, 2019) and indirectly (Debnath and Jain, 2020; Blunch and Gupta, 2020).

Additionally, long term economic inequality reinforces health disparities across generations. Many studies have documented that poor maternal health leads to worse health outcomes for children (Addo et al., 2013; Aizer and Currie, 2014; Chakrabarti et al., 2021). Furthermore, maternal health is correlated with household wealth, which is strongly differential across castes. Together, the health–wealth correlation for parents generates a channel for the inter-generational transmission of poor health within lower caste households, independent of any contemporaneous discriminatory practices (Dommaraju et al., 2008; Sanneving et al., 2013; Saikia et al., 2019; Blunch and Gupta, 2020; Hamal et al., 2020; Uddin et al., 2020).

In the next section, we offer a biologically-focused economic perspective on the process through which child health disparities arise across caste groups. To do so, we model the biological process of child growth and the economic factors that influence this process, providing clarity on the key temporal mechanisms through which disparities in child health come to exist and persist across castes in India.

3.4 Theoretical Framework

We approach the question of child growth differentials across caste groups from the perspective of health capital accumulation. In this section we sketch out the structure of health capital accumulation theory in order to define the roles of health endowments and investment streams in early life development (Grossman, 1972). We then discuss how HAZ is an especially appropriate measure of early life health capital and how the HAZ-age profile reflects the process of early life health capital accumulation in poor countries.

3.4.1 Health Capital

Households have preferences over the consumption and health outcomes of their members, and optimize an inter-temporal lifetime utility function representing those preferences subject to a budget constraint for their expenditures. Households can purchase consumption and health investments at a total cost that does not exceed the available budget in a period. We consider a household with a single newborn child.

Child health capital at birth is defined as H_0, a health endowment that is bestowed upon, and not chosen by, the newborn. In any given subsequent period, health at age A, H_A, is the result of a production function $f(H_{A-1}, I^*_{A-1})$ which takes as its inputs the health capital of the previous period (H_{A-1}) and optimally chosen health investments (I^*_{A-1}) in the previous period.

Households choose optimal consumption and health investment by maximizing their utility function subject to the budget constraints and the health capital production function. Choosing optimal health investment implies choosing an optimal health level, given health in the previous period (H_{A-1}). Furthermore, since health iterates from period to period beginning at birth, H_A can be expressed as $H^*_A(H_0, I^*)$, where I^* represents the stream of child health investments provided from birth up until a child is observed.

3.4.2 Endowments, Private Investments, and Public Health

The biological content of health capital theory resides in the health production function $f(H_0, I^*)$, which is realized as the health of a person of some age $H^*_A(H_0, I^*)$, and differences in health are attributed to differences in H_0 and/or I^*. That is, health capital theory conceives of two sets of potential causal channels that determine a child's realized health at any given point in their lives: health endowments (H_0) and health input streams (I^*). The legacy and contemporary reality of caste socioeconomic disparities make it likely a priori that both of these causal channels operate on caste HAZ disparities: on average, UC women are healthier when they give birth and have more resources to provide for their children after birth.

The health capital model defines the health endowment as the health of a child at birth, an initial store of health (energy, potential, genetics) bequeathed to a child as they enter the world. We proxy for this theoretical construct at a population level using caste-level estimates of implied birth length and very early life HAZ. Determinants of birth length, birth weight, or other very early life health measurements include maternal height, weight, age, and birth order (Currie, 2009; Ahmed and Ray, 2018; Chari et al., 2017; Özaltin et al., 2010; Maertens, 2013; Swaminathan et al., 2019; Von Grafenstein et al., 2023). Timely interventions on children born in the hospital can also play a role in ensuring that children are healthier at birth (Godlonton and Okeke, 2016; Daysal et al., 2015). We consider maternal health variables, birth order, and pre-natal and delivery care as observable characteristics related to the determinants of the health endowment, and thus a child's length at birth.

After-birth, health capital is determined jointly by this initial health endowment and the subsequent stream of health inputs a child experiences up until they are measured. Empirically, we thus consider differences in the population rate of growth of child HAZ as evidence of interactions between population-level initial health endowments and the subsequent stream of health inputs provided to the average child post-birth. This investment stream can be conceptually divided into two sub-types: private investments made by the household and public investments affecting the availability of public goods and the health environment. Private investments are made by individual households, and can be proxied most directly by household wealth or assets (land ownership, bathroom facilities) (Swaminathan et al., 2019; Attanasio et al., 2020). Alternatively, public goods like sanitation and health environment affect all children in an area (Spears, 2020; Geruso and Spears, 2018).

Health capital at any given age thus contains within it traces of an initial endowment component (H_0) and a component determined by the subsequent stream of health inputs consumed by the individual. While for adults we might expect the initial endowment effect to be wiped out by the subsequent health experiences of the person, for children the relative importance of the two is less clear. The health endowment is more recent, and the stream of privately and publicly provided health investments a child has received up until that point in their lives is relatively short.

3.4.3 Height-for-Age Z-score (HAZ)

A good measure of health capital for children, then, would be one that captures the cumulative health of a child from birth until they are measured. We argue that HAZ is precisely such a measure.

Children are born a certain length, and their growth trajectory is then determined by the interaction of this initial birth length and the stream of nutritional and medical health inputs the child experiences from that point on. That is, child length or height contains within it information on the cumulative health experience of children from birth until they are measured, in the same manner as health capital.

Child HAZ is an age- and gender-normalized measure of child height. The Z-score measures we employ have been standardized across developed and developing countries by the World Health Organization (WHO) using a reference population of relatively well-nourished children (WHO, 2006), with part of the sample coming from an affluent neighborhood in New Delhi, India. These well-nourished children grow, on average, at the same rates across the sampled countries, implying that population level deviations from these standards are not determined by genetic origin, but instead by the circumstances of children's birth and growth. This normalization allows for comparisons of the relative magnitude of child health outcomes as children age.

Other anthropometric measures of child health, such as those based on weight or arm circumference that change rapidly in the short term, cannot claim to be such acceptable proxies for health capital and its nature as a cumulative stock accrued as people age. Measures related to acute child morbidity are even shorter-term measures. Child mortality rates may contain information on average very early life health capital, but the infrequency of mortality at ages beyond the first year makes detecting differences in health levels of older children largely impossible.

3.4.4 HAZ-age Profile

Shrimpton et al. (2001) were the first to document the age-dynamics of HAZ as a consistent feature of child health in developing countries. They show that, across the developing world, children are born with HAZ that is slightly lower than healthy populations (slightly below 0). These children then grow much less quickly than the median child in the reference population up until the child is around 2 years old. Further work has shown that this pattern is consistent across social groups, and is not explained by observable covariates (Rieger and Trommlerová, 2016; Roth et al., 2017; Alderman and Headey, 2018). Focusing on the HAZ-age profile, as opposed to mean HAZ, centers our empirical perspective on the health capital accumulation process itself, as opposed to the stock of health capital at any particular moment in time.

Given this consistent and stable pattern in global child growth patterns, Aiyar and Cummins (2021) develop regression models to capture the effects of a key variable of interest on changes in the location and shape of the HAZ-age profile. They also develop the health capital accumulation interpretation we employ, relating changes in the HAZ-age profile intercept at age 0 (implied birth length z-score) to differences in health endowments, and changes in the slope of the HAZ-age profile to differences in the interaction between the health endowment and the post-birth health investment stream.

Our work extends this age-profile empirical perspective to the realm of decomposition methods. While Aiyar and Cummins (2021) were interested in estimating correlations between one key variable and changes in the shape of the HAZ-age profile, here we are interested in how suites of variables, defined in relation to theoretical economic mechanisms, can explain differences in HAZ-age profiles across socio-economic groups.

3.5 Data

3.5.1 Data Source

Our primary child-level dataset of outcomes and covariates come from the NFHS 2015-16 (NFHS-4), the fourth of the Indian NFHS series. The dataset is population representative at the district level. Our main estimation sample consists of information on 146,778 Hindu children below the age of five

Table 3.1 provides summary statistics.Indian children are on average 1.49 sd shorter than the WHO reference population median. Caste groups follow IPUMS-DHS recoding to include: Scheduled Caste (SC), Scheduled Tribe (ST), Other Backward Classes (OBC), and Upper Caste (UC). Overall, in our weighted sample, 23.7% of children are SC, 14.6% are ST, 43.7% are OBC, and 17.9% are UC. As expected, lower caste and tribal children have lower HAZ scores on average than their UC counter parts. SC children are 1.67 sd below, OBCs are 1.49 sd below, and ST children are around 1.72 sd below the reference height for their sex and age, while UC children are only 1.12 sd below.

3.5.2 Variable Groups

The NFHS-4 provides information on a large number of child, parent, household and community level characteristics. Motivated by health capital theory, we separate our observed covariates into three groups: endowment variables, and variables for private health inputs, and public health inputs. A table of representative summary statistics is provided in Table 3.1.

Our categorization of household variables as exclusive determinants of either health endowments or health investments, while not arbitrary, is imprecise. It is reasonable to argue that many variables, such as maternal height or household wealth, will affect both child health endowments and the subsequent stream of child health inputs (at least indirectly). Similarly, the total effects of private sanitation on health may nullify the effects of public sanitation on health. We stress that our goal is not to estimate the causal impact of any particular variable in explaining these caste disparities but rather to highlight two distinct channels of health disparities (health endowments and post-birth investment in health inputs) as determinants of the caste gaps in child HAZ.

An empirically equivalent definition of "endowment" and "investment" related variables would be variables that could (in theory) be acted upon either before (endowment) or after (input/investment) a child is born. Child birth order may have effects on the within-household distribution of resources post-birth, and maternal height may continue to operate post-birth, but by definition birth order cannot be altered after birth and maternal growth spurts after birth are unlikely to be important for child growth rates. Alternatively, household wealth is likely to have cumulative effects over the life-course by affecting the stream of post-birth child health inputs, and thus post-birth intervention or manipulation that alters the household budget constraint may be effective at improving child growth. When we frame the distinction in this manner, our theoretical distinction among metaphysical objects (endowments and investments) becomes an operational distinction regarding the locus of any potential intervention aimed at alleviating caste HAZ disparities (before or after a child is born). With this caveat in mind, we now proceed to define the contents of three families of explanatory variables.

Health Endowments

Endowment variables include birth order, maternal age at child's birth, maternal HAZ and WAZ, and delivery care. From Table 3.1, we see that Indian mothers are on average 24.23 years old, have around 2.14 children and are 2.03 sd shorter than the median height of healthy women across the world. UC mothers are on average healthier than lower caste mothers. They are slightly less likely to have more than two children, and more likely to report having given birth in a hospital. ST mothers are only slightly less tall than SC mothers but are much less likely to give birth in a hospital. OBC mothers are slightly healthier than SC and ST groups but less healthy than US mothers. About half of SC and OBC mothers report having their deliveries in a hospital, whereas 71% of UC mothers deliver in hospitals.

Private Investments

Private investment variables include household wealth index quintiles, maternal education, motorcycle ownership, ownership of agricultural land, access to treated water, owning a private toilet or shared toilet, vaccination status and post-natal care. 71% of mothers have at least a primary education. About 32% of the entire sample report access to clean drinking water and 45% of households own agricultural land. On average, UC households tend to be able to provide higher levels of private investments to their children relative to lower caste households. UC mothers are more likely to have some education (89%), and are less likely to be among the poorest asset quintile and more likely to be among the richest in the sample.

Among the disadvantaged groups, SC and ST households are much more likely to be in the poorest wealth quintile and less than 10% are in the richest quintile. OBC households, on the other hand, are slightly more likely than SC/ST households to be among the richest, though women from the UC dominate the richest category. Around one-third of lower caste and tribal women have no education, and less than 10% of mothers have higher education. Interestingly, OBCs own assets like motorcycles and land at a similar rate as UC mothers. ST households are more likely to own agricultural land but less likely to own means of transport than SC households. Treated drinking water is more prevalent for ST (38%) and UC (44%), while SC lag at 22% of households. About half of SC, ST, and OBC mothers have no access to household toilets. Nearly 90% of all children in these groups have completed their Bacille Calmette-Guérin (BCG) vaccinations for tuberculosis, with measles vaccine take-up the lowest at around 70%. In this dimension, children among ST groups fare the worst in terms of vaccination completion but all other groups are comparable.

Table 3.1: Summary Statistics (NFHS-4)

	SC mean/sd	ST mean/sd	OBC mean/sd	UC mean/sd	All mean/sd
HAZ (x 100)	-166.79	-171.84	-148.69	-112.61	-149.29
	(164.41)	(169.98)	(166.18)	(160.43)	(166.28)
Endowment Variables					
Mother's Age at Child's Birth	24.21	24.03	24.21	24.44	24.23
	(4.78)	(4.95)	(4.53)	(4.52)	(4.64)
Mother's HAZ (x 100)	-217.65	-211.85	-201.27	-182.60	-203.18
	(93.00)	(89.77)	(95.68)	(94.38)	(94.87)
Birth Order	2.29	2.29	2.12	1.86	2.14
	(1.46)	(1.43)	(1.31)	(1.04)	(1.33)
Delivery Care	0.51	0.47	0.56	0.71	0.56
	(0.50)	(0.50)	(0.50)	(0.45)	(0.50)
Private Investment Variables					
Poorest	0.33	0.50	0.23	0.08	0.26
	(0.47)	(0.50)	(0.42)	(0.28)	(0.44)
Maternal Educ at least Primary	0.65	0.55	0.71	0.89	0.71
	(0.48)	(0.50)	(0.45)	(0.31)	(0.45)
Treat Drinking Water	0.22	0.38	0.31	0.44	0.32
	(0.41)	(0.49)	(0.46)	(0.50)	(0.47)
Owns Agricultural Land	0.33	0.55	0.48	0.49	0.45
	(0.47)	(0.50)	(0.50)	(0.50)	(0.50)
No Toilet Facility	0.60	0.74	0.51	0.25	0.51
	(0.49)	(0.44)	(0.50)	(0.43)	(0.50)
Public Health Variables					
PSU has sewer system access	0.10	0.07	0.12	0.23	0.13
	(0.31)	(0.26)	(0.33)	(0.42)	(0.34)
Urban = 1	0.23	0.11	0.25	0.39	0.26
	(0.42)	(0.32)	(0.43)	(0.49)	(0.44)
N	34913	21441	64206	26218	146778

Notes: This table shows the summary statistics for endowment, private investment, and public health variables. It contains data from NFHS-4/2015 IPUMS-DHS in India that are used in the analyses. Results are weighted by sample weights.

Public Investments

Public health variables include access to a sewer system in the primary sampling unit (PSU), state-urban dummies, and whether distance to health facility is a barrier to health care access. On average, 13% have a sewer system, 24% live in urban areas, and 65% face barriers to access a health facility. UCs have more access to sewer systems and are more likely to live in urban areas, while STs are the least likely to have access to the system and about a third as likely to live in urban areas (11% for ST, 39% for UC).

These measures of public health inputs are limited relative to the richer and more predictive information we have on maternal and household characteristics. Sewer and health center access carry information about important channels, but they are rough, aggregate measures of key inputs such as the public health environment. We attempt to ameliorate this issue with a novel interpretation of a common regression adjustment. Regional or locational indicator variables by their nature capture the unobserved determinants of child health that are common to people in a specific place and time. In the context of child health, this could include access to health care, relative food prices, water and air quality, socio-cultural practices and any number of other unmeasured variables that affect every child in the community. We interpret these indicators as a kind of net public goods provision to children, determinants of HAZ that are non-rival and non-excludable for the local population, regardless of caste affiliation. If sorting across location (and thus public health inputs) statistically explains the caste HAZ gaps, regional variables should capture this effect.

3.6 Empirical Methods

We employ three main empirical methods to estimate and explain caste HAZ disparities. We use standard linear regression techniques, disaggregated by child age group, to estimate both the magnitude and age-dynamics of unconditional and conditional HAZ differentials across caste groups. To capture measures more closely aligned to our health capital concepts of endowment and investment effects, we then augment these non-parametric estimates with the parameterized age-profile methods described in Aiyar and Cummins (2021) that estimate caste gaps in (implied) birth length and the rate of growth of young children. Finally, we decompose the caste gaps using simple Oaxaca-Blinder (OB) decompositions to quantify the contribution of the relative influence of the three theoretical health capital channels towards explaining the HAZ gaps as children age: endowments, private investments, and the public health environment.

3.6.1 Estimating Unconditional and Conditional Caste Gaps

We first estimate age-specific mean HAZ differences across caste group (relative to UC children) using a standard linear regression model:

$$Y^A_{irvg} = Caste'^A_g \delta^A_g + X'^A_{1irvg}\beta^A_1 + X'^A_{2v}\beta^A_2 + \lambda^A_r + \epsilon^A_{irvg} \tag{3.1}$$

where Y^A_{irvg} is the HAZ of child i, aged A, living in region r in PSU v and belonging to caste g. $Caste'^A_{irvg}$ is a vector of indicator variables representing the caste to which the child belongs: SC, ST or OBC (with UC comprising the omitted reference group). X_1 and

X_2 are individual-level explanatory variables and village level variables respectively, and λ_r^A is a vector of indicator variables for each DHS state-by-urban location, each specific to child age group, A. The coefficients of interest, δ_g^A, are age-specific estimates of the HAZ gap between UC children and the other caste groups. We always estimate this equation separately for each 6-month child age group, allowing δ, β, and λ to vary by age.

We first estimate Equation (3.1) omitting X_1, X_2, and λ to estimate the unconditional HAZ gap between UC and other castes. The regressions are weighted by individual survey weights and standard errors are clustered at the primary sampling unit level. The resulting coefficients constitute our estimate of the true population gap in HAZ. We then include X_1, X_2, and λ and re-estimate Equation (3.1) to estimate the adjusted caste HAZ gaps given observable covariates of households and children. The estimates here are interpreted as estimates of the height gap that would exist in the population if the observed covariates included in the regression model were evenly distributed across the caste groups.

3.6.2 Estimating Proxy Parameters: Implied birth HAZ (α) and rate of loss of HAZ (β)

Aiyar and Cummins (2021) propose an alternative method to estimating how covariates of interest affect child health endowments and investments. As a complementary approach to estimating age-specific coefficients of the same model to trace out HAZ disparities across age, they attempt to more directly estimate the determinants of the HAZ-age profile using a two step quasi-structural approach. They first estimate the group-level parameters of a stylized structural HAZ-age profile, specifically the intercept (birth length) and slope (rate of growth) of the average HAZ-age profile for that group. Then, in a second stage regression, these estimates are used as observations to estimate the determinants of the group-level parameters themselves.

This allows the model to focus on two particular features of the HAZ-age profile: the y-axis intercept (the implied group-average birth length), which is an empirical counterpart to the health endowment; and the slope of the HAZ-age profile over the first two years of life (the average rate of loss of HAZ), which is related to the interaction of the health endowment and the subsequent stream of health inputs provided to a child. In contrast, when we bin regressions by age as above, we lose precision on the intercept itself (we estimate a single coefficient for 0-6 month olds) and on the rate of child growth (by failing to borrow information from observations across age-bins). By parameterizing the group-level HAZ-age profile as an intercept and a slope across age, we can potentially improve precision of parameter estimates, and simultaneously produce estimates that map more closely to the mechanics and predictions of health capital theory.

The model begins with the following equation, estimated only on children aged 0-2 and estimated separately by caste-region cells. We restrict these regressions to children under the age of 2 to capture the characteristic loss of HAZ over the first two years of life (Shrimpton et al., 2001; Victora et al., 2010), and we include only cells with at least 20 observations to ensure reasonable within-cell estimates of the intercept and slope.

$$Y_i^{rg} = \alpha^{rg} + \beta_{rg} * Age_i^{rg} + \epsilon_i^{rg} \tag{3.2}$$

Y_i^{rg} is the HAZ of child i from region r and caste-group g. $\hat{\alpha}^{rg}$, the estimate of the implied (sub-group) average birth-length z-score, is interpreted as a measure of the average health endowment for caste group g in geographic region r, defined in our main specifications as district-by-urban groups. Similarly, $\hat{\beta}^{rg}$ provides an estimate of the rate of loss of HAZ over the first two years of life for caste group g in region r. Slope coefficients are interpreted as the result of the interaction between the (subgroup average) initial health endowment and the (subgroup average) subsequent stream of consumed health inputs. Regressions in this first stage are weighted by survey sampling weights.

We then estimate caste level differences in $\hat{\beta}$ and $\hat{\alpha}$ with the following regression equation:

$$\hat{Z}_{rg} = Caste'_g\delta_g + Endowment'_{rg}\beta_1 + Private'_{rg}\beta_2 + Public'_{rg}\beta_3 + \epsilon_{rg} \tag{3.3}$$

Here, $\hat{Z}_{rg}$ is either the estimated health endowment, $\hat{\alpha}_{rg}$, or estimated rate of loss of HAZ, $\hat{\beta}_{rg}$, from Equation (3.2). δ^g represents the caste gap in health endowments or rate of growth.

As above in the individual-level regressions, we estimate unconditional versions of Equation (3.3) and conditional regressions that adjust the gap for observable covariates. For the conditional regressions, we include the district-urban-caste-group mean of our endowment and private investment variables. Public health variables are captured by state-urban cell fixed effects. These regressions are run at the district-urban-caste level and are weighted by cell-size of each district-urban-caste group. Standard errors are clustered by state-urban-caste group.

3.6.3 Decomposition

In previous studies and our own, the unconditional caste gap estimates are large, but the conditional caste gap differences are small. This motivates a decomposition exercise that attempts to quantify the contributions of the different covariates towards explaining the unconditional disparity estimates. We employ an Oaxaca-Blinder (OB) decomposition to answer that question.

In the OB decomposition framework, the difference in mean HAZ between caste group g and the upper caste group, uc, for a specific age group A can be represented as:

$$\overline{HAZ}_g^A - \overline{HAZ}_{uc}^A = \overline{\beta}_g^A \bar{X}_g^A - \overline{\beta}_{uc}^A \bar{X}_{uc}^A \tag{3.4}$$

The Oaxaca-Blinder decomposition then re-organizes those terms into:

$$\overline{HAZ}_g^A - \overline{HAZ}_{uc}^A = \hat{\beta}_g^A (\overline{X}_g^A - \overline{X}_{uc}^A) + \overline{X}_{uc}^A (\hat{\beta}_g^A - \hat{\beta}_{uc}^A) \tag{3.5}$$

The first and the second terms on the right-hand side are commonly referred to as the explained and the unexplained portions of the HAZ gap, respectively. In our case, the explained effect tells us the difference in mean HAZ due to the differences in the average level of covariates between lower caste children and UC children. The unexplained effect is interpreted as the difference in the returns to the covariates. In other words, if caste group g was given the mean level of observed covariates as that of the UC (uc) group ($\bar{X}_{uc}$), the remaining difference in HAZ between the groups would be apportioned to differences in the returns to those covariates, and ascribed to the unexplained portion of variation. For ease of interpretation, we present the explained share of variation from the OB decompositions in percentage points, calculated as the explained variation (in HAZ units) divided by the total gap in the unconditional regressions (also in HAZ units) across all the age groups, A.

3.7 Results

3.7.1 Unconditional and Conditional Estimates

Figure 3.2 (Top Panel) shows regression estimates from the unconditional model from Equation (3.1), tracing out mean HAZ differences across caste groups by age. Each point estimate is the coefficient of a caste group relative to the base category of UC children. The y-axis represents the coefficient estimates (and 95% confidence intervals) on the caste-group variables, and the x-axis separates the estimates into 6-month age-bins.

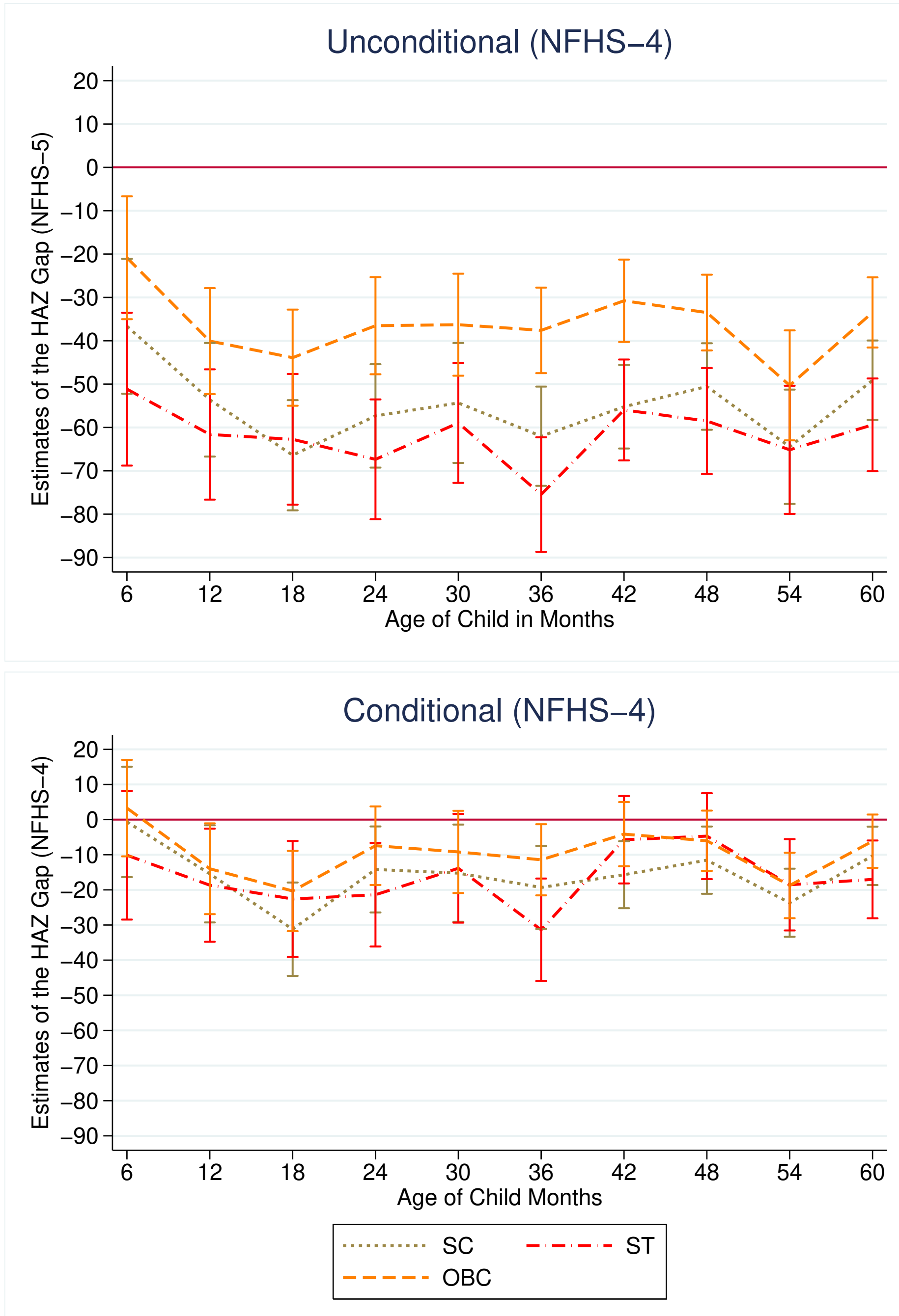


Figure 3.2: Regression Estimates of Caste HAZ Differentials by Age (0-60 months)

Notes: This figure presents regression estimates for both unconditional and conditional regressions in Equation (3.1). The y-axis provides the coefficient estimates (and confidence intervals) on the caste-group variables, and the x-axis separates the estimates into 6-month age-bins. The top panel presents regression estimates from the unconditional model. In the bottom panel, the conditional regression estimates include all controls related to health endowments, private investment, and public health. The regressions are weighted by survey weight and clustered at PSU level.

In the unconditional model, all children in lower caste and tribal minority groups are born smaller than UC children and experience post-birth growth rates that leave them much shorter than UCs by the time they have completed early childhood. OBC children start around 0.21 sd below UC children at birth. OBC children then grow less quickly than UC children after birth and average differences increase to around 0.38 sd by an OBC child's third birthday. These differences reduce to around 0.33 sd by the 4th year, providing some evidence of catch up in later years. SC children are born with HAZ scores around 0.37 sd below UC children. Over the first three years, they lose, on average, an additional 0.25 sd in HAZ, leading to a 0.62 sd difference by their third birthday. After that, heights improve slightly and by the child's fifth birthday they are on average 0.5 sd below a UC child. ST children experience the largest deficit of around 0.51 sd at birth. They face a large dip in HAZ by their 3rd birthday where differences grow to around 0.75 sd. By the time they are done with early childhood, ST children are 0.6 sd below UC children.

Figure 3.2 (Bottom Panel) provides conditional (regression adjusted) caste gap estimates controlling for covariates related to the health endowment, private investment, and public health variables described above. Including covariates reduces differences in HAZ gaps across all caste groups at all ages. STs, who face the largest unconditional HAZ gap relative to UCs, see reductions in the gap at birth of around 0.41 sd (to about 0.10 sd) once covariates are included. Adjusted differences in birth length differences are indistinguishable from zero. Adjusted differences increase to 0.3 sd by a child's third birthday, before settling to 0.2 sd by a child's fifth birthday. These estimates are consistent with the observable covariates explaining the ST birth length gap in its entirety, and between 65%–85% of the gap with UC children by age 5. Adjusted for observed covariates, SC and OBC children are also statistically insignificantly shorter than UC children at birth. However, as children age, differences begin to appear. Coefficients for OBCs children hover between 0 and 0.2 sd across most ages, with estimates for only 4 of the 10 periods statistically distinguishable from 0 at a 95% confidence level. Adjusted estimates for SCs show similar patterns with somewhat larger point estimates that hover between 0.05 to 0.31 sd, with 9 of the 10 estimates being significantly different from 0. Even still, covariate adjustment reduces the height gaps between OBCs and SCs with UCs by well over half.

One concern is that differential infant mortality across caste groups could be driving our estimates of the very early life caste gaps. Most children who die as children do so within the first six months of life. If lower caste children are more likely to die, and those that do die have differential potential HAZ across caste groups, this mortality effect would appear to us as part of a differential health endowment across caste groups. We provide two reasons this concern is not likely to prove fatal to our interpretation. First, in both theory and as documented around the world, child stunting is predictive of infant mortality (Olofin et al., 2013). In this case, our estimates of the caste HAZ deficits would likely be biased towards zero over the early period. Second, a recent paper by Panda (2020) argues that infant mortality risk is actually positively correlated with (potential) child HAZ in India. The study estimates that children who die would be approximately 17% taller (in units of HAZ) than the average child who lives, or approximately 0.34 sd at a mean HAZ of -2 sd. Meanwhile, Bora et al. (2019) estimates child mortality rates that are approximately 1 percentage point higher for SC/ST children than other children. Taken together, these would imply that absent differential mortality, mean HAZ for SC/ST children would be about 0.003 sd higher than we observe. This is approximately two orders of magnitude below our estimated caste gaps, and thus unlikely to greatly affect our inferences regarding the importance of health endowment deficits.

Regression Estimates by Gender and Location

The results above provide average correlations across the entire population, estimated as though the associations are similar for all groups of children. There are numerous reasons to believe this may not be the case: child growth faltering is different in rural and urban settings (Rieger and Trommlerová, 2016), gender differences in the effects of caste discrimination are assured (Islam et al., 2021; Deshpande, 2007), and different parts of India enforce and experience caste-based exclusionary social practices like untouchability in different ways (LoPalo et al., 2019; Coffey et al., 2019). **??** provides disaggregated estimates of unconditional and conditional caste gaps across three dimensions: child gender, rural/urban status, and across states of high and low density of UC Hindus.

The top row displays results when our models are estimated separately on girls and boys. Each graph shows unconditional (solid) and conditional (dashed) caste-gap estimates across child age for one gender, with girls on the left of the first row, and boys on the top right. There are large unconditional caste differentials for both boys and girls, on the order of 0.2–0.55 sd, and these gaps tend to grow in magnitude over the first two years of life. There is no noticeable difference in either the magnitude or the age-dynamics of the caste HAZ gaps across child gender. And as with the aggregate results, in both cases the unconditional estimates greatly attenuate and become, for the most part, statistically insignificant when controls are added.

The middle row divides the sample into rural (left graph) and urban (right graph) sub-samples according the NFHS definition. The results of the aggregate sample appear largely to represent attenuated dynamics in the rural areas. Rural children display the signature growth faltering dynamics seen in our aggregate results, but the growth faltering process is harder to characterize in the urban sample, in part due to noisier estimates from a smaller sample size. Urban and rural OBC children are born approximately 0.2 sd below UC children. Compared with UC children, SC and ST children in urban areas are born about 0.3 sd and 0.2 sd shorter respectively. In rural areas, these differences at birth are much larger, 0.35 sd (SC) and 0.55 sd (ST). The loss of HAZ over the first two years of life is common across all groups of children in rural areas, leading to HAZ gaps of around 0.6 sd for SC and ST children by age 2, and around 0.4 sd for OBCs. The loss of HAZ in the urban sample is more rapid over the first 18 months, dropping approximately an additional 0.4 sd for each group, but HAZ gaps immediately narrow in the next six months of life, and it is difficult to distinguish the extent to which massive changes from 18 to 24 months constitute real improvements or simply mean reversion in the estimates. In both areas, however, adjustment for covariates greatly attenuates the gaps towards zero at all ages, and most coefficients become statistically insignificant.

The final row of Figure 3.3 displays results across states with low (left graph) or high (right graph) share of UC children. This follows the intuition in Coffey et al. (2019), who divide their sample into regions with higher and lower concentrations of UC Hindus as a proxy for local discrimination, though we construct our sub-groups somewhat differently. We use the median of state-level shares of UC (∼24%) to divide the states into those with a high and low share of UC. While this process generates an almost equal number of states in each group, it does not generate equivalent sample sizes. Low UC share states contain almost 80% of the total sample observations, and the relatively noisy estimates for the high UC states are likely driven by this much smaller sample size.

Overall, the results for low UC states are more in line with the aggregate results as compared to the high UC states. Children from lower castes in low UC states are born 0.2–0.5 sd shorter than UC children. The growth faltering in low UC states is characteristic of the aggregate results, and these initial gaps increase by 0.2–0.3 sd over the first two years. The estimates also attenuate at every age after adjusting for covariates. On the other hand, in the high UC states, OBC children are no shorter than UC children at any age, even in the unconditional regressions. However, SC and ST children are between 0.2 sd and 0.6 sd shorter than UC children in the unconditional estimates, though the growth faltering process in these states is not visually apparent and the age-dynamics are indistinguishable from noise.

We note a few commonalities across the results and in relation to the aggregate results. First, the unconditional estimates for most sub-groups are of similar magnitude and statistically significant. UC children are born with better health than all types of sub-groups across India. Second, these differences also largely attenuate towards 0 in conditional regressions for all sub-groups, following the pattern in the full sample. Moreover, the growth faltering dynamics we focus on in our interpretation hold in the larger subsamples, while in the smaller sample sizes (Urban and High UC) we are unable to distinguish these dynamics from noise in our estimates.

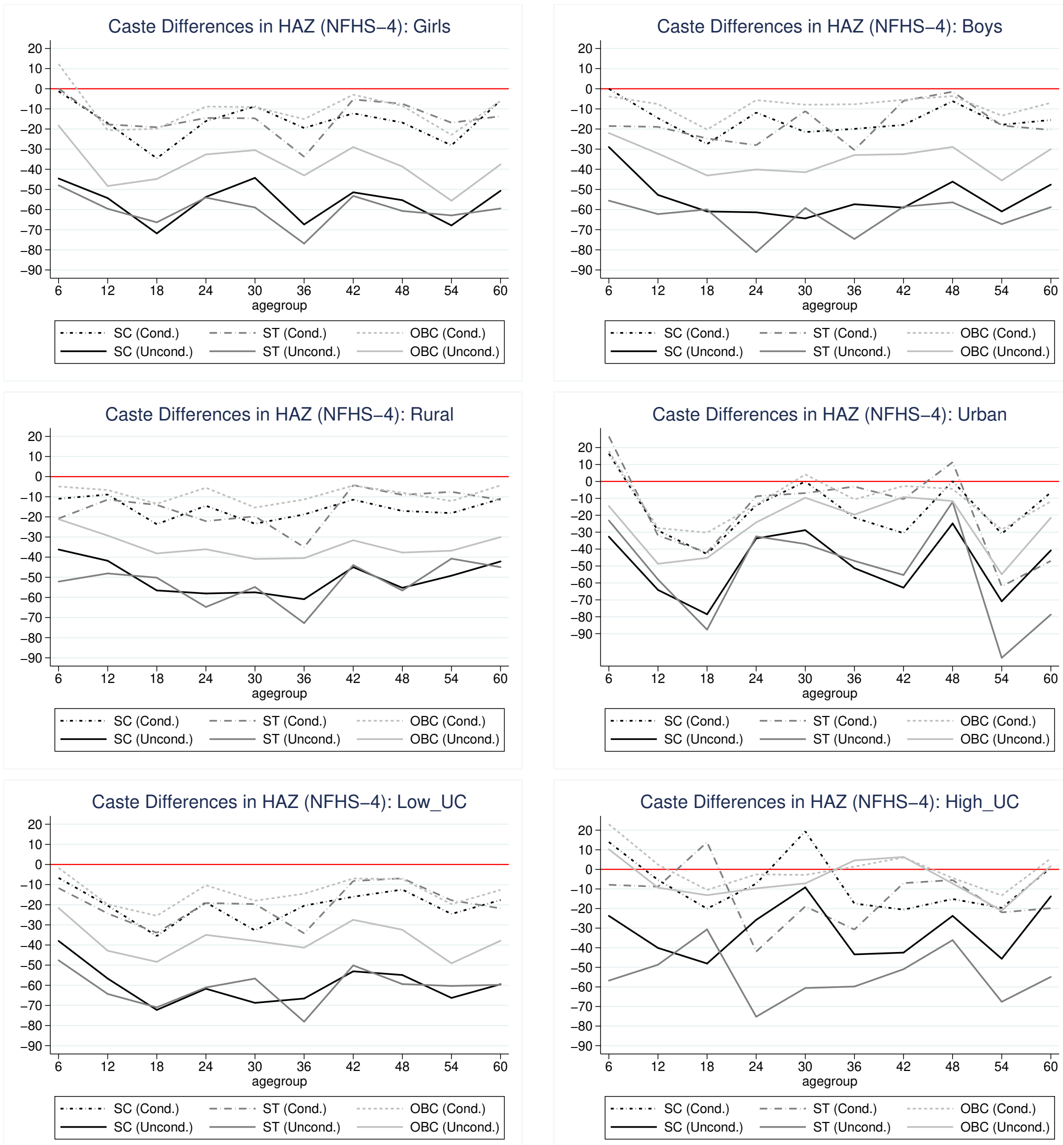


Figure 3.3: Replication of Individual-Level Results by Gender and Location (NFHS-4)

Notes: The graph on the top left panel plots the regression estimates for girls and the graph on the top right plots the regression estimates for boys. The graph in the middle panel on the left plots the regression estimates for rural children and the middle panel on the right plots the regression estimates for urban children. The bottom left panel has estimates from states with a below median share of UC children and the bottom right panel has estimates of the states with higher than median share. In each graph, the y-axis is the coefficient estimates on the caste-group variable, and the x-axis separates the estimates into 6-month age-bins. The solid lines presents regression estimates from the unconditional model and the dashed lines are estimates from the conditional model. Conditional regression estimates adjust for all controls related to endowment, private investment, and public health. The results are weighted by survey weight and clustered at PSU level.

Regression Estimates for Muslim Children

Our analysis thus far has focused exclusively on Hindu children. A natural question, though, is the extent to which the patterns we see in HAZ disparities among Hindu children resemble patterns in HAZ disparities with another marginalized group of Indians: Muslims. We thus replicate our analyses by comparing Muslim children with UC Hindu children. We provide a brief overview here, but elaborate further in Appendix A.4.

In Figure A.2 we see that the HAZ-age profiles demonstrate that Muslim children display a similar growth faltering dynamic as seen for low caste Hindu children. We then replicate our regression results in Figure A.3. Unconditional estimates in the top panel shows that Muslim children are around 0.2 sd smaller than UC children at birth. These differences increase to around 0.45 sd by age 5. Conditional estimates are small and statistically insignificant for the first 6 months. By age 2, Muslim children are around 0.25 sd shorter than UC children, and this gap maintains a relatively constant magnitude (and largely maintaining statistical significance) through age 5.

3.7.2 Intercept (α) and Slope (β) Estimates

An alternative way to investigate the age dynamics of HAZ caste disparities is to focus on the location and shape of the HAZ-age profile itself, and then estimate the determinants of that shape (Aiyar and Cummins, 2021). Results from Equation (3.3), estimating our measures of implied birth length and rate of growth, are presented in Table 3.2. Columns 1 through 4 in Table 3.2 provide our baseline estimates of caste differentials in the intercept (α) and slope (β) of the HAZ-age profile over the first two years of life. The first two columns present estimates for caste gaps in α and the second two columns provide estimates for β. Columns 1 and 3 present the unconditional estimates, where the second stage regression (Equation (3.3)) is estimated without covariates, and columns 2 and 4 provide estimates when district-caste-urban group level mean covariates and state-urban fixed-effects are included in the second stage.

Table 3.2: Rate of HAZ Loss and Caste (NFHS-4)

	(1) α b/se	(2) α(| X) b/se	(3) β b/se	(4) β(| X) b/se	(5) β(| α) b/se	(6) β(| X,α) b/se
SC	-46.65***	-4.72	-1.46**	-0.47	-3.90***	-0.74**
	(9.88)	(13.45)	(0.60)	(0.90)	(0.41)	(0.35)
ST	-54.80***	-8.68	-0.92	-0.83	-3.79***	-1.32***
	(15.23)	(16.31)	(0.86)	(1.03)	(0.49)	(0.39)
OBC	-30.67***	-1.53	-0.72	-0.36	-2.32***	-0.45*
	(8.32)	(10.63)	(0.57)	(0.70)	(0.35)	(0.24)
Mean	-37.3	-37.3	-7.8	-7.8	-7.8	-7.8
R Square	0.04	0.25	0.01	0.18	0.68	0.81
Weighted N	37959	37959	37959	37959	37959	37959
Real N	920	920	920	920	920	920

Notes: The results are weighted by numbers of individuals in each state-caste-urban cell and clustered at state-urban level. The covariates used include endowment, private investment, and public health variables. The state-urban fixed effects are included in the public health variables. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

Comparing results from columns 1 and 2 in Table 3.2 on birth length, we find that they are similar to those in the first age-bins of the individual-level regressions. SC children are on average born a statistically significant 0.47 sd shorter than the average UC child. Conditional on the inclusion of covariates, this difference reduces to 0.04 sd and is statistically insignificant. OBC children have a statistically significant average unconditional birth length deficit of 0.31 sd relative to UCs, a smaller gap compared to SC, but these differences reduce to 0.02 sd and are statistically insignificant once we condition on household covariates. Unconditionally, ST children have statistically significant birth length deficit estimated at 0.55 sd. Conditionally, this difference reduces to 0.09 sd and is statistically insignificant.

A similar picture emerges from the slope (β) estimates, where differences in growth rates are relatively large, and also largely attenuate when adjusted for observable group covariate means. Unconditional caste group point estimates in Column 3 are negative and statistically significant for SCs (-0.015 sd/month) but not statistically significant for OBCs and STs. The adjusted estimates in column 4 for SC and OBC estimates are insignificant and closer to 0.

Columns 5 and 6 in Table 3.2 provide a second set of estimates for β and represent the results of a slightly different motivation and thought experiment. We know from previous results that lower caste children are born shorter. If there is a natural relationship across all castes in which birth length affects rate of growth, then any attempt to capture a meaningful correlation between caste and relative rate of child growth (separate from a birth length effect) would need to condition on the birth length. In columns 5 and 6 we include $\hat{\alpha}$ as a control variable for β. The model implicitly allows the slope of the HAZ-age profile to vary based on the intercept, in a manner common across caste groups. This approach ensures that any differences in growth rates are not simply due to initial differences in birth length but reflect true variations in growth trajectories.

Column 5 provides estimates from a regression of $\hat{\beta}$ on caste dummy variables and group-specific $\hat{\alpha}$ and column 6 presents estimates conditional on including $\hat{\alpha}$ and our standard suite of covariates. Conditional on the intercept estimate, the slope estimates indicate large growth rate gaps between UC children and SC (-0.04 sd/month), OBC (-0.02 sd/month) and ST (-0.04 sd/month) children. These gaps would produce a cumulative effect of between -0.55 sd (for OBC) to -0.9 sd (for SC and ST) HAZ points by 24 months of age. Point estimates attenuate and remain statistically significant with the inclusion of group level covariates. Conditional on the intercept and covariates, the slope estimates are -0.007 sd/month for SC, -0.005 sd/month for OBC and -0.013 sd/ month for ST, a reduction on the order of 75% in magnitude relative to the unconditional estimates.

α and β Estimates by Gender and Location

In Table A.3 to Table A.8, we present the full sets of results from a disaggregation of the α and β estimates of caste gaps by gender and location. A caveat is that by restricting our analyses to district-urban cells with more than 20 observations, we lose a large portion of the sample within each sub-group analysis. We discuss this in more detail in Section 3.9, but we note that these results should be interpreted with some caution.

In relation to gender differences, we estimate a higher unconditional birth length for girls than boys (-0.32 sd compared to -0.53 sd) though the two groups have comparable rate of loss estimates of approximately -0.08 sd (Table A.5 and Table A.6). However, the unconditional caste gaps for both birth length and rate of loss are of a similar magnitude across gender. The conditional HAZ-caste gaps for α among girls range between 0.16 sd–0.27 sd but are not statistically different from UC children heights. The estimate of the growth rate for ST girls, conditional on both our usual covariates and the estimated birth length, is the only statistically significant conditional estimate and indicates a rate of loss of 0.002 sd per month (less than .05 sd cumulative by age 2), which is also the largest estimated group difference in β. For boys, conditional estimates for birth-length or rate of loss are not statistically different from 0.

As shown in Table A.5 and Table A.6, the caste gaps for rural areas are largely in line with the overall results, while again those for the urban areas are less consistent. Rural children are generally born shorter (mean $\hat{\alpha}$ of 0.38) than urban children (mean $\hat{\alpha}$ of 0.28), and also lose HAZ at a faster rate (mean $\hat{\beta}$ of -.08 sd/month for rural, relative to -.06 sd/month for urban). Unconditional HAZ-caste differences exist at birth in rural areas (0.3–0.5 sd) and urban areas (0.2–0.7 sd) but the urban estimates are generally imprecisely estimated. The rural birth length estimates attenuate greatly towards 0 and become insignificant when conditioning on observable variables and the urban sample birth length estimates become increasingly imprecise and in some cases increase in magnitude. The slope estimates for both rural and urban samples are similar to the overall results, with the largest implied gaps in rate of loss of HAZ appearing for urban SC and ST children. Conditioning on birth length, rural ST children grow more slowly than UC children (0.01 sd), but there is no measurable caste gap in growth rates for SC and OBC children. In the urban sample, SC children show significant growth rate gaps with UC children and these estimates are relatively large at around 0.02 sd/month.

Consistent with the results in subsection 3.7.1, the results for low UC states in Table A.7 are comparable with the aggregate results in Table 3.2, while those for the smaller sample in high UC states are not. A key difference is in the mean intercept results. Children in low UC states have a mean $\hat{\alpha}$ of -0.4 sd, but those in the high UC states have an estimated mean close to zero. This is likely a result of the relatively small sample size in high UC states, and not necessarily a feature of differential growth faltering. Estimates of the rate of loss of HAZ are similar across regions at around 0.07–0.08 sd/month for both groups. Unconditional HAZ caste gaps at birth vary from 0.14 sd to 0.25 sd in the low UC states, and are all statistically significant. In the high UC states, estimates of the birth length caste gaps are smaller and not statistically significant, but they are the opposite sign for ST children. Conditioning on covariates attenuates the estimates of birth length gaps by about half for the low UC regions, while in the high UC regions the estimates all become positive (but remain statistically indistinguishable from zero).

3.7.3 Decomposition Results

Figure 3.4 shows the Blinder-Oaxaca decomposition results, presented as the explained percentage of the unconditional height gap as described in Equation (3.4) and Equation (3.5). Child age binned into 6-month age groups is on the x-axis and the y-axis shows the share of the caste gap that can be explained by various groups of model covariates.

In general, well over half of the unadjusted caste HAZ differences can be explained by our observable covariate groups across all caste groups and all ages (top left panel). Birth length is almost fully explained, but during the critical year following, when child

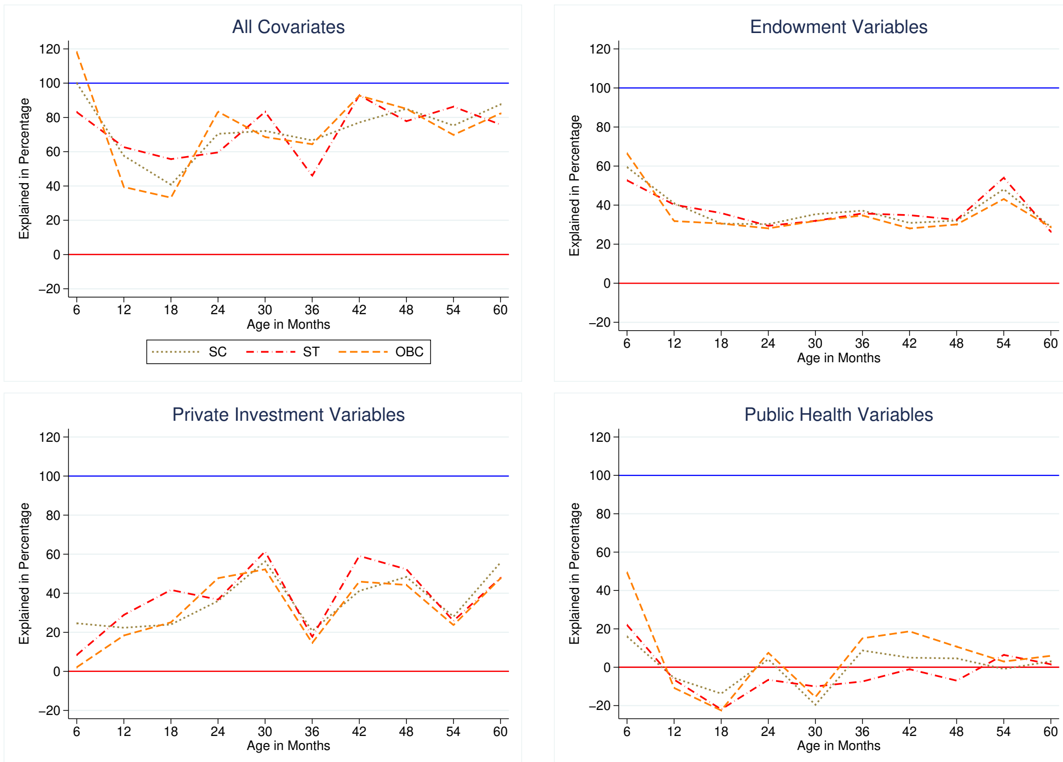


Figure 3.4: Blinder-Oaxaca Decomposition Results: Percent Explained

Notes: This figure presents Blinder-Oaxaca decomposition results. Child age measured in 6-month age groups is on the x-axis and the y-axis is the percent of the caste gap that can be explained by model covariates. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

HAZ drops most rapidly, the covariates lose some explanatory power. However, from 18 months onward, the model explains an increasing share of the caste HAZ gaps, so that over 80% of the gap with UC children is explainable by age 5 for every other caste group. Of additional interest is the fact that the dynamic effect of covariate explanatory power across age is consistent across caste groups as well — the functioning of the covariates on HAZ appears to be stable across all groups of children at any given age.

The remaining panels of Figure 3.4 decompose this explained variation into the percent of the total caste gap explained by each of our three families of explanatory variables.

As theory would predict, endowment variables explain the largest share of the explained variation in caste gaps for the youngest children in all caste groups. Private investment variables have almost no explanatory power over newborn caste HAZ gaps. As children age, the explanatory power of endowment variables remains relatively stable, decreasing only slightly to about 30% by age 5. The influence of private investment variables, though, begins to increase as children age. By age 5, half of caste HAZ differentials can be explained by the private investment related variables. Public health variables tend to have much smaller and inconsistent explanatory power over caste HAZ gaps. The only group for whom public health variables explain considerable fraction of the caste gap is for OBCs. The estimates, though, are difficult to interpret, given that location and public infrastructure effects are estimated to mostly exacerbate OBC deficits in the first two years.

In general, we find surprisingly small associations between public health variables and child HAZ, despite the well-known importance to child health of public goods like clean water and sanitation services. While public health environment is clearly an important determinant of child HAZ, it is not, according to our estimates, important to explaining caste HAZ gaps. There are two important caveats to this interpretation. The first is one of measurement: there is a general lack of high quality measures of public health inputs in our data, and we rely on regional indicator variables to capture much of any such effect. These regional variables are relatively large to allow for variation in our observable public health inputs at the community level, and thus may not capture imbalances in public health environment that are real in the world. The second is that even our direct local measures of public health – access to a sewer system and distance to health care facility – capture only the separate effect of having a community sewer system or health facility access, while the actual presence of a toilet in the house or health care visits for the child are ascribed to the private investment channel. Our work should not be interpreted as suggesting that public health is not an important determinant of child HAZ, but if it is important for explaining caste HAZ gaps, then it must be operating at either finer geographic levels and/or via inputs that are not observable in the data.

Decomposition Results by Gender and Location

We also provide decomposition results for the previously analyzed subgroups in the Appendix. The loss of sample size (about half in each sub-group analysis) leads to noisier estimates, but they largely mimic the aggregate results described above.

Figures A.5 and A.6 show that overall results differ slightly by gender. For both the groups, birth length is almost fully explained, after which the explanatory power of covariates falls up to the age of 18 months. By age 5, almost all of the caste HAZ gaps for girls and 60% of the caste HAZ gaps for boys are explained by included set of variables. Endowment variables explain the birth length entirely for girls, while accounting for 40%–65% of the caste HAZ gap in birth length for boys. Thereafter, the share explained remains stable at around 40% for girls and 20%–40% for boys at any age. The share explained by private investment variables starts low at birth and increases overtime for both genders. As before, the share of public health variables is null overall, with the exception that it explains a high share of the differences in the caste-height gap for OBC boys at their birth. These effects attenuate within the first 12 months.

Figures A.7 and A.8 present the results by rural and urban areas. Consistent with earlier results, the estimates from rural areas are similar to the aggregate results. The share of variation explained by endowment variables starts high and remains stable. The share of variation explained by private investment variables increases as children age. Public health variables continue to explain little of the HAZ caste gaps. Similar to the urban estimates of caste gaps themselves, our decomposition results do not reveal consistent patterns for urban children. This is likely attributable to both a decrease in sample size, and to the general differences in growth faltering patterns for urban children noted above.

In figures A.9 and A.10, we separate our results by the share of UCs in a state. Once again, consistent with findings in Section 3.7.1, the patterns for children in states with a low share of UCs (which accounts for 80% of the sample) are broadly consistent with the aggregate results. Birth length caste gaps are almost entirely explained by the included set of covariates up to 18 months. Endowment covariates have the highest explanatory share at birth and maintain a stable share at around 30% afterwards. The share explained for private investment begins at 20%–30% at birth and grows as children age. Public health variables, on the other hand, do not contribute in explaining the caste HAZ gaps. States with a high share of UC provide noisy estimates, driven by the relatively small sample size for these states.

Timing of Investments

One econometric concern about our estimates of the increasing influence of investment related variables is that, unlike endowment related variables such as maternal health, private investment variables are sometimes increasing in value, or probability, as children grow. Newborns do not receive many vaccinations, and children under the age of 6 months have only had so many health care visits. In order to determine the extent to which our increase in the explanatory power of private investment may be due simply to an increase in the magnitude or probability of age-determined covariates, we divide our Oaxaca-Blinder decomposition results for private investments into age-invariant and age-varying sub-types. We then graph out the relative contribution of each sub-type to the overall explanatory power of private investments across age in Figure 3.5.

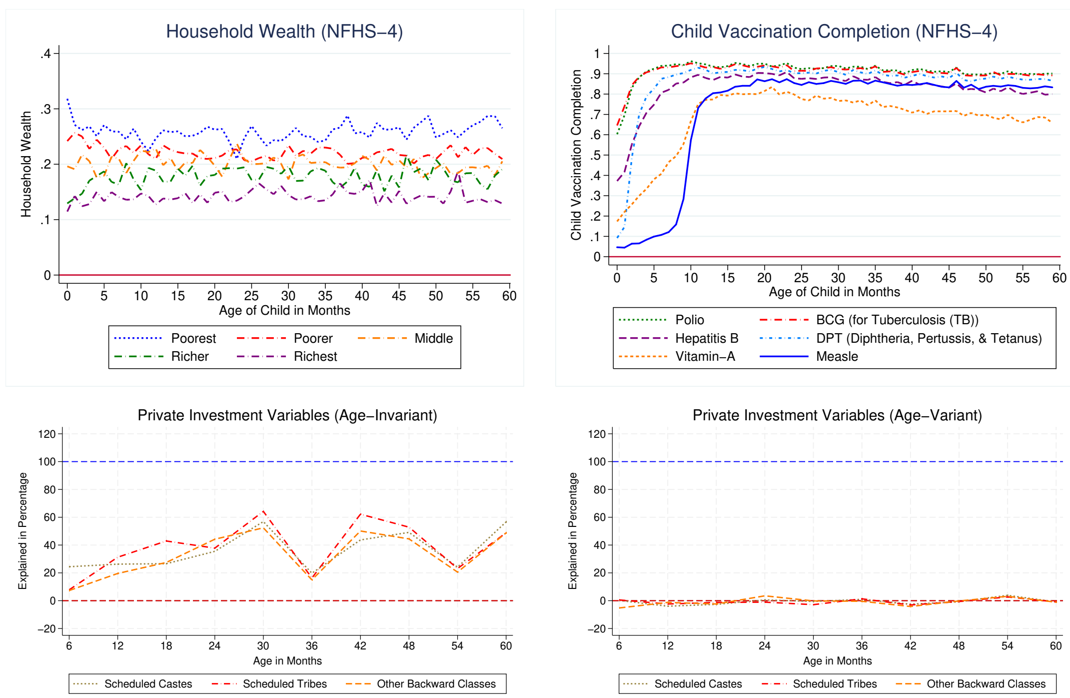


Figure 3.5: Age-Specific and Non-Age-Specific Private Investments

Notes: The top two panels of the figure shows the frequency of two particular private investment variables for children from 0 to 5 years. The top left panel shows the age-invariant nature of the household wealth measure of private investments across child age, while the top right panel shows the age-specific frequency of vaccination status by child age. The bottom row displays Blinder-Oaxaca decomposition results separately for age-varying and age-invariant private investment variables. Child age measured in 6-month age groups is on the x-axis and the y-axis is the percent of the caste gap that can be explained by model covariates. The bottom left panel presents the percent of the HAZ gap explained by the private investment variables that are age-invariant. The bottom right panel depicts the percent of the HAZ gap explained by the private investment variables that vary by age.

The top row of Figure 3.5 graphs the outcome-age profile for two representative variables. The upper left panel graphs the age-profile of an important age-invariant private investment, the fraction of children in households at each asset quintile across child age. Household wealth is not correlated with child age, at least for very young children. On the other hand, one can essentially infer the vaccination schedule for children from the top right panel, which graphs the age-profile of take-up of various vaccines, a representative age-varying private investment. If age-varying measures like vaccinations are driving the explanatory power of private investments, then the apparent increase in the explanatory power of private investment variables could, in theory, be simply the result of the increasing take-up of vaccines as children age. In that case, the estimates of increasing explanatory power of private investments would be driven by some of them simply turning on.

The bottom row of Figure 3.5 shows the Oaxaca-Blinder decomposition for the suites of age-invariant and age-varying private investment variables (estimated from the full model, but with their contributions accounted for separately). The left panel shows that the private investment effects are mostly driven by age-invariant private investment and that age-varying variables such as vaccinations and prenatal care do not explain much of the caste HAZ gaps.

3.8 Replication Using NFHS-5

We replicate the analyses above using the most recent wave of the NFHS conducted in 2019–20 (NFHS-5), which provides an independent sample of over 250,000 children. Our initial choices over the sample selection rules and regression model specifications employed in this paper, along with the economic interpretation of our results, were developed in 2021 and were presented at several conferences, using data exclusively from NFHS-4. Subsequently, data from the next round of the NFHS-5 was made available to researchers. We then replicated the results from the NFHS-4 on data from the newly available NFHS-5, using the same methods and (to the extent possible) the same regression specifications as before. That is, we used the methods, specifications and interpretation from our results using NFHS-4 as a kind of *de facto* pre-analysis plan for analyzing the NFHS-5 data.

We choose to present the main analysis of the paper using NFHS-4 because the data from the NFHS-5 wave is not complete for some of the variables. Specifically, NFHS-5 only provides information on health inputs (e.g. vaccination and pre-natal check status) for children up to the age of 3 years whereas we have complete information for children up to the age of 5 years in NFHS-4. The sample that answered the vaccination (Polio, DPT, Hepatitis B, Measles, BCG) question also changed in NFHS-5. Nevertheless, using this newer dataset, we can replicate a majority of the analyses conducted on the NFHS-4.

Figure 3.6 presents a comparison between the results from the NFHS-4 (left column) and NFHS-5 (right column). The patterns in the HAZ-profiles (first row) are quite similar between the two waves. Children from lower castes are shorter than upper caste children at birth and these differences get larger as children age. Similar to NFHS-4, SC and ST children in the NFHS-5 face the largest HAZ disparities. In the second row, we present the unconditional regression estimates of caste group differences in HAZ. Compared to NFHS-4, there appears to be a small secular decrease in unconditional caste gaps overall, with all lower caste groups seeing smaller unconditional gaps relative to UC children. In the NFHS-5 sample, differences in height at birth between OBC children and UC children have become statistically insignificant, while the gaps for SC and ST children remain significant but are relatively smaller than in the previous round, decreasing from 0.4–0.5 sd in the NFHS-4 to 0.2–0.3 sd in the NFHS-5. This upward level shift of the HAZ-age profile across rounds persists through the growth faltering process, and HAZ caste gaps from age 2 onwards are all about 0.1 sd smaller in the newer round of data. Conditional estimates, presented in the bottom panel, are more similar across rounds, and again indicate that caste differences in HAZ can be explained by observable characteristics of households, similar to the NFHS-4 results above and results from other research using NFHS-2 and NFHS-3 (Van de Poel and Speybroeck, 2009; Coffey et al., 2019).

Similarly, in Table 3.3, we replicate our earlier findings on implied birth length and rate of growth from Table 3.2 with results from NFHS-5 data. By and large, the patterns are similar across the two rounds in sign and magnitude but some differences emerge. Mean implied birth length (α) is actually lower in NFHS-5 (-.45 sd compared to -.37), but mean rates of loss (β) are lower as well (-0.06 sd/month compared to -0.08 sd in NFHS-4). On the other hand, the unconditional caste gaps in HAZ at birth decline over time (-0.31 sd to -0.55 sd in NFHS-4 as compared to -0.2 sd to -0.4 sd in NFHS-5). Conditional on covariates, the results from NFHS-5 follow those from NFHS-4 and all differences in birth-lengths become less than 0.1 sd and statistically insignificant. The estimated gaps for rate of loss of HAZ across caste groups are relatively stable across rounds. For ST children, the gaps in unconditional rate of loss increases from 0.009 sd in NFHS-4 to 0.012 sd in NFHS-5, while the unconditional slope gap estimates are small and statistically insignificant for SC and OBC children. Comparing columns 5 and 6, the slope estimates conditional on $\hat{\alpha}$, there is no meaningful change in the estimated gaps in β across rounds.

The striking similarity of results over the two waves indicates an amount of temporal stability in the child growth dynamics we originally observed in the NFHS-4, even when applying a *de facto* pre-defined regression model to the NFHS-5 data. This eases concerns related to cherry picking results or regression model specifications. This temporal stability exists over and above the similarities in dynamics across caste groups within each wave. Both of these layers of stability – within and between survey waves – lend credence to our argument that the differential growth processes across caste that we identified in the 2015 data are meaningful beyond just a particular sample at a particular moment in time.

3.9 Limitations

Our empirical analysis faces limitations, including our interpretation of results and our ability to estimate our models with sufficient precision.

First, to interpret our decomposition results as reflecting exact, true underlying contributions of each variable group, we would need a fully specified structural model of child health and measures of all the relevant inputs. We provide only a stylized growth model with three sets of key determinants, along with the relevant measures for such determinants that are observable in our data. In cases where our variable groups have rich information on key determinants, as for maternal health or household wealth, our estimates may be more reasonable. However, our measurement and empirical definition of public health inputs are both unorthodox and imperfect. We acknowledge that a large number of public health determinants of child HAZ may remain unmeasured and thus not accounted for in our decomposition estimates.

Second, data requirements for α/β slope and intercept models are high for both the number of geographic cells and the number of observations required per cell. This creates two important limitations for our work. The first is that sub-group estimates of slope and intercept coefficients we provide are imprecise and provide less insight than we would hope. The second is that we are unable to statistically decompose the changes in the intercept and slope coefficients as we can with the fixed-effects estimates. This is due to two factors. There is the large number of covariates, including state-urban fixed-effects, relative to the number of district-urban cells. But more fundamentally, these estimates rely almost exclusively on within-cell variation; they are designed to compare two HAZ-age profiles from the same region but from different caste groups. This puts tremendous importance on the locational fixed-effects, and makes it difficult to pin down the effects of other important covariates, including and in particular those associated with public health.

Table 3.3: Rate of HAZ Loss and Caste (NFHS-5)

	(1)	(2)	(3)	(4)	(5)	(6)
	α	α(| X)	β	β(| X)	β(| α)	β(| X,α)
	b/se	b/se	b/se	b/se	b/se	b/se
SC	-37.09***	-12.54	-1.35**	-0.56	-3.44***	-1.27**
	(8.02)	(12.75)	(0.60)	(0.83)	(0.55)	(0.50)
ST	-40.46***	-11.91	-1.20*	-0.69	-3.47***	-1.36**
	(11.40)	(14.26)	(0.69)	(0.92)	(0.51)	(0.60)
OBC	-20.38**	-4.03	-0.71	-0.37	-1.86***	-0.60
	(8.65)	(12.15)	(0.58)	(0.77)	(0.45)	(0.43)
Mean	-45.5	-45.5	-6.2	-6.2	-6.2	-6.2
R Square	0.02	0.13	0.00	0.16	0.69	0.77
Weighted N	33133	33133	33133	33133	33133	33133
Real N	836	836	836	836	836	836

Notes: The results are weighted by numbers of individuals in each district-caste-urban cell and clustered at state-urban level. The covariates used include endowment, private investment, and public health variables. The state-urban fixed effects are included in the public health variables. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

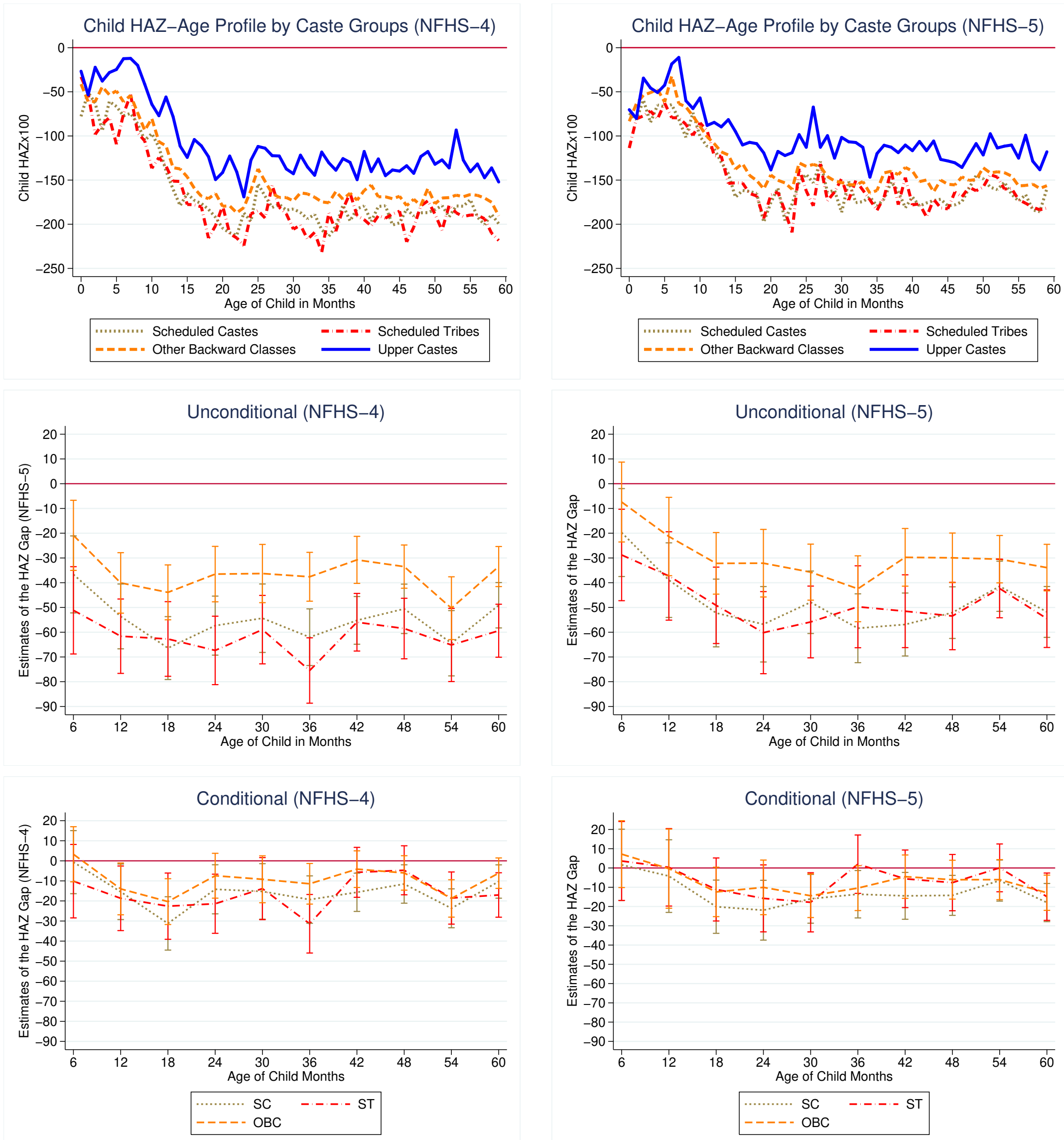


Figure 3.6: Replication of Individual-Level Results: NFHS-4 versus NFHS-5

Notes: The first row graphs mean child HAZ score (x100) by caste groups for children from 0 to 5 years. The left column shows estimates using the NFHS-4 (2015-2016) and the right columns shows the results from the NFHS-5 (2019-2021). The x axis represents age in months and the y axis is the weighted HAZ. The second and third rows graph the regression estimates for both unconditional and conditional regressions as well as the explanatory power. For the bottom two rows, the y-axis is the coefficient estimates (and confidence intervals) on the caste-group variable, and the x-axis separates the estimates into 6-month age-bins. The middle row presents regression estimates from the unconditional model. The gray dotted line is the difference of SC and UC children, the red dashed-dotted line represents differences between ST and UC children, and the orange dashed line represents differences between OBC and UC children. In the bottom row, the conditional regression estimates adjust for all controls related to endowment, private investment, and public health. The results are weighted by survey weight and clustered at PSU level.

3.10 Conclusion

Child HAZ gaps across caste groups in India are significant. In line with previous literature, we confirm that caste HAZ gaps remain large and are largely explainable by observable differences in households across caste groups (Coffey et al., 2019; LoPalo et al., 2019; Van de Poel and Speybroeck, 2009). Our framework, though, reveals new features of these caste gaps. We document that these caste gaps are present at birth and grow in size, particularly over the first two years of life. Our decomposition results suggest that observable characteristics of households related to both health endowments and (private) health investments matter significantly in explaining the differential child growth patterns across caste groups, and do so in a manner that changes as children age. Endowment related variables largely explain birth length HAZ gaps across castes, and private investments become increasingly important in explaining those gaps as children age. By the age of 5, the two variable groups each explain around half of the HAZ disparities across caste groups. These patterns are remarkably similar in both the fourth and fifth waves of the NFHS.

Our findings imply two things. First, persistent effects of endowment related variables as children age imply that historical factors affecting maternal physiology are likely to be nearly as important in generating caste HAZ gaps as contemporary factors (both economic and social). Second, the increasing importance of post-birth child investments as children age indicates that lower caste children face real disadvantages that disproportionately and negatively affect their growth and development even today. And while our results are consistent with both discriminatory and non-discriminatory, and both contemporary and historical factors, they also cast doubt on the ability of public policy to remediate HAZ caste gaps in the short-term. Health endowments do not change after birth, and remedying the health endowment disparities across castes is likely to be a multi-generational process.

Appendix

A.1 Data Availability Across NFHS-4 and NFHS-5

The National Family Health Survey 5 (NFHS-5) contains information on population, health, and nutrition for India from 2019-2021. Similar to NFHS-4, NFHS-5 includes district-level data for important variables. The total sample size of NFHS-5 is around 610,000 households. Variables collected in NFHS-5 are similar to the variables in NFHS-4, but with slight differences.

For endowment variables group, we use the same variables of birth order, maternal age, squared maternal age, maternal HAZ, maternal WAZ. However, 30% of delivery care data is missing. For the private investment variables, we include comparable variables such as wealth status, maternal education, motorcycle, own land, toilet type. To overcome the lack of data on water treatment status we utilize a water source variable. We divide this up into private water access and public water access at the home level. The major differences is the access to vaccination variables. This information was not available for children: 1. Who were born before 2016; 2. Who were not the "last birth, next to last birth, or second to last birth" child; 3. Who died during any stage of this. In other words, the NFHS-5 questionnaire only collect information on vaccine for last 3 births but has more restrictions than the NFHS-4. Thus vaccination information from many older children (33 months and above) was not available and hence comparable across surveys. For the public investment variables, we used sewer system access at PSU level, distance to health facilities, state-urban fixed effects.

Table A.1: Unconditional Caste Differences in HAZ (NFHS-4)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	0-6	6-12	12-18	18-24	24-30	30-36	36-42	42-48	48-54	54-59
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
SC	-36.65***	-53.61***	-66.41***	-57.34***	-54.34***	-62.02***	-55.22***	-50.55***	-64.45***	-49.10***
	(7.94)	(6.68)	(6.48)	(6.08)	(7.06)	(5.84)	(4.92)	(5.09)	(6.73)	(4.68)
ST	-51.16***	-61.61***	-62.73***	-67.36***	-58.96***	-75.47***	-55.96***	-58.51***	-65.15***	-59.40***
	(9.00)	(7.67)	(7.69)	(7.05)	(7.05)	(6.74)	(5.94)	(6.24)	(7.54)	(5.46)
OBC	-20.86***	-40.08***	-43.90***	-36.52***	-36.30***	-37.60***	-30.77***	-33.49***	-50.29***	-33.46***
	(7.23)	(6.24)	(5.66)	(5.71)	(6.00)	(5.03)	(4.85)	(4.46)	(6.47)	(4.13)
Observations	11811	15053	14470	14933	14493	14917	15546	15641	14705	15209

Notes: Age is measured in months and one age group is 6 months. p-values: * 0.10, ** 0.05, *** 0.01.

A.2 Alpha / Beta Estimates

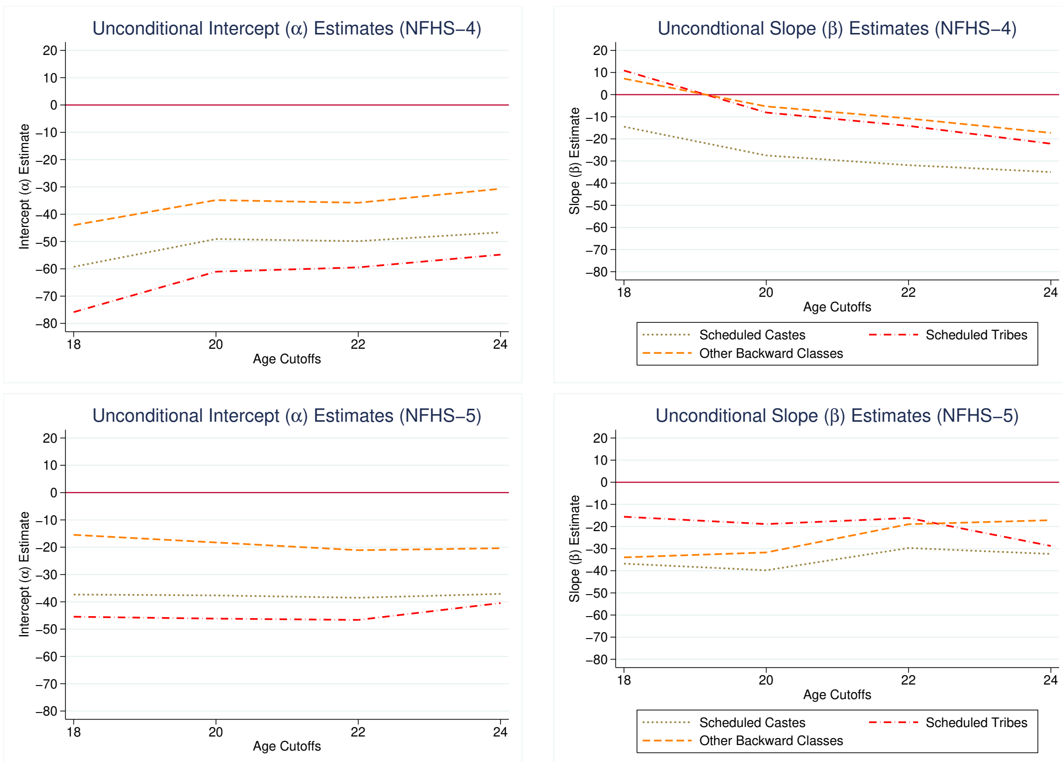


Figure A.1: α/β Estimates by Age Cutoffs

Notes: This figure shows the robustness of estimated coefficients on caste group dummies for the α and β regressions across in Equation (3.3) by age cutoff for NFHS 4 and NFHS 5 data. The x axis represents the age cutoff in months. The left panel presents the unconditional estimates for birth length ($\hat{\alpha}$). The right panel presents the unconditional estimates for the rate of loss ($\hat{\beta}$). The results are weighted by numbers of individuals in each state-caste-urban cell and clustered at state-urban level.

A.3 Oaxaca Blinder Decompositions

Table A.2: Explained in Percentage for Blinder-Oaxaca Decomposition

	All			Endowment			Private			Public		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	SC	ST	OBC	SC	ST	OBC	SC	ST	OBC	SC	ST	OBC
	mean	mean	mean	mean	mean	mean	mean	mean	mean	mean	mean	mean
Child Age = 6m	100.08	83.12	118.08	59.45	52.69	66.51	24.62	8.39	2.10	16.02	22.04	49.47
Child Age = 12m	57.61	62.67	39.41	40.89	40.23	31.83	22.33	28.90	18.37	-5.61	-6.46	-10.79
Child Age = 18m	40.77	55.66	33.18	30.52	35.92	30.57	23.98	41.69	25.11	-13.73	-21.95	-22.50
Child Age = 24m	70.44	59.59	83.35	30.25	29.39	28.09	36.03	36.75	47.71	4.16	-6.55	7.54
Child Age = 30m	72.13	83.31	68.57	35.34	31.93	31.89	56.34	61.37	52.22	-19.55	-10.00	-15.55
Child Age = 36m	66.59	46.00	64.36	37.18	35.74	34.71	20.69	17.67	14.55	8.72	-7.41	15.11
Child Age = 42m	77.08	92.93	92.72	30.89	34.85	28.06	41.20	59.11	45.95	5.00	-1.03	18.71
Child Age = 48m	85.04	77.80	85.06	32.09	32.50	30.10	48.35	52.28	44.24	4.60	-6.97	10.72
Child Age = 54m	75.28	86.28	69.80	48.16	54.01	43.12	28.01	25.84	23.70	-0.89	6.43	2.98
Child Age = 60m	87.66	75.81	82.35	28.84	26.38	28.73	55.75	47.89	47.67	3.07	1.54	5.96
Total	73.27	72.32	73.69	37.36	37.37	35.36	35.73	37.99	32.16	0.18	-3.04	6.17

Notes: This table shows the results of Blinder-Oaxaca Decomposition in terms of explained in percentage. Child age is measures in months.

A.4 HAZ differences between Muslim & UC children

Figure A.2 summarizes the mean HAZ across child age in months for Muslim children and upper caste children from the NFHS-4 data. Muslim children are, on average, born smaller than upper caste children and grow at a slower rate. In the first 6 months, UC children seem to recover rapidly.

In Figure A.3, the top and bottom panel shows regression estimates from the unconditional and conditional regression models. Each point estimate in Figure A.3 is the coefficient of Muslim children relative to upper caste children (UC). In the unconditional model, Muslim children are around 0.2 sd smaller than UC children at birth. These differences increase to around 0.45 sd by age 5. In the bottom panel, religion gap estimates lower after controlling for covariates related to the health endowment, private investment, and public investment. There are no differences in the first 6 months and these differences increase to 0.3 sd in the first 12 months. These changes support the issue of selective mortality that UC children face as discussed in Bhalotra et al. (2010). By age 2, Muslim children are around 0.1 sd shorter and this gap remains a constant over the rest of their age.

Figure A.4 shows the Blinder-Oaxaca decomposition results explained in percentage as described in Equation 3.4 and Equation 3.5 for Muslim children when compared to UC children. In the top left panel, we see that more than 50% of the religion gaps in birth length can be explained by covariates. One third of the height differences between Muslim children and UC children can be explained by their endowment covariates. These shares are decreasing over the child's age. In the bottom left panel, around 80% of the differences at birth can be explained by differences in private investment variables. These differences decrease by 12 months and then increase by age. By age 5, around two thirds of the differences can be explained by differences in private investment variables across groups. Public health variables explain almost no share of the differences.

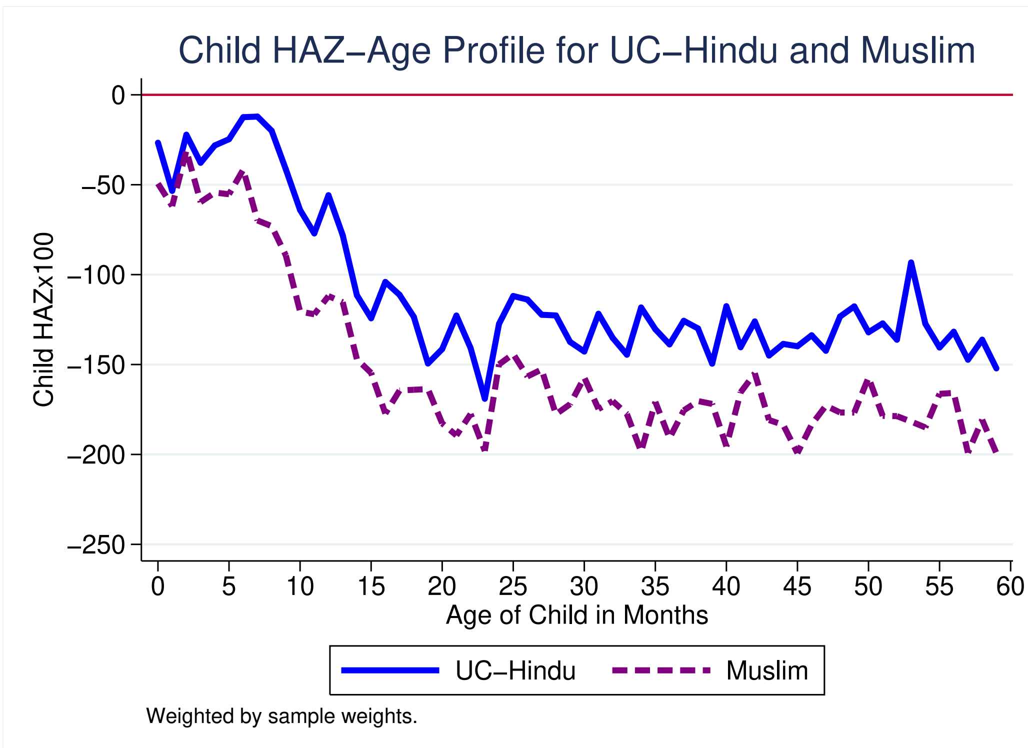
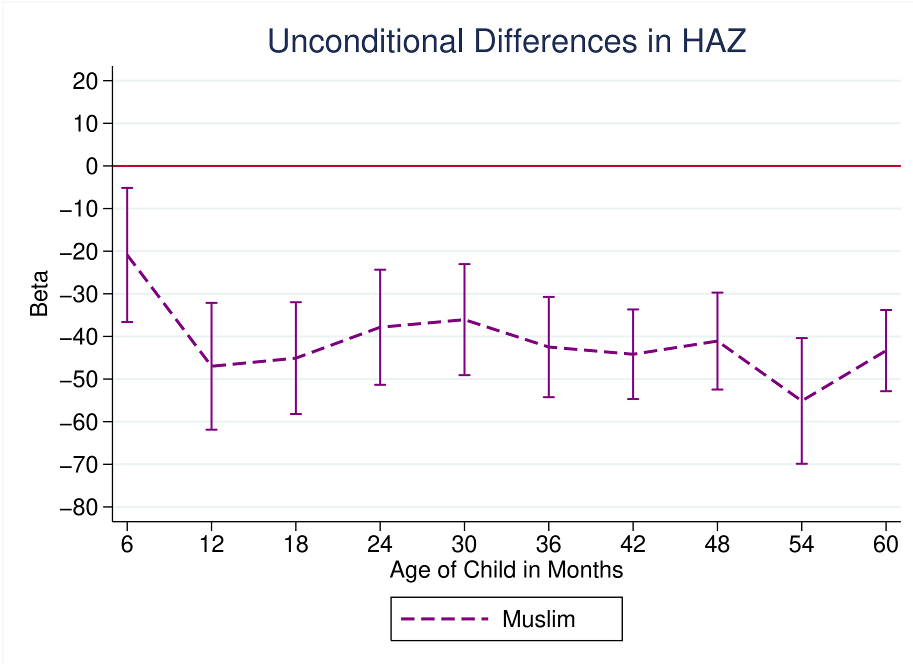


Figure A.2: Child HAZ for UC Hindu and Muslim

Notes: This figure presents child HAZ score by religion (UC-Hindus and Muslims). The x-axis represents age in months and the y-axis is the weighted mean HAZ. The results are weighted by survey weights.



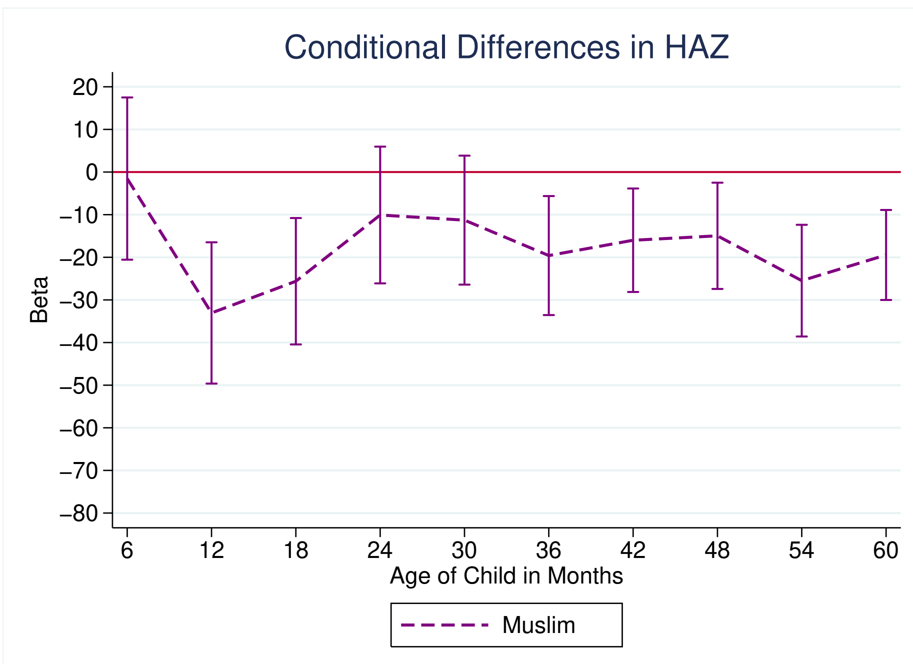


Figure A.3: Regression Estimates (UC Hindu vs. Muslims)

Notes: This figure presents regression estimates for both unconditional and conditional regressions in **??**, with non-UC Hindus excluded and Muslim children comprising the group of interest. The y-axis is the coefficient estimates (and 95% confidence intervals) on the caste-group variable, and the x-axis separates the estimates into 6-month age-bins. The top panel presents regression estimates from the unconditional model. In the bottom panel, the regression (conditional) estimates include all controls related to endowment, private investment, and public health. The results are weighted by survey weights and clustered at PSU level.

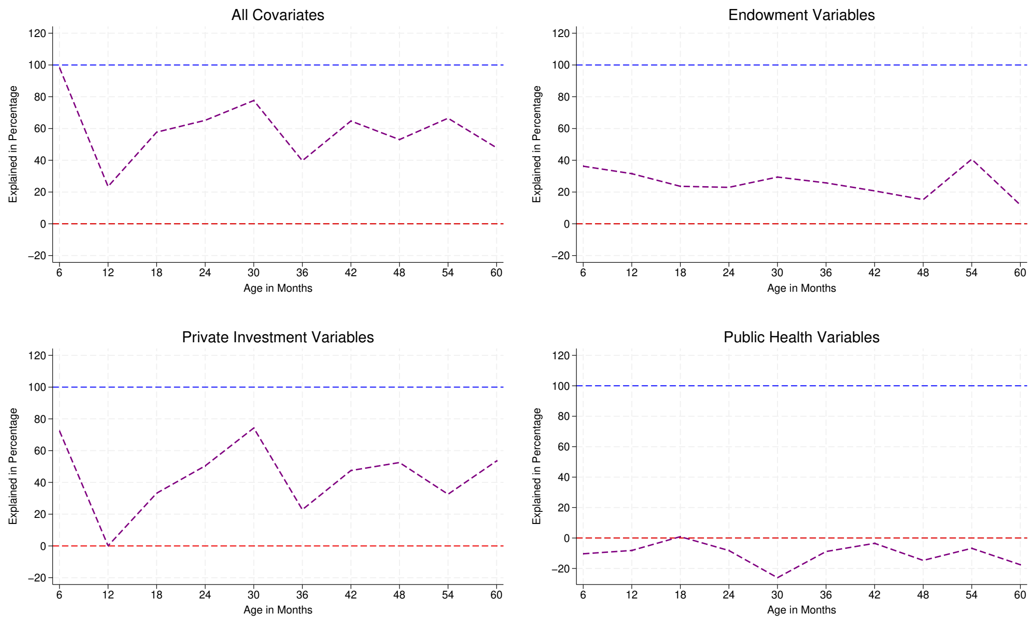


Figure A.4: Blinder-Oaxaca Decomposition Results (UC Hindu vs. Muslims)

Notes: This figure presents Blinder-Oaxaca decomposition results for Muslim children relative to UC-Hindu children. Child age measured in 6-month age groups is on the x-axis and the y-axis is the percent of the religion gap that can be explained by model covariates. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

A.5 Alpha / Beta (AB) Estimates: Additional Analyses

A.5.1 AB Estimates By Gender

Table A.3: Rate of HAZ Loss and Caste (NFHS-4): Girls

	(1)	(2)	(3)	(4)	(5)	(6)
	α	$\alpha(\mid X)$	β	$\beta(\mid X)$	$\beta(\mid \alpha)$	$\beta(\mid X,\alpha)$
	b/se	b/se	b/se	b/se	b/se	b/se
SC	-51.15***	26.27	-2.03*	-2.34	-4.78***	-0.84
	(17.95)	(36.08)	(1.12)	(2.44)	(0.71)	(0.83)
ST	-70.97***	18.62	-0.40	-2.73	-4.22***	-1.67*
	(20.62)	(34.84)	(1.03)	(2.25)	(0.61)	(0.85)
OBC	-48.57***	16.40	-0.34	-1.52	-2.95***	-0.59
	(14.03)	(30.53)	(0.75)	(1.91)	(0.66)	(0.77)
Mean	-32.0	-32.0	-8.0	-8.0	-8.0	-8.0
R Square	0.04	0.31	0.01	0.26	0.70	0.82
Weighted N	10576	10576	10576	10576	10576	10576
Real N	339	339	339	339	339	339

Notes: The results are weighted by numbers of individuals in each district-caste-urban cell and clustered at state-urban level. The covariates used include endowment, private investment, and public health variables. The state-urban fixed effects are included in the public health variables. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

Table A.4: Rate of HAZ Loss and Caste (NFHS-4): Boys

	(1)	(2)	(3)	(4)	(5)	(6)
	α	$\alpha(\mid X)$	β	$\beta(\mid X)$	$\beta(\mid \alpha)$	$\beta(\mid X,\alpha)$
	b/se	b/se	b/se	b/se	b/se	b/se
SC	-51.35*	-0.80	-2.32	-0.95	-5.23***	-1.00
	(25.69)	(23.76)	(1.67)	(1.51)	(0.55)	(0.62)
ST	-84.86***	-34.72	-0.40	1.19	-5.20***	-0.85
	(22.67)	(32.89)	(1.54)	(1.83)	(0.53)	(0.80)
OBC	-32.52	4.95	-1.30	-0.54	-3.14***	-0.24
	(19.24)	(23.64)	(1.37)	(1.29)	(0.43)	(0.67)
Mean	-53.4	-53.4	-7.8	-7.8	-7.8	-7.8
R Square	0.08	0.35	0.02	0.28	0.72	0.83
Weighted N	12817	12817	12817	12817	12817	12817
Real N	407	407	407	407	407	407

Notes: The results are weighted by numbers of individuals in each district-caste-urban cell and clustered at state-urban level. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

A.5.2 AB Estimates By Location

Table A.5: Rate of HAZ Loss and Caste (NFHS-4): Rural

	(1) α b/se	(2) α(| X) b/se	(3) β b/se	(4) β(| X) b/se	(5) β(| α) b/se	(6) β(| X,α) b/se
SC	-49.32***	-9.21	-1.06	0.10	-3.64***	-0.43
	(10.77)	(14.09)	(0.67)	(0.91)	(0.44)	(0.37)
ST	-56.20***	-11.34	-0.51	-0.49	-3.45***	-1.15***
	(16.10)	(17.33)	(0.94)	(1.07)	(0.54)	(0.37)
OBC	-30.19***	-1.80	-0.63	-0.17	-2.21***	-0.28
	(9.64)	(11.56)	(0.65)	(0.73)	(0.40)	(0.22)
Mean	-38.3	-38.3	-8.0	-8.0	-8.0	-8.0
R Square	0.04	0.25	0.00	0.16	0.68	0.80
Weighted N	33993	33993	33993	33993	33993	33993
Real N	790	790	790	790	790	790

Notes: The results are weighted by numbers of individuals in each district-caste-urban cell and clustered at state-urban level. The covariates used include endowment, private investment, and public health variables. The state-urban fixed effects are included in the public health variables. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

Table A.6: Rate of HAZ Loss and Caste (NFHS-4): Urban

	(1) α b/se	(2) α(| X) b/se	(3) β b/se	(4) β(| X) b/se	(5) β(| α) b/se	(6) β(| X,α) b/se
SC	-18.77	-8.02	-3.11*	-1.75	-4.10***	-2.17**
	(20.39)	(23.74)	(1.51)	(1.81)	(0.60)	(1.04)
ST	67.26	114.18	-5.97*	-5.55	-2.45***	0.30
	(69.47)	(88.62)	(3.23)	(3.68)	(0.77)	(1.95)
OBC	-35.67**	-39.92*	-0.11	1.06	-1.98***	-0.99
	(16.61)	(21.14)	(1.29)	(1.88)	(0.60)	(1.16)
Mean	-28.5	-28.5	-6.2	-6.2	-6.2	-6.2
R Square	0.04	0.50	0.05	0.52	0.76	0.87
Weighted N	3966	3966	3966	3966	3966	3966
Real N	130	130	130	130	130	130

Notes: The results are weighted by numbers of individuals in each district-caste-urban cell and clustered at state-urban level. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

A.5.3 AB Estimates By region

Table A.7: Rate of HAZ Loss and Caste (NFHS-4): Low UC

	(1) α b/se	(2) α(| X) b/se	(3) β b/se	(4) β(| X) b/se	(5) β(| α) b/se	(6) β(| X,α) b/se
SC	-44.03***	-20.59	-1.38	0.27	-3.69***	-0.89**
	(12.03)	(12.16)	(0.89)	(0.91)	(0.49)	(0.37)
ST	-54.07***	-25.61	-0.40	0.07	-3.24***	-1.37***
	(18.31)	(15.44)	(1.30)	(1.05)	(0.70)	(0.41)
OBC	-24.42**	-14.13	-0.52	0.31	-1.80***	-0.49
	(10.95)	(10.27)	(0.89)	(0.75)	(0.46)	(0.29)
Mean	-42.4	-42.4	-7.9	-7.9	-7.9	-7.9
R Square	0.04	0.23	0.01	0.18	0.68	0.80
Weighted N	32358	32358	32358	32358	32358	32358
Real N	750	750	750	750	750	750

Notes: The results are weighted by numbers of individuals in each district-caste-urban cell and clustered at state-urban level. The covariates used include endowment, private investment, and public health variables. The state-urban fixed effects are included in the public health variables. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

Table A.8: Rate of HAZ Loss and Caste (NFHS-4): High UC

	(1) α b/se	(2) α(| X) b/se	(3) β b/se	(4) β(| X) b/se	(5) β(| α) b/se	(6) β(| X,α) b/se
SC	-27.44	25.34	-0.71	-0.96	-2.22***	0.61
	(17.04)	(23.49)	(1.27)	(1.57)	(0.42)	(0.73)
ST	33.73	8.87	-4.96***	-1.06	-3.09**	-0.51
	(32.25)	(30.72)	(1.07)	(2.01)	(1.41)	(1.10)
OBC	-21.96	0.70	-0.14	-1.48	-1.36**	-1.43***
	(14.62)	(15.10)	(0.82)	(1.17)	(0.56)	(0.45)
Mean	-7.5	-7.5	-7.4	-7.4	-7.4	-7.4
R Square	0.04	0.39	0.05	0.33	0.73	0.88
Weighted N	5601	5601	5601	5601	5601	5601
Real N	170	170	170	170	170	170

Notes: The results are weighted by numbers of individuals in each district-caste-urban cell and clustered at state-urban level. Age Cutoff = Months. p-values: * 0.10, ** 0.05, *** 0.01.

A.6 OB Decompositions: Additional Analysis

A.6.1 OB Decompositions By Gender

Figure A.5 shows the Blinder-Oaxaca decomposition results for girls. In the top left panel, we see that more than 80% of the share gaps among lower and upper caste children can be explained by covariates. The caste gaps among girls is almost entirely explained at birth by differences in endowments but reduces to around 30% by age 3 and onwards. For girls, private investment covariates explain 60% of caste gaps and the rest is explained by differences in public investments. These trends are similar among the different caste groups.

Figure A.6 shows the Blinder-Oaxaca decomposition results for boys. In the top left panel, we see that the explanatory power decreases in the first 18 months and then increases to more than 80% , varying across the cohorts. Up to 60% of the caste gaps among boys is explained by differences in endowments at birth and this reduces to around 30% by age 3 and onwards. Private investments covariates explain 20% of differences at birth, increasing to 60% by age 3. Nearly the entire caste gap in birth length for boys is explained by differences in public investments. After that, these differences matter less as in the aggregate results and these trends are similar among different caste groups.

Figure A.5: Blinder-Oaxaca Decomposition Results: Explained in Percentage (Girls)

Notes: This figure presents Blinder-Oaxaca decomposition results. Results are expressed in percentage of the unconditional caste gap among children who live in regions (states) among girls. Child age measured in 6-month age groups is on the x-axis. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

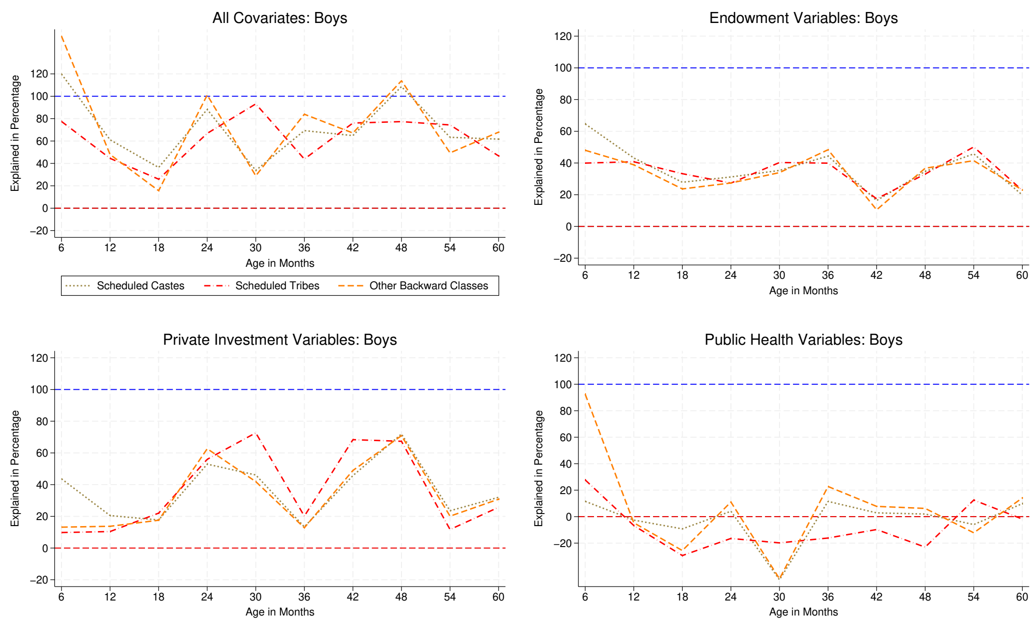


Figure A.6: Blinder-Oaxaca Decomposition Results: Explained in Percentage (Boys)

Notes: This figure presents Blinder-Oaxaca decomposition results. Results are expressed in percentage of the unconditional caste gap among children who live in regions (states) among boys. Child age is measured in 6 month bins. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

A.6.2 OB Decompositions By Location

Figure A.7 shows the Blinder-Oaxaca decomposition results for children living in rural areas. In the top left panel, we see that more than 80% of the caste gaps in HAZ among lower and upper caste children can be explained by covariates. In rural areas, the caste gaps are almost entirely explained at birth and see large drop in share explained at 18 months before it rises again. By age 5, almost the entire gap is explained by covariates. The endowment, investment, and public health variables follows the patterns of aggregate results. The share explained for endowment covariates starts high and remains stable, for private covariates increases after birth, and for public health variables remains around zero. Around 40% of the height differences at birth can be explained by endowment covariates when low caste children live in areas with lower shares of upper caste (top-right panel). These shares are slightly decreasing over the child's age. Endowment differences explain around 30% of differences by age 5. In the bottom left panel, less than 20% of the differences at birth can be explained by differences in private investment variables for ST children. Among SC and OBC children about 30% and for OBCs 20% of the HAZ differences can be explained by private investments. These differences increase and by age 5 explain around around 60% of the differences. In rural areas, public health variables explain around 20% of the differences at birth and up to age 5, with some years being irrelevant (bottom-right panel).

Figure A.8 shows the Blinder-Oaxaca decomposition results for children in urban areas. These estimates are noisy since less than 30% of the sample qualifies as part of the definition of being urban. In the top left panel, we see that all the gaps can be explained by covariates. Among OBC children (orange, dashed line), differences in endowment variables explain around 50% of the differences. This falls to 30% by age 5. Private investments play a large role in explaining differences in the first two years while public investments explain the differences between ages 2 and 4. Among ST children (red, dashed-dotted lines), endowment differences explain 40-60% of the differences over the child's age with the falling to 30% by age 5. Private investments explain differences when endowments matter less and public health variables do not explain differences for ST children HAZ gaps. SC children (grey dotted lines), follow similar patterns as the ST children with the difference that between years 3 and 4, public health variables explain some of differences HAZ gaps for SC children.

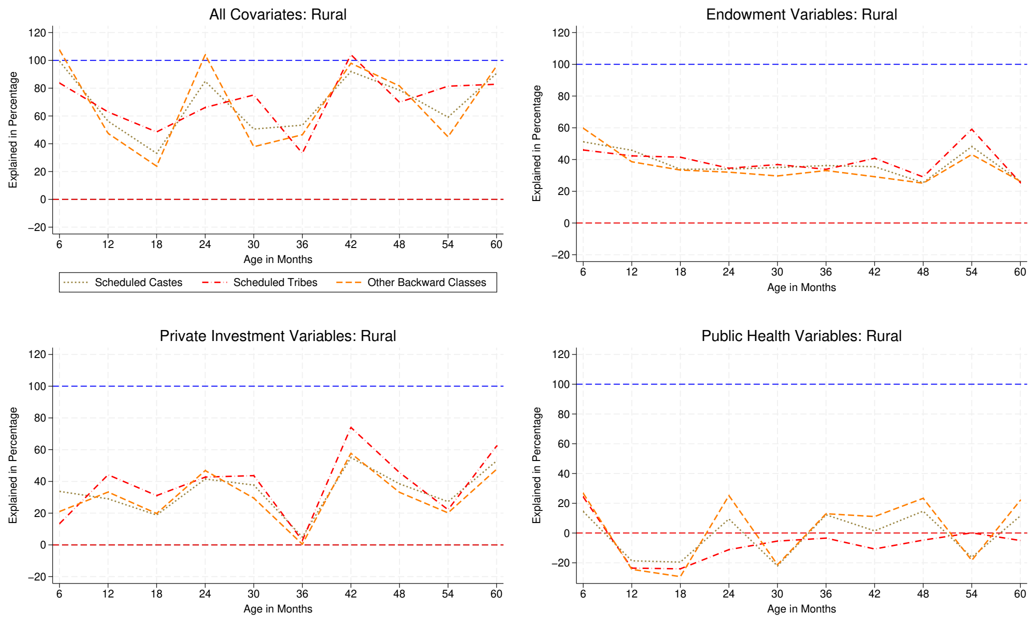


Figure A.7: Blinder-Oaxaca Decomposition Results: Explained in Percentage (Rural)

Notes: This figure presents Blinder-Oaxaca decomposition results. Results are expressed in percentage of the unconditional caste gap among children who live in regions (states) among rural children. Child age measured in 6-month age groups is on the x-axis. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

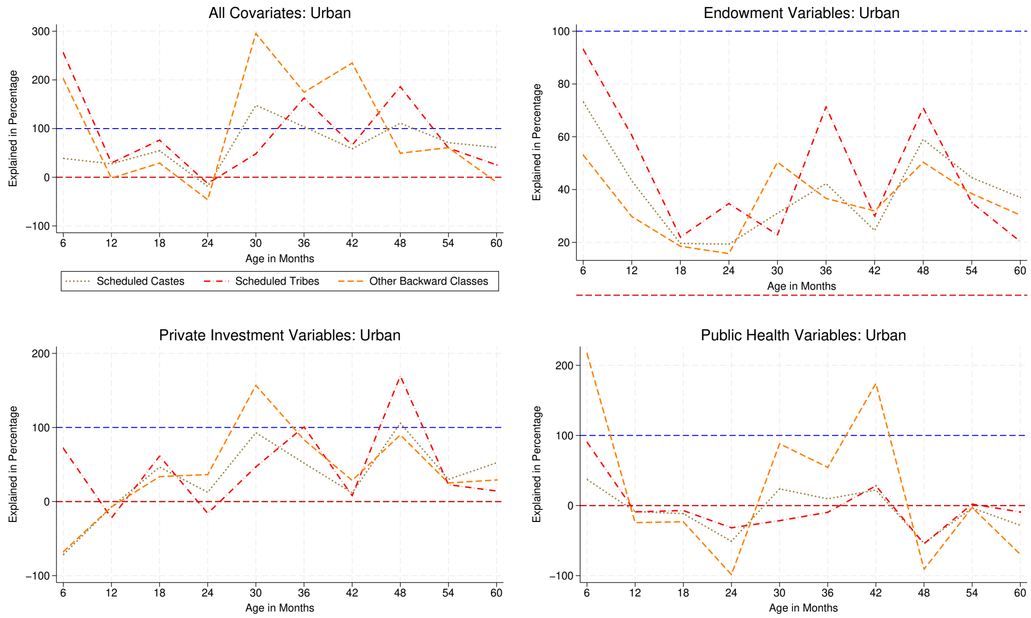


Figure A.8: Blinder-Oaxaca Decomposition Results: Explained in Percentage (Urban)

Notes: This figure presents Blinder-Oaxaca decomposition results. Results are expressed in percentage of the unconditional caste gap among children who live in regions (states) among gurban children. Child age is measured in 6 month bins. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

A.6.3 OB Decompositions By Region

The median share of UC households across the country is around 15%. Using this figure, we divide the sample into states with a high share of upper caste ($\geq$15%) and low share of upper caste (<15%).

Figure A.9 shows the Blinder-Oaxaca decomposition results for children living in areas with low shares of UC households. In the top left panel, we see that the share of caste gaps among lower and upper caste children that can be explained by covariates lies above 80%. This briefly decreases till age 3 after which all the differences can be explained by covariates in the model. Around 40% of the height differences at birth can be explained by endowment covariates when low caste children live in areas with lower shares of upper caste (top-right panel). These shares are slightly decreasing over the child's age and by age 5, endowment differences can explain around 30% of HAZ caste gaps. In the bottom left panel, about 20% of the differences at birth can be explained by differences in private investment variables for ST children. For SC and OBC children at birth, the explained share is about 50% and 35%, respectively. These differences increase and by age 5 explain around around 60% of the differences. In areas with low shares of UC households, public health variables explain almost no share of the differences in HAZafter birth (bottom-right panel).

Figure A.10 shows the Blinder-Oaxaca decomposition results explained in percentage for children living in regions with high shares of UC households. These estimates are noisy since 20% of the sample qualifies as part of the definition of high UC share states. A discerning reader, similar in the previous section, may take objection to placing weight on the interpretation of these share due to the small sample sizes on which these are estimated. In the top left panel, we see that all the share gaps can be explained by covariates. Hence we avoid presenting a more detailed analysis.

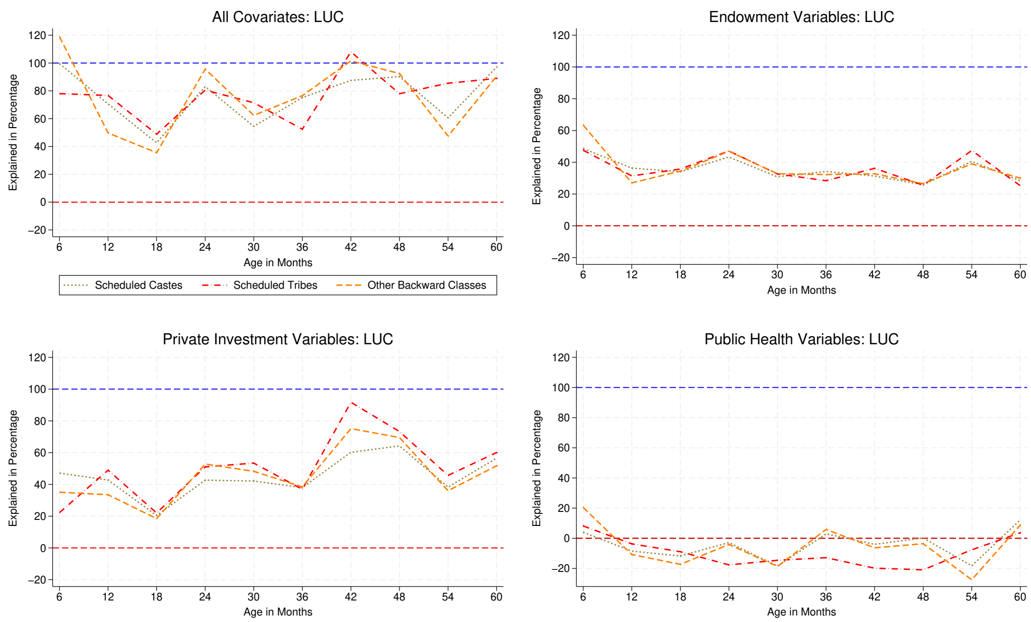


Figure A.9: Blinder-Oaxaca Decomposition Results (Low Share of Upper Caste by State)

Notes: This figure presents Blinder-Oaxaca decomposition results. Results are expressed in percentage of the unconditional caste gap among children who live in regions (states) with a *low* share of UC children. Child age is measured in 6 month bins. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

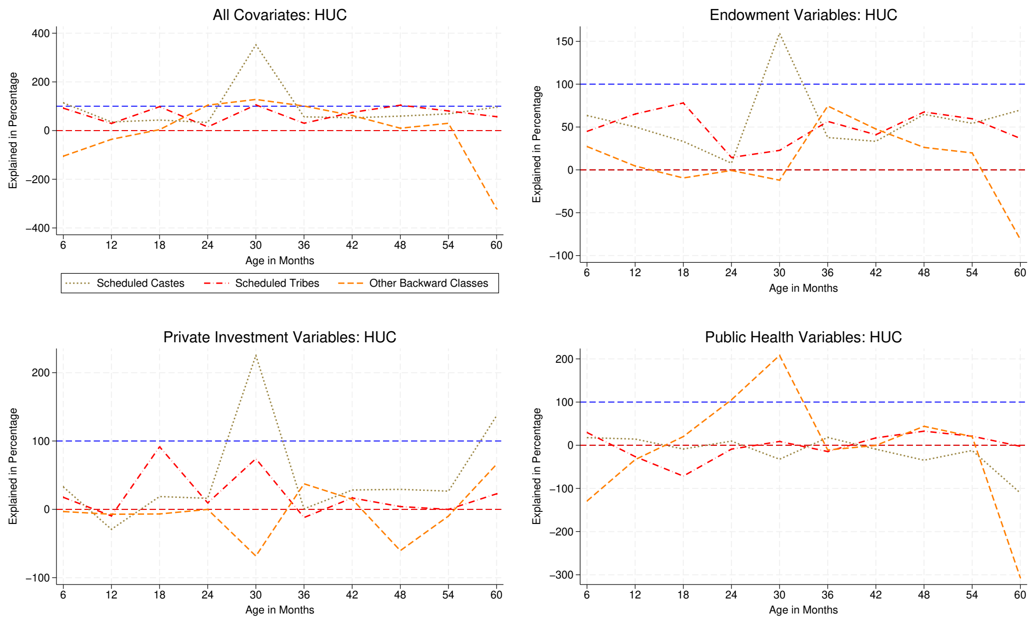


Figure A.10: Blinder-Oaxaca Decomposition Results (High Share of Upper Caste by State)

Notes: This figure presents Blinder-Oaxaca decomposition results. Results are expressed in percentage of the unconditional caste gap among children who live in regions (states) with a *high* share of UC children. Child age measured in 6-month age groups is on the x-axis. The top left panel presents the percent of the HAZ gap that is explained when all control variables are included. The top right panel is the the percent of the HAZ gap explained by endowment variables. The bottom left panel depicts the percent of the HAZ gap explained by the private investment variables. The bottom right panel presents the percent HAZ gap that is explained in percentage by public health variables.

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