

The Pennsylvania State University
The Graduate School

**EDUCATIONAL EXPANSION IN POST-WORLD WAR II AND THE DIABETES
EPIDEMIC:
THE RELATIONSHIP BETWEEN EDUCATION
AND TYPE 2 DIABETES MORBIDITY
DECOMPOSING CHANGE FOR COHORTS BORN BETWEEN 1935-1954**

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by

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ABSTRACT

We know that during the Epidemiological Transition (ET), countries experience large shifts in morbidity and mortality. Within ET, sudden and stark increases in population growth occur with mortality decline, as well as a releveling of population growth with fertility decline. With regards to health, morbidity and mortality from infectious diseases declines and morbidity and mortality for chronic disease increase. The increasing prevalence of chronic diseases is associated with increasing changes in food consumption, types of work, and lifestyles. These include increasing obesity, tobacco use, sedentary occupations, and other factors. Type 2 diabetes is a highly prevalent chronic disease in the United States, as well as a leading cause of death and linked to multiple complications. Rapid expansion of diabetes prevalence in the United States is associated with the ET, and forecast projections are bleak. Boyle et al. (2010) estimate that by 2050, approximately one-quarter of the adult US population will be diagnosed with diabetes. In tandem with this expanding chronic disease—and diabetes—prevalence in the United States is the expansion of mass education. In the aftermath of World War II, the G.I. Bill and other social and economic changes brought a rapid expansion in education and large increases in college enrollment and in the proportion of persons completing at least a bachelor degree. Graduating from high school--and especially college--not only became more common, but also viewed as a marker of individual success

The paradox is that while both diabetes prevalence and educational attainment have expanded in the United States, there is also an inverse relationship between educational attainment and diabetes. It is well established that persons with higher levels of education are at lower risk of being diagnosed with diabetes than their less educated counterparts. This begs the question, why has diabetes prevalence expanded despite increases in educational attainment? Little research has analyzed the social context of education expansion with diabetes disparities. It is unclear whether the relationship between educational attainment and diabetes has changed across cohorts, or the compositional effect of increasing education in a population.

Two types of analyses are conducted on four cohorts (ages 60-64 and born between 1935-1954) collected from 20 years of the National Health Interview Survey. The first set involves logit regression analysis predicting the odds of having ever been diagnosed with diabetes at age 15 or older, including the main independent variable (education) and population composition variables (gender, race-ethnicity, marital status, and region). The second set of analyses involves using the Fairlie (nonlinear) Decomposition procedure estimating the effect of the educational composition on the change in diabetes rate across cohorts controlling for population composition. The results indicate educational expansion acted to limit diabetes growth. The magnitude of the educational gradient did not change, but the compositional effect on diabetes change of the educational expansion was substantially and statistically significant from one cohort to the next across all cohorts. Namely, without educational expansion to limit diabetes growth, there would be approximately an additional million persons ages 60-64 born in 1950-54 diagnosed with diabetes.

TABLE OF CONTENTS

List of Tables.....	vi
List of Figures.....	vii
Acknowledgements	viii
Chapter 1. INTRODUCTION	1
Chapter 2. CONCEPTUAL AND THEORETICAL FRAMEWORKS	4
Introduction	4
Diabetes: Increasing Prevalence and Changing Disparities.....	4
Explanations for Chronic Disease Expansion.....	8
Diabetes and Educational Attainment.....	11
Education Expansion... ..	14
Research Questions and Contribution.....	20
Chapter 3: DATA AND RESEARCH METHODS	23
Summary of Datasets.....	23
Summary of Hypotheses	24
Defining Key Concepts.....	25
Summary of Research Method	28
Chapter 4. SAMPLE AND DESCRIPTIVE STATISTICS	33
Sample	33
Strengths of the Data	33
Limitations of the Data	34
Descriptive Statistics.....	35
Characteristics by Gender and by Race-Ethnicity Cohorts	38
Diabetes Prevalence by Educational Attainment and Cohort	51
Chapter 5. EDUCATIONAL GRADIENTS AND EDUCATIONAL COMPOSITION CHANGE IN DIABETES PREVALENCE ACROSS FOUR COHORTS	61
Introduction	61
Logistic Regression Results	61
Decomposition Results	69
Summary of Results	72
Additional Analysis (See Appendix).....	74
Chapter 6. GENDER DIFFERENCES IN EDUCATIONAL GRADIENTS AND EDUCATIONAL COMPOSITION CHANGE IN DIABETES PREVALENCE ACROSS FOUR COHORTS	75
Introduction	75
Logistic Regression Results	75
Decomposition Results	91
Summary of Results	100

Chapter 7. RACE-ETHNIC DIFFERENCES IN EDUCATIONAL GRADIENTS AND EDUCATIONAL COMPOSITION CHANGE IN DIABETES PREVALENCE ACROSS FOUR COHORTS	103
Introduction	103
Logistic Regression Results	103
Decomposition Results	119
Summary of Results	131
Chapter 8. DISCUSSION OF FINDINGS AND CONCLUSION	133
Introduction	133
Logistic Regression	134
Decomposition	135
Expansion of Education and Diabetes	137
Implications	140
Conclusion	140
References	144
Appendix: Additional Analyses.....	152

LIST OF TABLES

Table 3-1: How Composition Change Coefficients Are Interpreted In This Study	31
Table 4-1: N’s for Overall Sample and Age-Specific Cohort subsamples by Gender and by Race	33
Table 4-2: Descriptive Statistics of Persons Aged 60-64, by Cohort	35
Table 4-3: Descriptive Statistics of Males Aged 60-64, by Cohort	38
Table 4-4: Descriptive Statistics of Females Aged 60-64, by Cohort	41
Table 4-5: Descriptive Statistics of Whites Aged 60-64, by Cohort	44
Table 4-6: Descriptive Statistics of Hispanics Aged 60-64, by Cohort	46
Table 4-7: Descriptive Statistics of Blacks Aged 60-64, by Cohort	49
Table 4-8: Diabetes Prevalence for Persons Aged 60-64, by Education and Cohort	51
Table 4-9: Total Diabetes Prevalence for Males and Females Aged 60-64, by Cohort	53
Table 4-10: Total Diabetes Prevalence for Males and Females Aged 60-64, by Education and Cohort	53
Table 4-11: Total Diabetes Prevalence for Whites, Blacks, and Hispanics Aged 60-64, by Cohort	56
Table 4-12: Total Diabetes Prevalence for Whites, Blacks, and Hispanics Aged 60-64, by Education and Cohort	57
Table 5-1: Weighted Logit Regression for Diabetes among Persons Aged 60-64, by Cohort.....	62
Table 5-2: Decomposition of Cohort Disparities in Diabetes Prevalence	69
Table 6-1: Weighted Logit Regression for Diabetes among Males Aged 60-64, by Cohort.....	76
Table 6-2: Weighted Logit Regression for Diabetes among Females Aged 60-64, by Cohort.....	82
Table 6-3: Decomposition of Cohort Disparities in Diabetes Prevalence among Males	91
Table 6-4: Decomposition of Cohort Disparities in Diabetes Prevalence among Females	94
Table 7-1: Weighted Logit Regression for Diabetes among Whites Aged 60-64, by Cohort.....	104
Table 7-2: Weighted Logit Regression for Diabetes among Hispanics Aged 60-64, by Cohort	110
Table 7-3: Weighted Logit Regression for Diabetes among Blacks Aged 60-64, by Cohort.....	113
Table 7-4: Decomposition of Cohort Disparities in Diabetes Prevalence among Whites	119
Table 7-5: Decomposition of Cohort Disparities in Diabetes Prevalence among Hispanics.....	122
Table 7-6: Decomposition of Cohort Disparities in Diabetes Prevalence among Blacks	125
Table 8-1: Percentage Point Increase in Education and Diabetes Across Cohorts, 1935-39 versus 1950-54	137
Table 8-2: Gender Gaps in Education and Diabetes	137
Table 8-3: Race-Ethnicity Gaps in Education and Diabetes	138
Table A: Weighted Logit Regression for Diabetes among Persons Aged 60-64 Interactions between Education and Birth Cohort.....	152
Table B Weighted Logit Regression for Diabetes among Persons Aged 60-64 Interactions between Education and Gender, By Cohort	153
Table C: Weighted Logit Regression for Diabetes among Persons Aged 60-64 Interactions between Education and Gender, By Cohort	154

LIST OF FIGURES

Figure 2-1: Percent of US Population age 25-29 Completed College	14
Figure 2-2: Percent of US Population age 25-29 Completed College, by Gender	16
Figure 2-3: Percent of US Population age 25-29 Completed High School, by Race-Ethnicity	18
Figure 2-4: Percent of US Population age 25-29 Completed College, by Race-Ethnicity.....	19

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Chapter 1

Introduction

The largest research question leading this dissertation concerns temporal changes in the relationship between education and health in the United States. Namely, how has the relationship between education and health changed for those living through the last decades of the 20th Century and the first decades of the 21st century. We know that during the Epidemiological Transition (ET), countries experience large shifts in morbidity and mortality (Omran 1971; McKeown 2009). Within ET, sudden and stark increases in population growth occur with mortality decline, as well as a releveling of population growth with fertility decline. With regards to health, morbidity and mortality from infectious diseases declines and morbidity and mortality for chronic disease increase. The increasing prevalence of chronic diseases is associated with increasing changes in food consumption, types of work, and lifestyles (McKeown 2009). These include increasing obesity, tobacco use, sedentary occupations, and other factors (Flegal et al. 2010; McKeown 2009).

Type 2 diabetes is a highly prevalent chronic disease in the United States (Fishman et al. 2014; CDC 2017; Gregg et al. 2004; Mokdad et al. 2000), as well as a leading cause of death and linked to multiple complications (2018). There are distinct differentials in prevalence by gender, race-ethnicity, and socioeconomic factors (Dalstra et al. 2005; Borell et al. 2006; Maty et al. 2005; Menke et al. 2014; Cockerham 2016; Brancati et al. 1996; Robbins et al. 2001, 2000; CDC 2003). Additionally, rapid expansion of diabetes prevalence in the United States is associated with the ET (McKeown 2009; Omran 1971, 2005), and forecast projections are bleak. Boyle et al. (2010) estimate that by 2050, approximately one-quarter of the adult US population will be diagnosed with diabetes.

In tandem with this expanding chronic disease—and diabetes—prevalence in the United States is the expansion of mass education. In the aftermath of World War II, the G.I. Bill and other social and economic changes brought a rapid expansion in education and large increases in college enrollment and in the proportion of persons completing at least a bachelor degree (calculated using Barro and Lee 2013). Graduating from high school--and especially college--not only became more common, but also viewed as a marker of individual success (Baker 2014; Kao and Thompson 2003).

The paradox is that while both diabetes prevalence and educational attainment have expanded in the United States, there is also an inverse relationship between educational attainment and diabetes. It is well established that persons with higher levels of education are at lower risk of being diagnosed with diabetes than their less educated counterparts (Dalstra et al. 2005; Borell et al. 2006; Maty et al. 2005). This begs the question, why has diabetes prevalence expanded despite increases in educational attainment? Little research has analyzed the social context of education expansion with diabetes disparities. It is unclear whether the relationship between educational attainment and diabetes has changed across cohorts, or the compositional effect of increasing education in a population. Additionally, no research has examined differentials in both diabetes and educational expansion. There are distinct diabetes disparities by race and gender (Borrell et al 2006), as well as when educational expansion reached these subpopulations (US Bureau of the Census 1975; Jacobs 1996; US Dept of Education 1995; National Center for Educational Statistics 1994; Kao and Thompson 2003).

This dissertation seeks to understand how the effect of educational attainment and the composition of education has changed across generations with regard to type 2 diabetes prevalence. Chapter 2 discusses the conceptual and theoretical frameworks. Here, greater detail

is provided for type 2 diabetes, the expansion of both diabetes and education, and diabetes and educational differentials by gender and race-ethnicity. Chapter 3 presents the dataset, subsamples, and methods of analysis. There are three main sets of analyses, pertaining to 1) cohort analysis of the population, 2) cohort analysis by gender, and 3) cohort analysis by race-ethnicity (non-Hispanic white, non-Hispanic black, and Hispanic). Chapter 4 provides descriptive statistics of each sample, with univariate analyses finding greater diabetes prevalence and greater educational attainment in later born cohorts. Bivariate and multivariate analyses further parse out the samples to examine the relationship between educational attainment and diabetes by cohort for the entire sample, by gender, and by race-ethnicity.

Chapter 5 presents linear regression and decomposition models to examine the educational gradient for diabetes prevalence across four cohorts, and lightly touches on interactions between education and gender and race-ethnicity, justifying further analysis. Chapter 6 examines the gender differentials in the educational gradient for diabetes prevalence across the same four cohorts in Chapter 5. Chapter 7 examines three racial-ethnic groups to find differentials in diabetes prevalence across the four cohorts by race-ethnicity. Chapter 8 discusses the findings in Chapters 5-7, examining differences in the educational gradient on diabetes prevalence for subsamples. Chapter 9 provides conclusions, discusses limitations of the research, as well as suggestions for future research.

Chapter 2

Conceptual and Theoretical Frameworks

Introduction

This chapter discusses five topics. First, the increasing prevalence of type 2 diabetes and changing disparities by gender and race-ethnicity. Second, theoretical explanations and empirical evidence for the rise in chronic diseases, including diabetes. Third the relationship between education and diabetes and the differences by gender and race-ethnicity. Fourth, the disparate educational expansion among subpopulations. Finally, the research questions and contributions of the dissertation. It also gives a brief summary of the subsequent chapters in the dissertation.

Diabetes: Increasing Prevalence and Changing Disparities

Diabetes is a common condition in the United States. There are three main types of diabetes (Type 1, Type 2, and gestational), but the most common form is type 2 diabetes, developed later in life, in which the body fails to produce enough insulin to maintain normal levels of glucose in the blood (ADA 2005). The failure of the body to use insulin properly results in the overproduction of insulin by the pancreas, which in turn causes insulin resistance in organs, including the liver, skeletal muscles, kidneys, brain, small intestine, and adipose tissue (Holman et al 2008; Defronzo 2009). As organs become insulin resistant, the pancreas is no longer able to produce sufficient insulin levels. While medications and treatments for type 2 diabetes are available, no cure currently exists and, thus, it is a lifelong chronic condition requiring constant vigilance (Chatterjee et al. 2017). Type 2 diabetes can be diagnosed at any age, but it is most likely to be occur at older ages (Cowie 2010).

The prevalence of diagnosed diabetes has been increasing in the United States since the 1950s (Fishman et al. 2014; CDC 2017; Gregg et al. 2004; Mokdad et al. 2000). As of 2012, approximately 9 out of 100 adult Americans had been diagnosed with diabetes (29.1 million), of

which 1.7 million were diagnosed in that year (ADA 2005). Estimates indicate that an additional 8.1 million Americans who were diabetic in 2012 but had not been diagnosed, and many others are pre-diabetic, a condition placing individuals at greater risk of becoming diabetic. Overall, the prevalence rate has climbed from 0.91 percent in 1960, to 7.4 percent in 2015 (CDC 2017), and projection models indicate that by 2050, the prevalence of diabetes will more than double to at least 21 percent and perhaps as high as 28 percent of the adult US population (Boyle et al. 2010).

Previous research has sought to identify the causes of increasing diabetes prevalence. Broadly, the increases in diabetes might be attributed to the aging of the population, since diabetes is most commonly found in older persons, or to increasing obesity and sedentary lifestyles (NCD-RisC 2016). Increases in prevalence are not, however, entirely attributable to changing age distribution of the population (CDC 1997), nor differences in changing survival rates between persons with diabetes and the total population (Leibson et al. 1997).

The costs of diabetes are substantial in terms of reduced life expectancy, financial expenditures, and diminished quality-of-life. The risk of death for adults with diabetes is 50 percent greater than for adults who do not have diabetes (CDC 2014). Diabetics may also experience severe complications, including kidney failure, heart disease, blindness, stroke, or amputation. It is estimated that the cost of diabetes was \$327 billion in 2017, including both medical expenditures and lost productivity (ADA 2018). These costs had risen by \$82 billion in just five years, from \$245 billion in 2012 (CDC 2014). In addition, adults with diabetes are estimated to have average medical costs 2.3 times greater than expenditures for adults without diabetes. The average annual medical expenditures per person with diabetes was \$13,700 in 2012 (ADA 2013).

Diabetes results in emotional distress, and it has negative impacts on quality-of-life

(Cannon et al. 2018). Results from The Diabetes Attitudes, Wishes and Needs (Dawn) study indicate that 41 percent of diabetic patients reported poor psychological well-being (Peyrot et al. 2005). Additionally, health care providers (e.g. nurses, primary care physicians, and specialists) estimate that between 62 percent and 72 percent of diabetic patients experience psychological problems, such as depression, anxiety, stress, and burnout. The complexity of diabetes management, the burden of daily or more frequent self-treatments, such as injections, and need for regular glucose testing can result in diabetics feeling overwhelmed and frustrated by the daily hassles (Polonsky 2002). These stresses are exacerbated when additional complications occur. For example, analysis of data from the Study to Help Improve Early evaluation and management of risk factors Leading to Diabetes (SHIELD) found that diabetics with a higher cardiometabolic experience higher rates of moderate to severe depression (Grandy et al. 2008).

Data from the National Health and Examination Survey (NHANES) indicate that gender disparities in diabetes prevalence in the United States favored males in 1976-80, with rates of 4.7 percent and 5.7 percent, respectively, for males and females. But across the next thirty years (NHANES 1976-80 vs. NHANES 2007-2010), while the rate increased substantially, by about one-half, for females from 5.7 percent to 8.7 percent, the prevalence rate more than doubled for males from 4.7 percent to 11.2 percent (Menke et al. 2014). Thus, both genders experienced large increases in the prevalence of diabetes, but the female disadvantage in the late 1970s had become substantial advantage compared to males by the end of the first decade of the twentieth century. Prior research finds that males and females differ beyond biological and physiological characteristics, also processing information and solving intellectual problems differently (Amuta et al. 2016; Ruigrok et al 2014; Kautzky-Willer et al. 2015). Gender differences also exist in exposure to health risks and access and use of health information and services (Ostlin et al 2006).

For example, male health outcomes are more heavily influenced by substance use, exercise, and diet (Denton et al. 2004; Von Bothmer and Fridulund 2005), while female health is more influenced by stress, social support, critical life event, socioeconomic status, and age (Denton et al. 2004).

Prior research also finds disparities in diabetes prevalence by race-ethnicity (Cockerham 2016; Brancati et al. 1996; Golden et al. 2016; Robbins et al. 2001, 2000; CDC 2003). Raw percentages in 1999-2000 of NHANES reveal Non-Hispanic whites (5 percent) experience lower diabetes prevalence compared to non-Hispanic blacks (9.6 percent) and Mexican Americans (6.1 percent) (CDC 2003). Adjusting to represent the total U.S. population, these disparities increase between whites (4.8 percent), non-Hispanic blacks (11.7 percent) and Mexican Americans (9.6 percent). Multiple studies suggest that blacks are between 50 percent and 100 percent more likely to have diabetes than whites (Harris et al. 1998; Mokdad et al. 2003; CDC 2003; Carter et al. 1996; Cowie et al. 1993; Harris 1996). Additionally, there are growing racial-ethnic disparities in diabetes among normal weight groups and obese groups (Zhang et al. 2009; Harris 1996), indicating that obesity does not fully explain differences in diabetes prevalence for race-ethnicity.

There are also racial-ethnic disparities in the rising diabetes prevalence in the American population (Cowie et al. 2018). Using the National Health Interview Surveys (NHIS) and NHANES, studies reveal higher diabetes prevalence among minority groups (Zhang et al. 2009; CDC 1997). Diabetes prevalence increased by one-third for whites from 2.9 percent (NHANES I 1971-1975) to 5.5 percent (NHANES 1999-2004). In the same surveys, diabetes prevalence increased by nearly two-thirds for blacks (4.6 percent to 11 percent), and more than doubled for Mexican Americans (3.4 percent to 11 percent) (Zhang et al. 2009). Put another way, the

black/white ratio was 1.6 to 2 (one-quarter increase), and the Mexican/white ratio was 1.2 to 2 (two-thirds increase).

Researchers have explored a wide variety of possible explanations for disparities in diabetes prevalence, especially for the disparities separating whites and blacks --including environmental, socioeconomic, behavioral, physiological, and genetic factors (Sims et al. 2011; Signorello et al. 2007; Carter et al. 1996; Harris 1996; Abate and Chandalia 2003). For example, several studies argue greater genetic variation in blacks (i.e., allele frequency) may explain some of the racial-ethnic disparity (Klimentidis et al. 2011; Chen et al. 2012; Corona et al. 2013; Hanson et al. 2015), and it may persist after adjusting for socioeconomic factors (Cheng et al. 2012). Other researchers have attributed greater type 2 diabetes among blacks with increased urbanization, producing multigenerational overnutrition and physical inactivity (Ebrahim et al. 2010; Wells et al. 2016), or other environmental factors that are linked to greater risk of diabetes, such as exposure to arsenic, organic pollutants, and heavy metals (Navas-Acien et al. 2008; Kim et al. 2013; Lee and Jacobs 2006; Grice et al. 2017; Menke et al. 2016). Meanwhile, some researchers argue biomedical factors diabetes are overemphasized, and that disparities in socioeconomic factors better explain racial-ethnic differences in diabetes (Link and McKinlay 2009), specifically for black females (Robbins et al. 2000; Brancati et al. 2000).

Explanations for Chronic Disease Expansion

The theory of the epidemiological transition provides a broad conceptual framework for understanding why large increases have occurred in the prevalence of chronic diseases, generally, and hence in the prevalence of type 2 diabetes in particular. Omran (1971, 2005). Omran (2005: 732) "...focuses on the complex change in patterns of health and disease *and* on the interactions between these patterns and their demographic, economic and sociologic

determinants and consequences”. The theory consists of five propositions (Omran 2005; McKeown 2009).

First, “(t)he theory of the epidemiologic transition begins with the major premise that mortality is a fundamental factor in population dynamics” (Omran 2005: 733). “...this proposition is founded in the insight that declining mortality is typically followed by declining fertility, and it is a combination of lower birth rates as well as lower death rates and high life expectancy that produce the altered population distribution” (McKeown 2009), that is, the composition of the population shifts toward older age groups.

Second, “...a long-term shift occurs in mortality and disease patterns whereby pandemics of infection are gradually displaced by degenerative and made-made diseases as the chief form of morbidity and primary cause of death (Omran 2005: 737). In other words, as morbidity and mortality from infectious diseases decline and as the population ages, chronic diseases common among older people become more prevalent.

Third, “the most profound changes in health and disease patterns obtain among children and young women” (Omran 2005: 741). In other words, mortality declines are accounted for mainly by declining infant mortality, which was followed by declining fertility and led to the aging of the population, and by declining maternal mortality, which led to changes in gender disparities in mortality (McKeown 2009).

Fourth, “(t)he shifts in health and disease patterns that characterize the epidemiologic transition are closely associated with the demographic and socioeconomic transitions that constitute the modernization complex” (Omran 2005: 744). In discussing this proposition, Omran (2005) highlights the reciprocal nature of the relationship between the epidemiological and socioeconomic change, that socioeconomic improvements (and public health initiatives)

foster reductions in morbidity and mortality, but that reduced morbidity and mortality also tend to increase the effectiveness of workers and economic productivity (Oman 2005). Thus, modernization involves not only declines in the central demographic variables of mortality and fertility, but also numerous additional changes, such as, delayed childbearing, longer birth intervals, increased education, rising incomes, improved nutrition and sanitation, and changing health habits.

Fifth, “(p)eculiar variations in the pattern, the pace, and the determinants and the consequences of population change differentiate three basic models of the epidemiologic transition: the classical or western model, the accelerated model and the contemporary or delayed model (Omran 2005: 752) These models reflect broad differences in the historical periods and global regions of transitions as they occurred in specific countries.

The dissertation can be seen as linking and expanding upon the second and fourth propositions by focusing on how changes in disparities among gender and race-ethnic groups in prevalence of one of the major chronic diseases emerging in the twentieth century, type 2 diabetes, is connected to one of the most prominent socioeconomic changes in this century, the enormous expansion in secondary and college education. The next section of this chapter presents theoretical arguments regarding possible pathways, or mechanisms, by which higher educational attainments would be expected to influence the prevalence of diabetes, as well as summarizing empirical research which has found a strong relationship between educational attainment and diagnosis of diabetes, with sometimes large disparities by gender and race-ethnicity.

Diabetes and Educational Attainment

Prior research finds an inverse relation between education and diabetes (Dalstra et al. 2005; Borell et al. 2006; Maty et al. 2005). Education may affect diabetes prevalence and general health through four major pathways. The first pathway involves material/structural mechanisms. Educational credentials have become increasingly relevant and necessary to the changing types of jobs in the labor market, which then determine economic earnings and social status attainment (Bills 1987), even superseding the importance of family's socioeconomic status (Hout 1988, 2012). The ever-growing importance of education leads to importance in health status. For example, persons with higher levels of education have more stable jobs than their less educated counterparts (Catalano 1991; Ross and Wu 1995; Woolf and Braveman 2011) that are more likely to provide health insurance and higher disposable incomes to address health concerns (Woolf and Braveman 2011). Persons with higher education may be more proactive in spending their time and money testing for various health conditions, or be more likely to afford health treatments and medications. Additionally, educational attainment is associated with neighborhood selection (South et al. 2008), and neighborhoods or communities may be instrumental in persons' health through the availability of health care services, neighborhood characteristics that influence disease (e.g., toxin exposure) or promote health (e.g., access to health food stores, places to exercise), and the normative attitudes toward health and health behaviors within the neighborhood (Seeman and Crimmins 2001).

The second pathway involves social psychological mechanisms. Research has found formal education is associated with a greater sense of personal control and increased agency (Inkeles 1996) as individuals learn how to solve problems and to be more engaged in decision-making and planning their lives (Mirowsky and Ross 2003b, 2007; Wheaton 1980). With a

greater sense of personal control, individuals are more likely to seek information to guide and improve their personal outcomes, and to adopt positive health behaviors (Ross and Mirowsky 2010). Additionally, persons with higher education have more stable and supportive interpersonal relationships, including more likely to be married and less likely to divorce than persons with lower education (Mirowsky and Ross 2003a). Emotional support from marriage is associated with better psychological health which indirectly improves physical health.

The third pathway involves cognitive mechanisms. Lower educational attainment is also strongly associated with specifically worse health literacy (Baker et al. 1998; Gazmariarian et al 1999), and poor health literacy is linked to worse health (Street et al. 1993) and more hospital admissions (Baker et al. 1997). Research finds that a few years of schooling greatly improves decision making and effectively using information to solve problems (Baker et al. 2012; Duncan et al. 1996; Pascarella and Tenzini 1991). Schooling also more fully develops individuals' neurocognitive capacity (Baker et al. 2015), mediating the relationship between educational attainment and health (Cacioppo and Petty 1979; Herd 2010). As new research findings on diabetes and its prevention emerge and become available to the public, more highly educated individuals may more quickly process the information and understand how they can apply it in their own lives. Persons with higher education also tend to have a greater cognitive capacity to see connections between their immediate actions and important longer-term outcomes in their lives, a characteristic known as "learned effectiveness" (Mirowsky and Ross 1998, 2005). Thus, they may be more cognitively inclined to identify and avoid behaviors that could have negative health implications, as well as to adopt health behaviors that have positive implications.

All three pathways also can work through social and behavioral mechanisms. Skalamea and Hummer (2016) find health behaviors cluster by education level, with more highly-educated

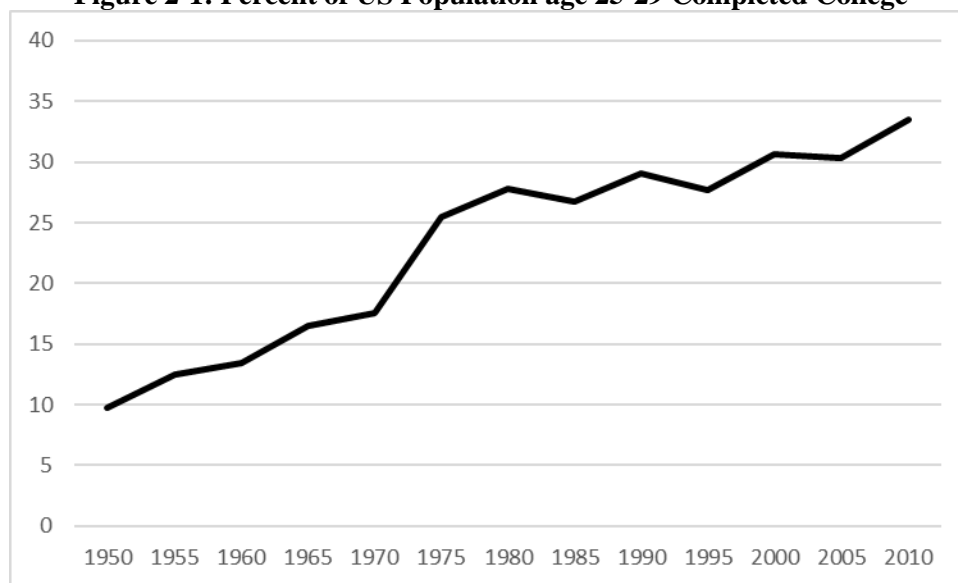
groups engaging in more healthy behaviors. Persons with higher education are more likely to exercise, walk, and drink alcohol moderately, and they are less likely to smoke cigarettes or be overweight than their less educated counterparts (Mirowsky and Ross 1998, 2003a, 2005; Ross and Bird 1994; Ross and Wu 1995). Each of these behaviors individually leads to improved health. Extensive research suggests educational attainment is associated with better health. Although good health can foster greater educational attainments, most previous research finds the positive relationship between educational attainment and health is largely due to the effect of education on health, and not the reverse (Doornbos and Kromhout 1990; Wilkinson 1986).

Borrell et al. (2006) find that the relationship between educational attainment and diabetes differs by gender and by race-ethnicity. The relationship is inverse for both males and females, but the impact of educational attainment on the odds ratio having diabetes is stronger for women than for men, 1.9 and 1.4 respectively (Borrell et al. 2006). Women with less than a high school diploma have nearly twice the odds of having diabetes as women with a bachelor's degree (Borrell et al. 2006). Researchers find educational attainment to be more strongly associated with better health outcomes among males than females (Ross et al. 2012; Masters et al. 2012; Pudrovska and Aniskin 2013), especially in later life (Hill and Needham 2006). Males and females not only differ in mechanisms of biological and physiological development and again, they also differ in their health exposures, health behaviors, and psychosocial resources, both within and across socioeconomic groups (Denney et al. 2010; Hayward and Gorman 2004; Liu and Hummer 2008; Masters et al. 2012; Ross et al. 2012; Pudrovska and Aniskin 2013). Furthermore, the inverse relationship between educational attainment and diabetes also exists for non-Hispanic whites and Hispanics, but not for blacks. For non-Hispanic whites and Hispanics with less than a high school diploma the odds of having diabetes are 70 percent and 60 percent

greater, respectively, than their counterparts with at least a bachelor’s degree (Borrell et al. 2006). There is no difference in the odds of higher education and diabetes for blacks. Possible explanations for the health association with socioeconomic factors among blacks include the “minority poverty” hypothesis and the “diminishing returns” hypothesis (Farmer and Ferraro 2005). The minority poverty hypothesis argues that blacks experience a unique disadvantage across the life course, facing threats to health and well-being due to the combined disadvantage of poverty and race (Willie 1979, 1989). The diminishing returns hypothesis argues that blacks do not experience the same returns to higher socioeconomic achievement as whites (e.g., educational achievement), and that disparities between blacks and whites are actually greatest at the highest socioeconomic levels (Bowles and Ginits 1976; Farley 1984).

Education Expansion

Figure 2-1: Percent of US Population age 25-29 Completed College



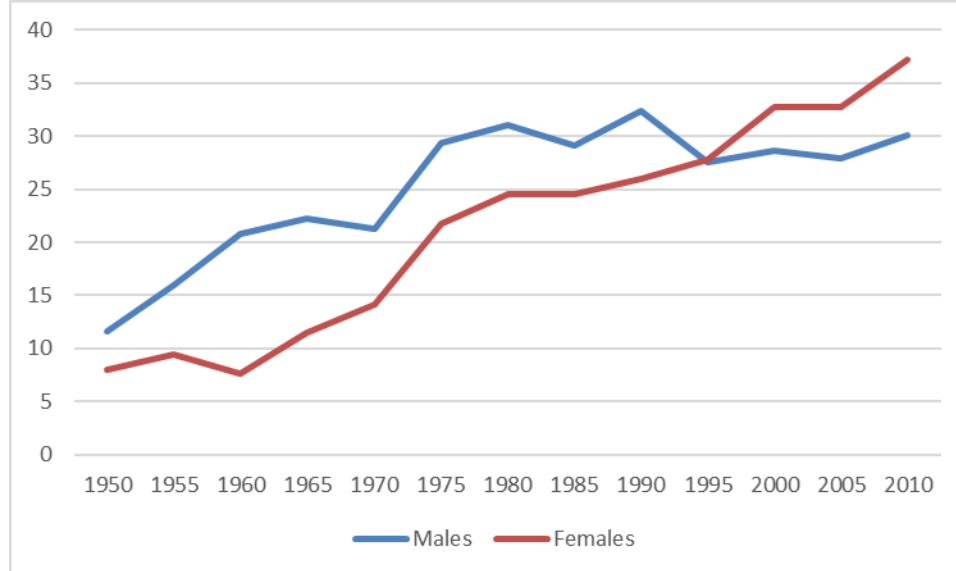
Source: Barro and Lee 2013

Immediately after World War II, the United States experienced enormous educational expansion, with greater proportions of people not only finishing high school, but enrolling in colleges and attaining higher education degrees. The G.I. Bill and a growing supply of colleges

and universities made this generation the first to widely benefit from advanced education. Figure 2-1 presents the percentage of the U.S. population age 25-29 that completed college from 1950 to 2010. Less than 10 percent of the U.S. age 25-29 population completed college in 1950. The percentage more than doubled by 1975 to 25.4 percent of the young adult population (i.e., the first Baby Boomer cohort). Following years continued to experience greater levels of college completion, increasing to 33.5 percent of the age 25-29 population in 2010. The percentage of young adults completing college more than tripled over 60 years.

But the expansion in educational opportunities did not occur equally for all groups. Among gender, males held greater initial advantage in educational achievement than females, although social and institutional shifts in the 20th century have led to a reversing gender gap in education. Research finds that a form of “educational egalitarianism” in the decades following the Great Depression influenced the gender gap (Buchmann and DiPrete 2005). Increasing female educational attainment first occurred among families in which both parents were college educated (specifically among white families), while male educational attainment was favored among families with less education (Buchmann and DiPrete 2005). As families became more educated, college graduation rates increased faster for females than males, beginning for the 1940 birth cohort. With later-born cohorts, however, females from less advantaged families gained traction in higher educational attainment. In 1940 during World War II, among persons ages 25-29, women represented 41.3 percent for college graduates, but this slipped to 23.9 percent in 1950, and 10 years later the rate had rebounded to 35 percent in 1960 (US Bureau of the Census 1975). Women continued to experience larger increases than men between the late 1970s and 2010 (Jacobs 1996; US Dept of Education 1995), with women surpassing men in the raw number of degrees earned (National Center for Educational Statistics 1994).

Figure 2-2: Percent of US Population age 25-29 Completed College, by Gender



Source: Barro and Lee 2013

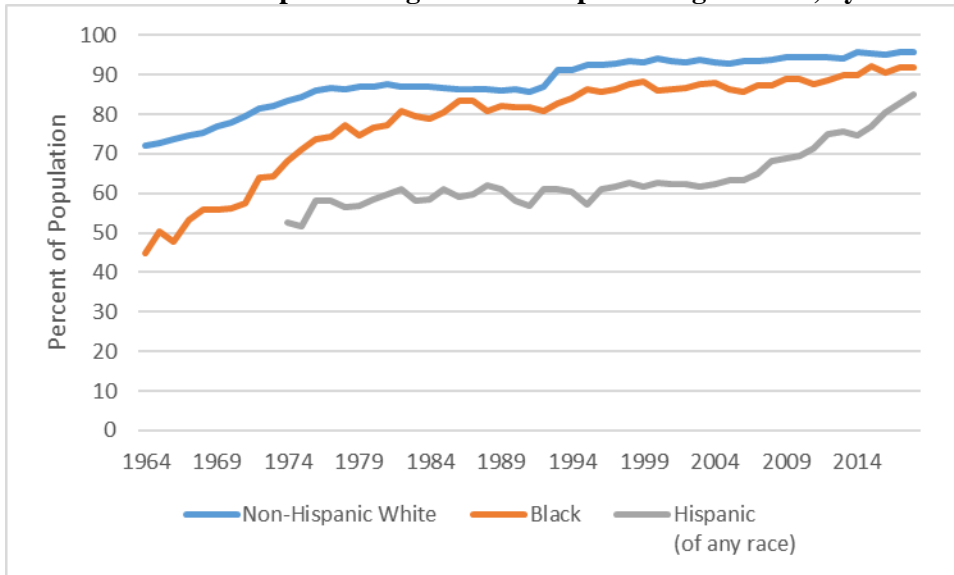
Put another way, the percentage of both males and females completing college increased over 60 years. Figure 2-2 presents the percentage of the U.S. population age 25-29 that completed college from 1950 to 2010 by gender. A greater percentage of males (11.7 percent) than females (8 percent) graduated from college in 1950, with a gender gap of 3.7 percentage points. Initially, college completion expanded faster for males than females, resulting in a peak gender gap of 13.1 percentage points in 1960. The gap began to narrow as more women were completing college, with the gender gap ranging from 4.6 to 7.6 percent between 1965 and 1990. The gender gap switched in 1995, with a greater percentage of females completing college than males. By 2010, a greater percentage of young females (37.2 percent) graduated from college than males (30 percent), with the gender gap in favor of females (7.2 percent). Educational opportunities did not expand as early nor as greatly for racial-ethnic minorities, specifically blacks. *Brown v. Board of Education* (1954), the landmark American Supreme Court decision challenging *Plessy v. Ferguson*'s (1896) "separate but equal" policy, mandated desegregation of

public schools in the United States (Patterson 2001). The *Brown* decision provided blacks access to educational opportunities in once all-white schools, integrating 11.5 million black and white students enrolled in nearly 11,200 American school districts. Additionally, *Brown* set precedent to increase diversity in higher education. integration of public universities provided blacks educational opportunities outside of attending historically black colleges and university (Wallenstein 2003).

Despite the constitutional law, however, integration policies were slow to take root, and many school districts remained segregated for years after (Wallenstein 2003). Approximately 14 states maintained virtually unchanged segregated school systems (e.g., North Carolina, Virginia), with some states operating two systems of higher education until the 1960s and beyond. Seventy percent of black students continued to attend historically black colleges and universities in the 1960s, although this proportion dropped to 20 percent by 2000.

Meanwhile, public schools were the main source of education for Hispanics by the 1930s, but these schools were segregated and poorly funded (Contreras 2004). Hispanic students were also likely to drop out due to pervasive poverty situations. After World War II, Hispanics gained greater access to public schools, spurred by factors such as the continuing expansion of public education, increasing economic status of communities to fund facilities, and new curricula aimed at educating working-class children.

Figure 2-3: Percent of US Population age 25-29 Completed High School, by Race-Ethnicity

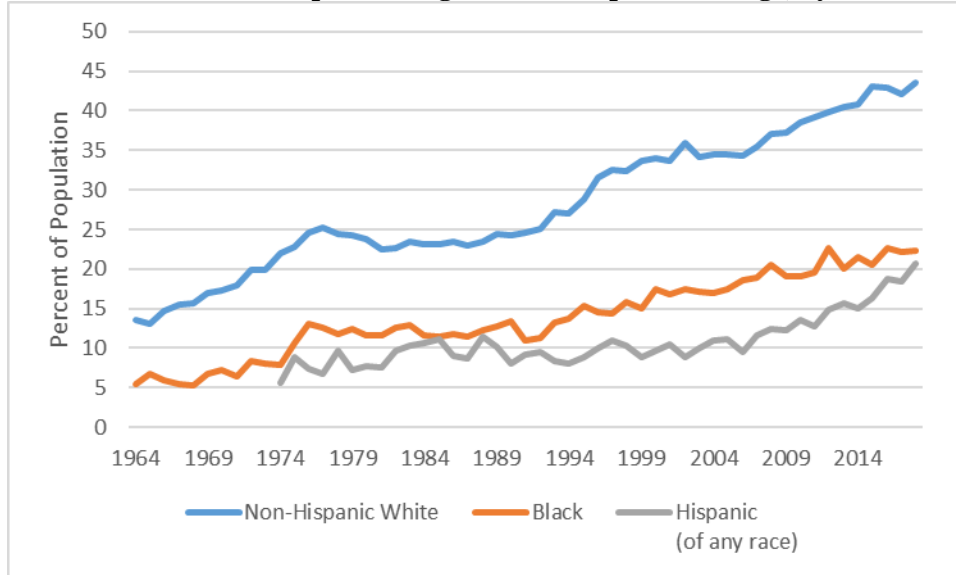


Source: U.S. Census Bureau, 1964 to 2002 March Current Population Survey, 2003 to 2018 Annual Social and Economic Supplement to the Current Population Survey (noninstitutionalized population, excluding members of the Armed Forces living in barracks)

Figure 2-3 presents the percentage of the U.S. population age 25-29 that completed high school from 1964 to 2018 by three racial-ethnic groups; Non-Hispanic whites, blacks, and Hispanics. A far greater percentage of whites (72.1 percent) graduated from high school in 1964 than blacks (45 percent). Hispanic education is first recorded by the Census in 1974, with the percentage of blacks (68.2 percent) and Hispanics (52.5 percent) graduating from high school lower than for whites (83.4 percent) in the same year. Rates of high school graduation increase steadily for whites and blacks across the 54 years, with the white-black gap in high school completion shrinking to only 3.6 percent in 2018. Meanwhile, approximately one-half to less than two-thirds of Hispanics graduate from high school from 1974 to 2000 with little or no educational expansion observed, unlike their white and black counterparts. The percent of Hispanics graduating high school begins to converge with whites and blacks after 2005. By

2018, high school graduation rates have converged, where whites (95.6 percent) and blacks (92 percent) are similar, and lower for Hispanics (85.2 percent).

Figure 2-4: Percent of US Population age 25-29 Completed College, by Race-Ethnicity



Source: U.S. Census Bureau, 1964 to 2002 March Current Population Survey, 2003 to 2018 Annual Social and Economic Supplement to the Current Population Survey (noninstitutionalized population, excluding members of the Armed Forces living in barracks)

The racial-ethnic disparity in education converges for high school graduation, but remains substantial for college graduation. Figure 2-4 presents the percentage of the U.S. population age 25-29 that completed college from 1964 to 2018 by the three racial-ethnic groups. A far greater percentage of whites (13.6 percent) graduated from college in 1964 than blacks (5.5 percent). By 1974, the college graduation rate for blacks (7.9) and Hispanics (5.7) is substantially lower compared to whites (22 percent). Rates of college graduation triples for whites across the 54 years, reaching 43.5 percent in 2018. The rates for quadruple for blacks and Hispanics across the same time period, but the rates in 2018 are substantially lower (22.3 and 20.7 percent, respectively) compared to whites. While all group experience high educational expansion, the racial-ethnic gaps between whites and non-whites increases. The white-black gap increases from

8.1 percent in 1964 to 21.2 percent in 2018 (more than doubling) and the white-Hispanic gap increases from 16.3 percent in 1974 to 22.8 percent in 2018 (one-third increase).

Research Questions and Contribution

The dissertation uses age-specific cohort-analysis to address the research questions. Cohort and age effects need to be simultaneously addressed to interacting effects with education, shifting the magnitude and significance of education when either age or cohort are omitted in health models (Lynch 2003). For example, educational health disparities in mortality shift with changing institutions, culture, technology, and demography (Masters et al 2012; Warren and Hernandez 2007). Additionally, research finds cohorts experience changing health exposures due to public health improvements, changing diet and nutrition, and policies providing compulsory schooling (Finch and Crimmins 2004; Masters et al. 2012).

While past cross-sectional research reports a strong linear and negative relationship between educational attainment and the overall risk of type 2 diabetes, little longitudinal analysis of this relationship has been conducted. Specifically, despite the large increases that occurred in both educational attainments and diabetes, overall, during the 20th century, past research has not studied how the relationship between education and diabetes has changed across successive cohorts. In addition, past research has not examined the effect of the changing educational composition on diabetes across cohorts. Finally, the effect of changing gender and race-ethnic disparities across cohorts in the magnitude of the relationship between educational attainments and diabetes, and the effect of changing gender and race-ethnic disparities across cohorts in the educational composition of the U.S. population on diabetes have not been systematically studied.

The gaps in research motivate four questions. First, has the magnitude of the relationship between education and diabetes changed across cohorts? How have the increases in the

education composition across cohorts influenced diabetes prevalence rates? Does education have unequal effects on diabetes by gender and by race-ethnicity across cohorts? And did unequal increases in education composition impact diabetes differently by gender and by race-ethnicity across cohorts? Questions 1 and 3 both pertain to the magnitude of the relationship, while question 2 and 4 focus on the educational compositional effect.”

We know that as the U.S. experienced increasing prosperity after World War II, that the prevalence of chronic diseases, including diabetes, increased, and that these changes were associated with a range of factors which included increasing obesity, tobacco use, and sedentary occupations (Flegal et al. 2010; McKeown 2009). There are substantial gender and racial-ethnic disparities in diabetes prevalence, with higher rates among males than females (Kautzky-Willer et al. 2015; CDC 2003; Menke et al. 2014; Sattar 2013), and higher rates among blacks and Hispanics than non-Hispanic whites (Cockerham 2016; Brancati et al. 1996; Robbins et al. 2001, 2000; CDC 2003).

Additional analyses find that among earlier generations in the U.S., the most highly educated were more likely to engage in risky health behaviors, such as cigarette smoking, which contribute to chronic disease (Baker et al. 2011), but that, as such behaviors became more prevalent, and knowledge of negative health effects reached the most educated, the relationship between education and risky health behavior reversed. It is not known, however, how the educational gradient differs by gender and race-ethnicity across generations with regard to the prevalence of and mortality from specific chronic diseases, like diabetes, that is, it is not known how gender and race-ethnic disparities in the relationship between education and the prevalence of diabetes changed across cohorts. Additionally, it is not known how compositional changes in education in gender and racial-ethnic groups (i.e., educational expansion) affected diabetes

prevalence for these subpopulations, that is, it is not known how changing gender and race-ethnic disparities in educational attainment affected gender and race-ethnic disparities in the prevalence of diabetes

This dissertation reports new results from three sets of analyses for four cohorts at ages 60-64 who were born in 1935-1939, 1940-1944, 1945-1949, and 1950-1954. In Chapter 5, the first set of results focus on the extent to which changes in the prevalence of diabetes across these cohorts can be accounted for by changes in the relationship between education and diabetes across cohorts and by changes in education composition across cohorts. In Chapter 6, the second set of results focus on the extent to which changes in gender disparities in the prevalence of diabetes across cohorts can be accounted for by changing gender disparities in the relationship between education and diabetes across cohorts and by changing gender disparities in education composition across cohorts. In Chapter 7, the third set of results focus on the extent to which changes in race-ethnicity disparities in the prevalence of diabetes across cohorts can be accounted for by changing race-ethnicity disparities in the relationship between education and diabetes across cohorts and by changing race-ethnicity disparities in education composition across cohorts.

Chapter 3

Data and Research Methods

Summary of Datasets

Data analyzed here are from the 1997-2016 National Health Interview Surveys (NHIS) obtained from the IPUMS Health Surveys (Blewett et al. 2018). The NHIS is a primary source of health data for the U.S. population, including general health, prevalence of acute and chronic illness, functional limitations, access to and usage of medical services, insurance coverage, and health behaviors such as exercise, diet, tobacco use, and alcohol consumption (Blewett et al. 2018). The survey interviews an annual average of 100,000 persons in 45,000 households. IPUMS provides variable codes that are identical across data sets to allow for consistent cross-time comparisons. In each year, the sample adult questionnaire is randomly administered to one adult age 18 or above in the household, with each eligible adult in a family having the same chance of being selected. Included for the dataset is an IPUMS-constructed sample weight (SAMPWEIGHT), based on the Final Annual Sample adult and Sample Child weights in the original NHIS public use files, representing the random selection of a sample person in the household to complete a supplement weight. The sample weights represent the inverse probability of selection into the sample, adjusted for non-response with post-stratification adjustments for sex, race-ethnicity, and age using Census population control totals. This allows researchers to calculate nationally representative estimates.

The current study created an IPUMS NHIS extract data set to analyze changes for four 5-year birth cohorts. Prior research indicates that diabetes diagnosis peaks between 60 and 74 years (Cowie et al. 2010). To maximize the available sample size during the ages of highest diabetes prevalence, persons age 60-64 selected for study here. The dataset analyzed in Chapter 6 focuses on all persons ages 60-64 for the four cohorts born between 1935-39 (N=7,019), 1940-44

(N=7,540), 1945-49 (N=9,311), and 1950-54 (N=10,231), a total sample of 34,101 respondents.

The dataset analyzed in Chapter 7 focuses on gender differences. The dataset analyzed in Chapter 8 focuses on race-ethnic differences. A more extensive discussion of sample and subsamples is provided in Chapter 4.

Summary of Hypotheses

Chapter 6 addresses two questions. **Research Question 1** is: *Has the magnitude of the relationship between education and diabetes changed across successive cohorts?* **Research Question 2** is: *How have increases in education levels across cohorts influenced the overall diabetes prevalence rate?* Research Question 1 pertains to the effect of educational attainment on diabetes for each cohort, while the second focuses on the effect of changes in educational composition across cohorts on the prevalence of diabetes. The first hypothesis is that the effect size of educational attainment on being diagnosed with diabetes has remained stable across cohorts between 1935 and 1954. Insofar as educational attainment is negatively related to being diagnosed with diabetes, and insofar as educational attainments have increased across cohorts, the second hypothesis is that if each successive cohort had experienced the educational composition of the previous cohort then the diabetes prevalence rate for the more recent cohort would have been higher than the rate actually experienced.

The analyses in Chapters 7 & 8 address the same questions, by gender (male vs. female) and for three specific race-ethnic groups (non-Hispanic whites, Hispanics, and non-Hispanic blacks). These results provide the basis for **Research Question 3**: *Does educational attainment have different effects on type 2 diabetes rates for various gender and race-ethnic groups?* The results will assess differences in effect size by gender and race-ethnicity within cohorts and differences in changes in effect size by gender and race-ethnicity across cohorts. **Research**

Question 4 focusses on differences within cohorts and changes across in education composition by gender and race-ethnicity: *Did differential changes in education composition by gender and race-ethnicity lead to changes in disparities in diabetes prevalence?*

Defining Key Concepts

Dependent Variable: Diagnosed with Diabetes

The main dependent variable is a binary variable indicating whether or not (yes=1, no=0) the respondent was diagnosed with diabetes at age 15 or older. This variable is created from two variables. The first source variable (DIABETICEV) identifies respondents who have ever been diagnosed with “diabetes or sugar diabetes by a doctor or other health professional”. Adult respondents could answer “yes,” “no,” or “borderline.” For the sake of creating conservative estimates, respondents for the current study are identified as having diabetes only if they answer “yes”. Validity of self-reported diabetes in the NHIS is high for those diagnosed with diabetes (Edwards et al. 1994)

The second source variable (DIABETICAGE) reports the age at first diagnosis only if respondents were told by a doctor or other health professional that they had “diabetes or sugar diabetes.” The goal is to identify persons diagnosed with type 2 diabetes, which occurs later in life (Cowie et al. 2010). This is not to be construed with type 1 diabetes. For the main dependent variable, diabetics who were first diagnosed at age 15 or later were coded as “yes,” and all other respondents were coded as “no.” The NHIS 2006 *Codebook* includes a warning about a possible source of inaccuracy in this variable, that some respondents may have given information about how long ago the condition was first diagnosed, rather than the age of first diagnosis. As there is no flag for which respondents answered this way, there is no clear way to adjust for this potential problem.

Main Independent variable: Educational Attainment

NHIS reports the highest level of schooling a respondent has completed (EDUC). For less than a high school degree, respondents are coded according to the highest grade completed (e.g., Grade 1, Grade 2, etc.). For high school graduates and those with higher education, respondents are coded in terms of their degree (e.g., AA degree, Bachelor's degree, etc.). Only "regular schooling which advances a person toward an elementary or high school diploma, or a college/university/professional school (such as law, medicine, dentistry)" counted as education. The EDUC variable was coded into four categories; 1) less than high school, 2) high school only (including persons with a GED), 3) some college and, 4) at least a bachelor's degree. The reference group in analysis is high school only, chosen because it is the largest of the four educational groups across for the sample as a whole.

Covariates

Covariates in analysis include population characteristics (gender, race-ethnicity, and marital status), family income, health insurance status, body mass index (BMI), and region.

Among population composition, gender is recoded as a binary variable, *female* (Male=0, Female=1). *Race* has been coded as six mutually exclusive categories; "Non-Hispanic White", "Non-Hispanic Black," "Hispanic," "Asian/Pacific Islander," and "Other (non-Hispanic)." Non-Hispanic whites serve as the reference group. *Marital status* has been coded into six categories; "Married," "Single/Never Married," "Cohabiting," "Separated," "Divorced," and "Widowed." Preliminary analysis finds these categories to be distinctive from one another with regard to the dependent variable, including between separated, divorced, and widowed.

The covariate, *family income*, is an intervening variable of education on improved health (Woolf and Bravemen 2011). With higher family incomes, persons have greater disposable

income to address health concerns, and prior research on Americans finds that lower family income is associated with worse health outcomes (Braveman et al. 2010). The variable is coded in \$1,000 increments. Since the dataset includes persons across a 20-year period, family incomes are adjusted to 2016 dollars using the Consumer Price Index (give reference).

Health insurance status is a second intervening variable of education on improved health (Woolf and Bravement 2011). Hadley (2003) reported an extensive literature review, finding uninsured persons are less likely to use preventive services and having timely diagnosis. Thus, since insured persons are more likely to get medical diagnosis, they may have greater odds of being diagnosed with diabetes. But currently having health insurance may also lead to better overall health and lower odds of having and being diagnosed with diabetes. Additionally, the health status variable identifies whether or not respondents have health insurance at the time of survey, and there is no data on the history of health insurance coverage. This variable determines health insurance status at time of survey, rather than history of health insurance coverage.

Body Mass Index (BMI) is linked to diabetes (Bo et al. 2001) and serves as a proxy for lifetime health behaviors among the respondents, as well as an intervening variable of education on improved health. While the NHIS measures health behaviors at time of survey, it does not include retrospective variables regarding food consumption in early life, healthy lifestyle in youth, or other behaviors that may differ by education. Such monumental shifts from the epidemiological transition make current health behavior variables inappropriate for study. BMI, however, is a proxy of such behaviors in early life.

Diabetes prevalence may differ by *region*, with different levels and types of food consumption or healthy behaviors. Region is coded into the Northeast, South, Midwest, and West. The South is the reference group.

Summary of Research Method

Research methods are described in Chapter 4, which first presents weighted descriptive statistics for all variables for the full sample, and trends across cohort. Next, weighted descriptive statistics are presented for specific subpopulations (gender and race-ethnicity) within and across cohorts. Chapter 4 then presents multivariate analyses of diabetes prevalence by educational attainment across cohorts. This involves analyses for the sample as a whole and for specific gender and race-ethnic groups.

Chapter 5 methods consist of two sets of multivariate analyses. The first set (testing Hypothesis 1) involves eight logit regression analyses (two for each cohort), predicting the odds of having ever been diagnosed with diabetes at age 25 older. The first regression model for each cohort includes the main independent variable (education) and population composition variables (gender, race, marital status, and region). Intervening variables are omitted from the first models to measure the overall effect of education on diabetes. The second model for each cohort includes intervening variables (family income, health insurance, and BMI) to examine how education's effect works through these variables. Odds ratios greater than one (>1.00) indicate an increased risk of being diagnosed with diabetes compared to a reference groups, while odds ratios less than one (<1) indicate a decreased risk of being diagnosed with diabetes compared to a reference group.

Interpretation begins with the 1935-39 birth cohort to establish the relationship between educational attainment and diabetes for the earliest cohort, controlling for all covariates. Based on previous research, educational attainment is expected to have an inverse relationship with diagnosis for diabetes (Dalstra et al. 2005; Borell et al. 2006; Maty et al. 2005; CDC 2014). Thus, in this analysis using with high school only as the reference group, the odds ratio for the

less than high school group is expected to have values greater than one (>1), while odds ratios for the some college and BA+ groups are expected to have values less than one (<1).

The second set of analyses in Chapter 5 (testing Hypothesis 2) involves using the Fairlie (nonlinear) decomposition procedure. This method similar to the Blinder-Oaxaca decomposition procedure, but address issues of nonlinearity when using a binary dependent variable (Fairlie 1999). In linear regression, the Blinder Oaxaca decomposition between two groups (in this case different cohorts) for the average value of the dependent variable (Y) is written as:

$$\bar{Y}^W - \bar{Y}^B = \left[(\bar{X}^W - \bar{X}^B) \hat{\beta}^W \right] + \left[\bar{X}^B (\hat{\beta}^W - \hat{\beta}^B) \right]$$

“where \bar{x}^j is the row vector of average values of the independent variables and $\hat{\beta}^j$ is a vector of coefficient estimates for cohort j ” (Fairlie 1999: 93). Such decomposition is widely used to measure the contributions of differences in observable variables (Fairlie 2017). The Blinder-Oaxaca decomposition, however, may provide misleading estimates for binary dependent variables. Nonlinear models are computationally more demanding than linear models, and user-written programs such as “decomp,” “decompose”, and “oaxaca” do not extend decomposition to linear models (Bartus 2006; Powers and Yun 2009). Instead, nonlinear programs are available, such as “fairlie” (Fairlie 1999, 2017), “gdecomp” (Bartus 2006), mvdcmp” (Powers et al. 2011) and “nldecompose” (Sinning et al. 2008).

The Blinder-Oaxaca decomposition model include three effects; the endowment effect, the coefficient effect, and the marginal effects (Bartus 2006). Fairlie decomposition focuses on the endowments, or the effect of changes in independent variables between two samples on the difference in the probability of the response of the dependent variable between the same two groups. Thus, the Fairlie decomposition (first in Fairlie 1999) is a simulation method of

performing a nonlinear decomposition, with estimates from logit, probit, or non-linear models.

The logit model is written as:

$$\bar{Y}^W - \bar{Y}^B = \left[\sum_{i=1}^{N^W} \frac{F(X_i^W \hat{\beta}^W)}{N^W} - \sum_{i=1}^{N^B} \frac{F(X_i^B \hat{\beta}^W)}{N^B} \right] + \left[\sum_{i=1}^{N^B} \frac{F(X_i^B \hat{\beta}^W)}{N^B} - \sum_{i=1}^{N^B} \frac{F(X_i^B \hat{\beta}^B)}{N^B} \right],$$

where N^j is the sample size for cohort j . This expression is used as an alternative for the decomposition because \bar{Y} does not necessarily equal $F(\bar{x} \hat{\beta})$ (Fairlie 1999). In both equations, the first bracketed term represents the portion of the difference between cohorts for the dependent variable that is due to group differences in distributions of X (population composition with regard to X) and the second term represents the portion of the difference for the dependent variables that is due to differences in group processes (rate effects of X on Y) determining levels of Y (Fairlie 1999).

Changes across two cohorts are examined in each of the four Fairlie models; 1) persons born in 1935-39 vs. 1940-44, 2) persons born in 1940-44 vs 1945-49, 3) persons born in 1945-49 vs 1950-54, and examining the change across the full study period 4) persons born in 1935-39 vs 1950-54. The estimation of each model begins with a pooled logit regression, followed by a decomposition analysis involving three estimates. First is the difference (change) in diabetes prevalence rates across cohorts. Second is the magnitude of the difference (change) in diabetes prevalence due to overall compositional changes in independent variables across cohorts. Third, a procedure provides for testing the statistical significance of the changing compositional effects for specific independent variables.

Table 3-1 describes the interpretation of results from the Fairlie analysis. For example, if the change in diabetes prevalence is positive, then the diabetes prevalence rate for the second cohort (C2) is greater than the prevalence for the first cohort (C1). If the change has a negative

value, however, then the opposite is true, diabetes prevalence declined across cohorts. For the difference explained by endowments and the individual coefficients for variables, a negative and significant value indicates the independent variable composition of C2 had a negative effect on diabetes prevalence compared to C1. A positive and significant value indicates that the independent variable composition of C2 had a positive effect on diabetes prevalence compared to C1.

Table 3-1: How Composition Change Coefficients Are Interpreted In This Study

DIRECTION OF COHORT DIFFERENCE	DIRECTION OF CHANGE IN EDUCATION EFFECT	GENERAL INTERPRETATION OF COMPOSITION CHANGE COEFFICIENT	DETAILED INTERPRETATION OF COMPOSITION CHANGE COEFFICIENT
POSITIVE GREATER DIABETES PREVALENCE IN COHORT 2	Positive	Composition change by itself would have increased the diabetes rate across cohorts	If Cohort 2 had experienced the composition of Cohort 1, then the increase in diabetes across cohorts would have been smaller
	Negative	Composition change by itself would have decreased the diabetes rate across cohorts	If Cohort 2 had experienced the composition of Cohort 1, then the increase in diabetes across cohorts would have been larger
NEGATIVE REDUCED DIABETES PREVALENCE IN COHORT 2	Negative	Composition change by itself would have reduced the diabetes rate across cohorts	If Cohort 2 had experienced the composition of Cohort 1, then the decrease in diabetes across cohorts would have been smaller
	Positive	Composition change by itself would have increased the diabetes rate across cohorts	If Cohort 2 had experienced the composition of Cohort 1, then the decrease in diabetes across cohorts would have been larger

The methods used to analyze data in Chapter 6 (gender) and 7 (race-ethnicity) are the same as the methods used in Chapter 5, but separate analyses are conducted for each gender and race-ethnic group. This provides the basis for a detailed understanding of changing disparities by gender and by race-ethnicity with regard to the effect of educational attainment on diabetes and

with regard to the nature and the effect of education composition changes for group-specific diabetes rates. Chapter 8 discusses interpretations of all models in Chapters 5-7. Chapter 9 discusses future research.

Chapter 4

Sample and Descriptive Statistics

Sample

Table 4-1: N's for Overall Sample and Age-Specific Cohort subsamples by Gender and by Race

	1935-39	1940-44	1945-49	1950-54	Full Sample
1) Overall Sample	7,019	7,540	9,311	10,231	34,101
2) Gender					
Males	3,236	3,443	4,355	4,682	15,716
Females	3,783	4,097	4,956	5,549	18,385
3) Race-Ethnic Groups					
Whites	5,201	5,491	6,608	7,278	24,578
Hispanics	796	808	967	1,025	3,596
Blacks	850	992	1,284	1,425	4,551

Table 4-1 provides sample sizes for the overall sample and for age-cohort-specific subsamples by gender and by race-ethnicity. This study analyzes data for persons interviewed at ages 60-64 years old for the 1997-2016 National Health Interview Survey (NHIS) IPUMS dataset, who were born between 1935 and 1954. The total sample of persons ages 60-64 born between 1935 and 1954 is 34,101 respondents. The sample is divided into four cohorts born in 1935-39 (N=7,019), 1940-44 (N=7,540), 1945-49 (N=9,311), and 1950-54 (N=10,231). Results for each 5-year cohort are based on data from five NHIS surveys. The second set of analyses distinguishes persons by gender—male (N=15,712) and female (N=18,385). The third set of analyses distinguishes persons by race-ethnicity— white (N=24,578) and non-Blacks (N=4,551) and Hispanics (N=3,596).

Strengths of the Data

First, the NHIS dataset has a sample substantial enough to provide estimates for many subpopulations (Backinger et al. 2008). The year 2000 interviewed sample consisted of roughly 39,000 households, resulting in data for 100,000 persons in all fifty states (Gentleman and Pleis 2002). This makes it possible to develop estimates for age-specific cohorts, by gender and by

race. Second, the NHIS uses in-person interviews, allowing for in-depth questioning. Third, the NHIS includes a wide variety of health variables, including specific diseases and the age at which they were diagnosed, health knowledge and health behaviors.

Limitations of the Data

The NHIS uses self-reported or proxy data, which is may be less accurate than other data sources. Some respondents may misremember retrospective information, or not answer honestly to questions about behaviors that they consider undesirable, all of which may lead to underestimates (Backinger 2008). More accurate data can be obtained from physical examinations conducted by National Health and Nutrition Examination Survey (NHANES). While the NHANES is the gold standard for health analysis, a preliminary analysis found the NHANES sample size insufficient to examine age-specific cohorts, and educational and diabetic trends did not match other information sources and research (Bauman 2016; Barro and Lee 2013; Cowie et al. 2010; De Boer et al. 2011; Caspersen et al. 2012; Fishman et al. 2014; CDC 2017).

The NHIS does not collect information on health behaviors at earlier time points in respondents' lives. All health behavior variables refer to respondents at the time of the survey. The Body Mass Index (BMI) variable serves as a proxy for life-time health behaviors. Therefore, and with its larger sample size, the NHIS better serves the research questions addressed in this study.

Descriptive Statistics

Specific Cohorts

Table 4-2: Descriptive Statistics of Persons Aged 60-64, by Cohort

	1935-39		1940-44		1945-49		1950-54		Full Sample	
	Mean/%	SD	Mean/%	SD	Mean/%	SD	Mean/%	SD	Mean/%	SD
Diabetes	12.6%		13.9%		16.4%		16.2%		15.1%	
EDUCATION										
Less than HS	21.7%		17.0%		12.7%		11.1%		14.9%	
HS Only	34.0%		33.1%		28.0%		24.5%		29.2%	
Some College	22.9%		24.1%		27.9%		30.5%		26.9%	
BA/BS+	21.5%		25.8%		31.5%		33.9%		29.1%	
% Any College	44.3%		49.9%		59.3%		64.3%		55.9%	
GENDER										
Male	50.3%		49.0%		50.7%		48.4%		49.6%	
Female	49.7%		51.0%		49.3%		51.6%		50.4%	
RACE-ETHNICITY										
Non-H White	81.7%		79.9%		78.9%		74.8%		78.4%	
Non-H Black	8.7%		9.3%		9.2%		11.2%		9.7%	
Hispanic	6.8%		7.2%		7.6%		9.1%		7.8%	
Asian/Pacific Islander	2.3%		2.8%		3.4%		4.2%		3.3%	
Native American	0.4%		0.7%		0.7%		0.5%		0.6%	
Other	0.1%		0.1%		0.3%		0.2%		0.2%	
MARTIAL STATUS										
Married	73.0%		69.6%		67.8%		65.3%		68.4%	
Single/Never Married	3.4%		4.2%		5.0%		7.0%		5.1%	
Divorced	10.7%		13.6%		15.4%		15.8%		14.3%	
Separate	1.8%		1.6%		1.9%		2.0%		1.9%	
Widowed	9.3%		8.3%		6.6%		6.3%		7.4%	
Cohabiting	1.8%		2.6%		3.3%		3.5%		2.9%	
MECHANISMS										
Family Income (1000s) in 2016 \$	55.52	0.46	56.11	0.45	65.12	0.48	64.65	0.50	61.23	0.25
Has Health Insurance	90.7%		89.4%		89.2%		91.2%		90.1%	
BMI	27.29	0.06	27.84	0.07	28.22	0.06	28.27	0.07	27.98	0.03
REGION										
Northeast	19.6%		19.0%		18.3%		18.2%		18.6%	
Midwest	25.1%		23.4%		23.6%		22.6%		23.5%	
South	37.4%		38.7%		36.3%		37.9%		37.5%	
West	17.9%		18.9%		21.9%		21.3%		20.3%	
N	7,019		7,540		9,311		10,231		34,101	

Table 4-2 presents weighted population estimates for all cohorts and each specific cohort. descriptive statistics for the sample as a whole and by cohort. The earliest-born cohort (1935-39) had the lowest prevalence rate at 12.6 percent. The diabetes prevalence rate was 1.3 percentage points higher for 1940-44 cohort at 13.9 percent, for an increase of about one-tenth. The 1945-49 cohort experiences a diabetes prevalence rate 2.5 percentage points greater than the preceding cohort at 16.4 percent (nearly one-fifth greater than the 1940-44 cohort). The diabetes prevalence rate was 0.2 percentage points lower for the 1950-54 cohort at 16.2 percent.

For each successive cohort, persons become more highly educated. Across cohorts, the percentage of persons with less than a high school degree declines by nearly half, from 21.7 percent for the 1935-39 cohort to 11.1 percent for the 1950-54 cohort. The percentage of persons with a high school degree declines by a somewhat smaller 9.5 percentage points, from 34.0 percent for the 1935-39 cohort to 24.5 percent for the 1950-54 cohort. Meanwhile, both categories of college experience increase. Persons with some college increases from 22.9 percent for the 1935-39 cohort to 30.5 percent for the 1950-54 cohort (a difference of 7.6 percentage points). Persons with at least a bachelor's degree increases by more than one-half, from 21.5 percent for the 1935-39 cohort to 33.9 percent for the 1950-54 cohort.

Examining gender, there are slightly more males for the 1935-39 and 1945-49 cohorts (50.3 percent and 50.7 percent, respectively), and fewer males for the 1940-44 and 1950-54 cohorts (49 percent and 48.4 percent, respectively).

Across cohorts, the percentage white declines by nearly 7 percentage points, from 81.7 percent for the 1935-39 cohort to 74.8 percent for the 1950-54 cohort. Meanwhile, the percentage of blacks increases from 8.7 percent (1935-39 cohort) to 11.2 percent (1950-54 cohort). The percentage Hispanic increases from 6.8 percent (1935-39 cohort) to 9.1 percent (1950-54 cohort). The percentage of Asians/Pacific Islanders increases from 2.3 percent (1935-39 cohort) to 4.2 percent (1950-54 cohort). The percentage who are Native Americans and Others increases only marginally.

Across cohorts, the percentage of persons married declines by nearly 8 percentage points between 1935-39 (73.0 percent) and 1950-54 (65.3 percent). Similarly, the percentage of persons who are widowed decreases from 9.3 percent for the 1935-39 cohort to 6.3 percent for the 1950-54 cohort. The percentage of persons single or never married more than doubles from 3.4 percent

for the 1935-39 cohort to 7.0 percent for the 1950-54 cohort. The percentage of persons divorced increases from 11 percent (1935-39 cohort) to 16 percent (1950-54 cohort). The percentage of persons cohabiting nearly doubles from 1.8 percent (1935-39 cohort) to 1.5 percent (1950-54 cohort). The percentage of persons separated remain unchanged.

Examining intervening variables that education may work through, later cohorts have higher family income, similar rates of health insurance, and higher BMI than earlier cohorts. The 1935-39 cohort experienced an average family income of \$55,520, with the 1940-44 cohort experiencing a slightly higher family income of \$56,110, which increased to \$65,120 for the 1945-49 cohort, and then decreased slightly to \$64,650 for the 1950-54 cohort. Across the four cohorts, between 89.4 and 91.2 percent had health insurance. BMI increased for each successive cohort. The 1935-39 cohort experienced an average BMI of 27.29, which increased to 28.27 for the 1950-54 cohort.

Turning to regional geographic distribution for the earliest-born cohort, the largest percentage of persons in each cohort lived for the South (37.4 percent), followed by the Midwest (25.1 percent), the West (17.9 percent), and the Northeast (19.6 percent). There are only small changes for the regional distribution across cohorts.

Characteristics by Gender and by Race-Ethnicity Cohorts

1) Males

Table 4-3: Descriptive Statistics for Males Aged 60-64, by Cohort

	1935-39		1940-44		1945-49		1950-54		Full Male Sample	
	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE
Diabetes	13.9%		15.4%		17.4%		17.2%		16.2%	
EDUCATION										
Less than HS	21.6%		16.2%		12.4%		10.8%		12.4%	
HS Only	29.6%		29.9%		25.0%		23.9%		25.0%	
Some College	21.5%		23.6%		26.7%		28.7%		26.7%	
BA/BS+	27.3%		30.3%		35.9%		36.6%		35.9%	
% Any College	48.8%		53.9%		62.6%		65.4%		58.9%	
RACE-ETHNICITY										
Non-H White	82.0%		80.6%		80.0%		75.1%		79.1%	
Non-H Black	8.3%		8.7%		8.8%		10.6%		9.2%	
Hispanic	6.8%		7.2%		7.1%		9.3%		7.7%	
Asian/Pacific Islander	2.4%		2.7%		3.1%		4.3%		3.2%	
Native American	0.5%		0.7%		0.7%		0.6%		0.6%	
Other	0.1%		0.2%		0.4%		0.1%		0.2%	
MARTIAL STATUS										
Married	80.4%		76.5%		73.6%		70.8%		74.7%	
Single/Never Married	3.7%		4.3%		4.5%		7.4%		5.1%	
Divorced	8.8%		10.8%		13.1%		13.7%		12.0%	
Separated	1.7%		1.6%		1.5%		1.7%		1.6%	
Widowed	3.1%		3.7%		3.2%		2.9%		3.2%	
Cohabiting	2.2%		3.2%		4.1%		3.5%		3.4%	
MECHANISMS										
Family Income (1000s) in 2016 \$	60.10	0.67	60.50	0.66	69.22	0.69	66.92	0.74	64.97	0.36
Has Health Insurance	91.7%		90.1%		90.5%		91.3%		90.8%	
BMI	27.56	0.08	28.05	0.09	28.29	0.08	28.51	0.09	28.17	0.04
REGION										
Northeast	19.6%		20.1%		17.8%		18.8%		18.9%	
Midwest	24.7%		23.6%		23.8%		23.0%		23.7%	
South	37.2%		36.9%		36.9%		36.5%		36.8%	
West	18.5%		19.4%		21.5%		21.7%		20.6%	
N	3,236		3,443		4,355		4,682		15,716	

Table 4-3 provides weighted population estimates for all males and by male cohort. The lowest diabetes rate is for the earliest-born cohort (1935-39) at 13.9 percent. The diabetes prevalence rate for the succeeding cohort (1940-44) increases by 1.4 percentage points, that is, by about one-tenth. The 1945-49 cohort experiences a diabetes prevalence that is 2 percentage points higher at 17.2 percent, which is more than one-tenth higher than among the 1940-44 cohort. The diabetes prevalence rate changes only slightly for the 1950-54 cohort to 17.2 percent. The diabetes prevalence of males only is slightly lower than the diabetes prevalence for the entire sample (as seen in Table 4-2).

Table 4-3 shows that for each successive cohort, males become more highly educated. Across cohorts, the percentage of males with less than a high school degree declines by one-half, from 21.6 percent for the 1935-39 cohort to 10.8 percent for the 1950-54 cohort. The percent of males with a high school degree declines by 5.7 percentage points, from 29.6 percent for the 1935-39 cohort to 23.9 percent for the 1950-54 cohort. Meanwhile, both categories of college experience increase. Males with some college increases from 21.5 percent for the 1935-39 cohort to 28.7 percent for the 1950-54 cohort (an increase of 7.2 percentage points). Males with at least a bachelor's degree increase by 9.3 percentage points, from 27.3 percent for the 1935-39 cohort to 36.6 percent for the 1950-54 cohort.

For each successive cohort, males become more racially-ethnically diverse. Across cohorts, the percentage of whites declines by nearly 7 percentage points, from 82.0 percent for the 1935-39 cohort to 75.1 percent for the 1950-54 cohort. Meanwhile, the percentage of blacks increases from 8.3 percent (1935-39 cohort) to 10.6 percent (1950-54 cohort). The percentage of males who are Hispanic increases from 6.8 percent (1935-39 cohort) to 9.3 percent (1950-54 cohort). The percentage who are Asians/Pacific Islanders increases from 2.4 percent (1935-39 cohort) to 4.3 percent (1950-54 cohort). The percentages for Native Americans and Others experience only marginal increases.

Table 4-3 shows that later-born cohorts have lower percentages of males who are married or widowed, and greater percentages who are single, divorced, and cohabiting. Across cohorts, the percentage of males who are married at ages 60-64 declines by 9.6 percentage points between the 1935-39 cohort (80.4 percent) and the 1950-54 cohort (70.8 percent). The percentage who are widowed decreases slightly from 3.1 percent for the 1935-39 cohort to 2.9 percent for the 1950-54 cohort. The percentage of males single or never married doubles from 3.7 percent for the

1935-39 cohort to 7.4 percent for the 1950-54 cohort. The percentage of males who are divorced increases from 8.8 percent (1935-39 cohort) to 13.7 percent (1950-54 cohort). The percentage of males cohabiting increases from 2.2 percent (1935-39 cohort) to 3.5 percent (1950-54 cohort). The percentage of males separated did not change significantly across cohorts.

Examining intervening variables that education may work through, later-born male cohorts have higher family income, similar rates of health insurance, and higher BMI than earlier-born cohorts. The 1935-39 male cohort experienced an average family income of \$60,100, similar to the 1940-44 cohort (\$60,500). Family income for males peaks for the 1945-49 cohort at \$69,220, with a small decline for the 1950-54 cohort to \$66,920. Across the four cohorts, between 90 and 92 percent of males have health insurance. BMI slightly increased for each successive cohort. The 1935-39 cohort experienced an average BMI of 27.56, which increased to 28.17 for the 1950-54 cohort.

Turning to regional geographic distribution for the 1935-39 cohort, the greatest percentage of males lived in the South (37.2 percent), followed by the Midwest (24.7 percent), Northeast (19.6 percent), and the West (18.5 percent). There are only small changes for the regional distribution across male cohorts.

2) Females

Table 4-4: Descriptive Statistics for Females Aged 60-64, by Cohort

	1935-39		1940-44		1945-49		1950-54		Full Female Sample	
	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE
Diabetes	11.4%		12.4%		15.4%		15.2%		14.0%	
EDUCATION										
Less than HS	21.7%		17.9%		13.0%		11.5%		15.2%	
HS Only	38.4%		36.1%		31.0%		25.2%		31.8%	
Some College	24.2%		24.7%		29.1%		32.1%		28.1%	
BA/BS+	15.7%		21.3%		26.9%		31.3%		24.9%	
% Any College	39.9%		46.0%		56.0%		63.3%		53.0%	
RACE-ETHNICITY										
Non-H White	81.4%		79.3%		77.7%		74.6%		77.8%	
Non-H Black	9.2%		9.9%		9.7%		11.7%		10.2%	
Hispanic	6.8%		7.3%		8.1%		9.0%		7.9%	
Asian/Pacific Islander	2.2%		2.8%		3.8%		4.1%		3.4%	
Native American	0.4%		0.7%		0.6%		0.5%		0.5%	
Other	0.1%		0.1%		0.2%		0.2%		0.2%	
MARTIAL STATUS										
Married	65.4%		63.1%		61.9%		60.2%		62.3%	
Single/Never Married	3.1%		4.1%		5.6%		6.6%		5.1%	
Divorced	12.7%		16.3%		17.7%		17.8%		16.5%	
Separated	1.9%		1.7%		2.3%		2.3%		2.1%	
Widowed	15.6%		12.8%		10.1%		9.6%		11.5%	
Cohabiting	1.3%		2.1%		2.4%		3.5%		2.5%	
MECHANISMS										
Family Income (1000s) in 2016 \$	50.91	0.61	51.90	0.60	60.90	0.66	62.52	0.68	57.55	0.34
Has Health Insurance	89.7%		88.6%		87.9%		91.2%		89.3%	
BMI	27.02	0.09	27.64	0.10	28.14	0.10	28.05	0.10	27.80	0.05
REGION										
Northeast	19.7%		17.9%		18.7%		17.6%		18.4%	
Midwest	25.4%		23.2%		23.4%		22.2%		23.3%	
South	37.5%		40.4%		35.6%		39.3%		38.1%	
West	17.4%		18.5%		22.3%		20.9%		20.2%	
N	3,783		4,097		4,956		5,549		18,385	

Table 4-4 provides weighted population estimates for all females and by female cohort. The lowest diabetes rate is for the earliest-born cohort (1935-39) at 11.4 percent. The diabetes prevalence rate for the succeeding cohort (1940-44) increases by 1.0 percentage points, that is, by a little less than one-tenth. The 1945-49 cohort experiences a diabetes prevalence that is 3.0 percentage points higher at 15.4 percent, which is nearly one-quarter higher than among the 1940-44 cohort. The diabetes prevalence rate changed little for the 1950-54 cohort to 15.2 percent. The diabetes prevalence of females only is slightly higher than the diabetes prevalence for the entire sample (as seen in Table 4-2).

Table 4-4 shows that for each successive cohort, females become more highly educated. Across cohorts, the percentage of females with less than a high school degree declines by one-half, from 21.7 percent for the 1935-39 cohort to 11.5 percent for the 1950-54 cohort. The percent of females with a high school degree declines by 13.2 percentage points, from 38.4 percent for the 1935-39 cohort to 25.2 percent for the 1950-54 cohort. Meanwhile, both categories of college experience increase. Females with some college increases from 24.2 percent for the 1935-39 cohort to 32.1 percent for the 1950-54 cohort (a difference of 7.9 percentage points). Females with at least a bachelor's degree doubles, from 15.7 percent for the 1935-39 cohort to 31.3 percent for the 1950-54 cohort.

For each successive cohort, females become more racially-ethnically diverse. Across cohorts, the percentage of whites declines by nearly 7 percentage points, from 81.4 percent for the 1935-39 cohort to 74.6 percent for the 1950-54 cohort. Meanwhile, the percentage of blacks increases from 9.2 percent (1935-39 cohort) to 11.7 percent (1950-54 cohort). The percentage of females who are Hispanic increases from 6.8 percent (1935-39 cohort) to 9.0 percent (1950-54 cohort). The percentage who are Asians/Pacific Islanders increases from 2.2 percent (1935-39 cohort) to 4.1 percent (1950-54 cohort). The percentages for Native Americans and Others experience only marginal increases.

Table 4-4 shows that later-born cohorts have lower percentages of females who are married or widowed, and greater percentages who are single, divorced, and cohabiting. Across cohorts, the percentage of females who are married at ages 60-64 declines by 5.2 percentage points between the 1935-39 cohort (65.4 percent) and the 1950-54 cohort (60.2 percent). The percentage who are widowed decreases slightly from 15.6 percent for the 1935-39 cohort to 9.6 percent for the 1950-54 cohort. The percentage of females single or never married more than

doubles from 3.1 percent for the 1935-39 cohort to 6.6 percent for the 1950-54 cohort. The percentage of females who are divorced increases from 12.7 percent (1935-39 cohort) to 17.8 percent (1950-54 cohort). The percentage of females cohabiting nearly doubles from 1.3 percent (1935-39 cohort) to 3.5 percent (1950-54 cohort). The percentage of females separated did not significantly change across cohorts.

Examining intervening variables that education may work through, later-born female cohorts have higher family income, similar rates of health insurance, and higher BMI than earlier-born cohorts. The 1935-39 female cohort experienced an average family income of \$50,910, similar to the 1940-44 cohort (\$51,900). Family income for females for the 1945-49 cohort increases to \$60,900, with another increase for the 1950-54 cohort to \$62,520. Across the four cohorts, between 88.6 and 91.2 percent of females had health insurance. BMI slightly increased for each successive cohort. The 1935-39 cohort experienced an average BMI of 27.02, peaks at 28.14 for the 1945-49 cohort, and is stable at 28.05 for the 1950-54 cohort.

Turning to regional geographic distribution, the greatest percentage of females born in the 1935-39 cohort lived in the South (37.5 percent), followed by the Midwest (25.4 percent), the Northeast (19.7 percent), and the West (17.4 percent). There are only small changes for the regional distribution across female cohorts.

3) Whites

Table 4-5: Descriptive Statistics for Whites Aged 60-64, by Cohort

	1935-39		1940-44		1945-49		1950-54		Full White Sample	
	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE
Diabetes	10.9%		11.9%		14.4%		13.5%		13.0%	
EDUCATION										
Less than HS	17.4%		12.7%		8.8%		6.7%		10.7%	
HS Only	35.6%		34.5%		28.0%		24.4%		30.0%	
Some College	24.2%		25.1%		28.6%		31.6%		27.8%	
BA/BS+	22.8%		27.7%		34.7%		37.3%		31.6%	
% Any College	47.0%		52.8%		63.2%		68.8%		59.3%	
GENDER										
Male	50.5%		49.4%		51.4%		48.5%		50.0%	
Female	49.5%		50.6%		48.6%		51.5%		50.0%	
MARTIAL STATUS										
Married	76.1%		72.4%		70.5%		68.5%		71.4%	
Single/Never Married	2.7%		3.6%		4.2%		5.9%		4.2%	
Divorced	10.2%		13.1%		14.8%		15.3%		13.7%	
Separated	1.0%		1.0%		1.1%		1.2%		1.1%	
Widowed	8.6%		7.3%		6.0%		5.7%		6.7%	
Cohabiting	1.4%		2.6%		3.3%		3.4%		2.8%	
MECHANISMS										
Family Income (1000s) in 2016 \$	58.10	0.51	58.76	0.50	68.46	0.55	69.16	0.58	64.48	0.28
Has Health Insurance	93.0%		91.7%		91.0%		93.2%		92.1%	
BMI	27.21	0.07	27.74	0.08	28.14	0.07	28.18	0.08	27.88	0.04
REGION										
Northeast	20.4%		19.3%		19.4%		19.5%		19.6%	
Midwest	27.3%		26.0%		26.5%		26.0%		26.4%	
South	35.3%		37.3%		34.2%		35.1%		35.4%	
West	17.0%		17.3%		19.9%		19.5%		18.6%	
N	5,201		5,491		6,608		7,278		24,578	

Table 4-5 provides weighted population estimates for all whites and by white cohort. The lowest diabetes rate is for the earliest-born cohort (1935-39) at 10.9 percent. The diabetes prevalence rate for the succeeding cohort (1940-44) increases by 1.0 percentage points, that is, a little less than one-tenth. The 1945-49 cohort experiences a diabetes prevalence that is 2.5 percentage points higher at 14.4 percent, which is nearly one-fifth higher than among the 1940-44 cohort. The diabetes prevalence rate is 0.9 percentage points lower for the 1950-54 cohort compared to the 1945-49 cohort. The diabetes prevalence of whites only is slightly higher than the diabetes prevalence for the entire sample (as seen in Table 4-2).

Table 4-5 shows that for each successive cohort, whites become more highly educated. Across cohorts, the percentage of whites with less than a high school degree declines by one-

third, from 17.4 percent for the 1935-39 cohort to 6.7 percent for the 1950-54 cohort. The percent of whites with a high school degree declines by 11.2 percentage points, from 35.6 percent for the 1935-39 cohort to 24.4 percent for the 1950-54 cohort. Meanwhile, both categories of college experience increase. whites with some college increases from 24.2 percent for the 1935-39 cohort to 31.6 percent for the 1950-54 cohort (a difference of 8.8 percentage points). whites with at least a bachelor's degree increases by 8.8 percentage points, from 22.8 percent for the 1935-39 cohort to 37.3 percent for the 1950-54 cohort.

Examining gender among whites, there are slightly more males for the 1935-39 and 1945-49 cohorts (50.5 percent and 51.4 percent, respectively), and fewer males for the 1940-44 and 1950-54 cohorts (49.4 percent and 48.5 percent, respectively).

Table 4-5 shows that later-born cohorts have lower percentages of whites who are married or widowed, and greater percentages who are single, divorced, and cohabiting. Across cohorts, the percentage of whites who are married at ages 60-64 declines by 7.6 percentage points between the 1935-39 cohort (76.1 percent) and the 1950-54 cohort (68.5 percent). The percentage who are widowed decreases slightly from 8.6 percent for the 1935-39 cohort to 5.7 percent for the 1950-54 cohort. The percentage of whites single or never married nearly triples from 2.7 percent for the 1935-39 cohort to 5.9 percent for the 1950-54 cohort. The percentage of whites who are divorced increases from 10.2 percent (1935-39 cohort) to 15.3 percent (1950-54 cohort). The percentage of whites cohabiting more than doubles from 1.4 percent (1935-39 cohort) to 3.4 percent (1950-54 cohort). The percentage of whites separated did not significantly across cohorts.

Examining intervening variables that education may work through, later-born white cohorts have higher family income, similar rates of health insurance, and higher BMI than

earlier-born cohorts. The 1935-39 cohort experienced an average family income of \$58,100, similar to the 1940-44 cohort (\$58,760). Family income for whites for the 1945-49 cohort increases to \$66,460, with another increase for the 1950-54 cohort to \$69,160. Across the four cohorts, between 91.0 and 93.2 percent of whites had health insurance. BMI slightly increased for each successive cohort. The 1935-39 cohort experienced an average BMI of 27.21, highest for the 1950-54 cohort at 28.18.

Turning to regional geographic distribution, the greatest percentage of whites in each cohort lived for the South (35.3 percent), followed by the Midwest (27.3 percent), the Northeast (20.4 percent), and the West (17.0 percent). There are only small changes for the regional distribution across white cohorts.

4) *Hispanics*

Table 4-6: Descriptive Statistics for Hispanics Aged 60-64, by Cohort

	1935-39		1940-44		1945-49		1950-54		Full Hispanic Sample	
	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE
Diabetes	18.9%		20.4%		25.5%		26.2%		23.6%	
EDUCATION										
Less than HS	49.9%		47.8%		43.8%		39.8%		44.3%	
HS Only	22.4%		23.0%		26.7%		22.3%		23.8%	
Some College	15.9%		17.8%		17.3%		23.1%		19.1%	
BA/BS+	11.9%		11.4%		12.2%		14.8%		12.9%	
% Any College	27.7%		29.3%		29.5%		37.9%		32.0%	
GENDER										
Male	50.4%		48.7%		47.4%		49.2%		48.7%	
Female	49.6%		51.3%		52.6%		50.8%		51.3%	
MARTIAL STATUS										
Married	65.1%		66.3%		62.6%		63.0%		63.9%	
Single/Never Married	5.8%		6.0%		6.6%		6.3%		6.3%	
Divorced	11.0%		12.2%		16.5%		14.3%		14.0%	
Separated	5.2%		3.9%		4.2%		5.2%		4.6%	
Widowed	9.3%		9.1%		6.7%		6.8%		7.7%	
Cohabiting	3.6%		2.5%		3.4%		4.3%		3.5%	
MECHANISMS										
Family Income (1000s) in 2016 \$	42.61	1.53	44.93	1.56	48.74	1.47	47.73	1.51	46.65	0.79
Has Health Insurance	74.8%		73.1%		75.8%		79.7%		76.4%	
BMI	27.94	0.18	28.56	0.22	28.73	0.20	28.91	0.19	28.63	0.10
REGION										
Northeast	15.5%		16.9%		15.0%		14.3%		15.3%	
Midwest	7.0%		6.4%		8.4%		8.5%		7.8%	
South	44.8%		38.3%		36.6%		38.0%		38.7%	
West	32.7%		38.4%		40.0%		39.1%		38.2%	
N	796		808		967		1,025		3,596	

Table 4-7 provides weighted population estimates for all Hispanics and by Hispanic cohort. The lowest diabetes rate is for the earliest-born cohort (1935-39) at 18.9 percent. The diabetes prevalence rate for the succeeding cohort (1940-44) increases by 1.5 percentage points, that is, a little more than one-twentieth. The 1945-49 cohort experiences a diabetes prevalence 5.1 percentage points higher at 25.5 percent, one-quarter higher than among the 1940-44 cohort. The diabetes prevalence rate for the 1950-54 cohort is 0.7 percentage points higher than the 1945-49 cohort. The diabetes prevalence of Hispanics is dramatically higher than the diabetes prevalence for the entire sample (as seen in Table 4-2).

Table 4-7 shows that for each successive cohort, Hispanics become more highly educated. Across cohorts, the percentage of Hispanics with less than a high school degree declines by nearly ten percentage points (one-quarter), from 49.9 percent for the 1935-39 cohort to 39.8 percent for the 1950-54 cohort. The percent of Hispanics with a high school degree is 22.4 percent for the earliest-born cohort, increases for the two middle cohorts (23.0 percent and 26.7 percent, respectively), and declines again for the final cohort (22.3 percent). Meanwhile, both categories of college experience increase. Hispanics with some college increases from 15.9 percent for the 1935-39 cohort to 23.1 percent for the 1950-54 cohort, that is, an increase of nearly half. Hispanics with at least a bachelor's degree increases by 3.9 percentage points, from 11.9 percent for the 1935-39 cohort to 14.8 percent for the 1950-54 cohort.

Examining gender among Hispanics, there are more males in the 1935-39 cohort (50.4 percent), and fewer males in the following three cohorts (48.7 percent, 47.4 percent, and 49.2 percent, respectively).

Table 4-7 shows that later-born cohorts have slightly lower percentages of Hispanics who are married or widowed, and slightly greater percentages who are single, divorced, and

cohabiting. Across cohorts, the percentage of Hispanics who are married at ages 60-64 declines by 2.1 percentage points between the 1935-39 cohort (65.1 percent) and the 1950-54 cohort (63.0 percent). The percentage of Hispanics single or never married increases by less than one percentage point from 5.8 percent for the 1935-39 cohort to 6.3 percent for the 1950-54 cohort. The percentage of Hispanics who are divorced increases from 11.0 percent (1935-39 cohort) to 14.3 percent (1950-54 cohort). The percentage of Hispanics separated decreases from 5.4 percent for the 1935-39 cohort to 3.9 percent for the 1940-44 cohort, increases to 4.2 percent for the 1945-49 cohort, and converges with the earliest-born cohort again by the 1950-54 cohort. The percentage who are widowed decreases from 9.3 percent for the 1935-39 cohort to 6.8 percent for the 1950-54 cohort. The percentage of Hispanics cohabiting increases from 3.6 percent (1935-39 cohort) to 4.3 percent (1950-54 cohort).

Examining intervening variables that education may work through, later-born Hispanics cohorts have higher family income, slightly higher rates of health insurance, and slightly BMI than earlier-born cohorts. The 1935-39 cohort experienced an average family income of \$42,610, similar to the 1940-44 cohort (\$44,930). Family income for Hispanics peaks for the 1945-49 cohort to \$48,740, with a slight decrease for the 1950-54 cohort to \$47,730. For the first cohort, 74.8 percent of Hispanics have health insurance. This slightly decreases to 73.1 percent for the 1940-44 cohort, and rises to 79.7 percent for the 1950-54 cohort. BMI also slightly increased for each cohort. The 1935-39 cohort experienced an average BMI of 27.94, which increased to 28.91 for the 1950-54 cohort.

Turning to regional geographic distribution, the greatest percentage of Hispanics for the full sample lived for the South (44.8 percent), followed by the West (32.7 percent), the Northeast

(15.5 percent), and the Midwest (7.0 percent). There are only small changes for the regional distribution across Hispanic cohorts.

5) Blacks

Table 4-7: Descriptive Statistics for Blacks Aged 60-64, by Cohort

	1935-39		1940-44		1945-49		1950-54		Full Black Sample	
	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE	Mean/%	SE
Diabetes	24.2%		23.9%		26.0%		24.9%		24.9%	
EDUCATION										
Less than HS	40.8%		30.4%		22.0%		17.7%		25.4%	
HS Only	31.2%		33.0%		30.4%		29.6%		30.8%	
Some College	17.3%		22.6%		30.6%		32.1%		27.2%	
BA/BS+	10.6%		14.0%		17.0%		20.7%		16.5%	
% Any College	27.9%		36.7%		47.6%		52.8%		43.7%	
GENDER										
Male	47.8%		45.8%		48.2%		46.1%		46.9%	
Female	52.2%		54.2%		51.8%		53.9%		53.1%	
MARTIAL STATUS										
Married	48.3%		47.6%		46.4%		42.6%		45.7%	
Single/Never Married	8.6%		8.2%		10.7%		15.2%		11.3%	
Divorced	16.6%		19.9%		22.6%		22.8%		21.1%	
Separated	6.5%		5.1%		7.1%		5.4%		6.0%	
Widowed	16.5%		16.2%		10.1%		10.1%		12.5%	
Cohabiting	3.4%		3.1%		3.1%		3.9%		3.4%	
MECHANISMS										
Family Income (1000s) in 2016 \$	40.14	1.38	41.42	1.33	47.03	1.31	45.83	1.29	44.31	0.68
Has Health Insurance	84.2%		84.6%		86.1%		87.9%		86.0%	
BMI	28.41	0.21	29.16	0.21	29.49	0.20	29.48	0.19	29.24	0.10
REGION										
Northeast	15.6%		16.3%		13.1%		13.6%		14.4%	
Midwest	20.5%		17.0%		16.6%		16.7%		17.3%	
South	57.8%		59.0%		63.2%		62.3%		61.1%	
West	6.1%		7.7%		7.1%		7.4%		7.2%	
N	850		992		1,284		1,425		4,551	

Table 4-6 provides weighted population estimates all blacks and by black cohort. The diabetes rate for blacks is fairly consistent across the four cohorts, with 24.2 percent for the earliest-born cohort. The diabetes prevalence rate for the succeeding cohort (1940-44) decreases by 0.3 percentage points, peaks at 26.0 percent for the 1945-49 cohort, and decreasing to 24.9 percent for the 1950-54 cohort. The diabetes prevalence of blacks is dramatically higher than the diabetes prevalence for the entire sample (as seen in Table 4-2).

Table 4-6 shows that for each successive cohort, blacks become more highly educated. Across cohorts, the percentage of blacks with less than a high school degree declines by over

one-half, from 40.8 percent for the 1935-39 cohort to 17.7 percent for the 1950-54 cohort. The percentage of blacks with a high school degree is 31.2 percent for the first cohort, increases to 30.4 percent for the second, declines to 30.4 percent for the third, and reaches 29.6 percent for the last cohort. Meanwhile, both categories of college experience increase. blacks with some college increases from 17.3 percent for the 1935-39 cohort to 32.1 percent for the 1950-54 cohort (a difference of 14.8 percentage points). blacks with at least a bachelor's degree doubles, from 10.6 percent for the 1935-39 cohort to 20.7 percent for the 1950-54 cohort.

Examining gender among blacks, there are fewer males in all four cohorts, ranging from 45.8 percent for the 1940-44 cohort to 48.2 percent for the 1945-49 cohort.

Table 4-6 shows that later-born cohorts have lower percentages of blacks who are married, separated, or widowed, and greater percentages that are single and divorced, with little change in cohabiting. Across cohorts, the percentage of blacks who are married at ages 60-64 declines by 1.7 percentage points between the 1935-39 cohort (48.3 percent) and the 1950-54 cohort (46.1 percent). The percentage of blacks single or never married increases by three-fourths, from 8.6 percent for the 1935-39 cohort to 15.2 percent for the 1950-54 cohort. The percentage of blacks who are divorced increases from 16.6 percent (1935-39 cohort) to 22.8 percent (1950-54 cohort). The percentage of blacks separated did not significantly across cohorts. The percentage who are widowed decreases from 16.5 percent for the 1935-39 cohort to 10.1 percent for the 1950-54 cohort. The percentage of blacks cohabiting does not significantly change.

Examining intervening variables that education may work through, later-born black cohorts have higher family income, similar rates of health insurance, and higher BMI than earlier-born cohorts. The 1935-39 cohort experienced an average family income of \$40,140,

similar to the 1940-44 cohort (\$41,420). Family income for blacks for the 1945-49 cohort increases to \$47,030, with a decrease for the 1950-54 cohort to \$45,830. Across the four cohorts, between 84.2 and 87.9 percent of blacks have health insurance. BMI slightly increased for the first three successive cohorts, and seems to stabilize for the 1950-54 cohort. The 1935-39 cohort experienced an average BMI of 28.4, highest for the 1945-49 and 1950-54 cohorts at 29.5.

Turning to regional geographic distribution, the greatest percentage of blacks in each cohort lived for the South (57.8 percent), followed by the Midwest (20.5 percent), the Northeast (15.6 percent), and the West (6.1 percent). There are only small changes for the regional distribution across black cohorts.

Diabetes Prevalence by Educational Attainment and Cohort

1) Full Sample

Table 4-8: Diabetes Prevalence for Persons Aged 60-64, By Education and Cohort

	1935-39	1940-44	1945-49	1950-54
Less than HS	19%	20%	25%	28%
HS Only	13%	14%	18%	19%
Some Col	11%	15%	17%	16%
BA/BS+	8%	9%	11%	11%
Educational Gap	11%	12%	15%	17%
Total	12.6%	13.9%	16.4%	16.2%

Table 4-8 presents weighted diabetes prevalence rates by education and cohort. From the earliest-born to latest cohorts, the diabetes prevalence rate for persons with less than high school degree increases by nearly one-half, that is, by 9 percentage points, from 19 percent for the 1935-1939 cohort to 20 and 25 percent, respectively, for the 1940-44 and 1945-49 cohorts, to 28 percent for the most recent, 1950-54 cohort. Diabetes prevalence for persons with only a high school degree also increase by one-half (6 percentage points), from 13 percent for the earliest-

born cohort to 14 and 18 percent, respectively, for the next two cohorts, to 19 percent for the most recent cohort.

Across these cohorts the diabetes prevalence rate for persons who attended college increase by 5 percentage points, from 11 percent for the earliest-born cohort to 15, 17, and 16 percent, respectively, for the birth cohorts of 1940-44, 1945-49, and 1950-54. Diabetes prevalence for persons with a bachelor's degree increases the least, by 3 percentage points from only 8 percent for the earliest-born cohort to 9 percent for the 1940-44 cohort and 11 percent for the next two cohorts.

Overall, diabetes rates increased for all groups, but the lower education groups experienced the largest increases, with 9, 6, 5, and 3 percentage point increases, respectively, for persons with less than high school, high school only, some college, and a bachelor's degree or more. Thus, the education gap in diabetes prevalence between the least and most educated groups expands by about one-half, increasing from 11 percentage points for the 1935-39 cohort to 12 percent and 15 percentage points, respectively, for the next two cohorts, and reaching a 17-percentage point difference for the 1950-54 cohort.

At the same time, as shown in Table 4-2 and discussed above, the completing less than high school or completing high school only declined across cohorts, while the proportions completing some college or a bachelor's degree increased. These two sets of changes, the education expansion and changes in education-specific diabetes prevalence rates both contributed to overall change in diabetes prevalence.

2) *Male vs. Female Cohorts*

Table 4-9: Total Diabetes Prevalence for Males and Females Aged 60-64, by Cohort

	1935-39	1940-44	1945-49	1950-54
Males	14%	15%	17%	17%
Females	11%	12%	15%	15%

Table 4-9 presents weighted total diabetes prevalence by gender and cohort. Across all cohorts, diabetes increases for both genders, and males are consistently more likely than females to have diabetes. For the earliest-born cohort, 14 percent of males and 11 percent of females have diabetes. For the second cohort, diabetes increases for males and females to 15 percent and 12 percent, respectively, and prevalence increases further for the third cohort to 17 percent and 15 percent, respectively. Diabetes prevalence was unchanged for the 1950-54 cohort for both genders, compared to the 1945-49 cohort.

Table 4-10: Diabetes Prevalence for Males and Females Aged 60-64, by Education and Cohort

		1935-39	1940-44	1945-49	1950-54
Less than HS	Male	19%	20%	26%	29%
	Female	18%	20%	25%	27%
HS Only	Male	14%	15%	19%	20%
	Female	12%	13%	17%	18%
Some College	Male	13%	19%	18%	17%
	Female	10%	12%	16%	15%
BA/BS+	Male	10%	10%	13%	12%
	Female	4%	6%	8%	9%

Table 4-10 presents the weighted diabetes prevalence by education and cohort for males and for females. For the earliest-born cohort (1935-39), males and females who did not graduate high school are nearly equally likely to have diabetes (19 percent and 18 percent, respectively). In every successively higher educational category for the 1935-39 cohort, males are more likely to have diabetes than females, and the gender diabetes differences increase for every successive educational category. The diabetes prevalence for males who finished high school is 14 percent,

while their female counterparts experience a diabetes rate of 12 percent. For males with some college experience, the diabetes rate is 13 percent, and only 10 percent for females with some college. The gender difference is largest for those with a bachelor's degree. Males with a bachelor's degree have a diabetes rate (10 percent) more than double the rate for their female counterparts.

Across these cohorts, both males and females experience diabetes increases in all education categories. Diabetes prevalence for males and females with less than a high school education increases from 19 percent and 18 percent, respectively, for the earliest-born cohort to 29 percent and 27 percent for the 1950-54 cohort. Across all except the 1940-44 cohort, female high school dropouts have slightly lower diabetes rates than their male counterparts. The gender difference in diabetes rates for high school graduates resembles a similar trend of increasing rates across cohorts, with females consistently two percentage points lower than males. The diabetes prevalence for males and females with a high school degree increases by 6 percentage points between the 1935-39 cohort (14 percent and 12 percent, respectively) and the 1950-54 cohort (20 and 18 percent).

For the earliest-born cohort, diabetes among males with some college is three percentage points higher (at 13 percent) than their female counterparts (10 percent). Both genders in this educational category experience higher diabetes rates in later-born cohorts, but among males with some college diabetes rates peaks for the 1940-44 cohort at 19 percent, while female college attendee diabetes rises to 12 percent for the same cohort. The greatest gender-diabetes difference is found for the 1940-44 cohort, with convergence for the last two cohorts. For the 1945-49 cohort, diabetes among males in this education group slightly declines to 18 percent, while diabetes among females increases to 16 percent. For the latest-born cohort, male and female

diabetes rates among males and females with some college are 17 percent and 15 percent, respectively.

Male and female college graduates for the earliest-born cohort experience diabetes prevalence of 10 percent and four percent, respectively. Ten percent of college educated males have diabetes for the first two cohorts. Meanwhile, diabetes prevalence rises for college females from 4 percent for the earliest-born cohort to six percent for the second cohort. For the 1945-49 cohort, diabetes among college males increases to 13 percent, while diabetes among college females increases to 8 percent. For the final cohort, college-educated male diabetes slightly decreases to 12 percent, while 9 percent of their female counterparts have diabetes.

Males are more likely to have diabetes than females at every education level in all cohorts. Across cohorts, diabetes prevalence increases for both genders, with similar trends for most education groups. For the earliest-born cohort, all except the most educated experience relatively similar diabetes differences between the genders. The largest gender diabetes gap by education is among college-educated males and females. For the latest-born cohort, the gender diabetes gap among the most educated shrinks, becoming nearly the same as the gender diabetes gap in other education categories, 3 vs. 2 percent

While the gender diabetes difference within education groups remained relatively stable, the education diabetes difference within gender increases. For the earliest-born cohort, the education diabetes gap for males (i.e., difference in diabetes prevalence between the least and most highly educated) is 9 percent for males and for 14 percent for females. These differences increase across cohorts, reaching a 17-percentage point gap for males and 18-percentage point gap for females for the latest-born cohort. Observed in tandem with the results in Table 4-3 and 4-5, as the education gap in diabetes grows across cohorts for males and females, a greater

proportion of males and females are more highly educated. In addition, the males and females become more similarly educated, although females are slightly less likely to complete college.

3) *White vs. Hispanic vs. Black Cohorts*

Table 4-11: Total Diabetes Prevalence for Whites, Blacks, and Hispanics Aged 60-64, by Cohort

	1935-39	1940-44	1945-49	1950-54
Whites	11%	12%	14%	14%
Hispanics	19%	20%	26%	26%
Blacks	24%	24%	26%	25%

Table 4-11 presents weighted total diabetes prevalence for three race-ethnic groups by cohort. Across all cohorts, diabetes increases for each race-ethnic group, and both blacks and Hispanics are consistently more likely than whites to have diabetes. For the 1935-39 cohort, the diabetes prevalence rate ranged from a low of 11 percent for whites to 19 and 24 percent, respectively, for Hispanics and blacks. Across the four cohorts, Hispanics experienced the largest increase, 7 percentage points, while the increase was about half as large for whites at 3 percentage points, and only 1 percentage point for blacks. For whites and Hispanics most of the increases occurred between the 1940-44 and 1945-49 cohorts. For the most recent cohort of 1950-54, the diabetes prevalence rate for whites was 14 percent, compared to 25-26 percent for Hispanics and blacks. Thus, in the 1950-54 cohort, the diabetes prevalence rates for Hispanics and blacks were 11-12 percentage points greater than for whites.

Table 4-12: Diabetes Prevalence for Non-Hispanic Whites, Blacks, and Hispanics Aged 60-64, by Cohort

		1935-39	1940-44	1945-49	1950-54
Less than HS	Whites	18%	16%	24%	23%
	Hispanics	19%	22%	27%	36%
	Blacks	22%	30%	30%	25%
HS Only	Whites	11%	13%	15%	17%
	Hispanics	20%	18%	30%	25%
	Blacks	25%	19%	28%	26%
Some College	Whites	10%	13%	16%	14%
	Hispanics	20%	27%	25%	15%
	Blacks	25%	26%	24%	27%
BA/BS+	Whites	6%	7%	10%	9%
	Hispanics	14%	8%	11%	20%
	Blacks	26%	19%	21%	19%

Table 4-12 presents the weighted diabetes prevalence rates for whites, Hispanics, and blacks by education for four successive cohorts. For the earliest-born cohort (1935-39), among those completing less than high school, the diabetes prevalence rates were nearly the same for whites and Hispanics, at 18-19 percent, and slightly higher for blacks at 22 percent. The patterns and magnitude of cohort-to-cohort change differed substantially by race. The largest increase between the earliest- and latest-born cohorts in diabetes prevalence for persons completing less than high school was for Hispanics, at 17 percentage points, with much smaller increases for whites and blacks at 5 and 3 percentage points, respectively. Thus, in the 1950-54 cohort, diabetes prevalence rates for whites and blacks were similar, at 23 and 25 percent, respectively, but much higher for Hispanics at 36 percent.

Unlike the less than high school group, among persons completing high school only, the diabetes prevalence rate was much lower for whites, at 11 percent, compared to Hispanics and blacks, at 20 and 25 percent respectively. Again, the patterns and magnitudes of cohort-to-cohort change differed substantially by race. For this education group, overall increases in diabetes prevalence between the earliest-born and the latest cohorts were similar for whites and

Hispanics, at 5-6 percentage points, with a very small increase of 1 percentage point for blacks. As of the 1950-54 cohort, whites completing high school only continued to have the lowest diabetes prevalence rate at 17 percent, with much higher and nearly equal rates of 25-26 percent for Hispanics and blacks.

Rates of diabetes prevalence by race among the 1935-39 cohorts completing some college were identical or nearly the same as the rates for persons completing high school only, at 10 versus 11 percent for whites, 20 percent for Hispanics at both education levels, and 25 percent for blacks at both education levels. Once again, the patterns and magnitudes of cohort-to-cohort change differed substantially by race. Comparing the 1935-39 and 1950-54 cohorts, the increase in diabetes prevalence for whites with some college was similar, but slightly smaller, than the increases for lower education groups, at 4 vs. 5-6 percentage points, respectively, and the increases for blacks were also quite similar at 2 vs. 1-3 percentage points, respectively. But Hispanics with some college experienced a decline of 5 percentage points. For the 1950-54 cohort, diabetes prevalence rates for whites and Hispanics with some college were nearly equal, at 14-15 percent, compared to the much higher rate of 27 percent for blacks.

Finally, as was the case for the three lower education groups in the 1935-39 cohorts, among persons in this cohort completing a bachelor's degree, the diabetes prevalence rate was lowest for whites, followed by Hispanics, and then blacks, at 6, 14, and 26 percent, respectively. Once again, as was the case for the lower education groups, the patterns and magnitudes of cohort-to-cohort change for the bachelor's degree group differed substantially by race. Comparing the 1935-39 and 1950-54 cohorts, the overall increase for whites completing a bachelor's degree was similar, but slightly smaller, than the increases for lower education groups, at 3 vs. 4-6 percentage points, respectively. Blacks completing a bachelor's degree, in

sharp contrast, experienced a substantial decline of 5 percentage points in diabetes prevalence, compared to the small increases among blacks in lower education groups of 1-3 percentage points. Among Hispanics completing a bachelor's degree, the increase in the diabetes prevalence rate was 6 percentage points, compared to a decline of 5 percentage points for those with some college, an increase of 5 percentage points for those completing high school only, and an increase of 17 percentage points for those not completing high school.

Across education groups, only whites experienced a clear inverse relationship between education and diabetes prevalence in both the earliest-born and latest cohorts, with rates lower for each successively higher education group in the 1935-39 cohort of 18, 11, 10, and 6 percent respectively, and for the 1950-54 cohort of 22, 17, 14, and 9 percent, respectively. For Hispanics in the 1935-39 cohort, the diabetes prevalence rates were about the same at 19-20 percent for the three lowest-education groups, and 14 percent for Hispanics with a bachelor's degree. But for the 1950-54 cohort, the rates were 36 percent for the lowest education group, 25 percent for the high school only group, 15 percent for the some college group, and 20 percent for the bachelor's degree group. Finally, for blacks in the 1935-39 cohort the diabetes prevalence rates were fairly similar across education groups, at 22 percent for the lowest education group, and 25-26 percent for the high school only, some college, and bachelor's degree groups. For the 1950-54 cohort of blacks, the diabetes prevalence rates were 25-27 percent for the less than high school, high school only, and some college group, compared to 19 percent for the bachelor's degree group.

These race group differences in patterns of diabetes prevalence across education groups, and in the magnitude and direction of change across cohorts, is quite complicated. Easy generalization regarding the impact of these changes on the overall diabetes prevalence rate is not possible. It is possible, however, to systematically analyze the extent to which change in the

overall diabetes prevalence rate can be accounted for by changes in the education composition of the population and by changes in education-specific diabetes rates for various race groups. This analysis is a primary goal of this dissertation.

Chapter 5

Educational Gradients and Educational Composition Change in Diabetes Prevalence across Four Cohorts

Introduction

As discussed in Chapter 2, historic increases in education could lead to changes in diabetes prevalence across cohorts either through changes in the educational gradient for diabetes or through changes in the composition of the population with regard to educational attainments. This chapter begins by presenting results from logistic regression models that estimate educational gradients across four cohorts. The chapter then presents results from Fairlie Decomposition models that estimate the extent to which changes in educational composition can account for change in diabetes prevalence across cohorts.

Logistic Regression Results

Table 5-1 presents weighted logit regression estimates for the effects of educational attainment, population composition (race-ethnicity, marital status, region), and intervening variables that education may act through (family income, BMI, health insurance status) on diabetic prevalence in each cohort (1935-39, 1940-44, 1945-49, and 1950-54). For each cohort, Model 1 (M1) presents odds ratios excluding the intervening variables, while Model 2 (M2) includes the intervening variables.

Table 5-1: Weighted Logit Regression for Diabetes among Persons Aged 60-64, by Cohort

	1935-39		1940-44		1945-49		1950-54	
	M1	M2	M1	M2	M1	M2	M1	M2
	O.R.	O.R.	O.R.	O.R.	O.R.	O.R.	O.R.	O.R.
Less Than High School	1.35 **	1.23	1.30 *	1.21	1.32 **	1.21	1.39 **	1.28 *
High School Only (omitted)								
Some College	0.88	0.88	1.08	1.09	0.94	0.98	0.87	0.91
Bachelor's Degree or Higher	0.57 ***	0.68 **	0.58 ***	0.72 *	0.57 ***	0.74 **	0.56 ***	0.72 **
Female	0.75 ***	0.75 ***	0.70 ***	0.65 ***	0.80 ***	0.72 ***	0.83 **	0.80 **
Non-Hispanic White (omitted)								
Non-Hispanic Black	2.37 ***	2.21 ***	1.97 ***	1.75 ***	1.70 ***	1.54 ***	1.69 ***	1.55 ***
Hispanic	1.65 ***	1.66 ***	1.62 ***	1.58 ***	1.64 ***	1.72 ***	1.87 ***	1.89 ***
Asian/Pacific Islander	1.06	1.68	1.83 *	2.94 ***	1.19	1.96 **	1.46	2.19 ***
Native American	1.63	1.87	1.48	1.48	1.47	1.21	1.84	1.60
Other	0.81	0.64	1.61	1.69	1.26	1.00	2.14	1.83
Married (omitted)								
Single/Never Married	0.65 *	0.59 **	1.11	0.91	1.07	0.85	1.41 **	1.15
Cohabiting	0.90	0.94	0.96	0.93	0.66	0.72	1.18	1.22
Separated	0.49 **	0.47 **	1.80 *	1.56	1.89 ***	1.64 **	1.36	1.13
Divorced	1.01	0.94	1.14	1.02	1.08	0.93	1.06	0.92
Widowed	0.98	0.83	1.34 *	1.18	1.07	0.88	0.96	0.81
Family Incomes (\$1000s) in 2016 dollars		0.996 **		0.993 ***		0.994 ***		0.994 ***
Has Health Insurance		1.43 *		1.34 *		1.53 ***		1.40 **
BMI		1.13 ***		1.13 ***		1.15 ***		1.12 ***
South (omitted)								
Northeast	0.91	0.92	0.77 *	0.74 *	0.75 **	0.80 *	0.84	0.87
Midwest	0.85	0.80 *	1.08	1.01	1.01	0.96	0.97	0.93
West	0.89	0.90	0.93	0.95	0.87	0.96	0.67 ***	0.69 ***
Constant	0.17 ***	0.01 ***	0.17 ***	0.01 ***	0.23 ***	0.00 ***	0.23 ***	0.01 ***
N	7,019		7,540		9,311		10,231	
Pseudo R-squared	0.0324	0.0816	0.0328	0.0947	0.0305	0.1135	0.0373	0.0990

***p<.001 **p<.01 *p<.05

In M1 for the earliest-born cohort (1935-39), the odds of being diagnosed with diabetes for persons who did not complete high school are 35 percent greater than persons who completes high school, controlling for population composition. The relationship is statistically significant (p<.01). Including intervening variables, the odds of being diagnosed for persons who did not complete high school are 23 percent greater than for persons who completed high school, reducing the effect size by nearly one-third. This relationship also is not statistically significant (p<.05). The odds of being diagnosed with diabetes for persons who went to college but did not graduate are 12 percent lower than for high school graduates, controlling only for population composition (M1), and the relationship is not statistically significant (p<.05). Including intervening variables (M2) does not change the estimate for persons with some college compared

to those who graduated high school, and is not statistically significant ($p < .05$). Finally, controlling for population composition, the odds of being diagnosed with diabetes for persons who graduate college are 43 percent lower than for persons with a high school education, and the relationship is highly statistically significant ($p < .001$). Introducing intervening variables, the odds for persons who graduate from college compared to persons with a high school education decreases to 32 percent (reduced by one-quarter), with a lower level of statistical significance ($p < .01$). Postestimation tests of the differences in odds ratios between M1 and M2 for the 1935-39 cohort are statistically significant for the persons with a bachelor's degree ($p < .01$).

Thus, the odds for diabetes prevalence for the 1935-1939 cohort, compared to persons with a high school education, becomes greater for the lowest educational level (23 percent greater), and becomes lower as educational levels rise, from 12, to 43 percent lower, respectively, for persons completing some college and graduating from college. In addition, while intervening variables account for between about one-fourth and one-third of the effect of education on diabetes prevalence (although there is no change for persons with some college experience), two-thirds to three-fourths of the effect of education is a direct effect not acting through the three intervening variables of family income, having health insurance, or BMI.

For the 1940-44 cohort, the odds of being diagnosed with diabetes for persons without a high school degree compared to high school graduates are 30 percent greater, controlling for population composition. The relationship is slightly statistically significant ($p < .05$). Including intervening variables, the odds decrease to 21 percent and are no longer significant ($p < .05$). The odds ratio for persons with some college experience is not statistically significant ($p < .05$) in either model for the 1940-44 cohort. Lastly, the odds of being diagnosed with diabetes for college graduates are 42 percent lower than for high school graduates, controlling for population

composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds decrease to 28 percent (reduced by one-third), with a lower level of statistical significance ($p < .05$). Postestimation tests of the differences in odds ratios between M1 and M2 for the 1940-44 cohort are statistically significant for the persons with at least a bachelor's degree ($p < .01$).

Thus, controlling for population composition, the odds of diabetes for the 1940-44 cohort for college graduates are 42 percent lower than for persons who complete high school, a value nearly identical to the 43 percent estimates for the 1935-39 cohort.

For the 1945-49 cohort, the odds of being diagnosed with diabetes for high school dropouts are 32 percent greater than high school graduates, controlling for population composition. The relationship is statistically significant ($p < .01$). With intervening variables in M2, the odds decrease to 21 percent, and are no longer statistically significant ($p < .05$). The odds ratios for persons with some college experience is not statistically significant ($p < .05$) in either model for the 1945-49 cohort. Meanwhile, the odds of being diagnosed with diabetes for college graduates are 43 percent lower than for high school graduates, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds of college graduates compared to high school graduates decrease to 26 percent, and some significance is lost ($p < .01$). Postestimation tests of high school dropouts' and college graduates' odds ratios between the two models is statistically significant ($p < .05$ and $p < .001$, respectively). Postestimation tests of the differences in odds ratios between M1 and M2 for the 1945-49 cohort are statistically significant for the least and most educated groups ($p < .05$ for less than high school and $p < .001$ for bachelor's degree).

Thus, controlling for population composition, the odds of diabetes for the 1945-49 for college graduates are 43 percent lower than for persons without a high school, a nearly identical value to the estimates of 43 and 42 percent for the two earlier-born cohorts. About one-third of these effect is accounted for the intervening variables introduced in M2.

For the fourth and final cohort (1950-54), the odds of being diagnosed with diabetes for high school dropouts are 39 percent greater than high school graduates, controlling for population composition. The relationship is statistically significant ($p < .01$). With intervening variables in M2, the odds decrease to 28 percent, but remains statistically significant ($p < .05$). The odds ratio for persons with some college experience is not statistically significant ($p < .05$) in either model for the 1950-54 cohort. Meanwhile, the odds of being diagnosed with diabetes for college graduates are 44 percent lower than for high school graduates, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds decrease to 28 percent. Postestimation tests of less than high school and bachelor's odds ratios between the two models is statistically significant ($p < .05$ and $p < .001$, respectively).

Thus, the odds of being diagnosed with diabetes for the 1950-54 cohorts for college graduates are 44 percent lower than for high school graduates, controlling for population composition, and about one-third of this effect is accounted for by the intervening variable. Thus, controlling for population composition, the odds of diabetes for various cohorts of college graduates are about half as large (42 to 44 percent lower) compared to persons with a high school, and about one-third of these effects are accounted for the intervening variables introduced in M2.

There is no statistical change in the effect of education on diabetes across cohorts (in either model). Postestimation tests find no statistical difference between odds ratios across

cohorts. It should be noted, however, that the odds ratio for persons with less than high school in the latest-born cohort (1950-54) is slightly significant ($p < .05$) when including intervening variables. This difference is not seen in the previous three cohorts in M2.

Turner to gender, there is little or no change in the effect size and statistical significance, either with and without intervening variables for each cohort. Similarly, there is little or no change in the effect size of gender of diabetes across cohorts. Controlling for population composition, the odds of being diagnosed with diabetes for females are between 17 and 25 percent lower than for males across cohorts. With intervening variables, the odds range from 20 to 35 percent across cohorts.

Turning to results for race-ethnic groups, without the intervening variables (M1), the odds ratios for the 1935-39 cohort are 137 percent greater for blacks than for white but this difference declines to 97 percent, 70 percent, and 69 percent, respectively, across the three later-born cohorts. Thus, the difference between blacks and whites is one-half as large for the latest-born cohort compared to the earliest-born cohort. Estimates for all four cohorts are highly statistically significant ($p < .001$). The odds ratios in the model that includes the intervening variables (M2) are slightly smaller for each cohort, and the pattern of change across cohorts for M2 models is similar to the M1 models. For Hispanics, without the intervening variables, the odds ratios are greater than for whites by 65 percent for the 1935-39 cohort, 62 percent for the 1945-49, and 64 percent for the 1945-49 cohort. All three estimates are highly statistically significant ($p < .001$). The difference is larger for the 1950-54 cohort at 87 percent and highly significant ($p < .001$). The introduction of the intervening variables (M2) has only a small effect on these coefficients. Nearly all estimates (20 out of 24) are not statistically significant for the Asian/Pacific Island, Native American, and Other groups. Chapter 7 presents a detailed analysis

of both the education gradient and education composition effects on diabetes separately for whites, blacks, and Hispanics.

Thus, the black disadvantage in odds compared to whites declined by about one-half across the cohorts from 137 percent and 121 percent higher (M1 and M2) for the earliest-born cohort to 69 percent and 55 percent higher (M1 and M2) for the latest-born cohort, while the Hispanic disadvantage increased by two-fifths from 65 percent and 66 percent higher (M1 and M2) for the earliest-born cohort to 87 percent and 89 percent higher (M1 and M2) for the latest-born cohort. Meanwhile, the odds for Asians/Pacific Islanders is significant in the 1940-44 cohort models, and M2 in the 1945-49 cohort and 1950-54 cohort.

There is no clear pattern linking marital status and diabetes across cohorts in either M1 or M2. Most odds ratios (32 out of 40) are not statistically significant ($p < .05$), and the direction of the effect is erratic across cohorts for most marital statuses. For example, in both models for the 1935-39 cohort, the odds of being diagnosed for persons who are separated are between 51-53 percent lower than for married persons. But the effect of being diagnosed with diabetes for the married person group comparison flips to higher odds, then again to lower odds across the three later-born cohorts.

Persons with high family income are consistently less likely than persons with lower family incomes to be diagnosed with diabetes for every cohort, and the odds ratios are highly statistically significant for three of the four cohorts ($p < .001$). In the 1935-39 cohort, for every \$1,000 increase in family income, the odds of being diagnosed with diabetes decrease by 0.4 percent. Meaning, a difference of \$25,000 would mean a 10 percent difference in the odds of being diagnosed with diabetes. The odds ratios for family income remain stable across cohorts, with the two latest-born cohorts (1945-49 and 1950-54) experiencing a 0.6 percent decrease in

the odds for everything \$1,000 increase in family income. Namely, a difference of \$25,000 would result in a 15 percent difference in the odds of diabetes.

Having health insurance is positively associated with diabetes across cohorts. In the 1935-39 cohort, the odds of being diagnosed with diabetes for persons with health insurance are 43 percent greater than for those without health insurance, controlling for other covariates. This relationship is slightly statistically significant for the 1935-39 and 1940-44 cohorts ($p < .05$), and more highly statistically significant for the 1945-49 and 1950-54 cohorts ($p < .001$ and $p < .01$, respectively), and the odds range between 34-53 percent greater for persons with health insurance compared to persons without health insurance. It may be the case that these coefficients overestimate the true differences in prevalence for these two groups, however, because persons with health insurance may be more likely to see a physician without health insurance, and as a result more likely to be diagnosed with diabetes.

Persons with a higher BMI also have greater odds of being diagnosed with diabetes, which is a consistent relationship across cohorts. Across cohorts, a one unit increase in BMI results in a 12-15 percent increase in the odds of being diagnosed with diabetes, controlling for other covariates. All four odds ratios are highly statistically significant ($p < .001$).

Most odds ratios (17 out of 24) are not significant ($p < .05$). Compared to the South, only one of eight odds ratios is significant for the Midwest, and only two of eight are significant for the West. Four odds ratios for the two middle cohorts are significant ($p < .05$ to $p < .01$), for the Northeast and are in the narrow range of 0.74 to 0.80 with erratically negative or positive odds ratios for persons not in the South across the four cohorts.

Decomposition Results

Table 5-2: Decomposition of Cohort Disparities in Diabetes Prevalence

	1935-39 to 1940-44		1940-44 to 1945-49		1945-49 to 1950-54		1935-39 to 1950-54	
Rates	12.64 to 13.86		13.86 to 16.43		16.43 to 16.16		12.64 to 16.16	
Percentage Point Change	1.22		2.57		-0.27		3.52	
Variables	Effect	S.E.	Effect	S.E.	Effect	S.E.	Effect	S.E.
Education Level	-0.59 ***	0.07	-0.72 ***	0.09	-0.47 ***	0.05	-1.46 ***	0.17
Gender	-0.05 *	0.02	0.05 ***	0.02	-0.07 ***	0.02	-0.03 *	0.02
Race-Ethnicity	0.26 ***	0.04	0.14 ***	0.03	0.40 ***	0.05	0.69 ***	0.08
Marital Status	0.04	0.04	0.07 *	0.03	0.12 **	0.04	0.25 *	0.10
Region	0.04	0.03	0.08 *	0.04	0.10 ***	0.03	0.02	0.03
N	14,559		16,851		19,542		17,250	

***p<.001 **p<.01 *p<.05

Table 5-2 presents results using the Fairlie Decomposition method to estimate the extent to which change across cohorts for the overall rate of diabetes prevalence can be accounted for by change in the education composition, controlling for changes in population compositions (gender, race-ethnicity, marital status, region). In order to measure the full effect of educational composition, the intervening variables are not included in the analysis discussed here. In addition, additional analysis that included intervening variables of family income, health insurance, and BMI are not report here, because they produce marginal changes in estimates of the education composition effect.

Table 5-2 shows the effect on diabetes prevalence of changes for the education composition and of changes for the composition of cohorts with regard to the control variables. The first decomposition model in Table 5-2 analyzes the contributions of education composition change and change in population composition (i.e., the control variables to the 1.22 percentage point increase for the diabetes prevalence that occurred across the 1935-39 and 1940-44 cohorts. The effects are highly statistically significant (p<.001) for changes in the education and race-ethnicity compositions, slightly statistically significant (p<.05) for changes in the gender composition, and not significant (p<.05) for changes in the marital status and region

compositions. Among these variables, education composition has the largest effect and it is negative. The results indicate that, controlling for population composition change (gender race-ethnicity, marital status, and region), if the educational composition had not changed across these two cohorts, then the overall diabetes rate would have increased by an additional 0.59 percentage points (from 1.22 percent to 1.81 percent). Thus, if educational attainment had not increased across these two cohorts, the diabetes rate would have been one-half greater than the actual increase of 1.22 percent.

The second model in Table 5-2 analyzes the contribution of education composition change, controlling for the composition change in the control variables, to the 2.57 percentage point increase for diabetes prevalence between the 1940-44 and 1945-49 cohorts. The effect of increasing education attainments and changes in gender and race-ethnicity are highly statistically significant ($p < .001$), slightly statistically significant ($p < .05$) for changes in marital status and region compositions. Again, education is the largest coefficient and has a negative effect on the change in diabetes. The results indicate that, controlling for population composition change, if the educational composition had not changed across the two cohorts, then the overall diabetes rate would have increased by an additional 0.72 percentage points (from 2.52 percent to 3.29 percent). In other words, the increase would have been nearly one-third greater than the actual increase.

Across the two most recent cohorts (1945-49 and 1950-54), the diabetes rate declined by a small 0.27 percentage points. The results in Table 5-2 indicate that if educational attainments had not increased, controlling for changes in population composition, the diabetes rate tended to increase by 0.47 percentage points, that is, instead of the actual decline of 0.27 percentage points, the diabetes rate for persons would have increased by 0.20 percentage points. Thus, instead of

declining slightly, the diabetes rate would have continued to increase across these two cohorts, as it had across the earlier-born cohorts.

The final model in Table 5-2 presents variable contributions to diabetes change across all four cohorts, that is, comparing the rate for 1950-54 cohort to the rate for 1935-39 cohort. Overall, the diabetes rate increased by 3.52 percentage points from 12.64 percent for the earliest-born cohort to 16.16 percent for the most recent cohort. If the education composition of the latest-born cohort had remained unchanged compared to the earliest-born cohort, diabetes prevalence would have been 1.46 percentage points higher, controlling for population composition. In other words, the change in diabetes prevalence would have been two-fifths greater (41.5 percent greater) in the 1950-54 cohort, increasing by 4.98 percentage points instead of 3.52 percentage points. That is, an additional 988,943 persons ages 60-64 in the 1950-54 cohort would have diabetes.

The changing gender composition has a positive effect in the second model, and a negative effect in the other three models, and is statistically significant in all four models. The gender composition explains little of the changes in diabetes prevalence across the models. Chapter 4 finds very little change in the gender composition, ranging from 49.7 percent male and 50.3 percent female for the 1935-39 cohort to 51.6 and 49.4 percent, respectively, for the 1950-54 cohort.

The changing racial-ethnic composition has a consistently positive and statistically significant effect in all four models. The positive direction of the effects of racial-ethnic composition changes indicate that changes in racial-ethnic composition contributed to the increases that occurred in the diabetes across cohorts. For example, of the 3.52 percentage point increase that occurred between the earliest and most recent cohorts, about 20 percent can be

accounted for the changes in the racial-ethnic composition, mainly because blacks and Hispanics experience higher diabetes rates than white, and blacks. Additionally, blacks and Hispanics increased as a percentage of the population from 15.1 percent in the earliest-born cohort to 20.3 percent in the most recent cohort (as seen in Chapter 4).

The change in diabetes prevalence explained by change in the marital composition is positive and slightly or slightly significant in the three later-born cohorts. The positive direction of effects of marital status changes indicate that changes in marital status contributed to the increases in diabetes for later-born cohorts. For example, of the 3.52 percentage point increase that occurred between the earliest and most recent cohorts, less than 10 percent can be accounted for the changes in marital status composition. Finally, regional composition explains little to none of the changes in diabetes prevalence across the models. Of the population composition variables, region is not significant in three of the four models, and the coefficients are null.

Summary of Results

The first important finding in this chapter is that higher educational attainment is strongly and negatively associated with diabetes prevalence in all four cohorts (Table 5-1). For example, in various model controlling for population composition, persons with a bachelor's degree have odds of being diagnosed with diabetes that are 42-44 percent lower than the odds for high school graduates. This relationship is significant for all cohorts, whether or not mediating variables (i.e., family income, health insurance, and BMI) are included. Although the introduction of mediating variables reduces the size of these estimates, even after controlling for mediating variables, persons with a bachelor's degree have much lower odds of being diagnosed with diabetes than persons who complete high school, 26-32 percent lower. Thus, the educational gradient is quite large, and while some of this effect acts through the mediating variables included in the models,

most of the educational gradient effect is a direct effect that does not act through the intervening variables. These findings support the previous literature of education's effect on diabetes. Additionally, the magnitude of the relationship between higher educational attainment and diabetes does not change across cohorts. Interaction models (see Appendix) and postestimation Wald tests find no statistical difference in odds ratios across cohorts. It should be noted, however, that the odds ratio for persons with less than high school in the latest-born cohort is slightly significant ($p < .05$) when including intervening variables. This difference is not seen in the previous three cohorts. It is currently unclear whether this difference in significance is statistical noise or a change within society. It may be that the effects of education on diabetes are beginning to stratify in the latest born cohort.

The second important finding is that the changing educational composition, that is, the educational expansion, has a negative effect on the growth in diabetes prevalence for later-born cohorts. If educational expansions had not occurred, that is, if education levels had remained constant at the levels for the 1935-39 cohort, the increase in the diabetes prevalence rate would have been two-fifths greater in the 1950-54 cohort (4.98 instead of 3.52 percentage points). That is, nearly an additional million persons ages 60-64 in the 1950-54 cohort would have diabetes.

Thus, educational expansion acted to limit diabetes growth. The magnitude of the educational gradient did not change, but the compositional effect on diabetes change of the educational expansion was substantially and statistically significant from one cohort to the next across all cohorts. These new findings move beyond the extant literature by estimating the magnitude and assessing the extent to which changes have occurred in the educational gradient for diabetes across four successive birth-cohorts. And by estimating the extent to which the educational expansion across these cohorts acted to limit increases in diabetes prevalence.

Additional Analysis (See Appendix)

In preparation for the next several chapters, additional models using gender and race-ethnicity interacting with were conducted. For the three earliest-born cohorts, gender and education interact, with females with higher education experiencing lower odds of being diagnosed with diabetes than their male counterparts. For the latest-born cohort, there is no statistical difference in the magnitude of gender-education interaction ($p < .05$). This may indicate a converging effect of education for gender across the four cohorts. Separate analyses of each gender's trajectory are valuable in further understanding this relationship.

For race-ethnicity, interactions with education were found for black and Hispanics for the earliest-born cohort model. For the 1935-39 cohort, blacks at high school or above have greater odds of being diagnosed with diabetes than their white counterparts. For the two middle cohorts, there are no clear patterns between race-ethnicity and education. For the latest-born cohorts, more highly educated blacks have higher odds of diabetes than highly educated whites. These findings suggest varying relationships between education and diabetes across cohorts by race-ethnicity.

Chapter 6

Gender Differences in Educational Gradients and Educational Composition Change in Diabetes Prevalence across Four Cohorts

Introduction

As discussed in Chapter 2, historic increases in education could lead to changes in diabetes prevalence across cohorts either through changes in the educational gradient for diabetes or through changes in the composition of the population with regard to educational attainments. This chapter begins by presenting results from logistic regression models that estimate educational gradients for males and females across four cohorts. The chapter then presents results from Fairlie Decomposition models that estimate the extent to which changes in educational composition for males and females can account for change in diabetes prevalence across cohorts.

Logistic Regression Results

1) Males Only

Table 6-1 presents weighted logit regression estimates for the effects of educational attainment, population composition (race-ethnicity, marital status, region), and intervening variables that education may act through (family income, BMI, health insurance status) on diabetic prevalence in each cohort (1935-39, 1940-44, 1945-49, and 1950-54) for males. For each cohort, Model 1 (M1) presents odds ratios excluding the intervening variables, while Model 2 (M2) includes the intervening variables.

Table 6-1: Weighted Logit Regression for Diabetes among Males Aged 60-64 by Cohort

	1935-39		1940-44		1945-49		1950-54	
	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.
Less Than High School (omitted)								
High School Only	0.74	0.82	0.80	0.85	0.73 *	0.81	0.73	0.77
Some College	0.64 **	0.72	1.03	1.12	0.70 *	0.79	0.68 *	0.73
Bachelor's Degree or Higher	0.51 ***	0.66 *	0.53 ***	0.71	0.46 ***	0.64 *	0.44 ***	0.59 **
Non-Hispanic White (omitted)								
Non-Hispanic Black	1.71 **	1.85 ***	1.54 **	1.60 **	1.33	1.36 *	1.30	1.36
Hispanic	1.29	1.34	1.50 *	1.50 *	1.29	1.41	2.06 ***	2.19 ***
Asian/Pacific Islander	0.79	1.18	1.53	2.17 *	1.11	1.67	1.11	1.67
Native American	1.17	1.18	1.76	1.73	1.45	1.19	1.34	1.32
Other	1.00	1.00	2.19	1.90	1.46	1.05	3.56	2.07
Married (omitted)								
Single/Never Married	0.61 *	0.61 *	1.29	1.15	0.79	0.78	1.24	1.12
Cohabiting	1.01	1.04	0.88	0.97	0.52 *	0.57	1.09	1.15
Separated	0.36 **	0.38 *	1.90 *	1.82	1.37	1.51	1.31	1.25
Divorced	0.96	0.87	1.02	0.93	0.99	1.00	0.92	0.88
Widowed	0.92	0.89	1.74 **	1.68 *	0.66	0.62 *	0.76	0.72
Family Incomes (\$1000s) in 2016 dollars		0.994 **		0.993 ***		0.994 ***		0.994 **
Has Health Insurance		1.71 *		1.14		1.53 *		1.66 *
BMI		1.10 ***		1.14 ***		1.17 ***		1.13 ***
South (omitted)								
Northeast	0.85	0.87	0.85	0.83	0.73 *	0.77	1.05	1.08
Midwest	0.72 *	0.69 *	1.12	1.11	0.98	0.95	1.03	1.00
West	0.93	0.93	1.06	1.11	0.93	1.02	0.70 **	0.73 *
Constant	0.25 ***	0.01 ***	0.19 ***	0.00 ***	0.34 ***	0.00 ***	0.30 ***	0.01 ***
N	3,232		3,443		4,355		4,682	
Pseudo R-squared	0.0209	0.0514	0.0235	0.0807	0.0205	0.0961	0.0297	0.8370

***p<.001 **p<.01 *p<.05

Note: race-ethnicity category "Other" dropped in 1935-39 cohort due to small N

In M1 for the earliest-born cohort (1935-39), the odds of being diagnosed with diabetes for males who graduated from high school are 26 percent lower than males who did not complete high school, controlling for population composition. The relationship is not statistically significant ($p < .05$). Including intervening variables, the odds of being diagnosed for males who graduated from high school are 18 percent lower than for males who did not complete high school, reducing the effect size by nearly one-third. This relationship also is not statistically significant ($p < .05$). The odds of being diagnosed with diabetes for males who went to college but did not graduate are 36 percent lower than for high school dropouts, controlling only for population composition (M1), and the relationship is statistically significant ($p < .01$). Including intervening variables (M2) reduced the estimate for males with some college compared to those

who did not graduate high school to 28 percent (reduced by nearly one-quarter), and is not statistically significant. Finally, controlling for population composition, the odds of being diagnosed with diabetes for males who graduate college are 49 percent lower than for males without a high school education, and the relationship is highly statistically significant ($p < .001$). Introducing intervening variables, the odds for males who graduate from college compared to males without a high school education decreases to 34 percent (reduced by nearly one-third), with a lower level of statistical significance ($p < .05$). Postestimation tests of the differences in odds ratios between M1 and M2 for the 1935-39 cohort are all statistically significant ($p < .05$ for high school only and some college, and $p < .001$ for bachelor's).

Thus, the odds for diabetes prevalence for the 1935-1939 cohort, compared to males without high school education, becomes lower as educational levels rise, from 26, to 36, to 49 percent lower, respectively, for males graduating from high school, completing some college, and graduating from college. In addition, while intervening variables account for between about one-fourth and one-third of the effect of education on diabetes prevalence, two-thirds to three-fourths of the effect of education is a direct effect not acting through the three intervening variables of family income, having health insurance, or BMI.

For the 1940-44 cohort, the odds of being diagnosed with diabetes for males with a high school degree compared to high school dropouts are not statistically significant, in either M1 or M2 ($p < .05$). The odds ratio for males with some college experience is also not statistically significant ($p < .05$) in either model for the 1940-44 cohort. Lastly, the odds of being diagnosed with diabetes for college graduates are 47 percent lower than for high school dropouts, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds decrease to 29 percent and are no longer significant

($p < .05$). Postestimation tests of the differences in odds ratios between M1 and M2 for the 1940-44 cohort are statistically significant for males with a bachelor's degree ($p < .001$).

Thus, controlling for population composition, the odds of diabetes for 1940-44 cohort for male college graduates are 47 percent lower than for males without high school, a value nearly identical to the 49 percent estimates for the 1935-1939 cohort.

For the 1945-49 cohort, the odds of being diagnosed with diabetes for high school graduates are 27 percent lower than high school dropouts, controlling for population composition. The relationship is slightly statistically significant ($p < .05$). With intervening variables in M2, the odds decrease to 19 percent, and are no longer statistically significant ($p < .05$). Controlling for population composition, the odds of being diagnosed with diabetes for males with some college experience are 30 percent lower than for males with less than a high school education, and statistically significant ($p < .05$). With intervening variables, the odds ratio for males with some college is no longer significant ($p < .05$). Meanwhile, the odds of being diagnosed with diabetes for college graduates are 54 percent lower than for high school dropouts, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds for college graduates compared to high school dropouts decrease to 36 percent, and some significance is lost ($p < .05$). Postestimation tests of bachelor's odds ratios between the two models is statistically significant ($p < .001$).

Thus, controlling for population composition, the odds of diabetes for the 1945-49 for male college graduates are 54 percent lower than for males without a high school, a value similar to the estimates of 47 and 49 percent for the two earlier-born cohorts. About one-third of these effects are accounted for the intervening variables introduced in M2.

For the fourth and final cohort (1950-54), the odds ratio for male high school graduates compared to high school dropouts is not statistically significant in either model. Controlling for population composition (M1), the odds of being diagnosed with diabetes for males with some college experience are 32 percent lower than for males without a high school education. The relationship is slightly statistically significant ($p < .05$). In M2 for the 1950-54 cohort, the odds are no longer significant ($p < .05$) when including intervening variables. Meanwhile, the odds of being diagnosed with diabetes for college graduates are 56 percent lower than for high school dropouts, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds for college graduates compared to high school dropouts decrease to 41 percent (reduced by one-quarter). Postestimation tests of bachelor's odds ratios between the two models is statistically significant ($p < .01$).

Thus, the odds of being diagnosed with diabetes for the 1950-54 cohorts for college graduates are 56 percent lower than for high school drop outs, controlling for population composition, and about one-fourth of this effect is accounted for by the intervening variable. Thus, controlling for population composition, the odds of diabetes for various cohorts of male college graduates are about half as large (47 to 56 percent lower) compared to males without a high school, and between about one-fourth and about one-third of these effects are accounted for the intervening variables introduced in M2.

There is no statistically significant change in the effect of education on diabetes across cohorts (in either model). Postestimation tests find no statistical difference between odds ratios across cohorts, except for the odds ratios for males with some college between the 1935-39 and 1940-44 cohort, which is only a weak statistical difference ($p < .05$). Thus, there is no statistically measurable difference in the education gradient for diabetes prevalence for males across cohorts.

Turning to results for race-ethnic groups, without the intervening variables (M1), the odds ratios for the 1935-39 cohort are 71 percent greater for black males than for white males but this difference declines to 54 percent, 33 percent, and 30 percent, respectively, across the three later-born cohorts. Thus, the difference between black males and white males is about one-half as large for the latest cohort compared to the earliest-born cohort. Estimates for the two earlier-born cohorts are statistically significant ($p < .01$), but not for the two later-born cohorts. The odds ratios in the model that includes the intervening variables (M2) are slightly larger for each cohort, and the pattern of change across cohorts for M2 models is similar to the M1 models. For Hispanic males, without the intervening variables, the odds ratios are greater than for white males by 29 percent for the 1935-39 and 1945-49 cohorts, but are not statistically significant. The difference is larger for the 1940-44 cohort at 50 percent and significant ($p < .05$), and the difference is about twice as large for the most recent cohort at 106 percent and significant ($p < .001$). The introduction of the intervening variables (M2) has only a small effect on these coefficients. Nearly all estimates (23 out of 24) are not statistically significant for the Asian/Pacific Island, Native American, and Other groups. Chapter 7 presents a detailed analysis of both the education gradient and education composition effects on diabetes separately for whites, blacks, and Hispanics.

Thus, the black male disadvantage in odds compared to whites declined by about one-half across the cohorts from 71 percent and 85 percent higher (M1 and M2) for the earliest-born cohort to 30 percent and 36 percent higher (M1 and M2) for the latest cohort, while the Hispanic disadvantage more than tripled from 29 percent and 36 percent higher (M1 and M2) for the earliest-born cohort to 106 percent and 109 percent higher (M1 and M2) for the latest cohort.

Meanwhile, the odds for Asians/Pacific Islanders is significant in only M2 for the 1940-44 cohort ($p < .05$).

There is no clear pattern linking marital status and diabetes across cohorts in either M1 or M2. Most odds ratios (32 out of 40) are not statistically significant ($p < .05$), and the direction of the effect is erratic across cohorts for most marital statuses. For example, in both models for the 1935-39 cohort, the odds of being diagnosed for males who are separated are between 52-54 percent lower than for married males. But the effect of being diagnosed with diabetes for the married males group comparison flips to higher odds, then again to lower odds, then again to higher odds across the three later-born cohorts.

Males with higher family income are consistently less likely than males with lower family incomes to be diagnosed with diabetes for every cohort, and the odds ratios are highly statistically significant ($p < .001$). In the 1935-39 cohort, for every \$1,000 increase in family income (in 2016 dollars), the odds of being diagnosed with diabetes decreases by 0.6 percent. Thus, an increase of \$25,000 (in 2016 dollars) would lead to a 15 percent decrease in the odds of being diagnosed with diabetes. The odds ratios for family income do not change across cohorts.

Having health insurance is positively associated with diabetes across cohorts. In the 1935-39 cohort, the odds of being diagnosed with diabetes for males with health insurance are 71 percent higher than for those without health insurance, controlling for other covariates. This relationship is slightly statistically significant for the 1935-39, 1945-49, and 1950-54 cohorts ($p < .05$), and the odds range between 53-71 percent greater for males with health insurance compared to males without health insurance. The results for the 1940-44 cohort are not significant ($p < .05$). It may be the case that these coefficients overestimate the true differences in prevalence for these two groups, however, because males with health insurance may be more

likely to see a physician than males without health insurance, and be more likely to be diagnosed with diabetes.

Males with a higher BMI also have greater odds of being diagnosed with diabetes, which is a consistent relationship across cohorts. Across cohorts, a one unit increase in BMI results in a 10-17 percent increase in the odds of being diagnosed with diabetes, controlling for other covariates. All four odds ratios are highly statistically significant ($p < .001$).

Most odds ratios for region (19 out of 24) are not significant ($p < .05$). Compared to the South, only one of eight odds ratios is significant for the Northeast, and only two of eight are significant for the Midwest and West.

2) Females Only

Table 6-2: Weighted Logit Regression for Diabetes among Females Aged 60-64 by Cohort

	1935-39		1940-44		1945-49		1950-54	
	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.
Less Than High School (omitted)								
High School Only	0.75	0.81	0.74 *	0.80	0.78	0.86	0.72 *	0.81
Some College	0.67 *	0.71	0.66 **	0.71 *	0.73 *	0.85	0.59 ***	0.69 *
Bachelor's Degree or Higher	0.25 ***	0.34 ***	0.34 ***	0.49 ***	0.37 ***	0.56 **	0.35 ***	0.52 ***
Non-Hispanic White (omitted)								
Non-Hispanic Black	3.35 ***	2.63 ***	2.54 ***	2.00 ***	2.12 ***	1.76 ***	2.11 ***	1.74 ***
Hispanic	2.18 ***	2.14 ***	1.77 **	1.69 **	2.03 ***	2.09 ***	1.69 ***	1.64 **
Asian/Pacific Islander	1.52	2.48	2.23 *	4.12 ***	1.27	2.33 *	1.83 *	2.78 ***
Native American	2.60	3.45 *	1.33	1.27	1.34	1.11	2.76 *	2.04
Other	3.04	2.61	1.00	1.00	0.85	0.81	1.75	1.59
Married (omitted)								
Single/Never Married	0.69	0.56	0.89	0.66	1.33	0.92	1.61 **	1.22
Cohabiting	0.77	0.88	1.23	0.94	1.03	1.14	1.31	1.34
Separated	0.64	0.53	1.67	1.34	2.36 **	1.73 *	1.41	1.05
Divorced	1.05	1.02	1.25	1.08	1.19	0.89	1.20	0.97
Widowed	0.96	0.80	1.17	1.00	1.29	0.96	1.05	0.85
Family Incomes (\$1000s) in 2016 dollars		0.997		0.992 **		0.992 ***		0.993 ***
Has Health Insurance		1.26		1.60 *		1.51 *		1.21
BMI		1.14 ***		1.12 ***		1.13 ***		1.11 ***
South (omitted)								
Northeast	0.96	1.00	0.67 *	0.64 *	0.77	0.84	0.65 **	0.69 *
Midwest	1.03	0.92	1.01	0.91	1.04	0.997	0.91	0.87
West	0.81	0.82	0.78	0.78	0.81	0.91	0.65 **	0.67 **
Constant	0.15 ***	0.003 ***	0.18 ***	0.01 ***	0.22 ***	0.01 ***	0.27 ***	0.01 ***
N	3,783		4,093		4,956		5,549	
Pseudo R-squared	0.0542	0.1227	0.0476	0.1150	0.0487	0.1379	0.0520	0.1193

*** $p < .001$ ** $p < .01$ * $p < .05$

Note: race-ethnicity category "Other" dropped in 1940-44 cohort due to small N

Table 6-2 presents weighted logit regression estimates for the effects of educational attainment, population composition (race-ethnicity, marital status, region), and intervening variables that education may act through (family income, BMI, health insurance status) on diabetic prevalence for each cohort (1935-39, 1940-44, 1945-49, and 1950-54) for females. For each cohort, Model 1 (M1) presents odds ratios excluding the intervening variables, while Model 2 (M2) includes the intervening variables.

In M1 for the earliest-born cohort (1935-39), the odds of being diagnosed with diabetes for females who graduated from high school are 25 percent lower than females who did not graduate from high school, controlling for population composition. The relationship is not statistically significant ($p < .05$). Including intervening variables, the odds of being diagnosed for females who graduated from high school are 19 percent lower than for females who did not complete high schools, reducing the effect size by nearly one-quarter. This relationship is not statistically significant ($p < .05$). The odds of being diagnosed with diabetes for females who went to college but did not graduate are 33 percent lower than for high school dropouts, controlling only for population composition (M1), and the relationship is slightly statistically significant ($p < .05$). Including intervening variables (M2) reduced the estimate for females with some college to 29 percent, and this estimate is not statistically significant ($p < .05$). Finally, controlling for population composition, the odds of being diagnosed with diabetes for females with a bachelor's degree are 75 percent lower than for females without a high school education, and the relationship is highly statistically significant ($p < .001$). Introducing intervening variables, the odds for females who graduate from college compared to females without a high school education decreases to 66 percent (reduced by more than one-tenth), and is highly statistically

significant ($p < .001$). Postestimation tests of the differences between M1 and M2 are statistically significant only for the bachelor's degree ($p < .01$).

Thus, the odds for diabetes prevalence for the 1935-39 cohort, compared to females without high school education, becomes lower as educational levels rise, from 25, to 33, to 75 percent lower, respectively, for females graduating from high school, completing some college, and graduating from college. In addition, while the intervening variables account for between about one-tenth to one-fourth of effect of education on diabetes, three-fourths to nine-tenths of the effect of education is a direct effect not acting through the three intervening variables of family income, having health insurance, or BMI.

For the second cohort (1940-44), the odds of being diagnosed with diabetes for females with a high school degree are 26 percent lower than for females who did not finish high school, controlling for population composition (M1), and the relationship is slightly statistically significant ($p < .05$). Including intervening variables with population composition (M2), the odds of being diagnosed with diabetes for female high school graduates compared to females who did not finish high school is reduced to 20 percent, and is no longer statistically significant ($p < .05$). In the first model for this cohort, the odds for females who did not complete college are 34 percent lower than for females who did not complete high school, controlling for population composition. The relationship is statistically significant ($p < .01$). Controlling for population composition and intervening variables (M2), the odds are reduced to 29 percent (reduced by one-seventh), and some significance is lost ($p < .05$). Postestimation tests find no difference between the two models for this odds ratio. Lastly, the odds of being diagnosed with diabetes for college graduates are 66 percent lower than for high school graduates, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in

M2, the odds decrease to 51 percent (reduced by one-quarter), remaining statistically significant. Postestimation tests find this odds ratio change between M1 and M2 to be statistically significant ($p < .001$).

Thus, the odds for diabetes prevalence for the 1940-44 cohort, compared to females without high school education, becomes lower as educational levels rise, from 25, to 34, to 66 percent lower, respectively, for females graduating from high school, completing some college, and graduating from college. In addition, while the intervening variables account for between about one-seventh to one-fourth of effect of education on diabetes, three-fourths to six-sevenths of the effect of education is a direct effect not acting through the three intervening variables of family income, having health insurance, or BMI.

For the 1945-49 cohort, the odds of being diagnosed with diabetes for females with a high school degree compared to high school dropouts are not statistically significant in either M1 or M2 ($p < .05$). Controlling for population composition, the odds of being diagnosed with diabetes for females with some college experience are 27 percent lower than for females with less than a high school education, and statistically significant ($p < .05$). Including intervening variables, the odds decrease to 15 percent and are not statistically significant ($p < .05$). Meanwhile, the odds of being diagnosed with diabetes for college graduates are 63 percent lower than for high school dropouts, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds decrease to by nearly one-third to 44 percent, and some significance is lost ($p < .01$). Postestimation tests of the least and most educated odds ratios between M1 and M2 for the 1945-49 cohort are statistically significant ($p < .05$ for high school only, and $p < .001$ for bachelor's degree).

Thus, the odds for diabetes prevalence for the 1940-49 cohort, compared to females without high school education, becomes lower as educational levels rise, from 22, to 27, to 63 percent lower, respectively, for females graduating from high school, completing some college, and graduating from college. In addition, while the intervening variables account for between about one-third of effect of education on diabetes, two-thirds of the effect of education is a direct effect not acting through the three intervening variables of family income, having health insurance, or BMI.

For the fourth and final cohort (1950-54), the odds of being diagnosed with diabetes for female high school graduates are 28 percent lower than high school dropouts, controlling for population composition (M1). Including intervening variables (M2), the odds decrease for female high school graduates compared to high school dropouts declines to 19 percent (reduced by one-third), and are no longer statistically significant. Controlling for population composition (M1), the odds of being diagnosed with diabetes for females with some college experience are 41 percent lower than for females without a high school education. The relationship is highly statistically significant ($p < .001$). In M2 for the 1950-54 cohort, the odds for females with some college compared to high school dropouts decreased to 31 percent (reduced by one-quarter) and some statistical significance is lost ($p < .05$) when including intervening variables. Lastly, the odds of being diagnosed with diabetes for college graduates are 65 percent lower than for high school dropouts, controlling for population composition. The relationship is highly significant ($p < .001$). Including intervening variables in M2, the odds for female college graduates compared to high school dropouts decrease to 48 percent (reduced by one-quarter). Postestimation tests of all three education odds ratios between M1 and M2 for the 1950-54 cohort are statistically significant ($p < .05$ for high school only and some college, and $p < .001$ for bachelor's).

Thus, the odds for diabetes prevalence for the 1950-54 cohort, compared to females without high school education, becomes lower as educational levels rise, from 28, to 41, to 65 percent lower, respectively, for females graduating from high school, completing some college, and graduating from college. In addition, while the intervening variables account for between about one-fourth to one-third of effect of education on diabetes, three-fourths to two-thirds of the effect of education is a direct effect not acting through the three intervening variables of family income, having health insurance, or BMI.

There is no statistical change in the effect of education on diabetes across cohorts (in either model). Postestimation tests find no statistical difference between odds ratios across cohorts. Thus, there is no statistically measurable difference in the education gradient for diabetes prevalence across female cohorts.

Turning to results for race-ethnic groups, all results for black and Hispanic females in both models (M1 and M2) are statistically significant (usually $p < .001$). Without the intervening variables (M1), the odds ratios for the 1935-39 cohort are 235 percent greater for black females than for white females but this difference declines to 154 percent, 112 percent, and 111 percent, respectively, across the three later-born cohorts. Thus, the difference between black females and white females is about one-half as large for the latest-born cohort compared to the earliest-born cohort. These differences are about three times larger than the corresponding differences in each cohort for black males compared to white males. The odds ratios in the model that includes the intervening variables (M2) are notably smaller for each cohort compared to the models without intervening variables (M1), but the pattern of change across cohorts for M2 models is similar to the M1 models. For Hispanic females, without the intervening variables, the odds ratios are greater than for white females by 118 percent for the 1935-39 cohorts, but this difference is

slightly smaller at 103 percent for the 1940-45 cohort, and smaller still for the 1940-44 and 1950-54 cohorts, at 77 and 69 percent, respectively. The introduction of the intervening variables (M2) has only a small effect on these coefficients. About one-half of the estimates (5 out of 8) for Asian/Pacific Islander females are statistically significant, but the differences compared to white females are erratic, jumping from 52 percent to 123 percent, respectively, across the two earlier-born cohorts, to 27 percent and then 83 percent respectively, for the two later-born cohorts. Most of the odds ratios (14 out of the 16) for Native American and Other females are not statistically significant. Chapter 7 presents a detailed analysis of both the education gradient and education composition effects on diabetes separately for whites, blacks, and Hispanics.

There is no clear pattern linking marital status and diabetes across cohorts in either M1 or M2. Most odds ratios (37 out of 40) are not statistically significant ($p < .05$), and the direction of the effect is erratic across cohorts. For example, in both models for the 1935-39 cohort, the odds of being diagnosed for females who are separated are between 36-47 percent lower than for married females, but not significant ($p < .05$). Meanwhile, the effect of being diagnosed for this same group comparison changes direction to higher odds in the three later-born cohorts, ranging from 34 percent to 136 percent greater for separate females than for married female, and estimates are significant only in the 1945-49 cohort.

Females with higher family income are consistently less likely than females with lower family incomes to be diagnosed with diabetes for the three later-born cohorts, and these estimates are statistically significant for the 1940-44 cohort ($p < .01$) and the 1949-50 and 1950-54 cohorts ($p < .001$). In the 1940-44 cohort, for every \$1,000 increase in family income, the odds of being diagnosed with diabetes decrease by 0.8 percent. Thus, an increase of \$25,000 would lead to a 20 percent increase in the odds of being diagnosed with diabetes. The odds ratios for family

income do not change across the three later-born cohorts, and are similar to the odds ratios for males at .992 or .993 vs. .993 or .994, respectively.

Having health insurance is positively associated with diabetes across cohorts, but is statistically significant only for the two-middle cohort. The odds of being diagnosed with diabetes for females born in the two-middle cohorts with health insurance are 40-51 percent higher than for those without health insurance, controlling for other covariates. This relationship is slightly statistically significant ($p < .05$). It may be the case that these coefficients overestimate the true differences in prevalence for these two groups, because females with health insurance may be more likely to see a physician than females without health insurance, and therefore more likely to be diagnosed with diabetes.

Females with a higher BMI also have greater odds of being diagnosed with diabetes, which is a consistent relationship across cohorts. Across cohorts, a one unit increase in BMI results in an 11-14 percent increase in the odds of being diagnosed with diabetes, controlling for other covariates. All four odds ratios are highly statistically significant ($p < .001$).

Most odds ratios for region (18 out of 24) are not significant ($p < .05$). Compared to the South, only two of eight odds ratios are significant for the West, and four of eight are significant for the Northeast.

3) Education Gradient Effect on Diabetes Prevalence: Males vs. Females

Results indicate that there is no statistically measurable difference in the education gradient for diabetes prevalence among either males or females across cohorts. Interactions and postestimation Wald tests indicate no pattern of change in the effect of education on diabetes across cohorts for either males or females.

Turning to differences across genders, the educational gradient is greater for females than for males. At the educational extremes, for the 1935-39 cohort, the odds of being diagnosed with diabetes for college graduates compared to high school dropouts are 49 percent and 75 percent lower, respectively, for males and females controlling for population composition. Postestimation Wald test indicate this difference to be slightly statistically significant ($p < .05$). Additionally, the odds for college graduates compared to high school dropouts are 34 percent and 66 percent lower, respectively, for males and females, controlling for population composition and intervening variables. Postestimation test indicates this difference is not statistically significant ($p < .05$). Results across cohorts are quite similar.

Thus, the odds of diabetes diagnosis for college graduates compared to high school dropouts, controlling for population composition, for various cohorts are 47 to 56 percent lower for males and 63 to 75 percent lower for females, with no clear trend across cohorts for either males or females. Similarly, the odds of diabetes diagnosis for college graduates compared to high school dropouts, controlling for population composition and the intervening variables, for various cohorts are 29 to 41 percent lower for males and 44 to 66 percent for females, with no clear trend across cohorts for either males or females.

In short, the odds of diabetes diagnosis for college graduates were substantially lower than for high school dropouts for males in each cohort, and still lower for female college graduates compared to female high school dropouts in each cohort, that is, the education gradient is steeper in each cohort for females than for males. Meanwhile, there is no statistically measurable change across cohorts for males in the education gradient for diabetes prevalence and no statistically measurable change across cohorts for females in the education gradient for diabetes prevalence.

As noted at the beginning of this chapter, gender differences in diabetes prevalence can be accounted for by gender differences in educational gradients, gender differences in educational composition, or both. The results from the regression models discussed above indicate that there are noteworthy gender differences between males and females in the education gradient for diabetes, but that neither males nor females experienced changes in the gradient across cohorts. The chapter now turns to results from Fairlie Decomposition models that estimate the extent to which changes in educational composition for males and females can account for change in diabetes prevalence across cohorts.

Decomposition Results

1) *Males Only*

Table 6-3: Decomposition of Cohort Disparities in Diabetes Prevalence among Males

	1935-39 to 1940-44		1940-44 to 1945-49		1945-49 to 1950-54		1935-39 to 1950-54	
Rates	13.88 to 15.37		15.37 to 17.39		17.39 to 17.20		13.88 to 17.20	
Percentage Point Change	1.49		2.02		-0.19		3.32	
Variables	Effect	S.E.	Effect	S.E.	Effect	S.E.	Effect	S.E.
Education Level	-0.37 ***	0.09	-0.54 ***	0.11	-0.27 ***	0.05	-1.11 ***	0.23
Race-Ethnicity	0.10 *	0.05	0.07	0.05	0.29 **	0.09	0.48 ***	0.12
Marital Status	0.05	0.06	0.00	0.04	0.10	0.07	0.08	0.13
Region	0.03	0.04	0.11 *	0.05	0.00	0.02	-0.05	0.04
N	6,679		7,798		9,037		7,918	

***p<.001 **p<.01 *p<.05

Table 6-3 presents results using the Fairlie Decomposition method to estimate the extent to which change across male cohorts in the overall rate of diabetes prevalence can be accounted for by change in the education composition of males, controlling of changes in population composition (race-ethnicity, marital status, region). In order to measure the full effect of educational composition, the intervening variables are not included in the analysis discussed here. In addition, the additional analysis that included the intervening variables of family income,

health insurance, and BMI are not reported here, because they produce only marginal changes in estimates of the education composition effect.

Table 6-3 shows the effect on diabetes prevalence of changes for the education composition and of changes for the composition of cohorts with regard to the control variables. The first decomposition model in Table 6-3 analyzes the contributions of education composition change and change in population composition (i.e., the control variables to the 1.49 percentage point increase for the diabetes prevalence that occurred across the 1935-39 and 1940-44 male cohorts. The effects are highly statistically significant ($p < .001$) for changes in the education composition, slightly statistically significant ($p < .05$) for changes in the race-ethnicity composition, and not significant ($p < .05$) for changes in the marital status and region compositions. Among these variables, education composition has the largest effect and it is negative. The results indicate that, controlling for population composition change (race-ethnicity, marital status, and region), if the educational composition had not changed across these two male cohorts, then the overall diabetes rate would have increased by an additional 0.37 percentage points (from 1.49 percent to 1.86 percent). Thus, if educational attainment had not increased across these two cohorts, the diabetes rate would have been one-quarter greater than the actual increase of 1.49 percent.

The second model in Table 6-3 analyzes the contribution of education composition change, controlling for the composition change in the control variables, to the 2.02 percentage point increase for diabetes prevalence between the 1940-44 and 1945-49 male cohorts. The effect of increasing education attainments is highly statistically significant ($p < .001$), slightly statistically significant ($p < .05$) for changes in region composition, and not significant ($p < .05$) for changes in the race-ethnicity and marital status compositions. Again, education is the largest

coefficient and has a negative effect on the change in diabetes. The results indicate that, controlling for population composition change, if the educational composition had not changed across the two male cohorts, then the overall diabetes rate would have increased by an additional 0.54 percentage points (from 2.02 percent to 2.56 percent). In other words, the increase would have been one-quarter greater than the actual increase.

Across the two most recent cohorts (1945-49 and 1950-54), the diabetes rate declined by a small 0.19 percentage points. The results in Table 6-3 indicate that if educational attainments had not increased, controlling for changes in population composition, the diabetes rate tended to increase by 0.27 percentage points, that is, instead of the actual decline of 0.19 percentage points, the diabetes rate for males would have increased by 0.08 percentage points. Thus, instead of declining slightly, the male diabetes rate would have continued to increase across these two cohorts, as it had across the earlier-born cohorts.

The final model in Table 6-3 presents variable contributions to diabetes change across all four male cohorts, that is, comparing the rate for 1950-54 cohort to the rate for 1935-39 cohort. Overall, the diabetes rate increased by 3.32 percentage points from 13.88 percent for the earliest-born cohort to 17.20 percent for the most recent cohort. If the education composition of the latest male cohort had remained unchanged compared to the earliest-born male cohort, diabetes prevalence would have been 1.11 percentage points higher, controlling for population composition. In other words, the increase in diabetes prevalence would have been one-third greater (33.4 percent greater) in the 1950-54 male cohort, increasing by 4.43 percentage points instead of 3.32 percentage points). That is, an additional 363,622 males ages 60-64 in the 1950-54 cohort would have diabetes.

The changing racial-ethnic composition has a consistently positive and statistically significant effect in three of the four models, although the effect across the 1940-44 and 1945-49 cohorts is not statistically significant. The positive direction of the effects of racial-ethnic composition changes indicate that changes in racial-ethnic composition contributed to the increases that occurred in the diabetes across cohorts. For example, of the 3.32 percentage point increase that occurred between the earliest and most recent male cohorts, about 15 percent can be accounted for by the changes in the racial-ethnic composition, mainly because black and Hispanic males experience higher diabetes rates than white males, and black and Hispanic males increased as a percentage of the population from 15.1 percent in the earliest-born cohort to 19.9 percent in the most recent cohort.

The change in diabetes prevalence explained by change in the marital composition is small and not significant in any of the models. Finally, regional composition explains little to none of the changes in diabetes prevalence across the models. Of the population composition variables, region is not significant in three of the four models, and the coefficients are null.

2) Females Only

Table 6-4: Decomposition of Cohort Disparities in Diabetes Prevalence among Females

	1935-39 to 1940-44		1940-44 to 1945-49		1945-49 to 1950-54		1935-39 to 1950-54	
Rates	11.38 to 12.41		12.41 to 15.44		15.44 to 15.18		11.38 to 15.18	
Percentage Point Change	1.02		3.03		-0.26		3.80	
Variables	Effect	S.E.	Effect	S.E.	Effect	S.E.	Effect	S.E.
Education Level	-0.75 ***	0.09	-0.84 ***	0.12	-0.78 ***	0.09	-1.91 ***	0.26
Race-Ethnicity	0.36 ***	0.06	0.21 ***	0.06	0.41 ***	0.07	0.84 ***	0.12
Marital Status	0.06	0.06	0.16 *	0.07	0.17 **	0.06	0.45 *	0.18
Region	0.07	0.04	-0.02	0.05	0.21 ***	0.06	0.10	0.06
N	7,880		9,035		10,505		9,332	

***p<.001 **p<.01 *p<.05

Note: race-ethnicity category "Other" dropped in 1940-44 cohort due to small N

Table 6-4 presents results using the Fairlie Decomposition method to estimate the extent to which change across female cohorts in the overall rate of diabetes prevalence can be

accounted for by change in the education composition of females, controlling of changes in population composition (race-ethnicity, marital status, region). In order to measure the full effect of educational composition, the intervening variables are not included in the analysis discussed here. In addition, the additional analysis that included the intervening variables of family income, health insurance, and BMI are not reported here, because they produce only marginal changes in estimates of the education composition effect.

Table 6-4 shows the effect on diabetes prevalence of changes for the education composition and of changes for the composition of cohorts with regard to the control variables. The first decomposition model in Table 6-3 analyzes the contributions of education composition change and change in population composition (i.e., the control variables to the 1.02 percentage point increase for the diabetes prevalence that occurred across the 1935-39 and 1940-44 female cohorts. The effects are highly statistically significant ($p < .001$) for changes in the education and race-ethnicity composition, and not significant ($p < .05$) for changes in the marital status and region compositions. Among these variables, education composition has the largest effect and it is negative. The results indicate that, controlling for population composition change (race-ethnicity, marital status, and region), if the educational composition had not changed across these two female cohorts, then the overall diabetes rate would have increased by an additional 0.75 percentage points (from 1.02 percent to 1.77 percent). Thus, if educational attainment had not increased across these two cohorts, the diabetes rate would have been three-fourths greater than the actual increase of 1.02 percent.

The second model in Table 6-3 analyzes the contribution of education composition change, controlling for the composition change in the control variables, to the 3.03 percentage point increase for diabetes prevalence between the 1940-44 and 1945-49 female cohorts. The

effect of increasing education attainments and changing racial-ethnic composition are highly statistically significant ($p < .001$), slightly statistically significant ($p < .05$) for changes in marital status composition, and not significant ($p < .05$) for changes in the region composition. Again, education is the largest coefficient and has a negative effect on the change in diabetes. The results indicate that, controlling for population composition change, if the educational composition had not changed across the two female cohorts, then the overall diabetes rate would have increased by an additional 0.84 percentage points (from 3.03 percent to 3.87 percent). In other words, the increase would have been one-quarter greater than the actual increase.

Across the two most recent cohorts (1945-49 and 1950-54), the diabetes rate declined by a small 0.26 percentage points. The results in Table 6-3 indicate that if educational attainments had not increased, controlling for changes in population composition, the diabetes rate tended to increase by 0.78 percentage points, that is, instead of the actual decline of 0.26 percentage points, the diabetes rate for females would have increased by 0.52 percentage points. Thus, instead of declining slightly, the female diabetes rate would have continued to increase across these two cohorts, as it had across the earlier-born cohorts.

The final model in Table 6-3 presents variable contributions to diabetes change across all four female cohorts, that is, comparing the rate for 1950-54 cohort to the rate for 1935-39 cohort. Overall, the diabetes rate increased by 3.80 percentage points from 11.38 percent for the earliest-born cohort to 15.18 percent for the most recent cohort. If the education composition of the latest female cohort had remained unchanged compared to the earliest-born female cohort, diabetes prevalence would have been 1.91 percentage points higher, controlling for population composition. In other words, the increase in diabetes prevalence would have been one-half greater (50.3 percent greater) in the 1950-54 female cohort, increasing by 5.71 percentage points

instead of 3.80 percentage points). That is, an additional 668,061 females ages 60-64 in the 1950-54 cohort would have diabetes.

The changing racial-ethnic composition has a consistently positive and statistically significant effect in all four models. The positive direction of the effects of racial-ethnic composition changes indicate that changes in racial-ethnic composition contributed to the increases that occurred in the diabetes across cohorts. For example, of the 3.80 percentage point increase that occurred between the earliest and most recent female cohorts, about 22 percent can be accounted for by the changes in the racial-ethnic composition, mainly because black and Hispanic females experience higher diabetes rates than white females, and black and Hispanic females increased as a percentage of the population from 16.0 percent in the earliest-born cohort to 20.7 percent in the most recent cohort.

The change in diabetes prevalence explained by change in the marital composition is positive and slightly or slightly significant in the three later-born cohorts. The positive direction of effects of marital status changes indicate that changes in marital status contributed to the increases in diabetes for later-born cohorts. For example, of the 3.80 percentage point increase that occurred between the earliest and most recent cohorts, nearly 12 percent can be accounted for the changes in marital status composition. Finally, regional composition explains little to none of the changes in diabetes prevalence across the models. Of the population composition variables, region is not significant in three of the four models, and the coefficients are null.

3) Education Composition Effect on Diabetes Prevalence: Males vs. Females

The diabetes rate for males was greater than the rate for females in the 1935-39 cohort, at 13.88 percent vs. 12.41 percent, respectively. The increase was greater for males than for females across the 1935-39 to 1940-44 cohorts (1.49 percentage points vs. 1.02 percentage

point), but this reversed and the increase between the 1940-44 and 1944-49 cohorts was greater for females than for males (3.03 vs. 2.02 percentage points). Females and males in the most recent cohort experienced slight and similar declines in the diabetes rate of -0.26 and -0.19 percentage points, respectively. Overall, across the four cohorts, females experienced a greater increase in the diabetes rate than males of 3.80 vs. 3.32 percentage points. Despite the greater increase for females, and the partial convergence, the overall diabetes rate for females remained lower than for males in the most recent cohort, at 15.18 percent vs. 17.20 percent.

The decomposition models indicate that the educational composition effect for females was about twice as great as for males across each successive set of cohorts. In the first models (1935-39 vs. 1940-44), the compositional effect of increasing education reduced the increase in the diabetes rate by 0.37 percentage points for males and 0.75 percentage points for females. Put another way, if educational attainment had remained unchanged between the 1935-39 and 1940-44 cohorts, then the increase in the diabetes rate for males would have been one-fourth greater (1.86 percentage points instead of 1.49 percentage points), and the increase would have been three-fourths greater for females (1.77 percentage points instead of 1.02).

In the second models (1940-44 vs. 1945-49), the compositional effect of increasing educational attainments reduced the increase in the diabetes rate by 0.54 percentage points for males and 0.84 percentage points for females. Thus, if the increase in educational attainments had not occurred, the diabetes rate for males would have increased by 2.56 percentage points instead of 2.02 percentage points, and the rate for females would have increased by 3.84 percentage points instead of 3.03 percentage points.

In the third models (1945-49 vs. 1950-54), the gender difference in the effect of compositional change in education continued, with effects sizes of 0.27 and 0.78 percentage

points, respectively, for males and females. In contrast to the changes across earlier-born cohorts, the, actual diabetes rates were lower for the 1950-54 cohort than for the 1945-1949 cohort. But if educational attainments had remained unchanged across the 1945-49 and 1950-54 cohorts, the diabetes rates for both males and females would have increased instead of decreased. For males the increase would have been 0.08 percentage points, instead of a decrease of -0.19 percentage points, and the increase for females would have been 0.54 percentage points, instead of a decrease -0.20 percentage points.

Overall, gender differences in the effect of education composition change on diabetes rate change between the earliest-born cohort (1935-39) and latest-born cohort (1950-54) is more nearly three-fourths as great for females as for males (1.91 and 1.11 percentage points, respectively). Thus, the greater increases in educational expansion experienced by females, compared to males, led to substantially larger education composition effects for females than for males. Overall, if educational attainment had remained unchanged between the earliest- and latest-born cohort (i.e., no educational expansion), diabetes prevalence increase for males would have been one-third greater (4.43 instead of 3.32 percentage points), and it would have been one-half greater for females (5.71 instead of 3.80 percentage points).

The effect of changes in the racial-ethnic composition is consistently positive, leading to increased diabetes prevalence, and statistically significant for females in all four models, and in three of four models for males. Increasing racial-ethnic diversity in later-born male and female cohorts tended to increase the diabetes prevalence rate. For all four models among both genders, the change in diabetes explained by change in race-ethnicity composition is smaller than the effect of change in education composition, except for the change across the 1945-1949 to 1950-54 cohorts of males, where the effect sizes are the same. Overall, comparing the earliest- and

latest-born cohorts, the changing racial-ethnic composition explained 0.48 percentage points of the total 3.32 percentage point increase in diabetes prevalence for males, and 0.84 percentage points of the total 3.80 percentage point increase in diabetes prevalence for females.

The change in diabetes prevalence explained by marital composition is not significant for males, but is positive, small, and slightly significant in three of the four models for females. Overall, comparing the earliest-born and latest-born cohorts, the changing marital composition explained only 0.08 percentage points for males (non-significant, $p < .05$) in the increase of diabetes prevalence, and 0.45 percentage points for females (significant, $p < .05$). Finally, regional composition change explains little to none of the changes in diabetes prevalence across the models for males, but is positive, small, and highly significant ($p < .001$) in the difference between females born in the 1950-54 cohort compared to the 1945-49 cohort. Of the population composition variables, region is the least significant and least influential, with no consistent pattern across cohorts for either gender.

Summary of Results

The first important finding presented in this chapter is that higher educational attainment is strongly and negatively associated with diabetes prevalence in all four cohorts for each gender (Tables 6-1 and 6-2). For example, in various models controlling for population composition, males with a bachelor's degree have odds of being diagnosed with diabetes that are 47-56 percent lower than the odds for males who are high school dropouts. Even more striking is that, controlling for population composition, the odds of being diagnosed with diabetes among females with a bachelor's degree are 65-75 percent less than for females who are high school dropouts. This relationship is significant for all cohorts, whether or not mediating variables (i.e., family income, health insurance, and BMI) are included. Although the introduction of the

mediating variables into the models reduces the size of these estimates, even after controlling for the mediating variables, persons with a bachelor's degree have much lower odds of being diagnosed with diabetes than persons who are high school dropouts, 29-41 percent lower for males and 48-66 percent for females. Thus, the educational gradient of education for diabetes is quite large, especially for females, and while some of this effect acts through the mediating variables included in the models, most of the educational gradient effect is a direct effect that does not act through the intervening variables. Additionally, the magnitude of the relationship between educational attainment and diabetes does not change across cohorts for either gender. Interaction models (see Appendix) and postestimation Wald tests find no pattern of statistical difference in odds ratios across cohorts for males or females.

The second important finding is that the changing educational composition, that is, the educational expansion, has a negative effect on the growth in diabetes prevalence for later-born cohorts for both genders. If educational expansions had not occurred, that is, if education had remained constant at the levels for the 1935-39 cohort, the increase in the diabetes prevalence rate would have been one-third greater for the 1950-54 male cohort (4.43 instead of 3.32 percentage points), and it would have been three-fifths greater for females (5.71 instead of 3.80 percentage points).

Thus, educational expansion acted to limit diabetes growth for both males and females. The magnitude of the educational gradient did not change across cohorts for males or females, but the education gradient was large for males, and even larger for females than for males for each cohort and across cohorts. The compositional effect on diabetes change of the educational expansion was substantial and statistically significant from one cohort to the next and across all cohorts for both males and females. These new findings move beyond the extant literature by the

estimating the magnitude and assessing the extent to which changes have occurred in the educational gradient for diabetes across four successive birth-cohorts, comparing results for males and females, and by estimating the extent to which the educational expansion across these cohorts acted to limit increases in diabetes prevalence, again comparing results for males and females.

Chapter 7

Race-Ethnic Differences in Educational Gradients and Educational Composition Change in Diabetes Prevalence across Four Cohorts

Introduction

As discussed in Chapter 2, historic increases in education could lead to changes in diabetes prevalence across cohorts either through changes in the educational gradient for diabetes or through changes in the composition of the population with regard to educational attainments. This chapter begins by presenting results from logistic regression models that estimate educational gradients for whites, Hispanics, and blacks across four cohorts. The chapter then presents results from Fairlie Decomposition models that estimate the extent to which changes in educational composition for whites, Hispanics, and blacks can account for change in diabetes prevalence across cohorts.

Logistic Regression Results

4) Whites Only

Table 7-1 presents weighted logit regression estimates for the effects of educational attainment, population composition (gender, marital status, region), and intervening variables that education may act through (family income, BMI, health insurance status) on diabetic prevalence in each cohort (1935-39, 1940-44, 1945-49, and 1950-54) for whites. For each cohort, Model 1 (M1) presents odds ratios excluding the intervening variables, while Model 2 (M2) includes the intervening variables.

Table 7-1: Weighted Logit Regression for Diabetes among Whites Aged 60-64 by Cohort

	1935-39		1940-44		1945-49		1950-54	
	M1	M2	M1	M2	M1	M2	M1	M2
	O.R.	O.R.	O.R.	O.R.	O.R.	O.R.	O.R.	O.R.
Less Than High School (omitted)								
High School Only	0.60 ***	0.70 **	0.81	0.88	0.61 ***	0.69 *	0.71 *	0.82
Some College	0.52 ***	0.59 ***	0.82	0.90	0.63 ***	0.74 *	0.60 ***	0.73
Bachelor's Degree or Higher	0.30 ***	0.43 ***	0.43 ***	0.61 **	0.37 ***	0.55 ***	0.34 ***	0.55 ***
Female	0.63 ***	0.66 ***	0.62 ***	0.60 ***	0.70 ***	0.64 ***	0.73 ***	0.73 ***
Married (omitted)								
Single/Never Married	0.73	0.65	1.07	0.88	1.03	0.80	1.74 ***	1.27
Cohabiting	1.14	1.17	0.85	0.77	0.72	0.81	1.55	1.59
Separated	0.39	0.37	2.74 **	2.24	2.69 **	2.20 *	1.43	1.17
Divorced	1.18	1.11	1.22	1.07	1.16	1.04	1.16	1.00
Widowed	0.93	0.80	1.39 *	1.19	1.28	1.05	1.14	0.94
Family Incomes (\$1000s) in 2016 dollars		0.995 *		0.993 ***		0.995 ***		0.994 ***
Has Health Insurance		1.31		1.56 *		1.45 *		1.43
BMI		1.14 ***		1.15 ***		1.16 ***		1.14 ***
South (omitted)								
Northeast	0.94	0.93	0.74 *	0.68 *	0.70 **	0.75 *	0.74 *	0.75 *
Midwest	0.87	0.79	1.05	0.96	1.04	0.99	0.81	0.75 *
West	0.95	0.92	0.80	0.80	0.81	0.92	0.61 ***	0.62 ***
Constant	0.28 ***	0.01 ***	0.24 ***	0.00 ***	0.37 ***	0.00 ***	0.37 ***	0.01 ***
N		5,201		5,491		6,608		7,278
Pseudo R-squared		0.0279		0.1035		0.0248		0.1165

In M1 for the earliest-born cohort (1935-39), the odds of being diagnosed with diabetes for whites who graduated from high school are 40 percent lower than for whites who did not complete high school, controlling for population composition. The relationship is strongly statistically significant ($p < .001$). Including intervening variables, the odds of being diagnosed for whites who graduated from high school are 30 percent lower than for whites who did not complete high school, reducing the effect size by nearly one-fourth. This relationship is statistically significant ($p < .01$). The odds of being diagnosed with diabetes for whites who went to college but did not graduate are 48 percent lower than for high school dropouts, controlling only for population composition (M1), and the relationship is strongly statistically significant ($p < .001$). Including intervening variables (M2) marginally reduced the estimate for whites with some college compared to those who did not graduate high school to 41 percent, and remains strongly statistically significant. Finally, controlling for population composition, the odds of being diagnosed with diabetes for whites who graduate college are 70 percent lower than for

whites without a high school education, and the relationship is strongly statistically significant ($p < .001$). Introducing intervening variables, the odds for whites who graduate from college compared to whites without a high school education decreases to 57 percent ($p < .001$), reduced by nearly two-fifths. Postestimation tests of the differences in odds ratios between M1 and M2 for the 1935-39 cohort are all statistically significant ($p < .05$ for some college, and $p < .001$ for high school only and bachelor's).

Thus, the odds for diabetes prevalence for the 1935-1939 cohort, compared to whites without high school education, becomes lower as educational levels rise, from 40, to 48, to 70 percent lower, respectively, for whites graduating from high school, completing some college, and graduating from college. In addition, while intervening variables account for between about one-fourth and two-fifths of the effect of education on diabetes prevalence, three-fifths to more than three-fourths of the effect of education is a direct effect not acting through the three intervening variables of family income, having health insurance, or BMI.

For the 1940-44 cohort, the odds of being diagnosed with diabetes for whites with a high school degree compared to high school dropouts are not statistically significant, in either M1 or M2 ($p < .05$). The odds ratio for whites with some college experience is also not statistically significant ($p < .05$) in either model for the 1940-44 cohort. However, the odds of being diagnosed with diabetes for college graduates are 57 percent lower than for high school dropouts, controlling for population composition. The relationship is strongly significant ($p < .001$). Including intervening variables in M2, the odds decrease to 39 percent and significant ($p < .01$). Postestimation tests of bachelor's odds ratios between the two models is statistically significant ($p < .001$).

Thus, controlling for population composition, the odds of diabetes for 1940-44 cohort for white college graduates are 57 percent lower than for whites without high school, a value similar to the 70 percent estimate for the 1935-1939 cohort. Postestimation Wald test of the difference in odds ratio for bachelor's compared to less than high school between these two cohorts are not statistically significant ($p < .05$)

For the 1945-49 cohort, the odds of being diagnosed with diabetes for high school graduates are 39 percent lower than high school dropouts, controlling for population composition. The relationship is strongly statistically significant ($p < .001$). With intervening variables in M2, the odds decrease to 31 percent, and statistical significance is slightly reduced ($p < .05$), a reduction of one-fifth. Controlling for population composition, the odds of being diagnosed with diabetes for whites with some college experience are 37 percent lower than for whites with less than a high school education, and strongly statistically significant ($p < .001$). With intervening variables, the odds ratio for whites with some college is 26 percent lower compared to white high school dropouts, and the relationship is slightly significant ($p < .05$). Meanwhile, the odds of being diagnosed with diabetes for college graduates are 63 percent lower than for high school dropouts, controlling for population composition. The relationship is strongly significant ($p < .001$). Including intervening variables in M2, the odds for college graduates compared to high school dropouts decrease to 45 percent, and some significance is lost ($p < .05$). Postestimation tests of the differences in odds ratios between M1 and M2 for the 1945-49 cohort are all statistically significant ($p < .05$ for high school only, $p < .01$ for some college, and $p < .001$ for bachelor's).

Thus, controlling for population composition, the odds of diabetes for the 1945-49 for white college graduates are 63 percent lower than for whites without a high school, a value

similar to the estimates of 57 and 70 percent for the two earlier-born cohorts. About one-fourth of these effects are accounted for the intervening variables introduced in M2.

For the fourth and final cohort (1950-54), the odds of being diagnosed with diabetes for white high school graduates are 29 percent lower than high school dropouts, controlling for population. The relationship is slightly statistically significant ($p < .05$). Including intervening variables, the odds ratio for high school graduates compared to high school dropouts is no longer significant ($p < .05$). Controlling for population composition (M1), the odds of being diagnosed with diabetes for whites with some college experience are 40 percent lower than for whites without a high school education. The relationship is strongly statistically significant ($p < .001$). In M2 for the 1950-54 cohort, the odds are no longer significant ($p < .05$) when including intervening variables. Meanwhile, the odds of being diagnosed with diabetes for college graduates are 66 percent lower than for high school dropouts, controlling for population composition. The relationship is strongly significant ($p < .001$). Including intervening variables in M2, the odds for college graduates compared to high school dropouts decrease to 45 percent (reduced by nearly one-third). Postestimation tests of the differences in odds ratios between M1 and M2 for the 1950-54 cohort are all statistically significant ($p < .05$ for high school only, $p < .01$ for some college, and $p < .001$ for bachelor's).

Thus, the odds of being diagnosed with diabetes for the 1950-54 cohorts for college graduates are 66 percent lower than for high school drop outs, controlling for population composition, and about one-third of this effect is accounted for by the intervening variable. Thus, controlling for population composition, the odds of diabetes for various cohorts of white college graduates are over one-half to nearly one-quarter larger (57 to 70 percent lower) compared to whites without a high school degree, and between about one-fourth and about one-third of these

effects are accounted for the intervening variables introduced in M2, although the differences between the models for are not statistically significant for high school only and some college groups. Thus, the results indicate that most of the effect of education on diabetes prevalence does not act through the three intervening variables.

There is no statistically significant change in the effect of education on diabetes across cohorts (in either model). Postestimation Wald tests find no consistent statistical difference between odds ratios across cohorts. Thus, there is no statistically measurable difference in the education gradient for diabetes prevalence for whites across cohorts

There is little or no change in the effect size and statistical significance of gender on diabetes across cohorts. Controlling for population composition, the odds of being diagnosed with diabetes for white females are between 27 and 38 percent lower than for white males in various cohorts. With intervening variables, the odds ratios range from 27 to 40 percent across cohorts.

There is no clear pattern linking marital status and diabetes across cohorts in either M1 or M2. Most odds ratios (35 out of 40) are not statistically significant ($p < .05$), and the direction of the effect is erratic across cohorts for most marital statuses. Across cohorts, none of the odds ratios cohabiting whites or for divorced whites are statistically significant ($p < .05$), and only one of eight odds ratios for single/married whites and for widowed whites are statistically significant ($p < .05$). Even for separated whites, fewer than one-half (3 out of 8) odds ratios are statistically significant.

Whites with higher family income are consistently less likely than whites with lower family incomes to be diagnosed with diabetes across cohorts. These estimates are slightly statistically significant for the 1935-39 cohort ($p < .05$), and strongly statistically significant for

the three later-born cohorts ($p < .001$). In the 1935-39 cohort, for every \$1,000 increase in family income, the odds of being diagnosed with diabetes decrease by 0.5 percent. Thus, an increase of \$25,000 would lead to a 15 percent increase in the odds of being diagnosed with diabetes. The odds ratios for family income are essentially the same across all four cohorts, in the range of 0.993 to 0.995

Having health insurance is positively associated with diabetes in the three later-born cohorts. The results for the 1935-39 cohort are not significant ($p < .05$). In the 1940-44 cohort, the odds of being diagnosed with diabetes for whites with health insurance are 56 percent higher than for those without health insurance, controlling for other covariates. This relationship is slightly statistically significant for the 1940-44 and 1945-49 cohorts ($p < .05$), and the odds are in the narrow range of 45-56 percent greater for whites with health insurance compared to whites without health insurance. The results for the 1950-54 cohort are not significant ($p < .05$). It may be the case that these coefficients overestimate the true differences in prevalence for these two groups, however, because whites with health insurance may be more likely to see a physician than whites without health insurance, and as a result more likely to be diagnosed with diabetes.

Whites with a higher BMI also have greater odds of being diagnosed with diabetes, which is a consistent relationship across cohorts. Across cohorts, a one unit increase in BMI results in a 14-16 percent increase in the odds of being diagnosed with diabetes, controlling for other covariates. All four odds ratios are strongly statistically significant ($p < .001$).

Most odds ratios for region (15 out of 24) are not significant ($p < .05$). Compared to the South, only one of eight odds ratios is significant for the Midwest, and only two of eight are significant for the West. The six odds ratios for the three later cohorts are significant ($p < .05$) for the Northeast and are in the narrow range of 0.68 to 0.75.

5) *Hispanics Only*

Table 7-2: Weighted Logit Regression for Diabetes among Hispanics Aged 60-64 by Cohort

	1935-39		1940-44		1945-49		1950-54	
	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.
Less Than High School (omitted)								
High School Only	1.07	1.11	0.74	0.80	1.12	1.12	0.61 *	0.61 *
Some College	1.05	1.09	1.28	1.28	0.85	0.85	0.32 ***	0.37 ***
Bachelor's Degree or Higher	0.70	0.85	0.29 *	0.34	0.33 ***	0.38 *	0.44 **	0.56
Female	1.01	1.01	0.85	0.75	1.11	1.00	0.75	0.72
Married (omitted)								
Single/Never Married	1.12	0.97	0.91	0.85	1.43	1.64	0.48 *	0.39 **
Cohabiting	0.72	0.61	1.75	1.55	0.44	0.49	0.41	0.46
Separated	0.38 *	0.34 *	1.31	1.19	1.41	1.28	1.05	0.76
Divorced	0.59	0.56	0.88	0.88	0.93	0.85	0.78	0.64
Widowed	1.58	1.34	0.79	0.82	0.59	0.56	0.71	0.58
Family Incomes (\$1000s) in 2016 dollars		0.994		0.994		0.995		0.990 **
Has Health Insurance		1.42		1.70		1.74 *		1.56
BMI		1.06 **		1.08 **		1.1 ***		1.09 ***
South (omitted)								
Northeast	0.76	0.82	0.56	0.59	0.90	0.92	1.39	1.32
Midwest	1.36	1.36	1.88	1.67	0.74	0.82	2.01	1.95
West	0.92	0.97	1.43	1.36	0.85	0.95	0.78	0.81
Constant	0.26 ***	0.05 ***	0.28 ***	0.03 ***	0.41 ***	0.02 ***	0.71	0.07 ***
N	796		808		967		1,025	
Pseudo R-squared	0.0187	0.0348	0.0421	0.0721	0.0256	0.0761	0.0602	0.1035

***p<.001 **p<.01 *p<.05

Table 7-2 presents weighted logit regression estimates for the effects of educational attainment, population composition (gender, marital status, region), and intervening variables that education may act through (family income, BMI, health insurance status) on diabetic prevalence in each cohort (1935-39, 1940-44, 1945-49, and 1950-54) for Hispanics. For each cohort, Model 1 (M1) presents odds ratios excluding the intervening variables, while Model 2 (M2) includes the intervening variables.

There is no statistically significant effect of educational attainment on diabetes in the 1935-39 cohort in either model ($p < .05$). For the 1940-44 cohort, the odds ratios for Hispanics with a high school education or some college experience compared to Hispanics who did not complete high school are not statistically significant in either model ($p < .05$). Meanwhile, the odds of being diagnosed with diabetes for college graduates are 71 percent lower than for high

school dropouts, controlling for population composition. The relationship is statistically significant ($p < .01$). Including intervening variables in M2, the odds ratio for college graduates compared to high school dropouts is no longer significant ($p < .05$). For the 1945-49 cohort, the odds ratios for Hispanics with a high school education or some college experience compared to Hispanics who did not complete high school are not statistically significant in either model ($p < .05$). Meanwhile, the odds of being diagnosed with diabetes for college graduates are 67 percent lower than for high school dropouts, controlling for population composition. Including intervening variables, the odds of being diagnosed with diabetes for Hispanic college graduates are 62 percent lower than for Hispanics without a high school education. The statistical significance is slightly lowered, but remains significant ($p < .05$).

For the fourth and final cohort (1950-54), the odds of being diagnosed with diabetes for Hispanic high school graduates are 39 percent lower than high school dropouts, controlling for population. The relationship is slightly statistically significant ($p < .05$). Including intervening variables, the odds ratio for high school graduates remains 39 percent lower compared to high school dropouts. The relationship is slightly statistically significant ($p < .05$). Controlling for population composition (M1), the odds of being diagnosed with diabetes for Hispanics with some college experience are 68 percent lower than for Hispanics without a high school education. The relationship is strongly statistically significant ($p < .001$). In M2 for the 1950-54 cohort, the odds of being diagnosed with diabetes for Hispanics with a high school education are 63 percent lower than Hispanics who did not complete high school. The relationship is strongly statistically significant ($p < .001$) when including intervening variables. Meanwhile, the odds of being diagnosed with diabetes for college graduates are 36 percent lower than for high school dropouts, controlling for population composition. The relationship is statistically significant

($p < .01$). Including intervening variables in M2, the odds ratio for college graduates compared to high school dropouts is no longer significant ($p < .05$). Postestimation tests of bachelor's odds ratios between the two models is statistically significant ($p < .01$).

Thus, educational attainment has an effect on diabetes only for the three later-born cohorts. For all three of these cohorts, the odds ratios, controlling for population composition, are significant for Hispanics with a bachelor's degree compared to high school dropouts, with values that are 71 percent lower compared to high school dropouts for the 1940-44 cohort ($p < .05$), 67 percent lower for the 1945-49 cohort ($p < .001$), and 56 percent lower for the 1950-54 cohort ($p < .01$). The odds ratios for the latest cohort (1950-54) also are significant for lower education levels, at 39 percent lower for Hispanics with high school only compared to high school dropouts ($p < .001$) and 68 percent lower for Hispanics with some college compared to high school dropouts ($p < .01$). Thus, the effect of education changes for the latest-born cohort of Hispanics.

There is no clear pattern in the effect of gender on diabetes for Hispanics across the four cohorts. All odds ratios are not statistically significant, and the direction of the effect is erratic. There also is no clear pattern linking marital status and diabetes across cohorts in either M1 or M2. Most odds ratios (36 out of 40) are not statistically significant ($p < .05$), and the direction of the effect is erratic across cohorts for most marital statuses.

The relationship between income and diabetes for Hispanics is not statistically significant ($p < .05$) in the 1935-39, 1940-44, and 1945-49 cohorts. The estimates are statistically significant for the 1950-54 cohort ($p < .01$). In the 1950-54 cohort, for every \$1,000 increase in family income, the odds of being diagnosed with diabetes decrease by 1.05 percent. Thus, an increase of \$25,000 would lead to a 25 percent increase in the odds of being diagnosed with diabetes.

Having health insurance is not linked with diabetes in three of the four cohorts. The odds ratios for Hispanics with health insurance compared to Hispanics without health insurance are not statistically significant ($p < .05$) for the 1935-39, 1940-44, and 1950-54 cohort. In the 1945-49 cohort, the odds of being diagnosed with diabetes for Hispanics with health insurance are 74 percent greater than for Hispanics without health insurance. The results are slightly statistically significant ($p < .05$).

Hispanics with a higher BMI have greater odds of being diagnosed with diabetes, which is a consistent relationship across cohorts. Across cohorts, a one unit increase in BMI results in a 6-10 percent increase in the odds of being diagnosed with diabetes, controlling for other covariates. The odds ratios are strongly statistically significant ($p < .001$) for the two later-born cohorts, and statistically significant for the two earlier born cohorts ($p < .01$). It may be the case that these coefficients overestimate the true differences in prevalence for these two groups, however, because Hispanics with health insurance may be more likely to see a physician than Hispanics without health insurance, and as a result more likely to be diagnosed with diabetes

There is no clear pattern between region and diabetes across cohorts in either model. All of the odds ratios are not significant ($p < .05$), with erratically negative or positive odds ratios compared to Hispanics not in the South across the four cohorts.

6) Blacks Only

Table 7-3: Weighted Logit Regression for Diabetes among Blacks Aged 60-64 by Cohort

	1935-39		1940-44		1945-49		1950-54	
	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.	M1 O.R.	M2 O.R.
Less Than High School (omitted)								
High School Only	1.19	1.13	0.56 **	0.60 *	0.87	1.00	1.02	1.10
Some College	1.20	1.21	0.84	0.92	0.72	0.84	1.08	1.14
Bachelor's Degree or Higher	1.16	1.29	0.59	0.70	0.60 *	0.94	0.68	0.80
Female	1.40	1.13	1.20	1.02	1.36	1.02	1.49 *	1.27
Married (omitted)								
Single/Never Married	0.36 **	0.37 **	0.85	0.73	0.73	0.55	1.04	1.01
Cohabiting	0.65	0.75	1.04	1.17	0.36 *	0.39 *	0.86	0.83
Separated	0.59	0.64	1.11	1.07	1.33	1.16	1.24	1.19
Divorced	0.62	0.60	0.94	0.89	0.81	0.65	0.70	0.66
Widowed	0.74	0.68	1.03	0.98	0.76	0.59	0.63	0.60
Family Incomes (\$1000s) in 2016 dollars		0.999		0.996		0.990 ***		1.00
Has Health Insurance		1.61		0.95		1.99 **		1.23
BMI		1.10 ***		1.06 ***		1.12 ***		1.08 ***
South (omitted)								
Northeast	0.91	1.00	1.05	1.09	0.96	1.03	1.16	1.31
Midwest	0.63	0.66	0.86	0.85	0.89	0.78	1.74 *	1.89 **
West	0.74	0.84	0.97	1.06	1.21	1.22	0.97	1.10
Constant	0.34 ***	0.02 ***	0.39 ***	0.09 ***	0.42 ***	0.01 ***	0.27 ***	0.03 ***
N	850		992		1,284		1,425	
Pseudo R-squared	0.0226	0.0627	0.0132	0.0306	0.0161	0.0907	0.0223	0.0472

Table 7-3 presents weighted logit regression estimates for the effects of educational attainment, population composition (gender, marital status, region), and intervening variables that education may act through (family income, BMI, health insurance status) on diabetic prevalence in each cohort (1935-39, 1940-44, 1945-49, and 1950-54) for blacks. For each cohort, Model 1 (M1) presents odds ratios excluding the intervening variables, while Model 2 (M2) includes the intervening variables.

There is no clear pattern between educational attainment and diabetes for blacks. The majority of odds ratios (21 out of 24) are not statistically significant ($p < .05$). For the 1935-39 cohort, none of the odds ratios for educational attainment are statistically significant ($p < .05$) in either model. For the 1940-44 cohort, the odds of being diagnosed with diabetes for blacks who are high school graduates are 44 percent lower than for blacks who did not complete high school, controlling for population composition. The relationship is statistically significant ($p < .01$). In

M2, the odds for high school graduates are 40 percent lower than high school dropouts, and statistical significance is lower ($p < .05$). The odds ratios for blacks with some college or college graduates, compared to blacks who did not finish high school, are not statistically significant ($p < .05$). For the 1945-49 cohort, the odds ratios for black high school graduates and blacks who attended college, compared to black high school dropouts, are not statistically significant ($p < .05$). Meanwhile, the odds of being diagnosed with diabetes for black college graduates are 40 percent lower than for black high school dropouts, controlling for population composition. The relationship is slightly statistically significant ($p < .05$). Including intervening variables, the odds ratio is not statistically significant ($p < .05$). For the fourth and final cohort (1950-54), the odds ratios for all educational attainment categories are not statistically significant ($p < .05$).

Thus, all the odds of being diagnosed with diabetes are not statistically significant for the educational categories in the earliest- and latest-born cohorts ($p < .05$). Additionally, the majority of odds ratios for educational attainment are not statistically significant in the two middle cohorts. There is no clear relationship linking educational attainment and diabetes for blacks across the four cohorts.

There is no clear pattern in the effect of gender on diabetes for blacks across the four cohorts. The majority of odds ratios (7 out of 8) are not statistically significant, although the direction of the effect is positive for all cohorts. There also is no clear pattern linking marital status and diabetes across cohorts in either M1 or M2. Most odds ratios (36 out of 40) are not statistically significant ($p < .05$), and the direction of the effect is erratic across cohorts for most marital statuses. For example, in both models for the 1935-39 cohort, the odds of being diagnosed for blacks who are single or never married are between 63-64 percent lower than for married blacks. The relationship is slightly statistically significant in both models ($p < .05$). But

the effect of being diagnosed with diabetes for the married blacks group comparison is not statistically significant in the three later-born cohorts ($p < .05$), and the direction of the effect flips to higher odds in the latest-born cohort.

Blacks with higher family income are as likely as blacks with lower family incomes to be diagnosed with diabetes in the three of the four born cohorts. The relationship is not statistically significant ($p < .05$) for blacks in the 1935-39, 1940-44, and 1950-54 cohorts. The estimates are strongly statistically significant for the 1945-49 cohort ($p < .01$). In the 1945-49 cohort, for every \$1,000 increase in family income, the odds of being diagnosed with diabetes decrease by 1.0 percent. Thus, an increase of \$25,000 would lead to a 25 percent increase in the odds of being diagnosed with diabetes.

Having health insurance is not linked with diabetes for blacks in three of the four cohorts. The odds ratios for blacks with health insurance compared to blacks without health insurance are not statistically significant ($p < .05$) for the 1935-39, 1940-44, and 1950-54 cohort. In the 1945-49 cohort, the odds of being diagnosed with diabetes for blacks with health insurance are 99 percent greater than for blacks without health insurance. The results are statistically significant ($p < .01$).

Blacks with a higher BMI have greater odds of being diagnosed with diabetes, which is a consistent relationship across cohorts. Across cohorts, a one unit increase in BMI results in a 6-12 percent increase in the odds of being diagnosed with diabetes, controlling for other covariates. The odds ratios are strongly statistically significant ($p < .001$) across cohorts.

There is no clear pattern between region and diabetes across cohorts in either model. The majority of odds ratios (22 out of 24) are not significant ($p < .05$), with erratically negative or positive odds ratios for blacks not in the South across the four cohorts.

7) *Education Effect on Diabetes Prevalence: Whites vs. Hispanics vs. Blacks*

Results indicate that there is no statistically measurable difference in the education gradient across cohorts for diabetes prevalence among whites and blacks, but a measurable difference among Hispanics born in the 1950-54 cohort compared to the three earlier cohorts. Interactions and postestimation Wald tests indicate no pattern of change in the effect of education on diabetes across cohorts for either whites or blacks, but an emergent effect of education on diabetes for the latest-born Hispanics.

Turning to differences across race-ethnic groups, the educational gradient is more consistent for whites than for Hispanics, and more consistent for Hispanics than for blacks. At the educational extremes, the educational gradient for whites which a bachelor's degree compared to high school dropouts, controlling for population composition, is statistically significant for all cohorts and does not change across cohorts. Whites with a bachelor's degree have odds of being diagnosed with diabetes that are 57-70 percent lower than for white high school dropouts.

Although the odds ratio for Hispanics with a bachelor's degree is not statistically significant for the earliest-born cohort (1935-39), the odds ratios for later-born cohorts, as is the case for whites, are statistically significant, controlling for population composition. Hispanics with a bachelor's degree in the later three cohorts have odds of being diagnosed with diabetes that are 56-71 percent lower than for Hispanic high school dropouts, a level similar to whites. Also similar to whites, who experienced no change in the educational gradient across all four cohorts, controlling for population composition, Hispanics experienced no change in the educational gradient across the three earlier cohorts, but, unlike whites, Hispanics did experience a change in the educational gradient, but only between the third cohort (1945-49) and the most

recent cohort (1950-54). The reason for this change is that for the 1945-49 Hispanic cohort, a statistically significant educational gradient existed only for the bachelor's degree, but for the most recent Hispanic cohort, the odds ratios were statistically significant for each of the three education levels (high school only, some college, and bachelor's degree) compared to high school drop outs.

For blacks, unlike Hispanics and especially unlike whites, only one odds ratio for the bachelor's degree compared to high school dropouts, controlling for population composition, was statistically significant, that is, for the 1945-49 cohort, and only one odds ratio for high school only compared to high school dropouts was statistically significant, that is, for the 1940-44 cohort. In contrast, among Hispanics the odds ratios for the bachelor's degree compared high school dropouts were statistically significant for all three of the most recent cohorts (1944-44, 1945-49, 1950-54), and for all three education levels for the 1950-54 cohort. In still sharper contrast, for whites the odds ratios for all cohorts for all three education levels, controlling for population composition, were statistically significant, with only two exceptions, namely, the high school only and some college groups for the 1940-44 cohort. Insofar nearly none of the odds ratios were statistically significant for blacks, the (lack of) educational gradient for blacks did not change across the four cohorts.

In short, at the educational extremes, all four white cohorts experienced a substantial educational gradient for the bachelor's degree compared to high school dropouts, and three of the four Hispanic cohorts experienced a similarly substantial educational graduate for the bachelor's degree compared to high school dropouts, while the odds ratios for the educational gradient for blacks with a bachelor's compared to high school dropouts were not statistically significant for three of the four cohorts. In addition, the educational gradient did not change for whites or for

blacks across the four cohorts, and the educational gradient for Hispanics also did not change across the three earliest cohort, although the educational gradient did change for Hispanics between the third cohort and the most recent cohort.

As noted at the beginning of this chapter, race-ethnicity differences in diabetes prevalence can be accounted for by race-ethnicity differences in educational gradients, race-ethnicity differences in educational composition, or both. The results from the regression models discussed above indicate that there are noteworthy race-ethnicity differences between whites, Hispanics, and blacks in the education gradient for diabetes, but that all three groups experienced no change in the educational gradient, with the exception of the most recent cohort of Hispanics. The chapter now turns to results from Fairlie Decomposition models that estimate the extent to which changes in educational composition for whites, Hispanics, and blacks can account for change in diabetes prevalence across cohorts.

Decomposition Results

4) *Whites Only*

Table 7-4: Decomposition of Cohort Disparities in Diabetes Prevalence among Whites

	1935-39 to 1940-44		1940-44 to 1945-49		1945-49 to 1950-54		1935-39 to 1950-54	
Rates	10.94 to 11.92		11.92 to 14.43		14.43 to 13.55		10.94 to 13.55	
Percentage Point Change	0.98		2.51		-0.88		2.61	
Variables	Effect	S.E.	Effect	S.E.	Effect	S.E.	Effect	S.E.
Education Level	-0.74 ***	0.09	-0.83 ***	0.11	-0.60 ***	0.07	-1.86 ***	0.22
Gender	0.14 ***	0.04	0.17 ***	0.04	-0.04 *	0.02	0.07 *	0.03
Marital Status	0.09	0.06	0.07 *	0.04	0.16 ***	0.05	0.52 ***	0.14
Region	0.05	0.03	0.07	0.03	0.15 ***	0.04	0.07	0.04
N	10,692		12,099		13,886		12,479	

***p<.001 **p<.01 *p<.05

Table 7-4 presents results using the Fairlie Decomposition method to estimate the extent to which change across white cohorts in the overall rate of diabetes prevalence can be accounted for by change in the education composition of whites, controlling of changes in population

composition (gender, marital status, region). In order to measure the full effect of educational composition, the intervening variables are not included in the analysis discussed here. In addition, the additional analysis that included the intervening variables of family income, health insurance, and BMI are not reported here, because they produce only marginal changes in estimates of the education composition effect.

Table 7-4 shows the effect on diabetes prevalence of changes for the education composition and of changes for the composition of cohorts with regard to the control variables. The first decomposition model in Table 7-4 analyzes the contributions of education composition change and change in population composition (i.e., the control variables) to the 0.98 percentage point increase for the diabetes prevalence that occurred across the 1935-39 and 1940-44 white cohorts. The effects are highly statistically significant ($p < .001$) for changes in the education and gender compositions ($p < .001$), and not significant ($p < .05$) for changes in the marital status and region compositions. Among these variables, education composition has the largest effect and it is negative. The results indicate that, controlling for population composition change (gender, marital status, and region), if the educational composition had not changed across these two white cohorts, then the overall diabetes rate would have increased by an additional 0.74 percentage points (from 0.98 percent to 1.72 percent). Thus, if educational attainment had not increased across these two cohorts, the diabetes rate would have been three-fourths greater than the actual increase of 0.98 percent.

The second model in Table 7-4 analyzes the contribution of education composition change, controlling for the composition change in the control variables, to the 2.51 percentage point increase for diabetes prevalence between the 1940-44 and 1945-49 white cohorts. The effect of increasing education attainments and changes in gender composition are highly

statistically significant ($p < .001$), slightly statistically significant ($p < .05$) for changes in marital status composition, and not significant ($p < .05$) for changes in the marital status composition. Again, education is the largest coefficient and has a negative effect on the change in diabetes. The results indicate that, controlling for population composition change, if the educational composition had not changed across the two white cohorts, then the overall diabetes rate would have increased by an additional 0.83 percentage points (from 2.51 percent to 3.34 percent). In other words, the increase would have been one-third greater than the actual increase.

Across the two most recent cohorts (1945-49 and 1950-54), the diabetes rate declined by a small 0.88 percentage points. The results in Table 7-4 indicate that if educational attainments had not increased, controlling for changes in population composition, the diabetes rate tended to increase by only 0.60 percentage points, that is, instead of the actual decline of 0.88 percentage points, the diabetes rate for whites would have declined by 0.28 percentage points. Thus, the white diabetes rate would have declined less.

The final model in Table 7-4 presents variable contributions to diabetes change across all four white cohorts, that is, comparing the rate for 1950-54 cohort to the rate for 1935-39 cohort. Overall, the diabetes rate increased by 2.61 percentage points from 10.94 percent for the earliest-born cohort to 13.55 percent for the most recent cohort. If the education composition of the latest white cohort had remained unchanged compared to the earliest-born white cohort, diabetes prevalence would have been 1.86 percentage points higher, controlling for population composition. In other words, the increase in diabetes prevalence would have been nearly three-fourths greater (71.3 percent greater) in the 1950-54 white cohort, increasing by 4.47 percentage points instead of 2.61 percentage points). That is, an additional 942,540 whites ages 60-64 in the 1950-54 cohort would have diabetes.

The changing gender composition has a positive and statistically significant effect in three of the four models, although the effect across the 1940-44 and 1945-49 cohorts is negative and slightly statistically significant. The gender composition explains little of the changes in diabetes prevalence across the models. Chapter 4 finds very little change in the gender composition, ranging from 51.4 percent male and 48.6 percent female for the 1945-49 cohort., and 48.5 and 51.5 percent, male and female, respectively, for the 1950-54 cohort.

The changing marital composition as a positive and statistically significant effect in three of the four models. The positive direction of effects of marital composition indicate that changes in marital composition contributed to the increase that occurred across the later-born cohorts. For example, of the 2.61 percentage point increase that occurred between the earliest and most recent white cohorts, about 20 percent can be accounted for by the changes in the marital composition. Finally, regional composition explains little to none of the changes in diabetes prevalence across the models. Of the population composition variables, region is not significant in three of the four models, and the coefficients are small.

5) *Hispanics Only*

Table 7-5: Decomposition of Cohort Disparities in Diabetes Prevalence among Hispanics

	1935-39 to 1940-44		1940-44 to 1945-49		1945-49 to 1950-54		1935-39 to 1950-54	
Rates	18.91 to 20.42		20.42 to 25.54		25.54 to 26.19		18.91 to 26.19	
Percentage Point Change	1.51		5.12		0.65		7.28	
Variables	Effect	S.E.	Effect	S.E.	Effect	S.E.	Effect	S.E.
Education Level	-0.16	0.16	-0.19	0.18	-1.20 ***	0.29	-1.56 ***	0.40
Gender	-0.02	0.08	0.00	0.06	0.07	0.12	0.03	0.08
Marital Status	-0.05	0.16	0.14	0.21	0.20	0.15	-0.17	0.19
Region	0.32	0.29	-0.13	0.20	0.14	0.15	0.27	0.26
N	1,604		1,775		1,992		1,821	

***p<.001 **p<.01 *p<.05

Table 7-5 presents results using the Fairlie Decomposition method to estimate the extent to which change across Hispanic cohorts in the overall rate of diabetes prevalence can be

accounted for by change in the education composition of Hispanics, controlling of changes in population composition (gender marital status, region). In order to measure the full effect of educational composition, the intervening variables are not included in the analysis discussed here. In addition, the additional analysis that included the intervening variables of family income, health insurance, and BMI are not reported here, because they produce only marginal changes in estimates of the education composition effect.

Table 7-5 shows the effect on diabetes prevalence of changes for the education composition and of changes for the composition of cohorts with regard to the control variables. The first decomposition model in Table 7-5 analyzes the contributions of education composition change and change in population composition (i.e., the control variables) to the 1.51 percentage point increase for the diabetes prevalence that occurred across the 1935-39 and 1940-44 Hispanic cohorts. The effects are not significant ($p < .05$) for changes in the education, gender, marital, and region compositions. Among these variables, education composition has the largest effect and it is negative, although the effect is not statistically significant ($p < .05$).

The second model in Table 7-5 analyzes the contribution of education composition change, controlling for the composition change in the control variables, to the 5.12 percentage point increase for diabetes prevalence between the 1940-44 and 1945-49 Hispanic cohorts. The effects are not significant ($p < .05$) for changes in the education, gender, marital, and region compositions. Among these variables, education composition has a slightly larger effect and it is negative, although the effect is not statistically significant ($p < .05$).

Across the two most recent cohorts (1945-49 and 1950-54), the diabetes rate increased by a small 0.65 percentage points. The effects are highly significant ($p < .001$) for changes in the education composition, and not significant ($p < .05$) for changes in the gender, marital, and region

compositions. The results in Table 7-5 indicate that, controlling for population composition change, if educational attainments had not increased across the two Hispanic cohorts, then the overall diabetes rate would have increased by an additional 1.20 percentage points (from 0.65 percent to 1.85 percent). In other words, the increase would have been nearly triple the actual increase.

The final model in Table 7-5 presents variable contributions to diabetes change across all four Hispanic cohorts, that is, comparing the rate for 1950-54 cohort to the rate for 1935-39 cohort. Overall, the diabetes rate increased by 7.28 percentage points from 18.91 percent for the earliest-born cohort to 26.19 percent for the most recent cohort. If the education composition of the latest Hispanic cohort had remained unchanged compared to the earliest-born Hispanic cohort, diabetes prevalence would have been 1.56 percentage points higher, controlling for population composition. In other words, the increase in diabetes prevalence would have been nearly one-fourth greater (24.0 percent greater) in the 1950-54 Hispanic cohort, increasing by 8.84 percentage points instead of 7.13 percentage points, and an additional 96,703 Hispanics ages 60-64 in the 1950-54 cohort would have diabetes.

The gender, marital, and region composition explain none of the changes in the diabetes prevalence across the models. The coefficients are not statistically significant in all four models ($p < .05$) and null.

6) Blacks Only

Table 7-6: Decomposition of Cohort Disparities in Diabetes Prevalence among Blacks

	1935-39 to 1940-44		1940-44 to 1945-49		1945-49 to 1950-54		1935-39 to 1950-54	
Rates	24.16 to 23.86		23.86 to 25.99		25.99 to 24.94		24.16 to 24.94	
Percentage Point Change	-0.30		2.13		-1.05		0.78	
Variables	Effect	S.E.	Effect	S.E.	Effect	S.E.	Effect	S.E.
Education Level	-0.28	0.32	-0.70 *	0.32	-0.50 *	0.22	-0.07	0.72
Gender	0.06	0.07	-0.07	0.08	0.08	0.08	0.00	0.08
Marital Status	-0.01	0.15	0.15	0.25	-0.13	0.18	0.00	0.35
Region	0.20	0.18	-0.03	0.14	0.08	0.09	-0.17	0.16
N	1,842		2,276		2,709		2,275	

***p<.001 **p<.01 *p<.05

Table 7-6 presents results using the Fairlie Decomposition method to estimate the extent to which change across black cohorts in the overall rate of diabetes prevalence can be accounted for by change in the education composition of blacks, controlling of changes in population composition (gender marital status, region). In order to measure the full effect of educational composition, the intervening variables are not included in the analysis discussed here. In addition, the additional analysis that included the intervening variables of family income, health insurance, and BMI are not reported here, because they produce only marginal changes in estimates of the education composition effect.

Table 7-6 shows the effect on diabetes prevalence of changes for the education composition and of changes for the composition of cohorts with regard to the control variables. The first decomposition model in Table 7-6 analyzes the contributions of education composition change and change in population composition (i.e., the control variables) to the 0.30 percentage point decrease for the diabetes prevalence that occurred across the 1935-39 and 1940-44 black cohorts. The effects are not significant (p<.05) for changes in the education, gender, marital, and region compositions. Among these variables, education composition has the largest effect and it is negative, although the effect is not statistically significant (p<.05).

The second model in Table 7-6 analyzes the contribution of education composition change, controlling for the composition change in the control variables, to the 2.13 percentage point increase for diabetes prevalence between the 1940-44 and 1945-49 black cohorts. The effects are slightly significant ($p < .05$) for changes in the education composition, and not significant ($p < .05$) for changes in the gender, marital, and region compositions. The results in Table 7-6 indicate that, controlling for population composition change, if educational attainments had not increased across the two black cohorts, then the overall diabetes rate would have increased by an additional 0.70 percentage points (from 2.13 percent to 2.83 percent). In other words, the increase would have been one-third larger than the actual increase and an additional 47,972 blacks ages 60-64 in the 1945-49 cohort would have diabetes.

Across the two most recent cohorts (1945-49 and 1950-54), the diabetes rate declined by a 1.05 percentage points. The effects are slightly significant ($p < .05$) for changes in the education composition, and not significant ($p < .05$) for changes in the gender, marital, and region compositions. The results in Table 7-6 indicate that, controlling for population composition change, if educational attainments had not increased across the two black cohorts, then the overall diabetes rate would have declined by 0.55 percentage points instead of 1.05 percentage points. In other words, the decline would have been about one-half as large as the actual decline, and an additional 41,565 blacks ages 60-64 in the 1950-54 cohort would have diabetes.

The final model in Table 7-6 presents variable contributions to diabetes change across all four black cohorts, that is, comparing the rate for 1950-54 cohort to the rate for 1935-39 cohort. Overall, the diabetes rate increased by a small 0.78 percentage points from 24.16 to 24.94 percent for the earliest-born cohort to 26.19 percent for the most recent cohort. The effects are not significant ($p < .05$) for changes in the education, gender, marital, and region compositions.

Among these variables, education composition has a negative effect, although the effect is not statistically significant ($p < .05$).

The gender, marital, and region composition explain none of the changes in the diabetes prevalence across the models. The coefficients are not statistically significant in all four models ($p < .05$) and null.

7) *Education Composition Effect on Diabetes Prevalence: Whites vs. Hispanics vs. Blacks*

The diabetes rate for the 1935-39 cohort was greatest for blacks (24.16 percent), followed by Hispanics (18.91 percent) and whites (10.94 percent). The increase was greater for Hispanics than for whites and blacks across the 1935-39 to 1940-44 cohorts (1.59 percentage points vs. 0.98 and a for blacks a decline of -0.30 percentage points), and this trend continued between the 1940-44 and 1944-49 cohorts for Hispanics, whites, and blacks who experienced increases of 5.12, 2.51, and 2.13 percentage points, respectively, and between the 1945-49 and 1950-54 cohorts who experienced an increase of 0.65, and declines of 0.88, and 1.05 percentage points, respectively. Overall, across the four cohorts, Hispanics experienced the greatest increase in the diabetes rate compared to whites and especially compared to blacks, at 7.28 vs. 2.61 and 0.78 percentage points, respectively.

Due to the especially large increase for Hispanics and the especially small increase for blacks, the direction of the gap in diabetes rates reversed, and the diabetes rate for Hispanics surpassed the rate for blacks in the most recent cohort (1950-54) at 26.19 percent versus 24.94 percent. Meanwhile, because whites experienced a smaller increase than Hispanics, the gap between the two expanded from 7.97 percentage points for the earliest cohort to 12.64 percentage points for the most recent cohort, as the diabetes rates for the most recent cohort were 13.55 percent and 26.19 percent, respectively, for whites and Hispanics. In contrast, because

whites experienced a larger diabetes rate increase than blacks, the gap narrowed somewhat from 13.22 percentage points for the earliest cohort to 11.39 percentage points for the most recent cohort, as the diabetes rates for the most recent cohort were 13.55 percent and 24.94 percent, respectively, for whites and blacks

The decomposition models indicate that the educational composition effect for whites was greater than Hispanics and black across each successive set of cohorts with one exception. In the first set of models (1935-39 vs. 1940-44), the compositional effect of increasing education reduced the increase in the diabetes rate by 0.74 percentage points for whites, 0.16 percentage points for Hispanics, and 0.28 percentage points for blacks. Put another way, if educational attainment had remained unchanged between the 1935-39 and 1940-44 cohorts, then the increase in the diabetes rate for whites would have been three-fourths greater (1.72 percentage points instead of 0.98 percentage points), one-tenth greater for Hispanics (1.67 percentage points instead of 1.51), and reduced to near zero for blacks (-0.02 percentage points instead of -0.30). The compositional effect of education on diabetes is statistically significant only for whites.

In the second set of models (1940-44 vs. 1945-49), the compositional effect of increasing educational attainments reduced the increase in the diabetes rate by 0.83 percentage points for whites, 0.19 percentage points for Hispanics, and 0.70 percentage points for blacks. Thus, if the increase in educational attainments had not occurred, the diabetes rate for whites would have increased by 3.34 percentage points instead of 2.51 percentage points, the rate for Hispanics would have increased by 5.31 percentage points instead of 5.12 percentage points, and the rate would have increased by 2.83 percentage points instead of 2.13. The compositional effect of education on diabetes is strongly statistically significant ($p < .001$) for whites, slightly statistically significant for blacks ($p < .05$), and not statistically significant ($p < .05$) for Hispanics.

In the third set of models (1945-49 vs.1950-54), the effect of compositional change in education was greatest for Hispanics, at 1.20 percentage points, followed by whites and blacks with effects sizes of 0.60 and 0.50 percentage points, respectively, and all are statistically significant. In contrast to the changes across 1940-44 and 1945-49 cohorts, actual diabetes rates were lower for the 1950-54 white and black cohorts than for the 1945-1949 white and black cohorts, and greater for the 1950-54 Hispanic cohort. But if educational attainments had remained unchanged across the 1945-49 and 1959-54 cohorts, the negative change in the diabetes rate for whites would have nearly been close to zero, the positive change in the diabetes rate for Hispanics would have nearly tripled, and the negative change in the diabetes rate for blacks would have been reduced by one-half. For whites the decline would have been -0.28 percentage points instead of -0.88, the increase for Hispanics would have been 1.85 percentage points instead of 0.65, and the decrease for blacks would have been -0.55 percentage points instead of -1.05.

Overall, race-ethnic differences in the effect of education composition change on diabetes rate change between the earliest-born cohort (1935-39) and latest-born cohort (1950-54) is largest for whites (-1.86 percentage points), followed by Hispanics (-1.56 percentage points), and nearly zero for blacks (-0.07 percentage points). Among whites, the enormous educational expansion accounts for the comparatively large education composition effect. Nearly as large is the educational composition effect for Hispanics, which is accounted for by their large educational expansion in conjunction with the large increase in the educational gradient experienced by the most recent cohort, compared to the earliest cohort. The near zero effect for blacks, however, occurred despite their enormous educational expansion, because the

educational gradient for blacks is small compared to the magnitude of the educational gradient for whites and Hispanics.

The effect of changes in the gender composition for diabetes change is positive for whites in three of the four models, leading to increased diabetes prevalence, and statistically significant for whites in all four models. Meanwhile, the effect of changes in gender composition in all four models are not statistically significant for Hispanics or blacks. Overall, comparing the earliest- and latest-born cohorts, the changing gender composition explained 0.07 percentage points of the total 2.61 percentage point increase in diabetes prevalence for whites, and 0.03 percentage points of the total 7.28 percentage point increase in diabetes prevalence for Hispanics (not statistically significant), and less than one-tenth of a percentage point in the increase in diabetes prevalence for blacks (not statistically significant).

The change in diabetes prevalence explained by marital composition is not significant for Hispanics and black, but is positive and small in the second model for whites, and positive and strongly significant in the last two models for whites. Overall, comparing the earliest-born and latest-born cohorts, the changing marital composition explained only 0.52 percentage points for whites (significant, $p < .001$) in the increase of diabetes prevalence, 0.17 percentage points for Hispanics (not significant, $p < .05$), and less than a tenth of a percentage point for blacks (not significant, $p < .05$). Finally, regional composition change explains little to none of the changes in diabetes prevalence across the models for whites (significant in only 1 out of 4 models), and no change in diabetes prevalence for Hispanics and blacks in all four models. Of the population composition variables, region is the least significant and least influential, with no consistent pattern across cohorts for any race-ethnicity.

Summary of Results

The first important finding presented in this chapter is that higher educational attainment is strongly and negatively associated with diabetes prevalence in all four white cohorts, negatively associated in the three later-born Hispanic cohorts, and slightly or not associated for blacks (Tables 7-1, 7-3, and 7-3). For example, in various models controlling for population composition change, whites graduating from high school and whites with some college have odds of being diagnosed with diabetes that are 18-48 percent lower than for white high school dropouts, and whites with a bachelor's degree are even less likely to be diagnosed with diabetes, with odds that are 57-70 percent lower than for white high school dropouts. For Hispanics in the three most recent cohorts with a bachelor's degree, the odds are similar to whites with a college degree, that is, these Hispanics with a college degree have odds of being diagnosed with diabetes that are 56-71 lower than for Hispanic high school dropouts. However, none of the odds ratios are statistically significant for Hispanics in the earliest cohort, and none of the odds rates are statistically significant for Hispanic high school graduates or Hispanics with some college for the 1940-44 and 1945-49 cohorts. Among blacks, controlling for population composition change, only 2 of the 12 odds ratios are statistically significant.

The second important finding is that the changing educational composition, that is, the educational expansion, has a negative effect on the growth in diabetes prevalence for later-born cohorts for whites and for Hispanics, but not for blacks. If educational expansions had not occurred, that is, if education had remained constant at the levels for the 1935-39 cohort, the increase in the diabetes prevalence rate would have been nearly three-fourths greater for the 1950-54 white cohort (4.47 instead of 2.61 percentage points, for an additional increase of 1.86 percentage points), and it would have been one-fifth larger for the 1950-54 Hispanic cohort 8.84

instead of 7.28 percentage points, for an additional increase of 1.56 percentage points). The effect for blacks, however, is not statistically significant and the effects size is only 0.07 percentage points.

Thus, educational expansion acted to limit diabetes growth for both whites and Hispanics. The magnitude of the educational gradient did not change across cohorts for whites, although it did increase for the most recent Hispanic cohort. For blacks the non-significant educational gradients did not change across cohorts, and the estimate direction of the effects was positive, not negative, for 5 of the 12 odds ratios. Since the educational gradients for blacks were small to negligible for most black education groups in all cohorts, the enormous educational expansion for blacks did not have an effect on diabetes growth for blacks.

These new findings move beyond the extant literature by the estimating the magnitude and assessing the extent to which changes have occurred in the educational gradient for diabetes across four successive birth-cohorts, comparing results for whites, Hispanics, and blacks, and by estimating the extent to which the educational expansion across these cohorts acted to limit increases in diabetes prevalence, again comparing results for whites, Hispanics, and blacks.

Chapter 8

Discussion of Findings and Conclusion

Introduction

Chapter 2 discussed the historic increases in education and the possible link to change in diabetes prevalence across cohorts, either through changes in the educational gradient for diabetes or through changes in the educational composition of the population. Chapter 3 and 4 discussed the data, methods, and univariate and bivariate analysis of the four 5-year cohorts born between 1935 and 1954 using 20 survey years of the NHIS. Chapter 5 presented results from logistic regression models estimating educational gradients for the four cohorts, as well as Fairlie Decomposition models estimating the extent to which changes in the educational composition are linked to changes in diabetes prevalence across cohorts. Chapter 6 presented gender differences in the results from logistic regression and Fairlie Decomposition models across cohorts. Lastly, Chapter 7 presented race-ethnicity differences in results from logistic regression and Fairlie Decomposition models across cohorts, with three-way comparisons.

This chapter begins by summarizing the results from logistic regression and Fairlie Decomposition for the overall sample, and substantive difference by gender and by race-ethnicity across four cohorts. The chapter then presents levels of education expansion and diabetes growth in the overall sample and subpopulations between the earliest- and latest-born cohorts. The chapter also examines gender and race-ethnicity gaps in education and diabetes expansion for males and females, and for whites, Hispanics, and blacks. Lastly, the chapter discusses the implications of the results in the number of persons positively affected by educational expansion, the unequal expansion of education and diabetes by subpopulations, and possible effect of education expansion on future cohorts.

Logistic Regression

Overall (Chapter 5), higher educational attainment is strongly and negatively associated with diabetes prevalence in all four cohorts. For example, persons with a bachelor's degree have odds of being diagnosed with diabetes that are 42-44 percent lower than the odds for high school graduates, controlling for population composition. Even after controlling for mediating variables, the odds ratios for college graduates are 26-32 percent lower than high school graduates. Thus, the educational gradient is large, and most of the educational gradient effect does not act through the intervening variables. Additionally, the magnitude of the relationship does not change across the four cohorts.

Higher educational attainment is also strongly and negatively associated with diabetes prevalence for both genders, and the magnitude is especially large for females (Chapter 6). For example, males with a bachelor's degree have odds of being diagnosed with diabetes that are 47-56 percent lower than the odds for males who are high school dropouts, controlling for population composition. Even more striking is that, the odds of being diagnosed with diabetes among females with a bachelor's degree are 65-75 percent less than for females who are high school dropouts, controlling for population composition. Although the introduction of the mediating variables into the models reduces the size of these estimates, even after controlling for the mediating variables, persons with a bachelor's degree have much lower odds of being diagnosed with diabetes than persons who are high school dropouts, 29-41 percent lower for males and 48-66 percent for females. Additionally, the magnitude of the relationship between educational attainment and diabetes does not change across cohorts for either gender.

With regards to race-ethnicity (Chapter 7), higher educational attainment is strongly associated with diabetes prevalence in all four white cohorts, negatively associated in the three

later-born Hispanic cohorts, and slightly or not associated for blacks. For example, whites graduating from high school and whites with some college have odds of being diagnosed with diabetes that are 18-48 percent lower than for white high school dropouts for the four cohorts, controlling for population composition. In the same models, the odds ratios for whites with a bachelor's degree are 57-70 percent lower than high school dropouts. Meanwhile, the educational gradient increases for later-born cohorts. The odds for Hispanic college graduates are similar to their white counterparts, meaning Hispanics with a college degree have odds of being diagnosed with diabetes that are 56-71 lower than for Hispanic high school dropouts. However, none of the odds ratios are statistically significant for Hispanics in the earliest cohort, and none of the odds rates are statistically significant for Hispanic high school graduates or Hispanics with some college for the 1940-44 and 1945-49 cohorts. Among blacks, controlling for population composition change, only 2 of the 12 odds ratios are statistically significant. The magnitude of the educational gradient did not change across cohort for whites, but did increase for the most recent Hispanic cohort. For blacks, the non-significant educational gradients did not change across cohort, and the direction of the effects was positive, not negative, for 5 of the 12 odds ratios.

Decomposition

Overall (Chapter 5), educational expansion has a negative effect on the growth in diabetes prevalence for later-born cohorts. For example, the Farilie Decomposition model of change between the earliest and most recent cohort indicate that if education levels had remained constant at the levels for the 1935-39 cohort, the increase in the diabetes prevalence rate would have been two-fifths greater in the 1950-54 cohort (4.98 instead of 3.52 percentage points). Thus, educational expansion acted to limit diabetes growth. The educational compositional effect on

diabetes change was substantially and statistically significant from one cohort to the next across all cohorts.

Educational expansion also has a negative effect on the growth in diabetes for later-born cohorts for both genders (Chapter 6). For example, if education had remained constant at the levels for the 1935-39 cohort, the increase in the diabetes prevalence rate would have been one-third greater for the 1950-54 male cohort (4.43 instead of 3.32 percentage points), and it would have been three-fifths greater for females (5.71 instead of 3.80 percentage points). Thus, educational expansion acted to limit diabetes growth for both males and females. The compositional effect on diabetes change of the educational expansion was substantial and statistically significant from one cohort to the next and across all cohorts for both males and females.

With regards to race-ethnicity (Chapter 7), educational expansion has a negative effect on the growth in diabetes prevalence for later-born white and Hispanic cohorts, but not for black cohorts. For example, if education had remained constant at the levels for the 1935-39 cohort, the increase in the diabetes prevalence rate would have been nearly three-fourths greater for the 1950-54 white cohort (4.47 instead of 2.61 percentage points, for an additional increase of 1.86 percentage points), and it would have been one-fifth larger for the 1950-54 Hispanic cohort (8.84 instead of 7.28 percentage points, for an additional increase of 1.56 percentage points). The effect for blacks, however, is not statistically significant and the effects size is only 0.07 percentage points. Thus, educational expansion acted to limit diabetes growth for both whites and Hispanics. Since the educational gradients for blacks were small to negligible for most black education groups in all cohorts, the enormous educational expansion for blacks did not have an effect on diabetes growth for blacks.

Expansion of Education and Diabetes

**TABLE8-1: Percentage Point Increase in Education and Diabetes Across Cohorts
1935-1939 versus 1950-54**

	BA/BS			Diabetes Rate		
	1935-39	1950-54	Change	1935-39	1950-54	Change
1) Overall Sample	21.46%	33.87%	12.41%	12.64%	16.16%	3.52%
2) Gender						
Males	27.28%	36.64%	9.36%	13.88%	17.20%	3.32%
Females	15.65%	31.26%	15.61%	11.38%	15.18%	3.80%
3) Race-Ethnicity						
Whites	22.80%	37.26%	14.46%	10.94%	13.55%	2.61%
Hispanics	11.85%	14.82%	2.97%	18.91%	26.19%	7.29%
Blacks	10.61%	20.70%	10.09%	24.16%	24.94%	0.78%

Table 8-1 presents the increases in higher education and the diabetes rate between the latest- and the earliest-born cohort. The latest-born cohort (1950-54) is more highly educated and has greater diabetes prevalence than the earliest born cohort (1935-39). In the full sample, the percentage with a bachelor’s degree is 12.41 percent greater for the 1950-54 cohort compared to the 1935-39 cohort, and the diabetes rate is 3.52 percent greater for the 1950-54 cohort. By gender, females experience greater expansion in both higher education and diabetes (15.61 and 3.80 percent, respectively) compared to males (9.36 and 3.32 percent, respectively). By race-ethnicity, whites experience the greatest higher educational expansion (14.46 percent), followed by blacks (10.09 percent), and Hispanics (2.97 percent). Meanwhile, Hispanics experience the greater diabetes expansion (7.29 percent), followed by whites (2.61 percent), and blacks (0.78 percent)

Gender and Race-Ethnicity Gaps in Education and Diabetes

TABLE 8-2: Gender Gaps in Education and Diabetes

	1935-39	1940-44	1945-49	1950-54
Male/Female Diff BA/BS+	11.63%	8.99%	9.01%	5.38%
Male/Female Diff Diabetes	2.49%	2.96%	1.95%	2.02%

Table 8-2 presents the changing gender gap (male versus female) in higher education and diabetes by cohort. The gender gap in higher education is reduced by over one-half across cohort, from 11.63 percent for the 1935-39 cohort to 5.38 percent for the 1950-54 cohort. The gender gap in education for the latest-born examined cohort indicates convergence, but previous research indicates that later-born, unexamined, cohorts experience a shift in the gender gap favoring females (Buchmann and DiPrete 2005). The current analysis details the beginning of educational expansion differentiated by gender, with substantial changes still to come. Meanwhile, the gender gap in diabetes is reduced by approximately two-fifths, from 2.49 percent for the 1935-39 cohort to 2.02 percent for the 1950-54 cohort. The results in diabetes prevalence differences by gender are consistent with recent research. While research indicates a female disadvantage in diabetes in the late 1970s, diabetes growth was greater for males, resulting in greater diabetes prevalence among males than females in subsequent time periods (Menke et al. 2014).

TABLE 8-3: Race-Ethnicity Gaps in Education and Diabetes

		1935-39	1940-44	1945-49	1950-54
Diff in BA/BS+	White/Hispanic	10.95%	16.25%	22.46%	22.44%
	White/Black	12.19%	13.65%	17.69%	16.56%
	Hispanic/Black	1.24%	-2.60%	-4.77%	-5.88%
Diff in Diabetes	White/Hispanic	-7.97%	-8.50%	-11.11%	-12.65%
	White/Black	-13.22%	-11.94%	-11.56%	-11.40%
	Hispanic/Black	-5.25%	-3.44%	-0.46%	1.25%

Table 8-3 presents the changing race-ethnicity gaps in higher education and diabetes by cohort. These results support previous literature in the widening gap in higher education between whites and racial minorities (U.S. Census Bureau 2018). The white/Hispanic gap in higher education doubles from the 1935-39 cohort (10.95) to the 1950-54 cohort (22.44 percent). The white/black gap expands by one-third, from 12.19 percent for the 1935-39 cohort to 16.65 percent for the 1950-54 cohort. Meanwhile, Hispanics in the earliest-born cohort are slightly

more likely to have a bachelor's degree than blacks (Hispanic/black gap at 1.24 percent), but blacks are more likely to have a bachelor's degree in the three later-born cohort, with the Hispanic/black the gap reversing to -5.88 percent in the 1950-54 cohort. Persons in the earliest-born cohort (1935-39) were in high school or nearly college age when the Supreme Court decision of *Brown v. Board of Education* (1954) was mandated. Blacks in the earliest-born cohort did not benefit from the constitutional law, as prior research has argued the American educational system remained segregated well into the 1960s. Hispanics also experienced large inequality in schooling compared to whites (Contreras 2004). Additionally, the low socioeconomic background of many Hispanics, with the exceptions of Cuban immigrants in the 1960s, has resulted in lower educational attainment compared to whites. Chapter 2 found higher educational expansion for all three racial-ethnic groups, but the gains have been more substantial for whites than Hispanics and blacks. The racial-ethnic convergence in educational attainment is found in high school graduation rates (U.S. Census 2018).

The white/Hispanic gap in diabetes increases by nearly three-fifths, from -7.97 percent for the 1935-39 cohort to -12.65 percent for the 1950-54 cohort. Meanwhile, the white/black gap in diabetes slightly decreases from -13.22 percent in the 1935-39 cohort to -11.40 in the 1950-54 cohort, with blacks more likely to have diabetes than whites. The Hispanic/black gap in diabetes changes direction, with blacks more likely to have diabetes than Hispanics in the 1935-39 cohort (-5.25 percent), but less likely to have diabetes than Hispanics in the 1950-54 cohort (1.25 percent). These findings support previous research in racial-ethnic disparities in diabetes and health (Cockerham 2016; Brancati et al. 1996; Golden et al. 2016; Robbins et al. 2001, 2000; CDC 2003). There are various potential mechanisms explaining this disparity in past research,

such as environmental, socioeconomic, behavioral, physiological, and genetic factors (Sims et al. 2011; Signorello et al. 2007; Carter et al. 1996; Harris 1996; Abate and Chandalia 2003).

Implications

Diabetes forecast models (i.e., Boyle et al. 2010), do not include the changing educational landscape. Educational expansion was most prominent among the Baby Boomer generation, but Chapter 2 indicates continued expansion for later-born cohorts, especially for females (Barr and Lee 2013). Final diabetes prevalence rates for younger cohorts cannot yet be known, but the continued educational expansion of later-born baby boomers and successive cohorts will further limit diabetes growth, and current forecasts may overestimate future diabetes increase. It remains unclear, however, whether there will be a changing effect of education on health for later-born cohorts; either because of differential change among education groups in risk behaviors (Baker et al. 2011) or growing inequality and greater significance in health mechanisms (Hayward et al. 2015).

The current analysis supports previous research on gender differences in the association between educational attainment and diabetes (Borrell et al. 2006). The magnitude of the relationship is significant for females than males. While there is little research directly testing the mechanisms of gender disparities in diabetes, there is a plethora explaining gender disparities in other health outcomes. There are gender differences in mechanisms of biological and physiological development and aging, as well as differences in their health exposures, health behaviors, and psychosocial resources, both within and across socioeconomic groups (Denney et al. 2010; Hayward and Gorman 2004; Liu and Hummer 2008; Masters et al. 2012; Ross et al. 2012; Pudrovska and Aniskin 2013).

The results also support previous literature on racial-ethnic disparities in the relationship between educational attainment and diabetes. Namely, educational attainment is strongly and negatively associated for whites and Hispanics, but not for blacks. Previous research suggests blacks have different contextual experiences in the United States. One argument is the “minority poverty” hypothesis, suggesting blacks experience unique disadvantages in health and well-being due to extreme poverty and race (Willie 1979, 1989). Meanwhile, the “diminishing returns” hypothesis suggests that blacks do not experience the same returns to higher socioeconomic achievement as whites, and the greatest disparities are at the highest socioeconomic levels (Bowles and Gintis, 1976; Farley 1984).

The magnitude of inequalities is precisely estimated for four successive five-year cohorts in Chapters 5-7 and has practical implications for research, health care planning and educational policy. Analysis of these four cohorts by gender and by race-ethnicity confirms educational attainment is negatively associated with diabetes for males and females, as well as whites and Hispanics, but not linked to diabetes prevalence for blacks. No prior research has explored the mechanisms of how educational attainment prevents diabetes by gender or for specific race-ethnic groups. The new research finds substantial disparities in effects by gender and for whites compared to Hispanics, but no effect for blacks. Thus, specific policies and strategies may need to be implemented in order to address the gender and racial-ethnic disparities in education and diabetes growth.

Conclusion

Previous research has presented cross-sectional reports on the strong linear and negative relationship between educational attainment, the risk and prevalence of type 2 diabetes. The previous literature, however, has never conducted a longitudinal analysis of the simultaneous

changing situation for education and diabetes in the United States. Despite the large increases that occurred in both educational attainments and diabetes, overall, during the 20th century, past research has not studied how the magnitude of the relationship between education and diabetes may or may not have changed across successive cohorts. In addition, past research has not examined the effect of the changing educational composition on diabetes prevalence growth across cohorts. Finally, the effect of changing gender and race-ethnic disparities across cohorts in the magnitude of the relationship between educational attainments and diabetes, and the effect of changing gender and race-ethnic disparities across cohorts in the educational composition of the U.S. population on diabetes have not been systematically studied.

Educational expansion acted to limit diabetes growth for the U.S. population ages 60-64 born between 1935 and 1954, as well as for subpopulations by gender and by race-ethnicity, although not for blacks. These new findings move beyond the extant literature by estimating the magnitude and assessing the extent to which changes have occurred in the educational gradient for diabetes across four successive birth-cohorts, comparing results by gender and by three race-ethnicity groups. These new findings also move beyond the literature by estimating the extent to which the educational expansion across these cohorts acted to limit increases in diabetes prevalence, again comparing results by gender and by race-ethnicity.

Overall, the Fairlie Decomposition models indicate that without the educational expansion that limited diabetes growth, there would be approximately an additional million persons ages 60-64 born in 1950-54 diagnosed with diabetes. Within this additional million, 363,622 would be male and 668,061 would be female. By race-ethnicity, an additional 942,540 whites, 96,703 Hispanics, and 47,972 blacks ages 60-64 in the 1945-49 cohort would have diabetes. The already substantial social and financial costs of diabetes would be even more

severe. Using annual medical expenditure per person with diabetes in 2012 (\$13,700, ADA 2013), approximately \$13.7 billion in medical expenditures were saved due to educational expansion.

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APPENDIX: Additional Analyses

**Table A: Weighted Logit Regression for Diabetes among Persons Aged 60-64
Interactions between Education and Birth Cohort**

	O. R.
Less Than High School	1.33 **
High School Only (omitted)	
Some College	0.88
Bachelor's Degree or Higher	0.57 ***
1935-39 (omitted)	
1940-44	1.09
1945-49	1.45 ***
1950-54	1.45 ***
LTH x 1940-44	0.99
LTH x 1945-49	0.98
LTH x 1950-54	1.05
SC x 1940-44	1.25
SC x 1945-49	1.08
SC x 1950-54	0.98
BA x 1940-44	1.03
BA x 1945-49	0.99
BA x 1950-54	0.96
Female	0.78 ***
Non-Hispanic White (omitted)	
Non-Hispanic Black	1.84 ***
Hispanic	1.70 ***
Asian/Pacific Islander	1.37 *
Native American	1.60 *
Other	1.48
Married (omitted)	
Single/Never Married	1.15
Cohabiting	0.91
Separated	1.43 ***
Divorced	1.07
Widowed	1.09
South (omitted)	
Northeast	0.81 ***
Midwest	0.99
West	0.82 ***
Constant	0.16 ***
<hr/>	
N	34,101
Pseudo R-squared	
***p<.001 **p<.01 *p<.05	

**Table B: Weighted Logit Regression for Diabetes among Persons Aged 60-64
Interactions between Education and Gender, by Cohort**

	1935-39	1940-44	1945-49	1950-54
	O. R.	O. R.	O. R.	O. R.
Less Than High School (omitted)				
High School Only	0.78	0.82	0.79	0.73
Some College	0.69 *	1.05	0.77	0.68 *
Bachelor's Degree or Higher	0.55 ***	0.56 ***	0.51 ***	0.46 ***
Female	0.88	0.90	0.95	0.93
HSONLY x Female	0.89	0.88	0.90	0.98
SC x Female	0.90	0.61 *	0.86	0.87
BA x Female	0.42 **	0.59 *	0.64 *	0.75
Non-Hispanic White (omitted)				
Non-Hispanic Black	2.39 ***	1.98 ***	1.70 ***	1.70 ***
Hispanic	1.64 ***	1.62 ***	1.64 ***	1.88 ***
Asian/Pacific Islander	1.04	1.81 *	1.18	1.45
Native American	1.64	1.56	1.48	1.87
Other	0.81	1.63	1.29	2.13
Married (omitted)				
Single/Never Married	0.66 *	1.12	1.07	1.41 **
Cohabiting	0.90	0.97	0.66	1.18
Separated	0.49 **	1.78 *	1.88 ***	1.35
Divorced	1.01	1.15	1.09	1.06
Widowed	0.96	1.34 *	1.06	0.95
South (omitted)				
Northeast	0.91	0.76 *	0.76 **	0.83
Midwest	0.85	1.08	1.01	0.97
West	0.89	0.93	0.87	0.67 ***
Constant	0.16 ***	0.16 ***	0.23 ***	0.22 ***
N	7,019	7,540	9,311	10,231
Pseudo R-squared				

***p<.001 **p<.01 *p<.05

**Table C: Weighted Logit Regression for Diabetes among Persons Aged 60-64
Interactions between Education and Race-Ethnicity, by Cohort**

	1935-39	1940-44	1945-49	1950-54
	O. R.	O. R.	O. R.	O. R.
Less Than High School (omitted)				
High School Only	0.60 ***	0.80	0.61 ***	0.69 *
Some College	0.52 ***	0.81	0.62 ***	0.58 ***
Bachelor's Degree or Higher	0.31 ***	0.42 ***	0.37 ***	0.34 ***
Non-Hispanic White (omitted)				
Non-Hispanic Black	1.41	2.06 ***	1.23	0.99
Hispanic	1.13	1.51 *	1.24	2.01 ***
Asian/Pacific Islander	0.70	1.43	0.62	0.72
Native American	0.76	2.51	0.70	2.92
Other	14.50	4.22	4.97	2.20
HS ONLY x Black	1.93 *	0.73	1.65	1.56
HS ONLY x Hispanic	1.76	0.96	1.89 *	0.87
HS ONLY x Asian/Pacific Islander	1.62	1.44	2.56	1.45
HS ONLY x Native American	2.36	0.45	2.80	0.45
HS ONLY x Other	Dropped	Dropped	0.41	Dropped
SC x Black	2.28 *	1.05	1.27	2.10 **
SC x Hispanic	2.03 *	1.67	1.43	0.56
SC x Asian/Pacific Islander	1.01	0.48	2.16	1.95
SC x Native American	0.79	0.53	1.82	0.27
SC x Other	Dropped	0.83	0.08	4.32
BA x Black	3.88 ***	1.40	1.74	2.25 **
BA x Hispanic	2.03	0.71	0.88	1.26
BA x Asian/Pacific Islander	2.18	1.93	1.75	2.90
BA x Native American	Dropped	Dropped	2.81	2.04
BA x Other	Dropped	Dropped	Dropped	0.72
Female	0.75 ***	0.70 ***	0.80 ***	0.82 **
Married (omitted)				
Single/Never Married	0.67 *	1.14	1.07	1.44 **
Cohabiting	0.89	0.97	0.65	1.21
Separated	0.52 *	1.83 **	1.92 ***	1.37
Divorced	1.00	1.13	1.08	1.06
Widowed	0.98	1.35 *	1.08	0.98
South (omitted)				
Northeast	0.90	0.76 *	0.76 **	0.84
Midwest	0.84	1.08	1.01	0.97
West	0.91	0.92	0.87	0.67 ***
Constant	0.16 ***	0.18 ***	0.22 ***	0.24 ***
N	7,010	7,527	9,306	10,226
Pseudo R-squared				
***p<.001 **p<.01 *p<.05				

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