

University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Sociology Theses, Dissertations, & Student
Research

Sociology, Department of

8-2014

WHAT YOU DON'T KNOW CAN HURT YOU: EARLY LIFE COURSE RACIAL HEALTH DISPARITIES IN UNDIAGNOSED DIABETES

Anna C. Bellatorre

University of Nebraska-Lincoln, acbellatorre@gmail.com

Follow this and additional works at: <http://digitalcommons.unl.edu/sociologydiss>



Part of the [Medicine and Health Commons](#)

Bellatorre, Anna C., "WHAT YOU DON'T KNOW CAN HURT YOU: EARLY LIFE COURSE RACIAL HEALTH DISPARITIES IN UNDIAGNOSED DIABETES" (2014). *Sociology Theses, Dissertations, & Student Research*. 33.

<http://digitalcommons.unl.edu/sociologydiss/33>

This Article is brought to you for free and open access by the Sociology, Department of at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Sociology Theses, Dissertations, & Student Research by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

WHAT YOU DON'T KNOW CAN HURT YOU: EARLY LIFE COURSE RACIAL
HEALTH DISPARITIES IN UNDIAGNOSED DIABETES

by

Anna Christine Bellatorre

A DISSERTATION

Presented to the Faculty of
The Graduate College at the University of Nebraska
In Partial Fulfillment of Requirements
For the Degree of Doctor of Philosophy

Major: Sociology

Under the Supervision of Professor Bridget J. Goosby

Lincoln, Nebraska

August 2014

WHAT YOU DON'T KNOW CAN HURT YOU: EARLY LIFE COURSE RACIAL
HEALTH DISPARITIES IN UNDIAGNOSED DIABETES

Anna Christine Bellatorre, Ph.D.

University of Nebraska, 2014

Adviser: Bridget J. Goosby

This dissertation addresses several issues related to racial health disparities in undiagnosed diabetes in American young adults in a three-article format. The first chapter examines rates of diabetes severity across age-matched samples of young adults from two large nationally representative studies. Although the purpose of this study was to explore the impact of nonresponse on prevalence estimates, I find that the prevalence discrepancies have less to do with which respondents are missing blood samples and more to do with the samples coming from initial samples that are not equivalent.

The second chapter uses an adaptation of the Stress Process Model to identify the effects of racial minority status, perceived discrimination, mastery, and risky coping strategies on diabetes severity in a race-stratified young adult sample. Data from the National Longitudinal Study of Adolescent Health were used to analyze diabetes risk severity using multinomial logistic regression analysis. Large disparities in diabetes risk severity were found by race, particularly for undiagnosed diabetes. Multivariate results show complex relationships between experiencing discrimination and diabetes risk severity by race, which suggest that discrimination effects diabetes risk severity differently for blacks and whites.

The final study examines the impact of help seeking and diagnosis allocation with diabetes diagnosis disparities. Andersen's Behavioral Model of Health Services Use (1995) is used to model diabetes diagnostic disparities among young adults with diabetes. Tests of Andersen's model using data from the National Longitudinal Study of Adolescent Health reveal no difference in help seeking across race/ethnic groups. Although all race/ethnic groups were equally likely to seek care, large diagnostic disparities persist for blacks. As a result, young adult black diabetics are significantly less likely to receive a diagnosis for diabetes even when they sought care in the previous three months.

Taken together, this dissertation reveals that racial health disparities in diabetes diagnoses are complex. Estimates of the prevalence, predictors, and pathways to diagnosis differ by race in meaningful and previously unexplored ways. This research serves to document this problem, provide foundational evidence of meaningful relationships, and shed light on the possible public health and policy implications associated with these disparities.

COPYRIGHT

© 2014, Anna C. Bellatorre

DEDICATION

To my parents, partner, professors, and mentors. I would not be here without you.

ACKNOWLEDGEMENTS

Writing a dissertation is not a solitary act. Although the work presented here is my own original research, it would not be possible without the guidance and support of several others. While I thank each of the members of my dissertation committee (Dr. Goosby, Dr. Olson, Dr. McQuillan, and Dr. Hibbing), I would like to offer special thanks to my adviser, Dr. Bridget Goosby. Bridget has helped me grow over the past five years enabling me to think broadly while cultivating a narrow focus. Bridget pushed me to succeed, provided support, and occasionally issued a reality check or two, which has shaped who I am as a scholar.

I came to the University of Nebraska-Lincoln with prior degrees from San Diego State University. The challenges and support I received at SDSU helped me to earn an Othmer Fellowship my first three years at the University of Nebraska-Lincoln. Later, with the challenges and support presented by my adviser, the Department of Sociology, the Department of Survey Research and Methodology, and my committee I was able to earn a Presidential Fellowship for my final year as a doctoral student. Now, as I finish my education and begin my postdoctoral fellowship at the National Institutes of Health, I want to acknowledge the path it took to get here.

Two of my biggest supporters at SDSU were Brian Finch and Jana Pershing. Sadly, Jana is not here to share this moment with, but I like to think that she is aware of the influence she has had on my life. Brian has continued to be one of my frequent co-authors since leaving San Diego State University. Both Brian and Bridget have helped me cultivate my peer and co-author networks. Without my advisers, both past and present, I

am sure I would not have the publication record I have, the colleague network I have amassed, nor the skill set I have achieved. I am eternally grateful for all your help.

I would also like to thank my loving partner, Stephen, and my family. Graduate school takes a lot of time, focus, and energy. All too often those closest to us are neglected in the pursuit of higher education. I hope that this dissertation is worthy of the time we have spent apart. Moreover, I hope my family can see that my dissertation is due in no small part to their sacrifices and support. I will forever love my “Nurse Mom” who came for my, many, surgeries, my dad for his daily check-ins, and Stephen for his constant love and support.

I have often said, “Graduate school is a team sport.” Although my struggle has been my own, my successes are shared with my department, professors, mentors, and graduate student peers. When any of us succeeds, we all rise together. I hope my dissertation can demonstrate to my peers that it is possible to finish if you dream big and persevere. Special thanks to Alian, Beth, Stacy, Megumi, Laura, and my officemates Harmoni, Deadric, and Scott, you were always there to lend an ear or talk through a complex model. Thank you!

DATA ACKNOWLEDGEMENT

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://non.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

CONTENTS

Contents

1	Introduction	1
2	Predicting diabetes severity comparing NHANES & Add Health: does the data used alter population inferences for young adult diabetes risk?	12
	2.1 Introduction	13
	2.2 Research Questions	16
	2.3 Literature Review	16
	2.4 Hypotheses	22
	2.5 Methods	22
	2.6 Results	27
	2.7 Discussion	32
	2.8 References	38
	2.9 Tables	42
3	Unequal exposure and unequal risk: race stratified diabetes risk severity in early adulthood	51
	3.1 Introduction	52
	3.2 Theoretical Framework and Literature Review	54
	3.3 Hypotheses	56
	3.3.1 Figure 3.1 Theoretical Stress Process Model for Diabetes Severity	56

3.3.2 Figure 3.2 Theoretical Stress Process Model for Race-Stratified Diabetes Severity	57
3.4 Methods	61
3.5 Results	67
3.5.1 Figure 3.3 Significant Interaction Effect for Perceived Discrimination by Race	69
3.5.2 Figure 3.4 Significant Interaction Effect for Mastery by Perceived Discrimination for NH Blacks	72
3.6 Discussion	72
3.7 References	80
3.8 Tables	86
3.9 Appendices	90
4 Testing the behavioral model of health services use: are disparities in diabetes diagnoses due to differences in help seeking or diagnosis allocation?	100
4.1 Introduction	101
4.2 Literature Review and Theoretical Model	103
4.2.1 Figure 4.1 Conceptual Map for the Behavioral Model of Health Services Use	107
4.2.2 Figure 4.2 Conceptual Map for the Behavioral Model of Health Services Use for Diabetes Diagnosis	109
4.3 Hypotheses	109
4.4 Methods	110

4.5 Results	116
4.6 Discussion	120
4.7 References	125
4.8 Tables	133
5 Discussion	137

CHAPTER 1

1.1 INTRODUCTION

The United States obesity epidemic is well documented in demographic, medical, and sociological literature. However, comparatively little attention is paid to the co-morbid conditions that can arise alongside obesity, like diabetes, in young adults who have completed the transition to adulthood, but have not yet reached midlife (James, Rigby and Leach 2004). Given the relationship between diabetes and obesity in the United States (Reilly and Kelly 2011), it is likely that the rise in diabetes will also enter into progressively younger age groups similar to the obesity trend. Although diabetes rates are increasing, it is less clear whether diabetes risk is being identified accordingly in young adults. Most national prevalence estimates for adults are aggregated for all adults over age 20 and adults over 65 using data from the National Health and Nutrition Examination Survey (NHANES), which may obscure variation across age groups (CDC 2011).

The national rates for combined diagnosed and undiagnosed diabetes for American adults over age 20 reveal clear disparities with a national diabetes rate of 11.3%, with a rate of only 10.2% for non-Hispanic whites compared to 18.7% for non-Hispanic blacks (American Diabetes Association Fact Sheet 2013; CDC 2011). If young adults have unique diabetes risk profiles, existing estimates may not capture this variation. Moreover, if undiagnosed diabetes is a hidden problem for young adults, it is important to appropriately identify the prevalence of the condition, predictors of risk, and impediments to diagnosis.

Although diabetes is increasing in the United States (Ciporen 2012), little research has examined how the social conditions of life in the United States may contribute to diabetes risk and morbidity and mortality risk across the life course. Part of the reason for this dearth of research could be the difficulty in tracking undiagnosed diabetes, which may be particularly difficult for young adults. The prevalence of undiagnosed diabetes is difficult to identify in young adults for a variety of reasons such as decreased perceptions of health risk for young adults (Van Osch, van den Hout, and Stiggelbout 2006; Vernon 1999; Walker et al. 2003), differential help seeking patterns (Guy & Gery 2010; Kullgren et al. 2012), and reduced survey participation for studies involving biological testing (Johnson et al. 2007).

Although challenges exist in assessing the prevalence of undiagnosed diabetes in young adults, the data that are available indicate that diabetes, particularly type II or insulin resistant diabetes, is shifting away from being viewed as a problem only for middle-aged adults. For example, type II diabetes, can no longer be referred to as “adult onset diabetes” to distinguish it from type I diabetes or insulin dependent diabetes, predominantly seen in juveniles. This is because type II diabetes is increasingly being diagnosed in American youth and is changing what diabetes “looks like” in young people (Ciporen 2012; Rosenbloom et al. 1999; Wei et al. 2010). This change is more than an alteration of nomenclature, it is indicative of a possible demographic shift in the risk profile for diabetes for young adults.

Although obesity is rising worldwide, studies involving children in Europe, such as the work of Neu and colleagues (2009), demonstrate that type II diabetes is much less prevalent in children in Germany and across Europe than in the United States after

accounting for socioeconomic differences. This may suggest that possible differences in the unique social environment of the United States- particularly the conditions relating to discrimination, may impact diabetes risk by early adulthood for Americans.

This dissertation seeks to address factors related to racial disparities in diagnoses of diabetes and provide a critical analysis of the social and demographic predictors of the risk of undiagnosed diabetes using data from the National Longitudinal Study of Adolescent Health (Add Health). These data may be better suited to address the gaps in the current social science literature regarding the prevalence and predictors of undiagnosed diabetes in young adults relative to the available measures in the NHANES data. The Add Health study is a nationally representative sample that includes biological measures, such as hemoglobin A1C, along with a wide variety of health and social measures providing information across the early life course for a nationally representative sample of adults aged 25-34 in 2009. Having hemoglobin A1C, in particular, is important because hemoglobin A1C is a reliable biomarker that measures the proportion of glucose containing hemoglobin molecules in red blood cells without requiring fasting (Krolewski et al. 1995; Cowie et al. 2010).

The Add Health sample may be uniquely suited to measure the problem of undiagnosed diabetes in young adults because the initial sample included an oversample of middle-class non-Hispanic blacks, a group with heightened risk based on current prevalence estimates (American Diabetes Association Fact Sheet 2013; CDC 2011). Moreover, since the Add Health study is longitudinal instead of cross-sectional, survey participation may be less unusual for Add Health participants than the comparative NHANES cohort, which may result in a significantly lower refusal rate among this

sample for the biomarker across race/ethnic groups due to familiarity with data collection procedures.

This dissertation is designed in a three-study format. Each analytic chapter presents a stand-alone problem related to undiagnosed diabetes and racial minority status. Together, these studies address issues in measuring the prevalence, predictors, and impediments to diagnosis of diabetes for young adults. The first analytic chapter compares the NHANES and Add Health samples and weighs the utility of these data sets in assessing the prevalence of undiagnosed diabetes in young adults. The second analytic chapter focuses on black-white differences in the effects of perceived discrimination on diabetes risk severity. The final chapter examines help seeking patterns among diabetics to discern if differences in diagnoses are primarily due to differences in help seeking or diabetes diagnosis allocation.

Predicting Diabetes Severity Comparing NHANES and Add Health

The first analytic chapter compares the differences in biomarker nonresponse in the NHANES and Add Health samples for assessing the prevalence of undiagnosed diabetes. The vast majority of research on undiagnosed diabetes relies on data from the National Health and Nutrition Examination Survey (NHANES), which uses mobile examination centers to collect a wide variety of health information including blood samples through venipuncture (Boltri et al. 2005; Cowie et al. 2009; Harris et al. 1998). However, few articles acknowledge the implications of this study's design in assessing the prevalence of undiagnosed conditions in young adults. The NHANES study requires a separate visit up to two weeks after the survey portion of the study. The delay between the survey and examination portions of the study may lead to high rates of refusal among

some age and racial groups. Differential biomarker nonresponse could impede the generalizability of this sample for subpopulations of young adults with undiagnosed conditions if those with undiagnosed conditions opt out of the medical exam at different rates than those who are healthy.

Differences in the rates of refusal for medical components of the NHANES study have been observed for biomarker participation across age and race categories (Crimmins et al. 2007). Specifically, Crimmins and colleagues (2007) noted that 27% of the non-Hispanic blacks selected for the examination portion of the 1999-2002 NHANES had to be dropped from their study due to refusal to participate in all biomarker and anthropometric data collection except being weighed compared to only 16% of non-Hispanic whites and Mexican Americans. The latest estimates of undiagnosed diabetes in all adults over age 20 indicate that undiagnosed diabetes is particularly high in non-Hispanic blacks (CDC 2011). If non-Hispanic blacks are more likely to have undiagnosed diabetes and more likely to opt out of biomarker testing, it is possible that estimates of the prevalence of undiagnosed diabetes may be biased downward. Differential nonparticipation rates are important to note because these rates may bias the estimates of the prevalence of conditions such as diabetes particularly among younger people and racial minorities known to opt out at higher rates than whites (Groves 2006).

This chapter presents four research questions: First, is there nonresponse bias in the NHANES and Add Health samples? Second, are the predictors of biomarker nonresponse the same across studies? Third, are levels of missing data and characteristics associated with missing cases equivalent across data sets? Finally, do the differences in

respondent biomarker nonresponse alter estimates of diabetes risk once the data is imputed?

Unequal Exposure and Unequal Risk

The second analytic chapter focuses on black-white differences in the effects of perceived discrimination on diabetes risk severity. Although medical literature has acknowledged increases in diabetes risk across the age spectrum (Narayan et al. 2003), social science literature has been slow to recognize the growing risk this condition may have on estimates of racial health disparities in the United States for young adults. Although diabetes has previously been linked to biological and behavioral risk factors, these factors alone do not explain disparities in diabetes prevalence across race/ethnic groups (Cowie 2006; Cowie et al. 2010). The unique circumstances involving exposure to discrimination in the United States could be a possible contributing factor for racial health disparities related to diabetes risk due to unequal stress exposure from structural and interpersonal sources of discrimination for non-Hispanic blacks (Clark et al. 1999).

Prior empirical research has demonstrated that chronic and acute life stressors can activate physiological stress responses of the hypothalamic-pituitary-adrenal (HPA) axis, which regulates hormone production involved in glucose control (DeSantis et al. 2007; Gunnar & Adam 2012; Sapolsky 2004). Exposure to recurrent social stressors, like discrimination, can lead to repeated activation of the HPA axis. Over time, this process could lead to dysfunction between the balance of cortisol, a stress hormone released during the activation of the HPA axis, and glucose, which could result in insulin resistance (pre-diabetes) and develop into diabetes (Eriksson et al. 2008).

If unequal social stress exposure does alter diabetes risk severity for racial minorities, this may partially explain disparities in diabetes risk severity not explained by behavioral or biological risk factors. Conversely, unequal stress exposure may also lead to differences in the development of empowering psychological resources and risky coping strategies (e.g.- drinking, smoking, poor diet, etc.) that could also alter diabetes risk onset and severity. Taken together, these factors motivate the following research questions: 1) is there an association between race and diabetes risk severity? 1a) If so, does perceived discrimination mediate this relationship? 1b) Alternatively, does racial minority status alter the effect of perceived discrimination on diabetes risk severity through moderation? 2) Do risky coping strategies mediate the effect of perceived discrimination on diabetes risk severity by race? Finally, 3.) Does mastery moderate the effect of perceived discrimination on diabetes risk severity by race?

Testing the Behavioral Model of Health Services Use

The final chapter examines help seeking patterns among diabetics to discern if differences in diagnoses are primarily due to differences in help seeking or diabetes diagnosis allocation. The extent of racial health disparities in diabetes diagnosis allocation remain unclear for young adults because many studies lack adequate sample sizes of both diagnosed and undiagnosed cases in younger cohorts to allow for comparisons across groups. Of the available research on undiagnosed diabetes, analyses tend to focus on the importance of increasing diabetes screening and identifying at-risk individuals, but little work has been done to examine risk across the life course for those who remain undiagnosed (Cowie 2006; Cowie et al. 2010; Harris et al. 1987; Hunt, Gebregziabher, and Egede 2012). Further, there has been little empirical work to discern

whether the disparity in diagnoses is due to lack of access to care, failure to seek care, or differences in symptom presentations across groups. It is important to know whether diabetes manifests differently in a young adult population than in an older adult population because delays in diagnosis can increase the risk of morbidity and mortality associated with prolonged exposure without appropriate treatment and increase the overall costs of diabetes care (Nichols, Arondekar, and Herman 2008; Nichols and Brown 2005; Zhang et al. 2009).

This study seeks to fill these gaps in the literature by asking: 1.) Do young adults with diabetes utilize healthcare equally by race? If not, 2.) Do differential patterns in help seeking explain diagnostic disparities for young adult diabetes? Conversely, if help seeking patterns are equivalent across demographic groups, 3.) Is diabetes diagnosis allocation equivalent among diabetics who seek care by race?

1.2 References

- American Diabetes Association .2013 <<http://professional.diabetes.org/Admin/UserFiles/0%20-%20Sean/FastFacts%20March%202013.pdf>> Accessed March 20, 2014
- Boltri JM, Okosun IS, Davis-Smith M, Vogel RL. 2005. "Hemoglobin A1c levels in diagnosed and undiagnosed black, Hispanic, and white persons with diabetes: results from NHANES 1999-2000." *Ethnicity and Disease*.15(4):562–567
- CDC 2011 < <http://non.cdc.gov/diabetes/pubs/estimates11.htm>> Accessed July 30, 2013
- Ciporen, Helaine. 2012. "Social Workers' Role in Combating the New Epidemic of Type 2 Diabetes in Children: Clinical Interventions at the Hall Family Center for Pediatric Edocrinology and Diabetes." *Social Work and Health Care*. 51(1): 22-35.
- Clark, Rodney, Norman B. Anderson, Vanessa R. Clark, and David R. Williams.1999. "Racism as a Stressor for Non-Hispanic blacks: A Biopsychosocial Model." *American Psychologist*. 54(10): 805-816.
- Cowie C. 2006. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health and Nutrition Examination Survey 1999-2002." *Diabetes Care*. 29(6):1263–1268.
- Cowie CC, Rust KF, Ford ES, Eberhardt MS, Byrd-Holt DD, Li C, et al. 2009. "Full accounting of diabetes and pre-diabetes in the U.S. population in1988-1994 and 2005-2006." *Diabetes Care*.;32(2):287–294
- Cowie CC, Rust KF, Byrd-Holt DD, Gregg EW, Ford ES, Geiss LS, et al. 2010. "Prevalence of diabetes and high risk for diabetes using A1C criteria in the U.S. population in 1988-2006." *Diabetes Care*.33(3):562-8.
- Crimmins, Eileen M., Jung Ki Kim, Dawn E. Alley, Arun Karlamangla, and Teresa Seeman. 2007."Hispanic paradox in biological risk profiles." *American Journal of Public Health* 97, (7): 1305-1310.
- DeSantis, Amy S., Emma K. Adam, Leah D. Doane, Susan Mineka, Richard E. Zinbarg, and Michelle G. Craske. 2007. "Racial/ethnic differences in cortisol diurnal rhythms in a community sample of adolescents." *Journal of Adolescent Health*.41(1): 3-13.
- Eriksson, A□K., A. Ekbohm, F. Granath, A. Hilding, S. Efendic, and C□G. Östenson. 2008."Psychological distress and risk of pre□diabetes and Type 2 diabetes in a prospective study of Swedish middle□aged men and women." *Diabetic Medicine* 25, no. 7: 834-842.

- Groves, Robert M. 2006. "Nonresponse Rates and Nonresponse Bias in Household Surveys." *Public Opinion Quarterly*. 70 (5): 646-675.
- Gunnar, Megan R., and Emma K. Adam. 2012. "THE HYPOTHALAMIC–PITUITARY–ADRENOCORTICAL SYSTEM AND EMOTION: CURRENT WISDOM AND FUTURE DIRECTIONS." *Monographs of the Society for Research in Child Development* 77, no. 2: 109-119.
- Guy, Jr., Gery P. 2010. "The Effects of Cost Sharing on Access to Care Among Childless Adults Effects Cost Sharing on Access to Care." *Health Services Research*. 45(6):1720-1739.
- Harris MI, Hadden WC, Knowler WC, Bennett PH. 1987. "Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in U.S. population aged 20-74 yr." *Diabetes*. 36(4):523–534
- Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, et al. 1998. "Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. The Third National Health and Nutrition Examination Survey, 1988-1994." *Diabetes Care*. 21(4):518–524
- Hunt, Kelly J., Mulugeta Gebregziabher, and Leonard E. Egede. 2012. "Racial and Ethnic Differences in Cardio-Metabolic Risk in Individuals with Undiagnosed Diabetes: National Health and Nutrition Examination Survey 1999-2008." *Journal of General Internal Medicine*. 27(8): 893-900.
- James PT, Rigby N, Leach R (2004). "The obesity epidemic, metabolic syndrome and future prevention strategies". *Eur J Cardiovasc Prev Rehabil* 11 (1): 3–8.
- Johnson, C., Lacher, D., Lewis, B., & McQuillan, G. 2007. "FEATURE PAPER: Challenges in Collecting Survey-Based Biomarker and Genetic Data: The NHANES Experience." *SESSION 1: EMERGENCY PREPAREDNESS AND SURVEILLANCE*, 139.
- Krolewski AS, Laffel LMB, Krowlewska M, et al. 1995. "Glycosylated hemoglobin and the risk of microalbuminuria in patients with insulin dependent diabetes mellitus." *New England Journal of Medicine*;332:1251-5
- Kullgren, Jeffrey, Catherine G. McLaughlin, Nandita Mitra, and Katrina Armstrong. 2012. "Nonfinancial Barriers and Access to Care for U.S. Adults." *Health Services Research*. 47(1): 462-485.
- Narayan, K. M. Venkat, James P. Boyle, Theodore J. Thompson, Stephen W. Sorenson, and David F. Williamson. 2003. "Lifetime Risk for Diabetes Mellitus in the United States." *JAMA*. 290 (14): 1884-1890.

- Neu, Andreas, Ltz Feldhahn, Stefan Ehehalt, Regine Hub, Michael B. Ranke. 2009. "Type 2 Diabetes Mellitus in Children and Adolescents is Still a Rare Disease in Germany: A Population-Based Assessment of the Prevalence of Type 2 Diabetes and MODY in Patients aged 0-20 Years." *Pediatric Diabetes*. 10(7):468-473.
- Nichols, Gregory A., Bhakti Arondekar, and William H. Herman. 2008. "Medical care costs one year after identification of hyperglycemia below the threshold for diabetes." *Medical care* 46, no. 3 :287-292.
- Nichols, Gregory A., and Jonathan B. Brown.2005. "Higher medical care costs accompany impaired fasting glucose." *Diabetes Care* 28, no. 9 : 2223-2229.
- Reilly, JJ and J Kelly. 2011. "Long-Term Impact of Overweight and Obesity in Childhood and Adolescence on Morbidity and Premature Mortality in Adulthood: Systematic Review." *International Journal of Obesity*. 35: 891-898.
- Rosenbloom, Arlan, J., Robert S. Young, Jennie R. Joe. And William E. Winter.1999. "Emerging Epidemic of Type 2 Diabetes in Youth." *Diabetes Care*. 22(2):345-354.
- Sapolsky, Robert M. 2004. *Why Zebras Don't Get Ulcers: An Updated Guide to Stress and Stress Related Diseases, and Coping* (3rd Edition). New York: Freeman & Co.
- van Osch, Sylvie MC, Wilbert B. van den Hout, and Anne M. Stiggelbout. 2006. "Exploring the reference point in prospect theory: Gambles for length of life." *Medical Decision Making* 26, (4): 338-346.
- Vernon S. 1999. "Risk Perception and Risk Communication for Cancer Screening Behaviors: a review." *Journal of the National Cancer Institute. Monographs*, 25, 101-119.
- Walker, Elizabeth A., Maria R. Kalten, C.K. Mertz, and James Flynn. 2003. "Risk Perception for Developing Diabetes: Comparative Risk Judgments by Physicians." *Diabetes Care*. 26: 2543-2548.
- Wei, Jung-Nan, Hung-Yuan Li, Yi-Chia Wang, Lee Ming Chuang, Mao-Shin Lin, Cheng-Hsin Lin, and Fung-Chang Sung. 2010. "Detailed Family History of Diabetes Identified Children at Risk of Type 2 Diabetes: A Population-Based Case-Control Study." *Pediatric Diabetes*. 11: 258-264.
- Zhang Y, Dall TM, Mann SE, Chen Y, Martin J, Moore V, et al.2009. "The economic costs of undiagnosed diabetes." *Population Health Management*. 12(2):95-101.

CHAPTER 2

Predicting Diabetes Severity Comparing NHANES & Add Health: Does the Data Used Alter Population Inferences for Young Adult Diabetes Risk?

Abstract

Population estimates of health conditions come from large national surveys. However, these estimates may vary across studies for groups with differing risk for diagnosable conditions like diabetes. The purpose of the current study is to investigate differences in prevalence estimates of diabetes risk for young adults between an age-matched subsample of the NHANES and the Add Health studies. This study seeks to determine whether differences in study designs alter estimates of diabetes risk for young adults. Focal analyses explore the impact of biomarker collection nonresponse in NHANES and Add Health on diabetes risk estimates. Results indicate that African Americans are disproportionately likely to be missing biomarker data despite completing the survey portion of both studies. When diabetes status is imputed for these individuals, increased odds of nonresponse do not change predicted risk of undiagnosed diabetes. However, the multivariate models predicting undiagnosed diabetes risk yield different conclusions across studies. This suggests that the NHANES estimates for undiagnosed diabetes may be less generalizable for young adults than the estimates from the Add Health data. Further study is needed to evaluate possible race-specific nonresponse bias of biomarker data collection across the two studies.

Key words: Diabetes, health disparities, NHANES, and Add Health

Abstract Word count: 190

Word Count: 6911

Acknowledgement: This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

2.1 Introduction

Undiagnosed health problems are difficult to measure because individuals who have undiagnosed conditions may not know they are at risk. Identifying the prevalence of undiagnosed conditions like diabetes is important because the earlier these conditions are caught and treated, the less likely it is that people will have irreversible health problems (Trull et al. 2002). Studies like the National Health and Nutrition Examination Survey (NHANES) collect extensive health information that is used by the Centers for Disease Control and Prevention (CDC) to create national prevalence estimates for undiagnosed health conditions (CDC 2011). Although these estimates provide an abundance of valuable health information about diagnosed and undiagnosed conditions, less attention has been paid to the use of this data to estimate prevalence rates for undiagnosed conditions in young adults specifically. Young adults may have increased risk of undiagnosed conditions due to having fewer health problems than older adults (Park et al. 2006) and underutilizing health care (Callahan and Cooper 2005). However, there may be elevated risk of biased estimates for the prevalence of undiagnosed conditions for young adults if those who opt out of biological data collection differ from those who do participate and those who are missing have increased risk of undiagnosed health problems (Peytcheva and Groves 2009).

The CDC aggregates NHANES estimates for health conditions by age into groups for youth 0-19, all adults over age 20, and adults over 65 (CDC 2011). The national prevalence estimates for these age groups have been useful, but recent studies have called into question the comparability of NHANES data to other studies of young adults with rich health information like the National Longitudinal Study of Adolescent Health (Add

Health) for finer age ranges (Chyu, McDade, and Adam 2011). In 2011, Chyu and colleagues uncovered differences in point estimates of the prevalence of undiagnosed hypertension for young adults in the Add Health and the NHANES studies. Their analyses revealed that Add Health had 4.5 times the rate of undiagnosed hypertension as NHANES, but the two studies had near equivalent rates of diagnosed hypertension. Chyu and colleagues (2011) found several differences between the studies including the timing and collection procedures of anthropometric data that make it difficult to discern which estimates are more reliable for young adults.

One possible reason for the difficulty in interpreting prevalence estimates across the two studies is that Chyu and colleagues (2011) compared only complete cases, but did not analyze the effects of missing data across studies as an alternative explanation for prevalence discrepancies with undiagnosed hypertension. The timing of biological data collection is a key difference between the two studies that may help explain the discrepancy between estimates of diagnosed and undiagnosed conditions for young adults. It is plausible that the delay in timing of anthropometric data collection in NHANES could bias the estimates of undiagnosed conditions that are evaluated using biomarkers by excluding those who are averse to medical testing or are otherwise unable to come back for a second visit that could correlate with increased risk of undiagnosed health conditions (Schafer et al. 1996). These factors could alter risk of nonresponse for participation in the medical exam portion of the study if the factors that increase nonresponse for the medical exam correlate with diabetes risk under the “common cause model” of nonresponse (Groves 2006).

If respondents have distrust in the medical community this could decrease participation in medical evaluations, which is of particular concern for racial minorities (Hammond 2010; Lyles et al. 2011; Vaccaro & Huffman 2012). However, it is possible that having biological data collected by non-medical professionals could reduce participation among those who perceive increased legitimacy or safety with medical practitioners over trained interviewers. Those who distrust biological data collected by non-professionals could opt out of biological data collection, which could be a problem with the Add Health study (Boerma, Holt, and Black 2001). Taken together, it is important to understand exactly who is missing from each study and assess whether the study designs contribute to bias in prevalence estimates of undiagnosed conditions for young adults.

A second reason why Chyu, McDade, and Adam (2011) could not conclusively determine the generalizability of undiagnosed hypertension prevalence estimates across the two studies involved the sensitivity of hypertension screening to timing of measurement. Blood pressure readings can vary greatly due to dehydration, stress, or “white coat” hypertension with people who fear medical testing (Ohkubo et al. 2005). Other undiagnosed conditions, like diabetes can be discovered using blood tests like hemoglobin A1C, which is accurate over a longer period of time that could make it easier to compare prevalence estimates across studies (Olson et al. 2010). The current study seeks to address these issues by examining prevalence estimates of undiagnosed diabetes using the biological marker (biomarker) hemoglobin A1C (A1C), which is used to diagnose diabetes and is accurate over a period of one to three months (Olson et al. 2010)

to minimize the effect of the timing discrepancy with biological data collection across studies.

2.2 Research Questions

Q1: Is there nonresponse bias in biomarker participation the NHANES and Add Health samples?

Q1a: Are the predictors of biomarker nonresponse the same across studies?

Q2: Do the differences in biomarker participation alter estimates of diabetes risk once the data sets are imputed?

2.3 Literature Review

The NHANES Sample

The NHANES study is administered every two years and is designed to capture a nationally representative random sample of civilian, non-institutionalized individuals (http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/generaldoc_e.htm). The NHANES study is unique in that in addition to a large survey portion, the study also includes a comprehensive health exam incorporating blood, urine, and other physical tests (http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/generaldoc_e.htm). The survey portion is administered to all selected participants, but the comprehensive medical examination portion is only conducted on participants who sign a second consent form for the examination portion and come to the testing location (http://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_lab.pdf; p.17). Glucose data was collected for selected participants age 12 and older (http://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/healthmeasurementlist07_eng%20.pdf).

The 2007-2008 and 2009-2010 NHANES studies had 2302 respondents age 25-34. Of those respondents, 2076 participated in the medical examination portion and had valid blood readings and 226 respondents (6.66% weighted) did not have values for the A1C portion. Of those who did not have values for the A1C portion, 75 were non-Hispanic white (7.87% of NH whites; weighted), 73 were non-Hispanic black (16.11% of NH blacks; weighted), 68 were Hispanic of any origin (8.87 % of all Hispanics; weighted) and 10 were from another race/ethnic group (7.75% of other race individuals; weighted). Sampling weights were created to address overall probability of selection, selection for the examination portion, and likelihood of participation in the examination portion if selected to account for unit nonresponse and representativeness.

One challenge to the NHANES data collection procedures is that NHANES asks respondents to participate in survey data collection and examinations that span more than one day, which may lead to decreased participation for the examination portion of the study. Missing biological data in the NHANES data has been cited in articles that have had to account for differential rates of non-response for medical information (Crimmins et al. 2007) and more directly as an illustrative example in a study focusing on solutions to missing data problems (Andridge and Little 2010). For example, Crimmins and colleagues (2007) noted that although respondents were selected with equal probability for the 1999-2002 NHANES, non-response rates for the medical portion differed across race/ethnic groups. Specifically, 27% of African Americans were missing at least one biomarker compared with 16% of Mexican Americans and 16% of Caucasians who were missing at least one biomarker when selected for the examination portion of the study

suggesting differential nonresponse by race/ethnic group for biomarker data collection could be a persistent problem with NHANES study design.

Other studies using NHANES data have demonstrated similar patterns of missing cases with biological data by gender (Pandya, Weinstein, and Gaziano 2011) that could potentially lead to underestimates of the prevalence of diabetes if those who opt out of the biomarker collection are at increased risk of undiagnosed diabetes and their risk is related to their likelihood of not completing the medical examination. For example, it is possible that those who are undiagnosed have health care access or utilization patterns associated with demographic characteristics that may influence underutilization of medical care due to structural problems, such as neighborhood segregation, that decrease opportunities for socialization into the medical system (Gary et al. 2007; White, Haas, and Williams 2012).

The Add Health Sample

The National Longitudinal Study of Adolescent Health (Add Health) is a longitudinal study of students in schools who were followed over time. The study was designed to be nationally representative of students in grades 7-12 in schools in 1994-1995 with oversamples of middle-income African Americans, siblings, and children with limb deformities (cf. Chantala and Tabor 1999; Harris 2009). The Add Health study assessed the social, emotional, and physiological well being of youths aged 11-22 in the first wave. Subsequent waves have followed the youths into the transition to adulthood. The most recent wave, collected in 2008 and 2009, included anthropometric and survey data on 15,701 of the original respondents (75.69% of the 20,745 from the first wave) who were now ages 25-34.

Wave IV of Add Health had 14,800 respondents with valid cross-sectional weights. Of those respondents, 13,499 had valid hemoglobin A1C readings and 1289 respondents (8.83% weighted) did not complete the biomarker portion. Of those missing A1C readings, 566 were non-Hispanic white (7.66% of NH whites; weighted), 358 were non-Hispanic black (12.14% of NH blacks; weighted), 232 were Hispanic (9.9 % of all Hispanics; weighted), 99 were Asian or Pacific Islanders (10.44% of all API; weighted) and 34 were from another race/ethnic group (10.59% of other race individuals; weighted).

The anthropometric data for the Add Health study were collected immediately following a 90-minute interview during the same visit by a trained interviewer (Harris et al. 2009). Trained interviewers collected physical measurements of height, weight, waist circumference, blood pressure, and several spots of blood for laboratory analysis (Harris 2009; Whitsel et al. 2012). Unlike the NHANES study, which uses mobile examination centers to collect biological data, the Add Health study collects survey and biological data in the same location as the interview (Harris et al. 2009). The Add Health biological data was collected by trained interviewers, but not medical professionals exclusively as was the case with the NHANES study (Harris et al. 2009; Whitsel et al. 2012). The Add Health study had a high cooperation rate for the anthropometric data collection and valid responses for 91.2% of respondents with cross-sectional weights (Whitsel et al. 2012).

The Add Health study relied on collecting whole blood spots obtained through finger-pricks instead of whole blood samples obtained through venipuncture like NHANES, because the Add Health biological and anthropometric data were collected on site during face-to-face interviews. The NHANES study's use of venipuncture has been noted as a challenge for collecting survey-based biological data particularly among

children, youths, and some adults who view the procedure as too invasive (Johnson et al. 2007). The use of dried blood spots versus venipuncture blood samples in large national health surveys is a contentious issue. Although the use of blood spots has been tested and verified using various assays since the 1980s (McDade, Williams, and Snodgrass 2007; Varnier et al. 1988; Williams and McDade 2009), universal acceptance for the practice remains elusive (Johnson et al. 2007). However, since whole blood spot collection is less expensive to conduct and does not require trained phlebotomists like venipuncture, more national studies are incorporating the use of less-invasive blood spot collection over venipuncture to reduce costs and minimize refusals while still collecting extensive biological information.

Young Adult Risk, Medical Distrust, and Diabetes

Diabetes is a chronic condition that arises from an inability of the pancreas to regulate the balance of glucose and insulin production, which leads to excessive levels of glucose in the blood (American Diabetes Association 2011; World Health Organization 2012). Unlike obesity that can be assessed at home using a scale, diabetes requires diagnosis by a medical professional to be revealed. Underutilization of health care may differ across race/ethnic groups and socioeconomic status, which could be particularly problematic if these groups are at increased risk of undiagnosed diabetes.

Underutilization of health care may be higher among young adults who do not think they are susceptible to chronic illness early in the life course (Walker et al. 2003; Van Osch, van den Hout, and Stiggelbout 2006; Vernon 1999). Others fearing the stigma of disease might be averse to seeking health care until they are very ill (Koszegi 2003). For people without insurance, the long-term effects of undiagnosed diabetes can cost

more than treatments for diabetes that is diagnosed early (Nichols, Arondekar, and Herman 2008; Nichols and Brown 2005; Zhang et al. 2009). Consequently, the initial costs of seeking care may put individuals with lower socioeconomic statuses at higher risk of avoiding care. Taken together, young adult fears and health perceptions may influence their risk of undiagnosed diabetes, but they could also alter the likelihood that they will participate in health screenings if they are afraid of the results, which could lead to nonresponse bias for the estimates of undiagnosed health conditions in national surveys.

Beliefs regarding the efficacy of the health care system and past interactions with doctors could alter the likelihood that someone would seek care based on past interactions with the medical community, but these beliefs may also reduce the likelihood that someone would participate in medical examinations as part of a survey. Historical mistreatment of racial minorities by the medical community is one possible factor that may contribute to increased undiagnosed conditions for minority individuals at risk. Research suggests that some racial minorities, particularly African Americans, avoid seeking care due to preconceived notions regarding discriminatory interactions with doctors (Hammond 2010; Lyles et al. 2011; Vaccaro & Huffman 2012). A recent study by Stepanakova (2012) found that when doctors are under time pressure, they are less likely to refer female patients perceived as African American for advanced testing than patients perceived as Caucasian. This finding may imply racial bias in the allocation of treatment even when patients make it into the office and present with the same symptoms. If diagnostic biases are present for young adults in the United States, it is

critically important that large-scale studies estimate the prevalence of undiagnosed diabetes accurately to reveal the magnitude of diagnostic disparities.

2.4 Hypotheses

H1a.1: The delay in timing of data collection with the NHANES study is expected to increase nonresponse among minorities, economically disadvantaged individuals, and those with reduced health care utilization.

H1a.2: The use of non-medical staff by the Add Health study is expected to increase nonresponse for racial minorities and those with regular health care utilization.

H2: Biomarker nonresponse is predicted to alter the estimates of diabetes risk on one or both studies.

2.5 Methods

Sample Comparability

The participants included in the Add Health and NHANES studies are different. Several decisions were made in order to make the samples comparable. Although both studies are racially diverse, the NHANES study does not explicitly sample Asian or Pacific Islander individuals. Moreover, one of the goals of the NHANES study is to include analyses of US born and foreign-born individuals of Hispanic origin, as a result the NHANES study has a very large proportion of foreign-born individuals. As such, the samples used for comparison were reduced to only include U.S. born non-Hispanic whites, non-Hispanic blacks, and Hispanic individuals. The samples were also restricted to exclude pregnant women, respondents only missing the A1C biomarker, and respondents without valid survey weights. These adjustments reduced the age-matched NHANES sample from 2302 to 1576 individuals with 166 individuals (7.07% weighted)

missing the A1C biomarker and reduced the Add Health sample from 14800 to 12483 individuals with 1039 individuals (7.35% weighted) missing the A1C biomarker.

Dependent Variables

The two dependent variables of interest in this study include not having a value for the A1C biomarker component of the medical exam and respondent diabetes status. NHANES respondents without a value for the A1C biomarker were determined to be missing if they participated in the survey, were selected for participation in the mobile exam unit, and were missing from the blood test components. Add Health respondents without a value for the A1C biomarker were determined to be missing if they participated in the survey, but were missing from the blood test components.

In both studies, respondent *diabetes status* was determined by cross-referencing measured A1C values and stated diagnostic history. Diabetes is clinically indicated if A1C levels exceed 6.5% of hemoglobin molecules. Pre-diabetes is indicated with A1C values between 5.7% and 6.49% of hemoglobin molecules (Olson et al. 2010). As such, *diabetes status* was classified into four categories with persons with A1C values 5.69% or below classified as normoglycemic (not diabetic), persons with A1C values between 5.7% and 6.49% classified as pre-diabetic, and persons with A1C values greater than 6.5% classified as undiagnosed diabetic. Any persons indicating that they had a prior diabetes diagnosis were reclassified into the fourth category of “diagnosed diabetic” regardless of their current A1C level. For the purposes of the comparative analysis, those who with no diabetes history were treated as the reference group.

Comparison Variables

Several demographic and socioeconomic variables were available in both studies and included to compare possible characteristics that could influence nonresponse to the medical portions of the studies. Additional variables assessing health behaviors and health status were also included to analyze whether factors that could influence risk of undiagnosed diabetes predict nonresponse to the biological data collection. The demographic variables compared in this study included single category race, sex, and age. All three variables were asked using the same or similar wording across studies. Race/ethnic variables retained in this study included non-Hispanic white, non-Hispanic black, and Hispanic. Sex was measured as male or female and age was defined as age in years at the time of interview.

Socioeconomic status (SES) measures were worded differently across the two studies. Three SES variables were compared across the two studies including income, education, and insurance access. Add Health measured income continuously in thousands of dollars whereas the NHANES study used income brackets. To ease comparisons across studies, income brackets were collapsed to: \$0 to \$19,999, \$20,000 to \$34,999, \$35,000 to \$54,999, \$55,000 to \$74,999, \$75,000 to \$99,999, and \$100,000 or more.

Education was also measured with different response categories across studies. In the NHANES study, response options included “less than 9th grade,” “9-11th grade,” high school/GED equivalent,” “some college or AA Degree” and “college graduate or above.” In the Add Health Study, response options included, “8th grade or less,” “some high school,” “high school graduate,” some vocational training,” “completed vocational training,” “some college,” “completed college,” “some graduate school,” “completed master’s degree,” some post-master’s training,” “completed doctoral degree,” “some post

baccalaureate professional study,” and “completed post baccalaureate professional degree.” In order to ease comparisons across studies, education was collapsed into three categories for “high school or less,” “some college or vocational training,” and “college or advanced degree.” The “some college” category was omitted as a reference category in multivariate models.

The Add Health study measured health insurance coverage with the question, “Which situation best describes your current health insurance situation?” with eleven response choices ranging from no coverage to several specific types of family or employment-based coverage. In the NHANES study, health insurance was measured with the question, “Are you covered by health insurance or some kind of other health care plan?” with only yes or no as response options. In order to simplify the measures for health insurance access, respondents in both studies who indicated having insurance access of any kind (e.g.-private, government, or military) were deemed to have insurance. Any respondent without coverage was deemed to have “no insurance.”

The two health behavior variables compared in this study include fast food consumption and not seeing a doctor in the past year. Both studies asked how many fast food meals had been consumed in the previous week, but the response choices varied. The Add Health study allowed respondents to list the number continuously whereas the NHANES study gave respondents the choice of “0 meals,” “1-21 meals” or “more than 21 meals.” Fast food consumption was measured as an indicator of having any fast food in the previous week (any fast food=1, 0 otherwise). NHANES measured utilization of health care using two questions each asked among half the respondents. One question asked, “During the past 12 months, how/How many times {have you/has SP} seen a

doctor or other health care professional about {your/his/her} health at a doctor's office, a clinic, hospital emergency room, at home or some other place? The other question asked, "About how long has it been since {you/SP} last saw or talked to a doctor or other health care professional about {your/his/her} health?" In the Add Health study, the health care utilization question was asked as, "How long ago did you last have a routine check-up?" For all questions it was possible to identify whether the respondent sought care within the past year versus longer than a year. Respondents who did not see a doctor in the past year were given a value of one and a zero otherwise.

Four health status variables were compared in this study. The variables included weight category, prior diagnosis of high cholesterol, prior diagnosis of high blood pressure, and self-rated health. Both studies included body mass index (BMI) measures. To compare BMI across studies, BMI was divided into four categories: underweight (BMI 0-18.49), normal weight (18.5-24.9), overweight (25-29.99), and obese (30+). Both diagnosed conditions were coded where presence of the condition was labeled with a one and a zero otherwise. Self-rated health was measured the same in both studies with response options for poor (1), fair (2), good (3), very good (4) or excellent (5).

Analytic Strategy

The research questions for this study focus on issues related to comparability, nonresponse bias with biomarker data, and inferential differences drawn from the data across the two studies. Several descriptive and multivariate analyses were tested order to assess the comparability and effects of biomarker nonresponse on the two studies. The first set of tables compares the pre-imputed descriptive statistics between the NHANES and Add Health data (Table 2.1). The next two tables compare the differences between

those with and without A1C values in the NHANES (Table 2.1a) and Add Health (Table 2.1b) studies. The second set of tables compares the pre-imputed descriptive statistics for those missing A1C values between the NHANES and Add Health data (Table 2.2).

Two sets of multivariate models were included in order to evaluate substantive differences in the inferences drawn from the data across the two studies. The first set of inferential models use logistic regression analysis to predict the odds of a respondent not having an A1C value despite participating in the survey portion of the study (Table 2.3). The second set of inferential models use multinomial logistic regression of imputed data to see if not having an A1C value predicts diabetes risk severity once missing cases were imputed (Tables 2.4a-2.4c).

Missing Data and Imputation Strategy

Missing values were addressed using multiple imputation with the “ice” command in Stata 11.2 to perform imputation through chained equations (Royston 2005). Missing values on variables were imputed to provide complete analytic data with ten imputed data sets (Ragunathan 2004). The inferential multivariate analyses were conducted on the ten imputed data sets that were combined and analyzed using “Rubin’s Combining Rules” (Little and Rubin 2002).

2.6 Results

Complete Case Comparisons

Table 2.1 displays the weighted and survey design adjusted descriptive statistics comparing the age-matched 2007-2010 NHANES to the 2008-2009 Add Health data. There are several statistically significant differences in means and proportions across the two studies. Prevalence estimates of diabetes vary significantly across the two studies

with NHANES having a very high proportion of individuals who are not diabetic compared to Add Health (82% vs. 63%) with no overlap of the 95% confidence intervals of the estimates. Conversely, Add Health has substantially larger prevalence estimates of pre-diabetes (25% vs. 8%) and undiagnosed diabetes (2.29% vs. 0.42%) than the NHANES study. Interesting, like Chyu, McDade and Adam (2011) found with diagnosed hypertension, the estimates for diagnosed diabetes are equivalent across the two samples. Unexpectedly, the amount of biomarker nonresponse is equivalent across the two samples.

Although the weighted means and proportions for all demographic variables are equivalent across studies, there are significant differences in estimates for most of the socioeconomic status variables. NHANES has more respondents with high school or less education (38% vs. 26%) and fewer respondents with some college or vocational training (34% vs. 44%) on average than the Add Health study participants. Both studies had equivalent proportions of participants with college or advanced degrees. NHANES and Add Health participants significantly differ on average for each of the income brackets with Add Health respondents overrepresented among the bottom three categories (\$0-54,999) and NHANES respondents overrepresented on the upper three categories (\$55,000-\$100,000+). Health insurance access was equivalent across studies.

Several of the health behavior and health status variables also differed across the two studies. NHANES respondents were significantly more likely to consume fast food in the previous week (82% vs. 76%) and less likely to have waited longer than a year to have a doctor's visit (20% vs. 42%) on average than Add Health respondents. On average, NHANES respondents were more likely to be normal weight (36% vs. 30%),

more likely to have diagnosed high cholesterol (19% vs. 8%), and report lower levels of self-rated health (3.52 vs. 3.65) than Add Health respondents. Estimates of diagnosed high blood pressure, underweight, overweight, and obesity rates were equivalent across studies.

Within and Across Sample Missing Case Comparisons

Table 2.1a compares the descriptive statistics for NHANES respondents with and without A1C values. The only statistically significant variable to vary between those with and without A1C values in the NHANES data was the proportion of non-Hispanic black respondents (15% of the whole sample, 29% of missing). Table 2.1b compares the descriptive statistics for Add Health respondents with and without A1C values. Four variables significantly differ between those with and without A1C values in the Add Health study including sex (Males 52% of the whole sample, 62% of missing; Females 48% of whole sample, 38% of missing), some college or vocational training (44% of the whole sample, 36% of missing), obesity (38% of the whole sample, 30% of missing), and self-rated health (3.65 for the whole sample, 3.87 of missing).

Table 2.2 compares the descriptive statistics of respondents who were missing A1C values across the NHANES and Add Health studies. Five variables significantly differed on average among those missing across the two studies. On average respondents who were missing from the NHANES study were significantly more likely to have high school or less education (50% vs. 27%) than missing Add Health respondents. However, those missing from the NHANES study were also more likely to report incomes of \$100,000 or more than Add Health respondents respectively (23% vs. 3%). Three health behavior and health status variables differed for those missing A1C values across the two

studies. Missing NHANES respondents were significantly more likely to consume fast food in the previous week (88% vs. 76%) and less likely to have waited longer than a year to have a doctor's visit (18% vs. 39%) on average than missing Add Health respondents. Self-rated health also significantly differed on average across the two studies for those missing A1C values with missing NHANES respondents reporting lower on average self-rated health (3.43 vs. 3.87) than missing Add Health respondents.

Across Sample Missing Biomarker Predictors

Table 2.3 reports the odds ratios predicting the likelihood that respondents will be missing A1C values across the two studies. The only significant predictor of being missing for the NHANES study was being non-Hispanic black compared to being non-Hispanic white (OR=2.65, 95%CI= 1.68, 4.16). Both non-Hispanic black (OR=1.82, 95%CI= 1.37, 2.40) and Hispanic (OR=1.49, 95%CI= 1.07, 2.09) Add Health respondents were statistically more likely to be missing than non-Hispanic whites.

Four other variables significantly predicted the odds of being missing in the Add Health study. Men in the Add Health study had higher odds of being missing than women (OR=1.54, 95%CI= 1.21, 1.96). Add Health respondents with college or advanced degrees had higher odds of being missing than those with some college or vocational training (OR=1.42, 95%CI= 1.14, 1.75). Add Health respondents who were underweight had higher odds of being missing (OR=2.15, 95%CI= 1.22, 3.81) while obese respondents were less likely to be missing (OR=0.75, 95%CI= 0.58, 0.97) than those who were normal weight. As self-rated health increased in the Add Health study, the odds of being missing also increased (OR=1.25, 95%CI= 1.11, 1.41).

Although the Add Health study had more variables predict the odds of being missing than the NHANES study, all of the confidence intervals for the estimates overlapped. Therefore the respondents who are missing across the two studies are not likely to be significantly different from each other on average.

Effect on Imputed Missing Cases on Diabetes Risk

Tables 2.4a-2.4c present the multinomial logistic regression results predicting diabetes risk relative to not having diabetes across the two studies. Table 2.4a compares pre-diabetes risk relative to no diabetes history across the two studies. Table 2.4b compares undiagnosed diabetes risk relative to no diabetes history across the two studies. Table 2.4c compares diagnosed diabetes risk relative to no diabetes history across the two studies. Being missing in the original data did not significantly predict diabetes risk in any model across the two studies.

Although being missing in the original data did not appear to alter the relative risk of pre, undiagnosed, or diagnosed diabetes, there were some differences in the size of the relative risk ratios for the predictions of diabetes risk across studies. Differences were found for pre and undiagnosed diabetes, but not diagnosed diabetes. In the NHANES data, being obese compared to not obese greatly increased the relative risk of pre-diabetes (RRR=3.38, 95%CI= 2.44, 4.67). Obesity also predicted pre-diabetes in the Add Health sample, but the predicted effect was significantly smaller (RRR=2.09, 95%CI= 1.86, 2.36).

There were more differences for predicting undiagnosed diabetes risk relative to no diabetes history than for pre-diabetes. Having a college or advanced degree compared to some college or vocational training (RRR=0.00, 95%CI= 0.00, 0.00), or an income of

\$75,000-\$99,999 (RRR=0.00, 95%CI= 0.00, 0.00) or \$100,000 or more (RRR=0.00, 95%CI= 0.00, 0.00) compared to an income of \$0-\$19,999 greatly reduced the relative risk of undiagnosed diabetes relative to no diabetes history in the NHANES study. These findings suggest that increased socioeconomic status is protective against undiagnosed diabetes with the NHANES data. However, that conclusion is not supported with the Add Health data where no socioeconomic status variables predict undiagnosed diabetes risk relative to no diabetic history.

Both studies support strong associations between race and obesity with increased relative risk of undiagnosed diabetes, but the Add Health data suggests men, older respondents, and those with either diagnosed high cholesterol or high blood pressure are also at elevated risk though the confidence intervals for the size of these effects overlapped across studies.

2.7 Discussion

This study makes several important contributions to the study of undiagnosed diabetes in young adults and estimates of young adult health. Although there are several factors that significantly predict the likelihood that an individual will be missing biological data across the two studies, these analyses do not provide support for nonresponse bias for biomarker participation. Therefore, hypothesis 1, which predicted that nonresponse bias would be a problem in one or both studies is not supported. Conversely, it appears that the differences in the prevalence estimates across the studies are more attributable to differences among those who are included in the survey portion of the studies (Table 2.1) as opposed to those who were missing from the biomarker portion of the studies (Table 2.2).

Hypothesis 1a proposed that the predictors of nonresponse would differ across studies is largely supported (Table 2.2) particularly by socioeconomic status, health behaviors, and self-rated health. However, hypothesis 1a.1 that predicted increased nonresponse by race, socioeconomic disadvantage, and those with lower health care utilization in the NHANES due to the increased burden of a second visit, only had support for increased nonresponse among non-Hispanic blacks (Table 2.1a). Hypothesis 1a.2 that proposed increased nonresponse for racial minorities and those with regular health care utilization in the Add Health study due to the use of non-medical professionals was not supported (Table 2.1b). Conversely, Add Health nonrespondents were more likely to vary by sex, less likely to be obese, and report higher self-rated health.

Although more variables differed between respondents and non-respondents with the Add Health data (Table 2.1b), the variables that predicted missing biomarker data did not significantly vary across studies (Table 2.3). Both the levels of missing data in Table 2.1 and the predictors of missing data in table 2.3 are equivalent across studies, which suggests that the timing delay of the NHANES study does not necessarily bias the estimates as initially suspected. Moreover, the indicators of being missing in the initial data were not statistically significant multivariate models in tables 2.4a-2.4c for either study. Taken together, this suggests that nonresponse to the biomarker data collection does not bias the estimates of undiagnosed diabetes for either study.

Even though these analyses do not support an argument for nonresponse bias in the samples for predicting undiagnosed diabetes, the multivariate models reveal additional concerns about comparing the data across studies when evaluating diabetes

risk. The relative risk ratios and resulting confidence intervals for the estimates are very large for the NHANES models predicting undiagnosed diabetes risk relative to no diabetes history especially when compared to the Add Health data. The most striking examples are seen with race and obesity in the models predicting undiagnosed diabetes.

Part of the extreme estimates with race and obesity are likely due to a “small n” problem with the NHANES data with too few cases of undiagnosed diabetes for meaningful comparison with only 11 undiagnosed cases in the NHANES study compared to 360 cases in the Add Health Study. However, very large relative risk ratios also exist for Add Health estimates of the relative risk of undiagnosed diabetes for non-Hispanic blacks that cannot be attributed to a “small n” problem. Strong correlations exist with undiagnosed diabetes and non-Hispanic black race/ethnic status (NHANES correlation = 0.06, $p=0.000$; Add Health correlation=0.20, $p=0.000$). The association between undiagnosed diabetes and non-Hispanic black race/ethnic status cannot be ignored, but would be impossible to interpret using the NHANES data alone.

Limitations

This study must be evaluated for both its strengths and its weaknesses. There are several limitations to the data that must be acknowledged as areas for further consideration. First, these analyses must be viewed with caution because only a small subset of the variables included in each study were tested here. Although I did not find support for nonresponse bias for the prevalence estimates of undiagnosed diabetes, that does not mean that nonresponse bias does not exist in either study for other variables of possible substantive interest.

Second, the inferences drawn from the multivariate models must also be viewed with caution as omitted variable bias may be a concern with this data. A relatively small number of variables that could impact diabetes risk were available in both studies. The NHANES data has a variety of health related variables that are not available in the Add Health data. Conversely, several social, behavioral, and family health history variables are available in the Add Health data that are not included in the NHANES data.

Third, although the relatively small subsample of young adults with undiagnosed diabetes is a challenge for multivariate analyses with the NHANES data, using the Add Health data also presents additional concerns about the generalizability of the data. Since the Add Health study is longitudinal, there is concern about the risk of attrition bias when using the Add Health data. Although exploring attrition bias was not within the scope of this study, it is a valid concern for future studies. This study did not find support for nonresponse bias for biomarker participation, but did observe substantial differences in estimates of weighted means and proportions for relevant variables for those included in both studies, which suggest that either these studies sample extreme groups of the same population or they do not sample the same population all together.

Fourth, supplementary sensitivity analyses presented in Table 2.5 explore the descriptive statistics of the non-Hispanic blacks missing in both studies. The only statistically significant difference in means for missing non-Hispanic blacks is found with high school or less education where 60% of NHANES compared to 35% of Add Health respondents were missing biomarkers. The sample sizes of missing non-Hispanic blacks were too small to allow for multivariate analyses to predict differences in biomarker nonresponse among non-Hispanic blacks. Given alternative hypothesized mechanisms

that could contribute to non-Hispanic black nonparticipation in biomarker research (Hammond 2010; Lyles et al. 2011; Schafer et al. 1996; Vaccaro & Huffman 2012), additional repeated study of biomarker nonresponse in the NHANES data may be warranted with larger pools of aggregated data.

Finally, although several studies have verified the utility of using dried blood spot analyses as comparable to venipuncture (McDade, Williams, and Snodgrass 2007; Varnier et al. 1988; Williams and McDade 2009), the fact that these two studies used the same biomarker (A1C) collected in different ways among similar samples and produced drastically different estimates is cause for concern. The Add Health data have prevalence estimates that were 3.09 times higher for pre-diabetes and 5.40 times as high for undiagnosed diabetes compared to the NHANES despite equivalent (1.07) levels of diagnosed diabetes. If venipuncture produces more reliable estimates of true hemoglobin A1C values or dried blood spots concentrate glycosylated hemoglobin differently, this could explain some of the difference seen here and would support the view point presented by Johnson and colleagues (2007) that more analysis is needed on the comparability of the measures. However, the similar prevalence estimate differences for diagnosed and undiagnosed hypertension by Chyu, McDade, and Adam (2011) coupled with the differences in means among complete cases in the analyses presented here suggest that these differences may more likely be due to sample composition differences as opposed to invalid or unreliable biomarkers. Both scenarios warrant future research, but are beyond the scope of this study.

Conclusions

Three key conclusions can be drawn from this study. Nonresponse bias does not appear to be an issue for estimates of undiagnosed diabetes either study, however the importance of race-based nonresponse remains unclear. The persistence of large differences in prevalence estimates for undiagnosed pre-diabetes and undiagnosed diabetes that cannot be explained by nonresponse suggests that either the studies sample different populations or the testing strategies used to generate the hemoglobin A1C values contribute to the differences in prevalence estimates. Both issues warrant further investigation. Finally, the sizeable relationship between undiagnosed diabetes for non-Hispanic blacks warrants considerable investigation to determine what influences undiagnosed diabetes in non-Hispanic black young adults at rates so much higher than non-Hispanic white and Hispanic young adults.

2.8 References

- American Diabetes Association .2013 <<http://professional.diabetes.org/Admin/UserFiles/0%20-%20Sean/FastFacts%20March%202013.pdf>> Accessed March 20, 2014
- Andridge, Rebecca R., and Roderick JA Little.2010."A Review of Hot Deck Imputation for Survey Nonresponse." *International Statistical Review* 78, (1): 40-64.
- Benyamini, Yael, Colleen S. McClain, Elaine A. Leventhal, and Howard Leventhal. 2003. "Living with the Worry of Cancer: Health Perceptions and Behaviors of Elderly People with Self, Vicarious, or No History of Cancer." *Psycho-Oncology*. 12:161-172.
http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/generaldoc_e.htm
http://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_lab.pdf; p.17).
- Chantala, Kim and Joyce Tabor. 1999. "National Longitudinal Study of Adolescent Health: Strategies to Perform a Design-Based Analysis Using the Add Health Data." Chapel Hill, NC: Carolina Population Center.
- Chakrabarti, Subrata. 2000."Diabetic Retinopathy in Experimental Animal Models and their Feasibility for Understanding the Human Disease." in Anders A.F. Sima and Eleazar Shafir (Eds.) *Chronic Complications in Diabetes: Animal Models and Chronic Complications*. Amsterdam, Netherlands. Pp.229-250.
- Chyu, Laura, Thomas W. McDade, and Emma K. Adam. 2011. "Measured Blood Pressure and Hypertension among Young Adults: A Comparison between Two Nationally Representative Samples." *Biodemography and social biology* 57, (2): 184-199.
- Crimmins, Eileen M., Jung Ki Kim, Dawn E. Alley, Arun Karlamangla, and Teresa Seeman. 2007."Hispanic paradox in biological risk profiles." *American Journal of Public Health* 97, (7): 1305-1310.
- Groves, Robert M., and Emilia Peytcheva. 2008. "The impact of nonresponse rates on nonresponse bias a meta-analysis." *Public opinion quarterly* 72.2: 167-189.
- Hammond, Wizdom Powell. 2010."Psychosocial correlates of medical mistrust among African American men." *American journal of community psychology* 45, no. 1-2: 87-106.
- Harris, K.M., C.T. Halpern, E. Whitsel, J. Hussey, J. Tabor, P. Entzel, and J.R. Udry. 2009. "The National Longitudinal Study of Adolescent Health: Research Design [WWW document]". URL: <http://www.cpc.unc.edu/projects/addhealth/design>.

- Johnson, C., Lacher, D., Lewis, B., & McQuillan, G. 2007. "FEATURE PAPER: Challenges in Collecting Survey-Based Biomarker and Genetic Data: The NHANES Experience." *SESSION 1: EMERGENCY PREPAREDNESS AND SURVEILLANCE*, 139.
- Juster, F. Thomas, and James P. Smith. 1997. "Improving the quality of economic data: Lessons from the HRS and AHEAD." *Journal of the American Statistical Association* 92.440: 1268-1278.
- Kalsbeek, William D, Morris, Carolyn, B., and Vaughn, Benjamin J. 2001. "Effects of nonresponse on the mean squared error of estimates from longitudinal study", ASA Proceedings of the Joint Statistical Meetings, 2001.
- Kash K.M., Holland J.C., Halper M.S. and Miller D.G. 1992. "Psychological Distress and Surveillance Behaviors of Women with a Family History of Breast Cancer." *Journal of the National Cancer Institute*, 84(1), 24-30.
- Kenen, Regina, Audrey Arden-Jones, and Rosalind Eeles. 2003. "Living with Chronic Risk: Healthy Women with a Family History of Breast/Ovarian Cancer." *Health, Risk, and Society*. 5(3):315-331.
- Köszegi, Botond. 2003. "Health anxiety and patient behavior." *Journal of health economics* 22, no. 6 1073-1084.
- Lim, Jennifer N. W., Jenny Hewison, Carol E. Chu, and Hamdan Al-Habsi. 2011. "Factors Influencing Consultation to Discuss Family History of Cancer by Asymptomatic Patients in Primary Care." *Journal of Community Genetics*. 2:19-26
- Lyles, Courtney, Andrew Karter, Bessie Young, Clarence Spingner, David Grembowski, Dean Schillinger, and Nancy Adler. 2011. "Patient-Reported Racial/Ethnic Healthcare Provider Discrimination and Medication Intensification in the Diabetes Study of Northern California (Distance)." *Journal of General Internal Medicine*.26(10):1138-1144.
- Olson DE, Rhee MK, Herrick K, Ziemer DC, Twombly JG, Phillips LS. 2010. "Screening for diabetes and pre-diabetes with proposed A1C-based diagnostic criteria." *Diabetes Care*. 33(10):2184–2189
- Nichols, Gregory A., Bhakti Arondekar, and William H. Herman. 2008. "Medical care costs one year after identification of hyperglycemia below the threshold for diabetes." *Medical care* 46, no. 3 :287-292.
- Nichols, Gregory A., and Jonathan B. Brown.2005. "Higher medical care costs accompany impaired fasting glucose." *Diabetes Care* 28, no. 9 : 2223-2229.

- Pandya, Ankur, Milton C. Weinstein, and Thomas A. Gaziano. 2011. "A comparative assessment of non-laboratory-based versus commonly used laboratory-based cardiovascular disease risk scores in the NHANES III population." *PloS one* 6, no. 5: e20416.
- Schafer, J., Ezzati-Rice, T. M., Johnson, W., Khare, M., Little, R., & Rubin III, D. 1996. "The NHANES III multiple imputation project." *Race/ethnicity*, 60(21.2), 15-5.
- Stepanakova, Irena. 2012. "Racial-Ethnic Biases, Time Pressure, and Medical Decisions." *Journal of Health and Social Behavior*. 53(3)329-343.
- Trull, Andrew, Lawrence M. Demers, David W. Holt, Atholl Johnston, J. Michael Tredger, and Christopher P. Price. 2002. *Biomarkers of Disease: An Evidence Based Approach*. Cambridge University Press: New York, New York.
- Vaccaro, Joan A., and Fatma G. Huffman. 2012. "Reducing health disparities: medical advice received for minorities with diabetes." *Journal of Health & Human Services Administration* 34, no.
- van Osch, Sylvie MC, Wilbert B. van den Hout, and Anne M. Stiggelbout. 2006. "Exploring the reference point in prospect theory: Gambles for length of life." *Medical Decision Making* 26, (4): 338-346.
- Varnier, Oliviero E., F B. Lillo, S Reina, A. De Maria, A. Terranga, and G. Schito 1988. "Whole blood collection on filter paper is an effective means of obtaining samples for human immunodeficiency virus antibody assay." *AIDS research and human retroviruses* 4, (2): 131-136.
- Vernon S. 1999. "Risk Perception and Risk Communication for Cancer Screening Behaviors: a review." *Journal of the National Cancer Institute. Monographs*, 25, 101-119.
- Walker, Elizabeth A., Maria R. Kalten, C.K. Mertz, and James Flynn. 2003. "Risk Perception for Developing Diabetes: Comparative Risk Judgments by Physicians." *Diabetes Care*. 26: 2543-2548.
- White, Kellee, Jennifer S. Haas, and David R. Williams. 2012. "Elucidating the role of place in health care disparities: the example of racial/ethnic residential segregation." *Health services research* 47, no. 3.2: 1278-1299.
- Whitsel, Eric A., Joyce W. Tabor, Quynh C. Nguyen, Carmen C. Cuthbertson, Mark H. Wener, Alan J. Potter, Ley A. Killeya-Jones, and Kathleen Mullan Harris. 2012. *Add Health Wave IV Documentation Report: Measures of Glucose Homeostasis*. Chapel Hill, NC: UNC Chapel Hill: Carolina Population Center
- Williams, Sharon R., and Thomas W. McDade. 2009. "The use of dried blood spot

sampling in the National Social Life, Health, and Aging Project." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 64, no. suppl 1 i131-i136.

World Health Organization. 2012. "Diabetes Fact Sheet."
<http://www.who.int/mediacentre/factsheets/fs312/en/index.html>. Accessed 11/4/2012.

Zhang Y, Dall TM, Mann SE, Chen Y, Martin J, Moore V, et al. 2009. "The economic costs of undiagnosed diabetes." *Population Health Management*. 12(2):95–101.

2.9 Tables

Table 2.1 Weighted Descriptive Statistics Comparing Age-Matched 2007-2010 NHANES to 2008-2009 Add Health

Variable	NHANES Mean	NHANES 95% CI	Add Health Mean	Add Health 95% CI	No Overlap of Intervals
<i>Diabetes Status</i>					
A1C Unknown	0.07	(0.05,0.09)	0.07	(0.06,0.08)	
Not Diabetic	0.82	(0.80,0.84)	0.63	(0.61,0.65)	#
Pre-Diabetic	0.08	(0.07,0.09)	0.25	(0.23,0.26)	#
Undiagnosed Diabetic	0.00	(0.00,0.01)	0.02	(0.02,0.03)	#
Diagnosed Diabetic	0.02	(0.02,0.03)	0.03	(0.02,0.03)	
<i>Demographics</i>					
Non-Hispanic White	0.75	(0.69,0.80)	0.74	(0.68,0.79)	
Non-Hispanic Black	0.15	(0.11,0.18)	0.16	(0.12,0.21)	
Hispanic	0.10	(0.07,0.14)	0.10	(0.07,0.13)	
Male	0.50	(0.47,0.53)	0.52	(0.50,0.53)	
Female	0.50	(0.47,0.53)	0.48	(0.47,0.50)	
Age	29.27 [^]	(29.06,29.49)	28.95 [^]	(28.71,29.19)	
<i>Socioeconomic Status</i>					
High School or Less	0.38	(0.34,0.42)	0.26	(0.23,0.28)	#
Some College	0.34	(0.31,0.37)	0.44	(0.42,0.46)	#
College or Advanced Degree	0.28	(0.23,0.32)	0.31	(0.27,0.34)	
<i>Income</i>					
\$0 to \$19,999	0.14	(0.11,0.17)	0.30	(0.28,0.32)	#
\$20,000 to \$34,999	0.17	(0.14,0.20)	0.28	(0.27,0.29)	#
\$35,000 to \$54,999	0.19	(0.17,0.22)	0.25	(0.24,0.27)	#
\$55,000 to \$74,999	0.16	(0.13,0.19)	0.10	(0.09,0.11)	#
\$75,000 to \$99,999	0.17	(0.14,0.19)	0.04	(0.03,0.05)	#
Over \$100,000	0.17	(0.13,0.20)	0.02	(0.02,0.03)	#
Has Health Insurance	0.73	(0.70,0.77)	0.78	(0.76,0.80)	
No Health Insurance	0.27	(0.23,0.30)	0.22	(0.20,0.24)	
<i>Health Behaviors</i>					
Fast food consumption	0.82	(0.79,0.84)	0.76	(0.74,0.78)	#
Hasn't seen a doctor in past year	0.20	(0.17,0.23)	0.42	(0.40,0.43)	#
<i>Health Status</i>					
Underweight	0.02	(0.01,0.02)	0.01	(0.01,0.02)	
Normal Weight	0.36	(0.33,0.38)	0.30	(0.28,0.31)	#
Overweight	0.29	(0.26,0.32)	0.31	(0.30,0.32)	
Obese	0.33	(0.31,0.36)	0.38	(0.36,0.40)	
Diagnosed High Cholesterol	0.19	(0.11,0.27)	0.08	(0.07,0.09)	#
Diagnosed High Blood Pressure	0.12	(0.10,0.14)	0.11	(0.10,0.12)	
Self-Rated Health	3.52 [^]	(3.44,3.60)	3.65 [^]	(3.62,3.69)	#
N	1576		12483		

Notes: a. NHANES means and proportions are calculated using data from the 2007-2008 and 2009-2010 cohorts of individuals 25-34 at time of interview; b. Add Health means are calculated using data from Wave IV when respondents were 25-34; c. Both samples only include individuals with complex survey design adjustment weights; d. Other race, immigrants, and pregnant women excluded from analyses; e. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history.; f. pre-imputation means and confidence intervals reported; g. [^] indicates mean instead of proportion; h.# indicates no overlap of confidence intervals of weighted means

Table 2.1a Weighted Descriptive Statistics Comparing 2007-2010 NHANES Respondents With and Without A1C Readings

Variable	NHANES Mean	NHANES 95% CI	NHANES Missing Mean	NHANES Missing 95% CI	No Overlap of Intervals
<i>Diabetes Status</i>					
A1C Unknown	0.07	(0.05,0.09)			
Not Diabetic	0.82	(0.80,0.84)			
Pre-Diabetic	0.08	(0.07,0.09)			
Undiagnosed Diabetic	0.00	(0.00,0.01)			
Diagnosed Diabetic	0.02	(0.02,0.03)			
<i>Demographics</i>					
Non-Hispanic White	0.75	(0.69,0.80)	0.63	(0.53,0.73)	
Non-Hispanic Black	0.15	(0.11,0.18)	0.29	(0.19,0.39)	#
Hispanic	0.10	(0.07,0.14)	0.07	(0.03,0.12)	
Male	0.50	(0.47,0.53)	0.52	(0.44,0.60)	
Female	0.50	(0.47,0.53)	0.48	(0.40,0.56)	
Age	29.27 [^]	(29.06,29.49)	29.63 [^]	(28.86,30.40)	
<i>Socioeconomic Status</i>					
High School or Less	0.38	(0.34,0.42)	0.50	(0.37,0.63)	
Some College	0.34	(0.31,0.37)	0.31	(0.16,0.45)	
College or Advanced Degree	0.28	(0.23,0.32)	0.20	(0.05,0.34)	
<i>Income</i>					
\$0 to \$19,999	0.14	(0.11,0.17)	0.15	(0.08,0.22)	
\$20,000 to \$34,999	0.17	(0.14,0.20)	0.14	(0.04,0.25)	
\$35,000 to \$54,999	0.19	(0.17,0.22)	0.19	(0.09,0.29)	
\$55,000 to \$74,999	0.16	(0.13,0.19)	0.14	(0.02,0.25)	
\$75,000 to \$99,999	0.17	(0.14,0.19)	0.15	(0.05,0.26)	
Over \$100,000	0.17	(0.13,0.20)	0.23	(0.10,0.35)	
Has Health Insurance	0.73	(0.70,0.77)	0.78	(0.67,0.88)	
No Health Insurance	0.27	(0.23,0.30)	0.22	(0.12,0.33)	
<i>Health Behaviors</i>					
Fast food consumption	0.82	(0.79,0.84)	0.88	(0.81,0.96)	
Hasn't seen a doctor in past year	0.20	(0.17,0.23)	0.18	(0.10,0.26)	
<i>Health Status</i>					
Underweight	0.02	(0.01,0.02)	0.01	(-0.01,0.02)	
Normal Weight	0.36	(0.33,0.38)	0.36	(0.27,0.44)	
Overweight	0.29	(0.26,0.32)	0.31	(0.21,0.40)	
Obese	0.33	(0.31,0.36)	0.33	(0.25,0.42)	
Diagnosed High Cholesterol	0.19	(0.11,0.27)	0.13	(0.02,0.23)	
Diagnosed High Blood Pressure	0.12	(0.10,0.14)	0.16	(0.08,0.24)	
Self-Rated Health	3.52 [^]	(3.44,3.60)	3.43 [^]	(3.20,3.67)	
N	1576		166		

Notes: a. NHANES means are calculated using data from the 2007-2008 and 2009-2010 cohorts of individuals 25-34 at time of interview; b. NHANES missing means calculated using the subsample of respondents who did not have hemoglobin A1C values; c. Both samples only include individuals with complex survey design adjustment weights; d. Other race, immigrants, and pregnant women excluded from analyses; e. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history.; f. pre-imputation means and confidence intervals reported; g. [^] indicates mean instead of proportion; h.# indicates no overlap of confidence intervals of weighted means

Table 2.1b Weighted Descriptive Statistics Comparing 2008-2009 Add Health Respondents With and Without A1C Readings

Variable	Add Health Mean	Add Health 95% CI	Add Health Missing Mean	Add Health Missing 95% CI	No Overlap of Intervals
<i>Diabetes Status</i>					
A1C Unknown	0.07	(0.06,0.08)			
Not Diabetic	0.63	(0.61,0.65)			
Pre-Diabetic	0.25	(0.23,0.26)			
Undiagnosed Diabetic	0.02	(0.02,0.03)			
Diagnosed Diabetic	0.03	(0.02,0.03)			
<i>Demographics</i>					
Non-Hispanic White	0.74	(0.68,0.79)	0.64	(0.56,0.72)	
Non-Hispanic Black	0.16	(0.12,0.21)	0.24	(0.17,0.31)	
Hispanic	0.10	(0.07,0.13)	0.12	(0.08,0.16)	
Male	0.52	(0.50,0.53)	0.62	(0.56,0.67)	#
Female	0.48	(0.47,0.50)	0.38	(0.33,0.44)	#
Age	28.95[^]	(28.71,29.19)	28.95[^]	(28.65,29.26)	
<i>Socioeconomic Status</i>					
High School or Less	0.26	(0.23,0.28)	0.27	(0.20,0.35)	
Some College	0.44	(0.42,0.46)	0.36	(0.31,0.41)	#
College or Advanced Degree	0.31	(0.27,0.34)	0.37	(0.30,0.43)	
<i>Income</i>					
\$0 to \$19,999	0.30	(0.28,0.32)	0.27	(0.22,0.33)	
\$20,000 to \$34,999	0.28	(0.27,0.29)	0.23	(0.19,0.27)	
\$35,000 to \$54,999	0.25	(0.24,0.27)	0.29	(0.24,0.33)	
\$55,000 to \$74,999	0.10	(0.09,0.11)	0.12	(0.09,0.14)	
\$75,000 to \$99,999	0.04	(0.03,0.05)	0.06	(0.04,0.08)	
Over \$100,000	0.02	(0.02,0.03)	0.03	(0.02,0.04)	
Has Health Insurance	0.78	(0.76,0.80)	0.76	(0.72,0.80)	
No Health Insurance	0.22	(0.20,0.24)	0.24	(0.20,0.28)	
<i>Health Behaviors</i>					
Fast food consumption	0.76	(0.74,0.78)	0.76	(0.71,0.80)	
Hasn't seen a doctor in past year	0.42	(0.40,0.43)	0.39	(0.34,0.43)	
<i>Health Status</i>					
Underweight	0.01	(0.01,0.02)	0.03	(0.01,0.04)	
Normal Weight	0.30	(0.28,0.31)	0.34	(0.30,0.38)	
Overweight	0.31	(0.30,0.32)	0.33	(0.28,0.37)	
Obese	0.38	(0.36,0.40)	0.30	(0.26,0.35)	#
Diagnosed High Cholesterol	0.08	(0.07,0.09)	0.06	(0.04,0.08)	
Diagnosed High Blood Pressure	0.11	(0.10,0.12)	0.10	(0.07,0.13)	
Self-Rated Health	3.65[^]	(3.62,3.69)	3.87[^]	(3.78,3.95)	#
N	12483		1039		

Notes: a. Add Health means are calculated using data from Wave IV when respondents were 25-34; b. Add Health missing means calculated using the subsample of respondents who did not have hemoglobin A1C values; c. Both samples only include individuals with complex survey design adjustment weights; d. Other race, immigrants, and pregnant women excluded from analyses; e. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history.; f. pre-impputation means and confidence intervals reported; g. [^] indicates mean instead of proportion; h.# indicates no overlap of confidence intervals of weighted means

Table 2.2 Weighted Descriptive Statistics Comparing Age-Matched 2007-2010 NHANES to 2008-2009 Add Health Missing A1C Status

Variable	NHANES Mean	NHANES 95% CI	Add Health Mean	Add Health 95% CI	No Overlap of Intervals
<i>Demographics</i>					
Non-Hispanic White	0.63	0.53 0.73	0.64	0.56 0.72	
Non-Hispanic Black	0.29	0.19 0.39	0.24	0.17 0.31	
Hispanic	0.07	0.03 0.12	0.12	0.08 0.16	
Male	0.52	0.44 0.60	0.62	0.56 0.67	
Female	0.48	0.40 0.56	0.38	0.33 0.44	
Age	29.63[^]	28.86 30.40	28.95[^]	28.65 29.26	
<i>Socioeconomic Status</i>					
High School or Less	0.50	0.37 0.63	0.27	0.20 0.35	#
Some College	0.31	0.16 0.45	0.36	0.31 0.41	
College or Advanced Degree	0.20	0.05 0.34	0.37	0.30 0.43	
<i>Income</i>					
\$0 to \$19,999	0.15	0.08 0.22	0.27	0.22 0.33	
\$20,000 to \$34,999	0.14	0.04 0.25	0.23	0.19 0.27	
\$35,000 to \$54,999	0.19	0.09 0.29	0.29	0.24 0.33	
\$55,000 to \$74,999	0.14	0.02 0.25	0.12	0.09 0.14	
\$75,000 to \$99,999	0.15	0.05 0.26	0.06	0.04 0.08	
Over \$100,000	0.23	0.10 0.35	0.03	0.02 0.04	#
Has Health Insurance	0.78	0.67 0.88	0.76	0.72 0.80	
No Health Insurance	0.22	0.12 0.33	0.24	0.20 0.28	
<i>Health Behaviors</i>					
Fast food consumption	0.88	0.81 0.96	0.76	0.71 0.80	#
Hasn't seen a doctor in past year	0.18	0.10 0.26	0.39	0.34 0.43	#
<i>Health Status</i>					
Underweight	0.01	-0.01 0.02	0.03	0.01 0.04	
Normal Weight	0.36	0.27 0.44	0.34	0.30 0.38	
Overweight	0.31	0.21 0.40	0.33	0.28 0.37	
Obese	0.33	0.25 0.42	0.30	0.26 0.35	
Diagnosed High Cholesterol	0.13	0.02 0.23	0.06	0.04 0.08	
Diagnosed High Blood Pressure	0.16	0.08 0.24	0.10	0.07 0.13	
Self-Rated Health	3.43[^]	3.20 3.67	3.87[^]	3.78 3.95	#
N	166		1039		
Proportion	0.07		0.07		

Notes: a. NHANES means are calculated using data from the 2007-2008 and 2009-2010 cohorts of individuals 25-34 at time of interview; b. Add Health means are calculated using data from Wave IV when respondents were 25-34; c. Both samples only include individuals with complex survey design adjustment weights; d. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history; e. [^] indicates mean instead of proportion; f. # indicates no overlap of confidence intervals of weighted means

Table 2.3 Weighted Predictors of Missing Hemoglobin A1C values Comparing Age-Matched 2007-2010 NHANES to 2008-2009 Add Health Odds Ratios Reported

Variable	NHANES		Sig.	Add Health		No Overlap Sig. of Intervals
	Odds Ratio	95% CI		Odds Ratio	95% CI	
<i>Demographics</i>						
Non-Hispanic Black	2.65	(1.68,4.16)	***	1.82	(1.37,2.40)	***
Hispanic	1.04	(0.61,1.77)		1.49	(1.07,2.09)	*
Male	1.11	(0.77,1.61)		1.54	(1.21,1.96)	***
Age	1.02	(0.96,1.09)		0.99	(0.93,1.05)	
<i>Socioeconomic Status</i>						
High School or Less	1.43	(0.77,2.67)		1.19	(0.83,1.70)	
College or Advanced Degree	0.89	(0.37,2.14)		1.42	(1.14,1.75)	**
<i>Income</i>						
\$20,000 to \$34,999	1.14	(0.43,3.00)		0.92	(0.70,1.22)	
\$35,000 to \$54,999	1.37	(0.61,3.10)		1.22	(0.91,1.63)	
\$55,000 to \$74,999	1.07	(0.36,3.22)		1.24	(0.87,1.75)	
\$75,000 to \$99,999	1.55	(0.56,4.28)		1.53	(0.95,2.44)	
Over \$100,000	2.28	(0.87,5.96)		1.27	(0.74,2.21)	
No Health Insurance	0.71	(0.36,1.41)		1.15	(0.87,1.54)	
<i>Health Behaviors</i>						
Fast food consumption	1.59	(0.81,3.13)		0.96	(0.76,1.20)	
Hasn't seen a doctor in past year	0.83	(0.44,1.58)		0.83	(0.68,1.02)	
<i>Health Status</i>						
Underweight	1.44	(0.21,9.92)		2.15	(1.22,3.81)	**
Overweight	0.86	(0.51,1.45)		0.88	(0.69,1.13)	
Obese	0.76	(0.46,1.25)		0.75	(0.58,0.97)	*
Diagnosed High Cholesterol	0.40	(0.15,1.09)		0.88	(0.61,1.27)	
Diagnosed High Blood Pressure	1.63	(0.86,3.11)		0.99	(0.70,1.41)	
Self-Rated Health	0.88	(0.61,1.29)		1.25	(1.11,1.41)	***

Notes: a. NHANES means are calculated using data from the 2007-2008 and 2009-2010 cohorts of individuals 25-34 at time of interview; b. Add Health means are calculated using data from Wave IV when respondents were 25-34; c. Both samples only include individuals with complex survey design adjustment weights; d. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history.; e. Odds Ratios Reported; f. * p<0.05, ** p<0.01, *** p<0.001; g. # indicates no overlap of confidence intervals of odds ratios

Table 2.4a Relative Risk Ratios and Confidence Intervals Comparing Age-Matched 2007-2010 NHANES to 2008-2009 Add Health Missing A1C Status (Imputed) Predicting Pre-Diabetes Relative to No Diabetic History

Variable	NHANES Risk Ratio	NHANES 95% CI	Sig.	Add Health Risk Ratio	Add Health 95% CI	Sig.	No Overlap of Intervals
<i>Missing A1C</i>							
A1C Unknown (pre-imputed)	0.65	(0.21,2.05)		0.98	(0.66,1.47)		
<i>Demographics</i>							
Non-Hispanic Black	3.40	(2.10,5.51)	***	3.33	(2.85,3.89)	***	
Hispanic	1.46	(0.82,2.62)		1.91	(1.56,2.35)	***	
Male	1.26	(0.79,2.02)		1.71	(1.47,1.99)	***	
Age	1.06	(0.98,1.14)		1.06	(1.02,1.11)	***	
<i>Socioeconomic Status</i>							
High School or Less	1.47	(1.02,2.11)	*	1.06	(0.91,1.23)		
College or Advanced Degree	0.72	(0.36,1.45)		0.73	(0.62,0.87)	***	
<i>Income</i>							
\$20,000 to \$34,999	1.27	(0.69,2.35)		1.15	(0.96,1.38)		
\$35,000 to \$54,999	0.95	(0.40,2.23)		1.05	(0.87,1.27)		
\$55,000 to \$74,999	0.66	(0.26,1.65)		0.91	(0.67,1.23)		
\$75,000 to \$99,999	0.78	(0.34,1.81)		1.12	(0.68,1.85)		
Over \$100,000	0.46	(0.20,1.07)		0.79	(0.51,1.21)		
No Health Insurance	1.05	(0.64,1.72)		1.04	(0.90,1.19)		
<i>Health Behaviors</i>							
Hasn't seen a doctor in past year	0.82	(0.50,1.35)		0.93	(0.82,1.06)		
<i>Health Status</i>							
Obese	3.38	(2.44,4.67)	***	2.09	(1.86,2.36)	***	#
Diagnosed High Cholesterol	1.67	(0.60,4.67)		1.34	(1.09,1.65)	**	
Diagnosed High Blood Pressure	1.03	(0.51,2.10)		1.06	(0.85,1.33)		
Self-Rated Health	0.94	(0.74,1.20)		0.91	(0.85,0.98)	*	

Notes: a. NHANES estimates are calculated using data from the 2007-2008 and 2009-2010 cohorts of individuals 25-34 at time of interview; b. Add Health estimates are calculated using data from Wave IV when respondents were 25-34; c. Both samples only include individuals with complex survey design adjustment weights; d. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history.; e. weight categories truncated to Obese vs. not obese due to too few cases for analysis with four weight categories; f. Fast food consumption omitted because NHANES models would not converge with it included; g. Relative Risk Ratios Reported; h. * p<0.05, ** p<0.01, *** p<0.001; i. # indicates no overlap of confidence intervals of odds ratios

Table 2.4b Relative Risk Ratios and Confidence Intervals Comparing Age-Matched 2007-2010 NHANES to 2008-2009 Add Health Missing A1C Status (Imputed) Predicting Undiagnosed Diabetes Relative to No Diabetic History

Variable	NHANES Risk Ratio	NHANES 95% CI	Sig.	Add Health Risk Ratio	Add Health 95% CI	No Overlap Sig. of Intervals
<i>Missing A1C</i>						
A1C Unknown (pre-imputed)	0.00	(0.00,1.63E+10)		0.96	(0.36,2.56)	
<i>Demographics</i>						
Non-Hispanic Black	14.73	(1.97,110.10)	*	29.03	(17.99,46.84)	***
Hispanic	7.68	(1.34,44.17)	*	6.61	(3.62,12.07)	***
Male	3.25	(0.77,13.64)		1.74	(1.26,2.40)	**
Age	1.01	(0.89,1.16)		1.12	(1.02,1.23)	*
<i>Socioeconomic Status</i>						
High School or Less	1.65	(0.29,9.49)		0.96	(0.66,1.38)	
College or Advanced Degree	0.00	(0.00,0.00)	***	0.87	(0.60,1.27)	#
<i>Income</i>						
\$20,000 to \$34,999	0.81	(0.12,5.55)		0.86	(0.55,1.33)	
\$35,000 to \$54,999	1.82	(0.28,12.02)		0.79	(0.49,1.26)	
\$55,000 to \$74,999	1.70	(0.14,21.09)		0.82	(0.43,1.59)	
\$75,000 to \$99,999	0.00	(0.00,0.00)	***	0.69	(0.24,1.97)	#
Over \$100,000	0.00	(0.00,0.00)	***	0.55	(0.17,1.77)	#
No Health Insurance	1.61	(0.29,9.05)		0.95	(0.63,1.43)	
<i>Health Behaviors</i>						
Hasn't seen a doctor in past year	1.28	(0.31,5.23)		1.15	(0.81,1.62)	
<i>Health Status</i>						
Obese	17.45	(1.66,183.72)	*	3.18	(2.28,4.43)	***
Diagnosed High Cholesterol	2.48	(0.26,23.79)		1.89	(1.06,3.34)	*
Diagnosed High Blood Pressure	1.13	(0.11,11.77)		1.52	(1.00,2.29)	*
Self-Rated Health	0.69	(0.30,1.58)		0.90	(0.73,1.10)	

Notes: a. NHANES estimates are calculated using data from the 2007-2008 and 2009-2010 cohorts of individuals 25-34 at time of interview; b. Add Health estimates are calculated using data from Wave IV when respondents were 25-34; c. Both samples only include individuals with complex survey design adjustment weights; d. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history; e. weight categories truncated to Obese vs. not obese due to too few cases for analysis with four weight categories; f. Fast food consumption omitted because NHANES models would not converge with it included; g. Relative Risk Ratios Reported; h. * p<0.05, ** p<0.01, *** p<0.001; i. # indicates no overlap of confidence intervals of odds ratios

Table 2.4c Relative Risk Ratios and Confidence Intervals Comparing Age-Matched 2007-2010 NHANES to 2008-2009 Add Health Missing A1C Status (Imputed) Predicting Diagnosed Diabetes Relative to No Diabetic History

Variable	NHANES Risk Ratio	NHANES 95% CI	Sig.	Add Health Risk Ratio	Add Health 95% CI	No Overlap Sig. of Intervals
<i>Missing A1C</i>						
A1C Unknown (pre-imputed)	1.19	(0.11,12.79)		1.39	(0.57,3.42)	
<i>Demographics</i>						
Non-Hispanic Black	1.29	(0.54,3.08)		2.36	(1.65,3.37)	***
Hispanic	1.35	(0.47,3.81)		1.69	(1.02,2.80)	*
Male	1.48	(0.63,3.47)		1.13	(0.76,1.68)	
Age	1.04	(0.91,1.19)		1.11	(1.01,1.22)	*
<i>Socioeconomic Status</i>						
High School or Less	0.66	(0.26,1.68)		1.61	(1.10,2.34)	*
College or Advanced Degree	0.74	(0.22,2.53)		0.75	(0.50,1.13)	
<i>Income</i>						
\$20,000 to \$34,999	0.95	(0.36,2.48)		0.89	(0.57,1.40)	
\$35,000 to \$54,999	0.61	(0.23,1.66)		1.19	(0.75,1.90)	
\$55,000 to \$74,999	0.76	(0.19,3.11)		0.95	(0.43,2.09)	
\$75,000 to \$99,999	0.52	(0.12,2.26)		0.35	(0.09,1.34)	
Over \$100,000	0.10	(0.02,0.58)	*	0.45	(0.11,1.91)	
No Health Insurance	0.81	(0.37,1.80)		1.03	(0.69,1.55)	
<i>Health Behaviors</i>						
Hasn't seen a doctor in past year	0.34	(0.07,1.64)		0.65	(0.46,0.91)	*
<i>Health Status</i>						
Obese	2.64	(1.19,5.82)	*	2.87	(1.97,4.18)	***
Diagnosed High Cholesterol	6.16	(1.54,24.67)	*	3.25	(2.18,4.84)	***
Diagnosed High Blood Pressure	0.57	(0.10,3.27)		2.14	(1.44,3.20)	***
Self-Rated Health	0.77	(0.43,1.39)		0.52	(0.43,0.62)	***

Notes: a. NHANES estimates are calculated using data from the 2007-2008 and 2009-2010 cohorts of individuals 25-34 at time of interview; b. Add Health estimates are calculated using data from Wave IV when respondents were 25-34; c. Both samples only include individuals with complex survey design adjustment weights; d. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history; e. weight categories truncated to Obese vs. not obese due to too few cases for analysis with four weight categories; f. Fast food consumption omitted because NHANES models would not converge with it included; g. Relative Risk Ratios Reported; h. * p<0.05, ** p<0.01, *** p<0.001; i. # indicates no overlap of confidence intervals of odds ratios

Table 2.5 Descriptive Statistics of Non-Hispanic Blacks Missing A1C Status in 2007-2010 NHANES vs. 2008-2009 Add Health

Variable	NHANES Mean	NHANES 95% CI	Add Health Mean	Add Health 95% CI	No Overlap of Intervals
<i>Demographics</i>					
Male	0.48	(0.31,0.66)	0.68	(0.58,0.78)	
Female	0.52	(0.34,0.69)	0.32	(0.22,0.42)	
Age	28.88 ^a	(28.39,31.37)	29.12 ^a	(28.51,29.74)	
<i>Socioeconomic Status</i>					
High School or Less	0.60	(0.53,0.67)	0.35	(0.25,0.46)	#
Some College	0.32	(0.19,0.44)	0.35	(0.28,0.42)	
College or Advanced Degree	0.08	(-0.03,0.20)	0.30	(0.20,0.40)	
<i>Income</i>					
\$0 to \$19,999	0.24	(0.06,0.43)	0.41	(0.30,0.52)	
\$20,000 to \$34,999	0.13	(0.00,0.26)	0.19	(0.13,0.24)	
\$35,000 to \$54,999	0.25	(0.07,0.42)	0.26	(0.18,0.34)	
\$55,000 to \$74,999	0.07	(-0.07,0.20)	0.11	(0.06,0.16)	
\$75,000 to \$99,999	0.09	(-0.04,0.23)	0.03	(0.01,0.05)	
Over \$100,000	0.22	(-0.07,0.51)	0.01	(-0.01,0.02)	
Has Health Insurance	0.75	(0.64,0.86)	0.66	(0.57,0.74)	
No Health Insurance	0.25	(0.14,0.36)	0.34	(0.26,0.43)	
<i>Health Behaviors</i>					
Fast food consumption	0.82	(0.65,0.99)	0.85	(0.79,0.91)	
Hasn't seen a doctor in past year	0.17	(-0.03,0.37)	0.28	(0.21,0.35)	
<i>Health Status</i>					
Underweight	0.03	(-0.04,0.11)	0.01	(0.00,0.03)	
Normal Weight	0.25	(0.08,0.42)	0.27	(0.19,0.35)	
Overweight	0.23	(0.09,0.38)	0.31	(0.22,0.39)	
Obese	0.48	(0.23,0.73)	0.41	(0.34,0.48)	
Diagnosed High Cholesterol	0.24	(-0.12,0.60)	0.07	(0.03,0.12)	
Diagnosed High Blood Pressure	0.10	(-0.05,0.24)	0.18	(0.09,0.27)	
Self-Rated Health	3.18 ^a	(2.72,3.64)	3.76 ^a	(3.61,3.91)	
N	40		332		

Notes: a. NHANES means are calculated using data from the 2007-2008 and 2009-2010 cohorts of Non-Hispanic Black individuals aged 25-34 and missing A1C data at the time of interview; b. Add Health means are calculated using data from Non-Hispanic Black respondents in Wave IV when respondents were aged 25-34; c. Both samples only include individuals with complex survey design adjustment weights; d. A1C Unknown determined by individuals with survey responses, but missing hemoglobin A1C values and no diabetic history; e. 23 NHANES cases not included in mean calculations because confidence intervals could not be computed due to too few cases per stratum to calculate variances. f. ^a indicates mean instead of proportion; g. # indicates no overlap of confidence intervals of weighted means

CHAPTER 3**Unequal Exposure and Unequal Risk:
Race Stratified Diabetes Risk Severity in Early Adulthood****Abstract**

Discrimination is pervasive in America. Despite this, few studies have examined how inequitable exposure to the social and emotional stress of discrimination impacts racial health disparities for diabetes onset and severity in young adults. This study uses an adaptation of the Stress Process Model to identify the effects of racial minority status, perceived discrimination, mastery, and risky coping strategies on diabetes severity in a race-stratified young adult sample (N=10,723). Biomarker and survey data from the National Longitudinal Study of Adolescent Health were used to analyze diabetes risk severity using multinomial logistic regression analysis. Descriptive results demonstrate large disparities in the distribution of diabetes risk severity by race, particularly for undiagnosed diabetes. Multivariate results show complex relationships between experiencing discrimination and diabetes risk severity by race, which suggest that discrimination effects diabetes risk severity differently for non-Hispanic blacks and non-Hispanic whites. Further study is needed to assess how these factors affect health trajectories over the life course.

Key words: Diabetes, health disparities, social stressors, and Add Health

Abstract Word count: 156

Word Count: 6,805

Acknowledgement: This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://non.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

3.1 Introduction

Although perceived discrimination has been associated with a variety of health and mental health disparities over the life course (Pascoe and Richman 2009; Williams & Mohammed 2009), it is less clear how discrimination impacts disparities in diabetes onset risk and severity for young adults. The Centers for Disease Control and Prevention (CDC) list the current prevalence rate of pre-diabetes in adults age 20 or older in the United States at 35%, which is the same both for non-Hispanic blacks and non-Hispanic whites (CDC 2011). The rates for combined diagnosed and undiagnosed diabetes for American adults over age 20 reveal clear disparities with a national diabetes rate of 11.3%, but a rate of only 10.2% for non-Hispanic whites compared to 18.7% for non-Hispanic blacks (American Diabetes Association Fact Sheet 2013; CDC 2011). Although there appears to be no difference in the rate of pre-diabetes by race for all adults over age 20, it is less clear whether young adults (25-34) who have completed the transition to adulthood, but have not yet reached mid-life, share equivalent risk leading up to diabetes by race. Existing literature remains unclear regarding whether early life course disparities exist for pre-diabetes that shift toward disparities in diabetes diagnoses later in life or if disparities only exist with clinical diabetes and only appear at midlife or later.

Although diabetes has previously been linked to biological and behavioral risk factors, these factors alone do not explain disparities in diabetes prevalence across race/ethnic groups (Cowie 2006; Cowie et al. 2010). The unique circumstances involving exposure to discrimination in the United States could be a possible contributing factor for racial health disparities related to diabetes risk due to unequal stress exposure from structural and interpersonal sources of discrimination for non-Hispanic blacks (Clark et

al. 1999). Prior empirical research has demonstrated that chronic and acute life stressors can activate physiological stress responses of the hypothalamic-pituitary-adrenal (HPA) axis, which regulates hormone production involved in glucose control (DeSantis et al. 2007; Gunnar & Adam 2012; Sapolsky 2004). Exposure to recurrent social stressors, like discrimination, can lead to repeated activation of the HPA axis. Over time, this process could lead to dysfunction between the balance of cortisol, a stress hormone released during the activation of the HPA axis, and glucose, which could result in insulin resistance (pre-diabetes) and develop into diabetes (Eriksson et al. 2008).

If unequal social stress exposure does alter diabetes risk severity for racial minorities, this may partially explain disparities in diabetes risk severity not explained by behavioral or biological risk factors. Conversely, unequal stress exposure may also lead to differences in the development of empowering psychological resources and risky coping strategies (e.g.- drinking, smoking, poor diet, etc.) that could also alter diabetes risk onset and severity. Mastery is a type of psychological resource that measures the degree of control one feels over his or her life that can alter the emotional impact and interpretation of negative life events (e.g.- “There is little I can do to change the important things in my life”) (Pearlin & Schooler 1978). Since people may develop risky coping strategies or empowering psychological resources as a result of exposure to discrimination, it is important to isolate the effects of discrimination-based stress exposure from the negative impact of risky coping strategies and emotionally protective impact of mastery that may affect diabetes risk through discrimination exposure.

Taken together, the current sociological literature lacks an in depth analysis of the role of perceived discrimination as a pathway through which disparities in diabetes onset

and severity emerge in young adults. This study seeks to fill this gap in the literature by testing the *Stress Process Model* (Pearlin et al. 1981) using data from the National Longitudinal Study of Adolescent Health (Add Health) (Harris 2009). The purpose of this research is to explore how social characteristics, perceived discrimination, and intervening processes contribute to diabetes severity for young adults controlling for emotionally protective and health risk behaviors for diabetes. The focal research questions of this study are: 1) is there an association between race and diabetes risk severity? 1a) If so, does perceived discrimination mediate this relationship? 1b) Alternatively, does race alter the effect of perceived discrimination on diabetes risk severity through moderation? 2) Do risky coping strategies mediate the effect of perceived discrimination on diabetes risk severity by race? Finally, 3.) Does mastery moderate the effect of perceived discrimination on diabetes risk severity by race?

3.2 Theoretical Framework and Literature Review

The Stress Process Model

The *Stress Process Model* (SPM), first identified by Pearlin and colleagues in 1981, is a sociological theory that evaluates the sources, mediators, and manifestations of stress on health and mental health outcomes. In its initial iteration, the SPM was designed to evaluate how major life strains impact manifestations of stress focusing on mental health and depression as specific outcomes. Subsequent adaptations of the SPM have focused less on specific traumatic life events in favor of exploring constellations of stressors (Pearlin 1999) and more concrete ties to physiological outcomes based on structural inequality (Pearlin et al. 2005).

Foundational propositions of the SPM include the existence of social conditions earlier in the life course that can create chronic emotional or economic strain that may alter exposure to future stressors (Aneshensel et al. 1991; Turner 2013). In this respect, prior social conditions may alter the effects of future stress by regulating both exposure and responses to future stressors. Another key component of the SPM is the idea that intervening processes related to *psychological resources* and *coping mechanisms* can mitigate or exacerbate some of the effects of stress on general health outcomes by developing social and emotional tools. Mastery, or the sense of control one feels over his or her life, is one such example of a positive emotional tool whereas the adoption of risky coping strategies like excessive drinking, smoking or eating for comfort could contribute to worse health outcomes (Avison and Gotlib 1994; Pearlin 1989).

The SPM (Pearlin 1989; Pearlin et al. 2005) provides a theoretical framework to connect structural inequality to discrimination exposure as a testable pathway for diabetes risk severity. The United States has a long and complex history of institutionalized racial discrimination that has led to generations of structural inequality for racial minorities resulting in inequities in education, housing, wealth accumulation, and employment (Charles, Dinwiddie, and Massey 2004; Collins and Williams 1999; Do 2009; Massey and Fisher 2000; Williams et al. 2010). Structural inequality may lead non-Hispanic blacks and non-Hispanic whites to experience discrimination differently both in frequency and severity of exposure. Under this framework, it is possible to examine how structural inequality, discrimination exposure, mastery, and risky coping strategies provide an additional previously unexplored pathway to understand disparities in early adult diabetes onset and severity. The goal of this study is to examine whether there is a

biopsychosocial connection between social characteristics of structural inequality, experiencing discrimination, and an observable health outcome, diabetes severity (Clark et al. 1999; Collins and Williams 1999).

3.3 Hypotheses

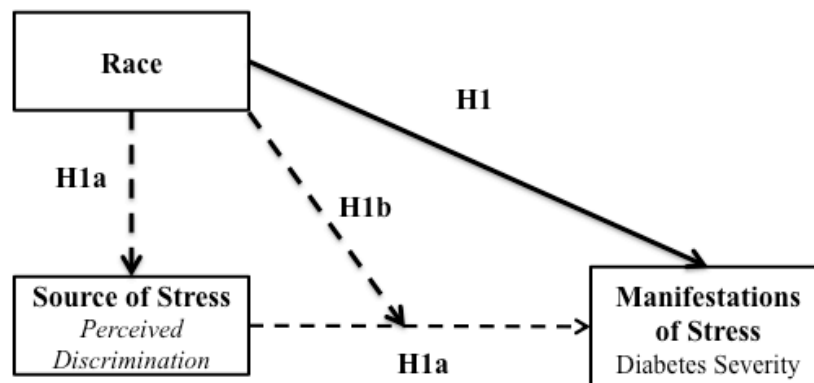
General Hypotheses

H1: Racial minority status is associated with increased diabetes risk severity.

H1a: Perceived discrimination mediates the relationship between racial minority status and the severity of diabetes risk.

H1b: Perceived discrimination moderates the relationship between racial minority status and the severity of diabetes risk.

3.3.1 Figure 3.1- Theoretical Stress Process Model for Diabetes Severity

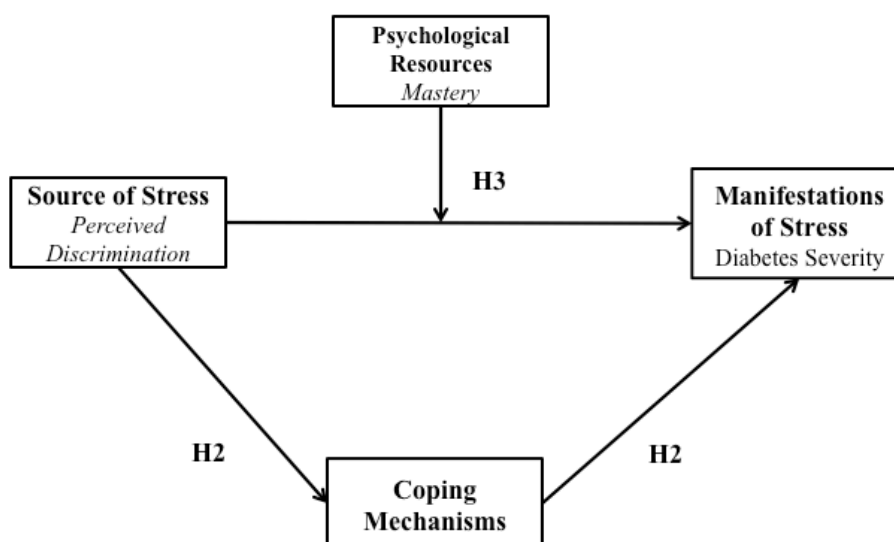


Race-Stratified Hypotheses

H2: Risky coping strategies partially mediate the effect of perceived discrimination on diabetes risk by race.

H3: Mastery moderates the effect of discrimination on diabetes risk by race.

3.3.2 Figure 3.2- Theoretical Stress Process Model for Race-Stratified Diabetes Severity



The fundamental theoretical argument presented here is that social and structural inequality is present throughout the life course for non-Hispanic blacks, which provides a foundation for *chronic strain* through continued exposure to discrimination that historically differs in quality and quantity than for non-Hispanic whites. The relationship between race and discrimination exposure may be so intertwined that frequent differential exposure to discrimination by race could explain some of the association between racial minority status and diabetes risk in early adulthood (H1). The specific relationship between racial minority status and the effect of perceived discrimination on diabetes risk severity is unknown in existing literature. Therefore, it is proposed that the resulting relationship between racial minority status and perceived discrimination on diabetes risk will result in either mediation (H1a) or moderation (H1b). Further, it is proposed that people may adopt of *risky coping strategies* after experiencing discrimination, which could mediate some of the effect of perceived discrimination on diabetes risk severity

(H2) by race. Finally, *mastery* is proposed to interact with perceived discrimination to provide additional risk of or protection from increased diabetes risk severity (H3) by race.

Structural Conditions for Chronic Strain and Perceived Discrimination

Structural inequality, social disadvantage, and perceived discrimination are all chronic stressors that could impair the process of achieving the balance of stress on the body called “allostasis” (McEwen 1998). There is reason to suspect that young adults, particularly those from minority and socioeconomically disadvantaged backgrounds (Cohen et al. 2007; Geronimus et al. 2006), may have elevated risk for pre-diabetes or diabetes due to disproportionate exposure to economic stressors, residential segregation, health risk behaviors, and concentrated poverty (Boardman 2004; Gary et al. 2006; Gresenz, Rogowski, and Escarce 2009). A proposed connection between stress and diabetes risk severity is strengthened by empirical evidence suggesting connections between autonomic reactivity and diabetes correlates such as hypertension (Anderson, McNeilly, and Meyers 1990), coronary artery calcification (Lewis et al 2006), and accelerated aging (Geronimus et al. 2010) for non-Hispanic blacks experiencing distress. Although there appears to be a strong association between autonomic stress responses and hypertension, until now, there has not been an effort to test whether similar relationships exist for diabetes risk.

Poor social circumstances and perceptions of unfair treatment can lead to stress proliferation (Pearlin, Aneshensel, and Leblanc 1997). In the United States, *race and ethnicity* (Williams et al. 2010) is strongly associated with exposure to future health risk by regulating access to protective and disproportionate exposure adverse social conditions even for well-off racial minorities (Do 2009). Specific examples include

reduced access to safe places to live (Collins and Williams 1999; Macintyre and Ellaway 2000) and higher education (Charles, Dinwiddie, and Massey 2004; Kawachi and Berkman 2000), which can provide opportunities for, or protection from, stress (Gary, Stark, and LaVeist 2007; Lin and Ensel 1989), *perceived discrimination* (Clark et al. 1999) and adoption of *risky coping strategies* (Jackson, Knight, and Rafferty 2010; Williams et al. 2010).

Structural inequality creates disadvantage at a macro level that encompasses multiple aspects of the lived environment including structural forms of discrimination. Illustrative examples of the co-occurrence of various types of structural inequality include reduced access to services (Clampet-Lundquist and Massey 2008; White, Haas, and Williams 2012), fewer parks and outdoor paths (Kaplan 1981; 1983), increased exposure to environmental toxins (Morelo-Frosch and Jesdale 2006), increased crime exposure (Robert and House 2000) and residential segregation (Bellatorre et al. 2011) by those who are the most socioeconomically disadvantaged and racial minorities. However, higher income non-Hispanic blacks are not immune from the effects of structural inequality as they have higher risk of living in or near lower quality areas than equally well off whites (Do 2009). As a result, increased social status may not translate into better health in the same way for middle class non-Hispanic blacks as for equally well-off non-Hispanic whites due to the long-term effects of historical residential segregation (Massey and Fischer 2000; Williams et al. 2010; Williams and Jackson 2005).

Prior research on the physiological responses to chronic stress (Cohen et al. 2007; Anderson, McNeilly, and Meyers 1990; Lewis et al. 2006; Geronimus et al. 2010) and allostatic load (McEwen 1998); motivate the hypotheses (H1-H1b) that the relationship

between race and discrimination exposure will influence diabetes risk through physiological processes (Pascoe and Richman 2009). If true, it is possible (H1b) that race *moderates* or provides an additional combined effect for non-Hispanic blacks that experience discrimination differently than their non-Hispanic white counterparts on diabetes risk (Clark et al. 1999; Williams and Mohammed 2009).

Psychological Resources, Coping Mechanisms, and Diabetes Risk

Psychological resources and coping mechanisms are key components of the SPM. Being cognizant of inequality may also affect agency and emotional coping mechanisms through the development of personal resources such as *mastery*, or an individual's perception that they have the ability to control the things that they experience (Pearlin and Schooler 1978). Although increased feelings of mastery have been shown to have protective health effects (Mirowsky and Ross 1990), impediments to the health protective effects of mastery have been noted particularly for non-Hispanic blacks (Lincoln 2007; Lincoln, Chatters and Taylor 2003), which motivates the hypothesis that mastery will moderate the relationship between perceived discrimination and diabetes severity by race.

Although developing a sense of mastery is one way to combat the effects of stressful life events, people can also adopt several other coping strategies to alleviate stress-including risky ones. For example, positive coping behaviors like exercise may relieve stress and reduce diabetes risk. It has been demonstrated that higher socioeconomic status is associated with increased use of exercise and physical activity for stress relief, which yields positive metabolic effects on glucose regulation (Chang, Brown, and Nitzke 2008). However, reduced access to safe places to exercise outdoors

may impede the ability of persons with lower socioeconomic statuses to benefit in the same ways (Kaplan 1996).

Several risky coping strategies, such as *smoking*, *drinking*, and *poor diet*, have been linked to stress exposure, which may exacerbate the effects of stress exposure on diabetes risk by worsening the health of those already at risk. Specifically, Williams and colleagues (2010) noted *increased use of alcohol* and *cigarette smoking* for men, racial minorities, and people with lower levels of education. Possible selection effects for adopting risky coping strategies have also been suggested by Jackson, Knight, and Rafferty (2010) who argue that some of the race differences in obesity rates could be explained by *eating as a coping strategy* for racial minorities facing persistent racial discrimination. Although the adoption of risky coping strategies have a general negative impact on physical health, these activities provide emotional relief and yield positive dopamine responses that can relax some of the activation of the HPA axis (Sapolsky 2004). As such, it is possible that the adoption of risky coping strategies may mediate some of the effects of perceived discrimination on diabetes risk severity for racial minorities (H2).

3.4 Methods

Data and Sample

The data for this study come from Waves I and IV of the National Longitudinal Study of Adolescent Health (Add Health) (Harris 2009). The Add Health study was designed to be a nationally representative sample of students in schools in grades 7-12 in 1994-1995 (Chantala and Tabor 1999). During the first wave of data collection, parents or legal guardians were also interviewed. In the most recent wave of data collection,

biological markers were collected on the focal respondents in addition to survey data when the respondents are now 25-34 years old.

There were 15,701 respondents who participated in Wave IV. The analytic sample for this study was restricted to only non-Hispanic whites (whites) and non-Hispanic blacks (blacks) to allow for race-stratified comparisons across models. The restricted sample for this study included 10,723 respondents who were not pregnant at the time of interview. Approximately 71% of the restricted sample respondents (N=7,599) were white and the remaining 29% (N=3,124) were black. Due to significant oversamples of middle-class blacks, twins, siblings, and persons with limb deformities, it is not possible to generalize this sample to the young adult population without applying complex survey design weights (Chantala and Tabor 1999; <http://non.cpc.unc.edu/projects/addhealth/design/wave4>). Cross-sectional survey design weights from Wave IV were used because all respondents who participated in the most recent wave also participated in Wave I. All analyses are also adjusted for complex survey design features such as clustering and strata. The cross-sectional weights allow for meaningful inferences of non-Hispanic black and non-Hispanic white young adults in 2008-2009.

Dependent Variable

Respondent *diabetes status* was determined by measured hemoglobin A1C (A1C) values, which measure the proportion of glucose-containing hemoglobin molecules in red blood cells (Krolewski et al. 1995; Olson et al. 2010), and stated diagnostic history of diabetes. Diabetes is clinically indicated if A1C levels exceed 6.5% of hemoglobin molecules. Pre-diabetes is indicated with A1C values between 5.7% and 6.49% of

hemoglobin molecules (Olson et al. 2010). As such, *diabetes status* was classified into four categories with persons with A1C values 5.69% or below classified as normoglycemic (not diabetic), persons with A1C values between 5.7% and 6.49% classified as pre-diabetic, and persons with A1C values greater than 6.5% classified as undiagnosed diabetic. Any persons indicating that they had a prior diabetes diagnosis were reclassified into the fourth category of “diagnosed diabetic” regardless of their current A1C level in order to capture diabetics with glucose levels currently under control. For the purposes of this analysis, those who were normoglycemic were treated as the reference group.

Independent Variables

Race and Perceived Discrimination

Respondent single category race/ethnic identification was used to determine race. *Race* categories included non-Hispanic white or non-Hispanic black, with Hispanic, Asian, and other race or biracial respondents excluded from the analyses. Non-Hispanic white was used as the reference category in these analyses. *Perceived discrimination* was measured using a single question, “In your day-to-day life, how often do you feel you have been treated with less respect or courtesy than other people?” which is a component of the *Everyday Discrimination Scale* (Krieger et al. 2005; Sternthal, Slopen, & Williams 2011; Williams et al. 1997) and was the only discrimination measure available in the Add Health data. An attribution variable was available for this measure, but it was not used because it was only asked for respondents with extreme responses and did not provide clarity for less extreme responses. The response categories for this question included,

“never,” “rarely,” “sometimes,” and “often.” This variable was dummied into four categories. Never experiencing discrimination was omitted as the reference category.

Risky Coping Strategies and Mastery

Four risky coping strategies were included to test a proposed mediating pathway between discrimination exposure and diabetes risk severity. Possible risky coping strategies included *fast food* (Feldstein & Tucker 2007) and *sugary drink* (Hallfrisch 1990) consumption in the previous week as continuous measures to gauge how poor diet affects diabetes risk severity. *Regular smoking* was assessed using an indicator variable for the number of days in the previous month a respondent smoked (1= 15-30 days; 0 otherwise). *Daily drinking* was assessed using a question that asked, “During the past 12 months, on how many days did you drink alcohol?” Respondents who indicated that they drank every day were coded as daily drinkers. Those who did not drink or drank less frequently were coded with zeros for this measure. This coding was chosen to capture drinking that may be used as a coping strategy.

Mastery was measured using a five-item version of Pearlin’s *Mastery Scale* (Pearlin and Schooler 1978) that was available in the Add Health data. The five questions asked how much the respondent agreed or disagreed with each statement on a 5-item bipolar Likert scale. The questions included- 1.) There is little I can do to change the important things in my life; 2.) Other people determine most of what I can and cannot do; 3.) There are many things that interfere with what I want to do; 4.) I have little control over the things that happen to me; and 5.) There is really no way I can solve the problems I have. In all cases, higher values indicate greater disagreement with these statements and therefore higher mastery or the sense of control over one’s life. These items were

combined using confirmatory factor analysis into a predicted factor score where higher values indicated higher levels of mastery (Cronbach's $\alpha = 0.77$).

Two interactions were tested in these analyses. The interactions *included race by perceived discrimination* and *perceived discrimination by mastery score*.

Control Variables

The control variables used in this study included both demographic and health variables. The demographic variables included: age at interview, sex, nativity status, education level, and natural logarithm adjusted income in 2009 dollars. *Age* was measured continuously in years. Both nativity status (*immigrant*=1 and *U.S. born*=0) and sex (*male*=1 and *female*=0) were dichotomously measured. Education was collapsed into four categories: high school or less education, some college or vocational training, college graduate, and advanced degree. Some college or vocational training was omitted as the reference category.

Four health measures associated with diabetes risk were also included to control for possible predisposition to diabetes risk. These measures included current obesity, parent diabetes history, birth weight, and an indicator of walking for exercise. A dichotomous measure was used for *current obesity* (BMI 30.0+ =1, zero otherwise) and a dichotomous indicator of *parent diabetes history* was included if the reporting parent indicated that one or both biological parents had diabetes at Wave I. Birth weight extremes were also included to control for possible epigenetic predisposition to diabetes risk (Barker 1995; Sapolsky 2004). Having a parent-reported *microsomic* (5.5 pounds or less) or a *macrosomic* (above 9.9 pounds) birth weights were recorded as separate dichotomous indicators. Values representing normal birth weights (5.5-9.9 pounds) were

omitted as the reference category. Walking for exercise has been associated with positive effects for regulating glucose levels (Kaplan 1996; Lake and Townsend 2006).

Respondents who indicated that they did not take at least one walk in the previous week were given a value of one and those who did were given a zero for this measure to control for those who do not exercise.

Analytic Plan and Missing Data

In order to test the theoretical model, multinomial logistic regression was used to determine whether variables of interest altered diabetes risk severity. Two sets of models were included in these analyses to test the theoretical model. The first set of analyses included three models on the full analytic sample. Model 1 included race and all control variables. Model 2 built off of Model 1 and included perceived discrimination to test hypothesis 1. Model 3 built off of model 2 and included race by perceived discrimination interactions to test hypothesis 1a controlling for risky coping strategies and mastery.

The second set of models included four race-stratified models. Model 1a included perceived discrimination and all control variables. Model 2a included risky coping strategies and all control variables. Model 3a included perceived discrimination, risky coping strategies, mastery, and all control variables to test hypothesis 2. Finally, Model 4a builds off of Model 3a and includes the perceived discrimination by mastery interaction to test hypothesis 3.

Missing data were addressed through multiple imputation using the “ice” command in Stata 11.2 (Royston 2005). Due to methodology norms regarding imputation, missing values on the dependent variables (233 cases) were deleted (Von Hippel 2007). Pregnant women (519 cases) were excluded from these analyses due to the

risk of misclassifying cases of gestational diabetes with general diabetes risk. Hispanic, Asian or Pacific Islander, and other race individuals (3688 cases) were also excluded due to the focus on race-stratified models and too few cases for analysis of interaction effects. The analytic sample was further reduced by cases missing survey weights (901 cases). In total, 4,978 cases were dropped for one or more of the aforementioned reasons yielding a final analytic sample of 10,723 individuals. Missing values on all independent variables were imputed to provide complete analytic data (Ragunathan 2004). Analyses were conducted on ten imputed data sets that were combined and analyzed using “Rubin’s Combining Rules” (Little and Rubin 2002).

3.5 Results

Descriptive Results

Table 3.1 reports the weighted means and proportions for the analytic sample and the race-stratified sub-samples. Significant differences in weighted proportions were observed for three of the four categories of diabetes risk severity by race, but did not differ for diagnosed diabetes. Approximately 73% of the sample had no diabetes history, but the figure is heavily comprised of non-Hispanic whites (whites), as roughly 77% of whites were not diabetic compared to 45% of non-Hispanic blacks (blacks) in the sample. Although 22% of the analytic sample is pre-diabetic, 41% of blacks in the sample are pre-diabetic compared to only 19% of whites. The most striking difference is observed with undiagnosed diabetes where 3% of the analytic sample is comprised of undiagnosed diabetics, but an astounding 11% of blacks were undiagnosed diabetics compared to only 2% of whites.

The only statistically significant difference in perceived discrimination by race was observed with those who “sometimes” experienced discrimination. Approximately 26% of blacks reported “sometimes” experiencing discrimination compared to 18% of whites and 19% of the full analytic sample. Of the remaining key independent variables, only regular smoking (33% of whites; 22% of blacks) significantly differed by race.

Table 3.1 about Here

Multivariate Results

Race, Perceived Discrimination, and Diabetes Risk Severity

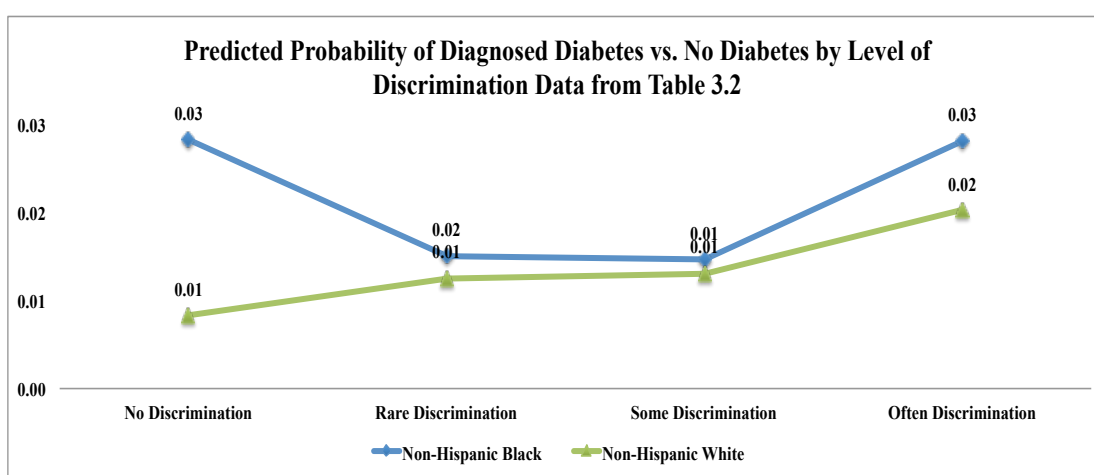
Table 3.2 shows the final model results predicting diabetes risk severity for the full sample. The full model sequences are included in Appendices 3.1-3.3. In the full model, blacks have elevated relative risk of having pre-diabetes (RRR=3.52, 95% CI 2.72, 4.55), undiagnosed diabetes (RRR=11.04, 95% CI 6.65, 18.33), and diagnosed diabetes (RRR=3.99, 95% CI 2.27, 7.00) relative to no diabetes history compared to whites. This finding supports hypothesis 1, that racial minority status has a strong association with elevated diabetes risk severity.

Table 3.2 illustrates a complex relationship between race and perceived discrimination. Rarely experiencing perceived discrimination had no effect on any of the three diabetes risk categories. Sometimes experiencing discrimination elevated the relative risk of pre-diabetes (RRR=1.30, 95% CI 1.02, 1.65) relative to not having diabetes, but had no effect on either diabetic category. Often experiencing discrimination elevated the relative risk of diagnosed diabetes (RRR=2.47, 95% CI 1.07, 5.69), but did not predict pre-diabetes or undiagnosed diabetes relative to not having diabetes. Although some categories of perceived discrimination increased the relative risk of categories of

diabetes risk severity, including perceived discrimination into the model (Model 2 of Appendices 3.1-3.3) did not attenuate the strong relationship between race and diabetes risk severity. Therefore, hypothesis 1a, which proposed a mediating relationship between race and perceived discrimination on diabetes risk severity, cannot be supported with this data.

Table 3.2 about Here

3.5.1 Figure 3.3- Significant Interaction Effect for Perceived Discrimination by Race



The race by perceived discrimination interaction was significant for blacks experiencing rare (RRR=0.35, 95% CI 0.15,0.84) or some (RRR=0.32, 95% CI 0.13,0.83) discrimination (Figure 3.3), but not often discrimination (RRR=0.40, 95% CI 0.13,1.49) for predicting diagnosed diabetes relative to no diabetes history, which was a counterintuitive finding. The interaction effects did not significantly predict pre-diabetes or undiagnosed diabetes risk relative to no diabetes history (Appendices 3.7-3.8). These findings provide partial support for hypothesis 1b that race moderates the relationship between perceived discrimination and diabetes risk severity, however the interaction effect occurs in the opposite direction. Specifically, racial minorities who experience

perceived discrimination “rarely” or “sometimes” were significantly less likely to be diagnosed with diabetes.

Race Stratified Results

Table 3.3 lists the final model results predicting diabetes risk for the race-stratified models. The full model sequences for the race-stratified models are included in Appendices 3.4-3.6. The race-stratified results elucidate the effects for experiencing discrimination by race. Blacks that often experience discrimination had reduced risk of undiagnosed diabetes (RRR=0.20, 95% CI 0.05, 0.82) relative to no diabetic history, but had no effect on pre-diabetes or diagnosed diabetes risk. Conversely, whites that often experienced discrimination had elevated risk of diagnosed diabetes (RRR=2.55, 95% CI 1.15, 5.53) relative to no diabetic history, but no effect for pre-diabetes or undiagnosed diabetes. Experiencing discrimination rarely or sometimes did not significantly predict any level diabetes risk severity for either blacks or whites.

Table 3.3 about Here

Risky Coping Strategies

Risky coping strategies yield different effects for black and white diabetes risk. No risky coping strategies predicted undiagnosed diabetes risk relative to no diabetes risk for either group in the race-stratified models. Blacks had unexpected statistically significant effects regarding risky coping strategies for sugary drink consumption, daily drinking, and smoking. Blacks that were regular smokers had lower risk of pre-diabetes relative to not being diabetic (RRR=0.72, 95% CI 0.52, 1.00) when compared to non-smokers. Blacks that had higher levels of sugary drink consumption (RRR=0.96, 95% CI 0.93, 0.99) or were daily drinkers (RRR=0.10, 95% CI 0.10, 0.98) were less likely to be

diagnosed diabetics relative to not diabetic. The use of these risky coping strategies by diagnosed diabetics is significantly different than blacks with increased risk of pre-diabetes or undiagnosed diabetes relative to not having diabetes. This finding may reflect alterations to negative health behaviors after diagnosis. Said another way, blacks who receive diabetes diagnoses may elect to change their health risk behaviors after diagnosis.

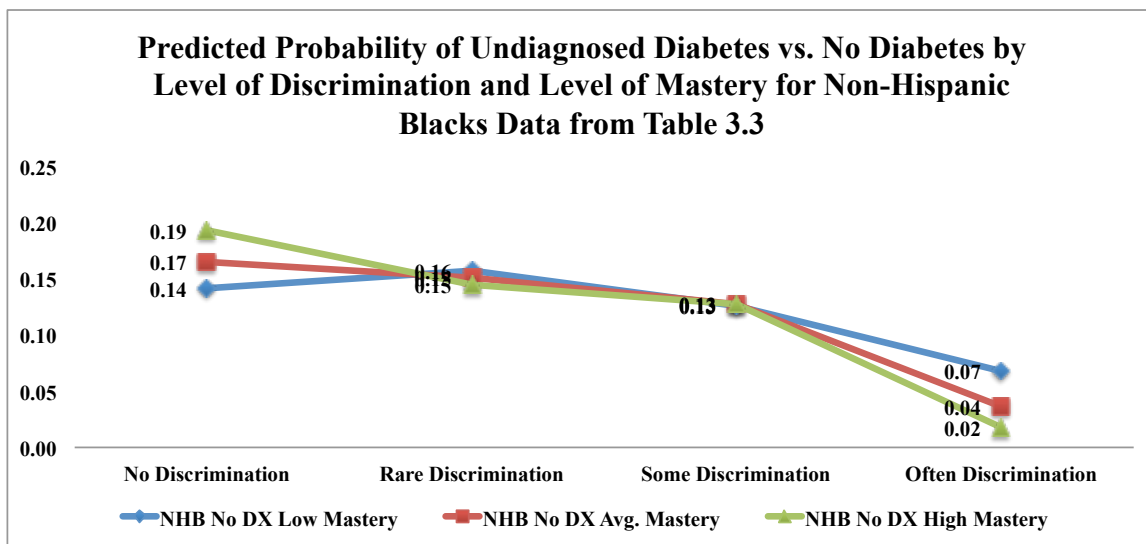
Whites that were daily drinkers had a reduced risk of pre-diabetes (RRR=0.54, 95% CI 0.33, 0.89) relative to no diabetic history, but had no effect for either of the diabetic categories. Whites that had increased sugary drink consumption had increased risk of pre-diabetes (RRR=1.01, 95% CI 1.00, 1.01) relative to no diabetes history. Although independent effects were observed for risky coping strategies in both the full sample and the race-stratified models, including risky coping strategies had no effect on the relationships between perceived discrimination and diabetes risk severity. As a result, hypothesis 2, which posited that risky coping strategies would mediate some of the effect of perceived discrimination on diabetes risk by race, cannot be supported with this data. However, there is some support for alterations in risky coping strategies after diagnosis for blacks.

Mastery

Mastery was not a statistically significant predictor of diabetes risk severity in either the full or race-stratified models. However, the perceived discrimination by mastery interaction was statistically significant for blacks that often experienced discrimination (RRR=0.40, 95% CI 0.18, 0.92) for undiagnosed diabetes risk relative to no diabetes risk in the race-stratified models (Figure 3.4). This suggests that increased mastery is protective against risk of undiagnosed diabetes for blacks that experience

frequent discrimination. Figure 3.4 (below) displays the predicted probability of undiagnosed diabetes for non-Hispanic blacks by level of discrimination at high (one standard deviation above average), low (one standard deviation below average), and average levels of mastery. The figure illustrates that the predicted probability of undiagnosed diabetes decreases as perceived discrimination increases more for those with higher levels of mastery. This finding provides support for hypothesis 3, which proposed that mastery would moderate the effect of discrimination on diabetes risk by race. Neither mastery nor the mastery by perceived discrimination interaction effect were statistically significant predictors of diabetes risk for whites (Appendix 3.11).

3.5.2 Figure 3.4 Significant Interaction Effect for Mastery by Perceived Discrimination for NH Blacks



3.6 Discussion

The results of this research demonstrate complex relationships between perceived discrimination and diabetes risk severity by race. The most striking revelation of this study is the large disparity in diabetes onset and severity by race for young adults. In this sample of young adults, having some type of diabetes risk (55%) was more common than

not having diabetes (45%) for non-Hispanic blacks (blacks), which is alarming considering the opposite is true by a wide margin for non-Hispanic whites (whites) (77% not diabetic vs. 23% some kind of diabetes risk). Undiagnosed diabetes has an even more concerning trend with 11% of blacks having undiagnosed diabetes compared to 2% of whites and 3% of the full sample. These descriptive findings yield wider disparities for young adults than those observed for all adults over age 20 from the CDC (American Diabetes Association Fact Sheet 2013; CDC 2011). These findings also document disparities in pre-diabetes rates as well, which were not observed when looking at all adults over age 20 (CDC 2011).

These discrepancies may provide foundational evidence of racial health disparities in diabetes risk severity for young adults that differs compared to all adults over age 20. Future research should repeat these tests and explore how early these trends emerge. If diabetes is indeed increasing for non-Hispanic black young adults at rates significantly higher than non-Hispanic white young adults at ages earlier than expected, this may contribute to later racial health disparities in diabetes-related morbidity and mortality for those who remain undiagnosed for longer than necessary.

Key findings presented here include preliminary evidence that perceived discrimination is experienced differently by race and those differences may affect diabetes risk severity for young adults in measurable ways. Strong direct associations were observed for both racial minority status and perceived discrimination with diabetes risk severity. However, hypothesis 1a, which proposed a mediating relationship between racial minority status, perceived discrimination, and diabetes risk severity was not supported. Alternatively, these data support hypothesis 1b, which proposed that racial

minority status would moderate the effect of perceived discrimination on diabetes risk severity. Specifically, blacks who reported “rarely” or “sometimes” experiencing discrimination were significantly less likely to be diagnosed diabetics, which was an unexpected finding.

The race-stratified models of series 2, revealed that blacks who reported experiencing discrimination “often” were significantly less likely to be undiagnosed diabetics. Although this finding is counterintuitive, at least one possible explanation exists that is supported with these data. It is possible that blacks who experience the highest level of discrimination are less likely to be undiagnosed diabetics because at the highest level of discrimination they begin to suffer more severe symptoms of diabetes that lead to their diabetes being revealed through diagnosis.

Appendices 2.9 and 2.10 show an overlay of the predicted probabilities of diagnosed and undiagnosed diabetes for blacks (Appendix 2.9) and whites (Appendix 2.10). There is a pronounced drop in the predicted probability for undiagnosed diabetes for both blacks and whites as discrimination increases from “sometimes” to “often,” however while the predicted probabilities begin to converge for blacks, they cross over for whites. Since blacks are disproportionately likely to be undiagnosed if they are diabetic when compared to whites, there could be dueling processes occurring that lead to this finding. For whites, increased discrimination is associated with increased odds of diagnosis if diabetic, but blacks do not see a corresponding increase in diagnosis despite increased discrimination. This could be due to imprecise measurement of discrimination or there could be additional structural impediments to diagnosis for blacks that obscure the relationship between discrimination and diabetes severity for some, but not all blacks.

In order to understand the complexity of the relationship between racial minority status, perceived discrimination, and diabetes risk severity, predicted probabilities were plotted for Model 3 of Series 1 by level of discrimination for race and discrimination exposure for undiagnosed and diagnosed diabetes in Appendices 3.9-3.10. When all variables are held at their means and only discrimination is allowed to vary, a clearer picture emerges for the impact of discrimination on diabetes risk severity by race, particularly when comparing diagnosed and undiagnosed diabetics. In general, as discrimination level increases, relative risk of undiagnosed diabetes decreases, but drops off most between “sometimes” and “often” experiencing discrimination. Conversely, risk of diagnosed diabetes increases considerably between “sometimes” and “often” experiencing discrimination. For both non-Hispanic blacks and non-Hispanic whites, the lines cross at the highest level of discrimination, which suggests that at the highest level of discrimination, both blacks and whites are more likely to be diagnosed than undiagnosed if they are diabetic. Although the lines cross at lower levels of discrimination for whites, non-Hispanic blacks have higher risk of diagnosed and undiagnosed diabetes at every level of discrimination (Appendices 3.9-3.10).

If the imprecise nature of the perceived discrimination variable is capturing acute, but not chronic stress this could explain why whites see more harmful effects from this type of discrimination than blacks. Conversely, if the process works in the same way for blacks and whites in increasing diabetes risk severity, but there are additional barriers to diagnosis for blacks this could explain why a crossover effect is not observed for blacks, but is seen for whites. Further research is needed to disentangle these relationships.

Hypothesis 2, which proposed a mediating relationship between experiencing discrimination and risky coping strategies as a pathway to diabetes risk severity in the race-stratified models, was not supported. There was partial support for hypothesis 3, which proposed that mastery would moderate the effect of discrimination on diabetes risk by race, but this was only supported for blacks and undiagnosed diabetes risk relative to no diabetes history (Figure 3.4).

If one were to think of having undiagnosed diabetes as a temporary state where all diabetics will have diabetes for some amount of time before they receive a diagnosis, it is possible to see undiagnosed diabetes as a stepping stone to diabetes that may be more severe, which is then diagnosed. Future research should explore the possibility that the difference between diabetes that is diagnosed and diabetes that is undiagnosed relates to differences in the severity of the condition at the time of diagnosis. If true, the findings presented here demonstrating increased risk of diagnosed diabetes for those who often experience discrimination may coincide with more severe diabetes symptoms, which would support the initial theoretical mechanism presented here based on the *Stress Process Model* that views exposure to discrimination and diabetes risk severity with a dose-response relationship.

Limitations

Although this study makes several contributions to document foundational relationships between perceived discrimination and diabetes risk severity by race, the strengths of this study must also be viewed in light of its limitations. The data used in this study has several strengths, but also some important weaknesses. The National Longitudinal Study of Adolescent Health is one of a few health studies to include

biological markers for diabetes and a wide variety of health and social variables in a racially diverse sample, but the extensive list of measures included leaves out some areas that would strengthen the arguments made here.

Having only one measure of discrimination likely weakened the association between perceived discrimination and diabetes risk severity due to low content validity. However, even with a less than perfect measure of discrimination, foundational relationships were still established between perceived discrimination and diabetes risk severity by race, particularly for whites, which suggests that further study is warranted. As biological data collection continues to increase, it would be beneficial to repeat this study with a more complete version of the *Everyday Discrimination Scale* (Krieger et al. 2005; Sternthal, Slopen, & Williams 2011; Williams et al. 1997). Moreover, it is important for future research to distinguish between the effects of racial discrimination specifically as opposed to more general discrimination on diabetes risk severity, which was not possible to do with this data. Given the strong associations with “often” experiencing discrimination and diagnosed diabetes for whites, the discrimination measure may reflect generalized unfair treatment or social marginalization as well as discrimination. Further study is warranted with a wider array of discrimination measures to discern whether racial discrimination yields the same effects on diabetes risk by race.

Although the inclusion of the hemoglobin A1C biomarker made it possible to classify different categories of diabetes risk, having additional hormonal biomarkers to test the physiologic stress response of the HPA axis (e.g.-cortisol, epinephrine, and norepinephrine) would have been a great improvement. The theory behind the proposed relationship between discrimination and diabetes is the belief that repeated experiences of

discrimination constantly activate the HPA axis and lead to dysregulation of hormones related to glucose control. If more of these hormonal measures were included, it would be possible to test the findings from Eriksson and colleagues (2008) regarding psychological distress and diabetes for those who experience discrimination in the United States. Cortisol measures would be of great importance in this regard as cortisol is closely related to glucose regulation and has been associated with race differences in sleep and stress exposure (DeSantis et al. 2007; Gunner and Adam 2012). If repeated exposure to discrimination does affect the production and regulation of cortisol in response to perceived threats, microaggressions, and other stressors (Sapolsky 2004) and cortisol dysregulation affects diabetes risk, this would be a way to connect discrimination to diabetes through a biopsychosocial mechanism. Although this study could not make that connection due to too few measures, the exploratory findings here justify future research to directly test that connection.

Conclusion

The fundamental theoretical argument presented by this study was that social and structural inequality is present throughout the life course for racial minorities, which provides exposure to chronic strain through multiple forms of discrimination that differ for whites. This study revealed that the relationship between race and discrimination is complex and affects both blacks and whites. Race was shown to moderate the effect of perceived discrimination on diabetes risk severity independent of the adoption of risky coping strategies and mastery, which suggests that further study is needed.

Although the focus of this study was not to determine why undiagnosed diabetics lack diagnoses, these findings suggest that this is a serious problem particularly for non-

Hispanic blacks. Further study is needed to determine if additional structural or behavioral patterns contribute to the dramatic under diagnosis of young black diabetics. If diagnoses are allocated differently by race, this could create the conditions for future racial health disparities as people age that may contribute to increased morbidity and mortality. Moreover, if the racial disparities in undiagnosed diabetes reflect structural problems, reduced access to care, or access to health insurance, it is important that policies be put in place to address these issues. Conversely, if under diagnosis occurs due to differences in quality of care (Lutfey and Freese 2005) or biases that affect doctor-patient interactions (Mouton et al 2010; Stepanikova 2012; Shavers et al. 2012) it is important that policies be put in place to address these issues regarding management of care.

3.7 References

- American Diabetes Association. Standards of medical care in diabetes -- 2012. *Diabetes Care*. 2012; 35 Suppl 1:S11-S63.
- American Diabetes Association .2013 <<http://professional.diabetes.org/Admin/UserFiles/0%20-%20Sean/FastFacts%20March%202013.pdf>> Accessed March 20, 2014
- Anderson, N. B., M. McNeilly, and H. Myers. 1990. "Autonomic reactivity and hypertension in blacks: a review and proposed model." *Ethnicity & disease* 1, no. 2: 154-170.
- Aneshensel, Carol S., Carolyn M. Rutter, and Peter A. Lachenbruch.1991. "Social structure, stress, and mental health: Competing conceptual and analytic models." *American sociological review* : 166-178.
- Avison, William R., and Ian H. Gotlib. 1994. "Future prospects for stress research." In *Stress and Mental Health*, pp. 317-332. Springer US.
- Barker, D.J.P. 1995. "Fetal Origins of Coronary Heart Disease." *British Medical Journal* 311(6998): 171-174.
- Bellatorre, Anna, Brian K. Finch, D. Phuong Do, Chloe E. Bird, and Audrey N. Beck. 2011. "Contextual predictors of cumulative biological risk: Segregation and allostatic load." *Social Science Quarterly* 92, no. 5: 1338-1362.
- Boardman, Jason D. 2004. "Stress and physical health: the role of neighborhoods as mediating and moderating mechanisms." *Social Science & Medicine* 58, no. 12: 2473-2483.
- CDC 2011 < <http://non.cdc.gov/diabetes/pubs/estimates11.htm>> Accessed July 30, 2013
- Chang, Mei-Wei, Roger Brown, and Susan Nitzke. 2008. "Scale development: factors affecting diet, exercise, and stress management (FADESM)." *BMC public health* 8, no. 1: 76.
- Chantala, Kim and Joyce Tabor. 1999. "National Longitudinal Study of Adolescent Health: Strategies to Perform a Design-Based Analysis Using the Add Health Data." Chapel Hill, NC: Carolina Population Center.
- Charles CZ, G. Dinwiddie, and DS. Massey. 2004. "The Continuing Consequences of Segregation: Family Stress and College Academic Performance." *Social Science Quarterly*. 85(5): 1353-73.
- Clark, Rodney, Norman B. Anderson, Vanessa R. Clark, and David R. Williams.1999.

“Racism as a Stressor for Non-Hispanic blacks: A Biopsychosocial Model.”
American Psychologist. 54(10): 805-816.

Clampet-Lundquist S, and DS. Massey. 2008. “Neighborhood Effects on Economic Self-Sufficiency: A Reconsideration of the Moving to Opportunity Experiment.”
American Journal of Sociology. 114(1): 107-43. Cohen et al. 2007

Collins CA, Williams DR. 1999. “Segregation and Mortality: The Deadly Effects of Racism?” *Sociological Forum*, 14:495-523.

Cowie C. 2006. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health and Nutrition Examination Survey 1999-2002.”
Diabetes Care. 29(6):1263–1268.

Cowie CC, Rust KF, Byrd-Holt DD, Gregg EW, Ford ES, Geiss LS, et al. 2010.
“Prevalence of diabetes and high risk for diabetes using A1C criteria in the U.S. population in 1988-2006.” *Diabetes Care*.33(3):562-8.

(<http://non.cpc.unc.edu/projects/addhealth/design/wave4>;

DeSantis, Amy S., Emma K. Adam, Leah D. Doane, Susan Mineka, Richard E. Zinbarg, and Michelle G. Craske. 2007. "Racial/ethnic differences in cortisol diurnal rhythms in a community sample of adolescents." *Journal of Adolescent Health*.41(1): 3-13.

Do, D. Phuong. 2009. "The dynamics of income and neighborhood context for population health: Do long-term measures of socioeconomic status explain more of the black/white health disparity than single-point-in-time measures?." *Social Science & Medicine* 68, no. 8 1368-1375.

Eriksson, A□K., A. Ekblom, F. Granath, A. Hilding, S. Efendic, and C□G. Östenson. 2008. "Psychological distress and risk of pre□diabetes and Type 2 diabetes in a prospective study of Swedish middle□aged men and women." *Diabetic Medicine* 25, no. 7: 834-842.

Feldeisen SE, Tucker KL (2007). "Nutritional strategies in the prevention and treatment of metabolic syndrome". *Appl Physiol Nutr Metab* 32 (1): 46–60.

Gary, Tiffany L., Sarah A. Stark, and Thomas A. LaVeist.2007. "Neighborhood characteristics and mental health among African Americans and whites living in a racially integrated urban community." *Health & Place* 13, no. 2 569-575.

Geronimus, Arline T., Margaret Hicken, Danya Keene, and John Bound. 2006.
““Weathering” and age patterns of allostatic load scores among blacks and whites in the United States." *Journal Information* 96, no. 5.

- Graham, Non. and C. Power. 2004. "Childhood disadvantage and health inequalities: a framework for policy based on lifecourse research." *Child Care, Health, & Development*. 30 (6) 671-678.
- Gresenz, Carole Roan, Jeannette Rogowski, and José J. Escarce. 2009. "Community demographics and access to health care among US Hispanics." *Health services research* 44, no. 5p1 1542-1562.
- Gunnar, Megan R., and Emma K. Adam. 2012. "THE HYPOTHALAMIC–PITUITARY–ADRENOCORTICAL SYSTEM AND EMOTION: CURRENT WISDOM AND FUTURE DIRECTIONS." *Monographs of the Society for Research in Child Development* 77, no. 2: 109-119.
- Hallfrisch J.1990. "Metabolic effects of dietary fructose". *FASEB J* 4 (9): 2652–2660.
- Harris, Kathleen Mullan. 2009. The National Longitudinal Study of Adolescent Health (AddHealth), Waves I & II, 1994–1996; Wave III, 2001–2002; Wave IV, 2007–2009 [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill. DOI: 10.3886/ICPSR27021.v9
- Jackson, James S., K. M. Knight, and J. A. Rafferty. 2010. "Race and unhealthy behaviors: chronic stress, the HPA axis, and physical and mental health disparities over the life course." *American Journal of Public Health* 100:933-939.
- Kaplan GA. 1996. "People and Places: Contrasting Perspectives on the Association between Social Class and Health." *Int J Health Serv*, 26:507-519.
- Kawachi, I., and L. Berkman. 2000. "Social Cohesion, Social Capital, and Health." in L. Berkman and I. Kawachi, eds., *Social Epidemiology*. Oxford, UK: Oxford University Press. 174-90.
- Krieger, Nancy, Kevin Smith, Deepa Naishadham, Cathy Hartman, and Elizabeth M. Barbeau. 2005. "Experiences of discrimination: validity and reliability of a self-report measure for population health research on racism and health." *Social science & medicine* 61, (7): 1576-1596.
- Krolewski AS, Laffel LMB, Krowlewski M, et al. 1995. "Glycosylated hemoglobin and the risk of microalbuminuria in patients with insulin dependent diabetes mellitus." *New England Journal of Medicine*;332:1251-5
- Lake, Amelia and Tim Townshend. 2006. "Obesogenic Environments: Exploring the Built and Food Environments." *Journal of the Royal Society for the Promotion of Health*. 126(6):262-267.
- Lincoln, Karen D. 2007. "Financial strain, negative interactions, and mastery: Pathways

- to mental health among older African Americans." *Journal of Black Psychology* 33, no. 4: 439-462.
- Lincoln, Karen D., Linda M. Chatters, and Robert Joseph Taylor. 2003. "Psychological distress among Black and White Americans: Differential effects of social support, negative interaction and personal control." *Journal of Health and Social Behavior* 44, no. 3: 390.
- Little, Roderick and Donald Rubin. 2002. *Statistical Analysis with Missing Data*. Hoboken, NJ: Wiley Interscience. Macintyre and Ellaway 2000
- Massey DS, and MJ. Fischer. 2000. "How Segregation Concentrates Poverty." *Ethnic and Racial Studies*. 23(4):670-91.
- McEwen BS. 1998. "Protective and Damaging Effects of Stress Mediators." *NEJM*, 338(3):171-179.
- Mirowsky, John, and Catherine E. Ross. 1990. "Control or defense? Depression and the sense of control over good and bad outcomes." *Journal of health and social behavior*: 71-86.
- Morello-Frosch R, and BM. Jesdale. 2006. "Separate and Unequal: Residential Segregation and Estimated Cancer Risks Associated with Ambient Air Toxics in U.S. Metropolitan Areas." *Environmental Health Perspectives*. 114(3):386-93.
- Mouton, Charles P., Pamela L. Carter-Nolan, Kepher H. Makambi, Teletia R. Taylor, Julie R. Palmer, Lynn Rosenberg, and Lucile L. Adams-Campbell. 2010. "Impact of perceived racial discrimination on health screening in black women." *Journal of health care for the poor and underserved* 21(1): 287.
- Olson DE, Rhee MK, Herrick K, Ziemer DC, Twombly JG, Phillips LS. 2010. "Screening for diabetes and pre-diabetes with proposed A1C-based diagnostic criteria." *Diabetes Care*. 33(10):2184-2189.
- Pascoe, Elizabeth A., and Laura Smart Richman. 2009. "Perceived discrimination and health: a meta-analytic review." *Psychological bulletin* 135, no. 4: 531.
- Pearlin, L. I., and C. Schooler. 1978. "The structure of coping." *Journal of health and social behavior* 19, no. 1: 2-21.
- Pearlin, L. I., M. A. Lieberman, E. G. Menaghan, and J. T. Mullan. 1981. "The stress process." *Journal of health and social behavior* 22, (4): 337-356.
- Pearlin, Leonard I. 1989. "The sociological study of stress." *Journal of health and social behavior* : 241-256.

- Pearlin, Leonard I., Carol S. Aneshensel, and Allen J. Leblanc. 1997. "The forms and mechanisms of stress proliferation: the case of AIDS caregivers." *Journal of Health and Social Behavior*. 3 : 223-236.
- Pearlin, Leonard I. 1999. "The stress process revisited." In *Handbook of the sociology of mental health*, Springer US, pp. 395-415.
- Pearlin, Leonard I., Scott Schieman, Elena M. Fazio, and Stephen C. Meersman. 2005. "Stress, health, and the life course: Some conceptual perspectives." *Journal of Health and Social Behavior* 46, (2): 205-219.
- Raghunathan, T. E. 2004. "What do we do with missing data? Some options for analysis of incomplete data." *Annual Review of Public Health*, 25, 99–117.
- Robert SA, House JS. 2000. "Socioeconomic Inequalities in Health." In G Albrecht, R Fitzpatrick, S Scrimshaw (eds.), *Handbook of Social Studies in Health and Medicine*. Thousand Oaks, CA: Sage Publications, pp. 115-135.
- Royston, Patrick. 2005. "Multiple Imputation of Missing Values: Update of Ice." *The Stata Journal*. 5(4):527-536.
- Sapolsky, Robert M. 2004. *Why Zebras Don't Get Ulcers: An Updated Guide to Stress and Stress Related Diseases, and Coping* (3rd Edition). New York: Freeman & Co.
- Shavers, Vickie L., Pebbles Fagan, Dionne Jones, William MP Klein, Josephine Boyington, Carmen Moten, and Edward Rorie. 2012. "The state of research on racial/ethnic discrimination in the receipt of health care." *American journal of public health* 102(5): 953-966.
- Stepanikova, Irena. 2012. "Racial-ethnic biases, time pressure, and medical decisions." *Journal of health and social behavior* 53, (3): 329-343.
- Sternthal, Michelle J., Natalie Slopen, and David R. Williams. 2011 "Racial disparities in health." *Du Bois Review: Social Science Research on Race* 8, (1): 95-113.
- Tsigos, C; Chrousos, GP; October 2002. "Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress". *J Psychosom Res.* **53** (4): 865–71.
- Turner, R. Jay. 2013. "Understanding Health Disparities The Relevance of the Stress Process Model." *Society and Mental Health* 3 (3)170-186.
- Von Hippel, Paul T. 2007. "Regression with Missing Y's: An Improved Strategy for Analyzing Multiply Imputed Data." *Sociological Methodology*, 37:83-117.
- White, Kellee, Jennifer S. Haas, and David R. Williams. 2012. "Elucidating the role of

place in health care disparities: the example of racial/ethnic residential segregation." *Health services research* 47, no. 3(2):1278-1299.

Williams, David R., Yan Yu, James S. Jackson, and Norman B. Anderson. 1997. "Racial differences in physical and mental health socio-economic status, stress and discrimination." *Journal of health psychology* 2, no. 3 (1997): 335-351.

Williams David R. and Pamela. Braboy Jackson.2005. "Social Sources Of Racial Disparities in Health." *Health Affairs*. 24(2):325-34.

Williams, David R. and Selina A. Mohammed. 2009. "Discrimination and Racial Disparities in Health: Evidence and Needed Research." *Journal of Behavioral Medicine*. 32(1): 20-47.

Williams, David R., Selina A. Mohammed, Jacinta Leavell, and Chiquita Collins. 2010. "Race, socioeconomic status, and health: Complexities, Ongoing Challenges, and Research Opportunities." *New York Academy of Sciences*. 1186. 69-101.

World Health Organization. 2012. "Diabetes Fact Sheet." <http://non.who.int/mediacentre/factsheets/fs312/en/index.html>. Accessed 11/4/2012.

3.8 Tables

Table 3.1: Descriptive Statistics, Weighted Means or Proportions by Race (N=10,723)

	Full Sample	NH White	NH Black	Significant Difference
<i>Diabetes Status</i>				
No Diabetic History	0.73	0.77	0.45	*
Pre-Diabetic	0.22	0.19	0.41	*
Undiagnosed Diabetic	0.03	0.02	0.11	*
Diagnosed Diabetic	0.02	0.02	0.03	
<i>Race</i>				
Non-Hispanic White	0.71			
Non-Hispanic Black	0.29			
<i>Sources of Stress</i>				
No Experiences of Discrimination	0.30	0.30	0.27	
Rarely Experiences Discrimination	0.48	0.49	0.43	
Sometimes Experiences Discrimination	0.19	0.18	0.26	*
Often Experiences Discrimination	0.04	0.04	0.05	
<i>Risky Coping Strategies</i>				
Fast Food Consumption	2.19	2.05	3.14	
Sugary Drink Consumption	11.73	11.60	12.61	
Daily Drinker	0.04	0.04	0.04	
Regular Smoker	0.31	0.33	0.22	*
<i>Personal Resources</i>				
Mastery Factor Score	0.05	0.05	0.08	
<i>Control Variables</i>				
Male	0.53	0.53	0.51	
Female	0.47	0.47	0.49	
Adult Income 2009	9.67	9.70	9.52	
High School or Less	0.22	0.21	0.27	
Some College	0.43	0.43	0.43	
College Degree	0.22	0.23	0.16	*
Advanced Degree	0.13	0.13	0.13	
Age in 2009	28.80	28.77	28.99	
Immigrant	0.00	0.00	0.01	*
Microsomic Birth Weight	0.05	0.04	0.08	*
Normal Birth Weight	0.86	0.86	0.87	
Macrosomic Birth Weight	0.09	0.10	0.05	*
Currently Obese	0.35	0.33	0.43	*
Doesn't Walk for Exercise	0.47	0.47	0.45	
Parent Diabetic	0.07	0.06	0.14	*

Note: Descriptives are reported on the pre-imputed sample.

Table 3.2- Final Model Results Predicting Diabetes Risk-Relative to being Normoglycemic (N=10,723)

	Pre-Diabetes	Undiagnosed Diabetes	Diagnosed Diabetes
<i>Race</i>			
Non-Hispanic Black	3.52 ***	11.04 ***	3.99 ***
<i>Sources of Stress</i>			
Rarely Experiences Discrimination	1.13	1.29	1.55
Sometimes Experiences Discrimination	1.30 *	1.09	1.60
Often Experiences Discrimination	0.98	0.43	2.47 *
<i>Risky Coping Strategies</i>			
Fast Food Consumption	1.01	0.93 *	1.05 *
Sugary Drink Consumption	1.01 **	1.00	0.99
Daily Drinker	0.72	0.73	0.27
Regular Smoker	1.08	0.88	1.12
<i>Personal Resources</i>			
Mastery Factor Score	1.03	1.02	1.06
<i>Interaction Effects</i>			
Rare Discrimination x African American	0.77	0.67	0.35 *
Some Discrimination x African American	0.76	0.64	0.32 *
Often Discrimination x African American	0.87	0.53	0.40
Rare Discrimination x Mastery Score	0.96	0.76	0.69
Some Discrimination x Mastery Score	1.14	1.01	0.88
Often Discrimination x Mastery Score	0.99	0.59	1.11
<i>Control Variables</i>			
Male	1.48 ***	1.35 *	0.90
Adult Income 2009	1.02	1.05	0.98
High School or Less	1.03	1.20	1.65 **
College Degree	0.66 ***	0.77	0.55 *
Advanced Degree	0.71 *	0.83	0.78
Age in 2009	1.05 **	1.11 **	1.10 *
Immigrant	1.30	0.70	0.00 ***
Microsomic	1.11	1.19	1.12
Macrosomic	0.84	0.61	1.36
Currently Obese	2.21 ***	3.01 ***	4.15 ***
Doesn't Walk for Exercise	1.14	1.19	0.98
Parent Diabetic	1.20	1.67	1.85 **

Notes: a. Pre-diabetic refers to having a hemoglobin A1C level of 5.7-6.49 and no diagnostic history of diabetes when the respondent was not pregnant; b. Undiagnosed diabetic refers to having a hemoglobin A1C level 6.5+ and no prior diabetes diagnosis when the respondent was not pregnant; c. Diagnosed diabetic refers to the respondent ever being diagnosed with diabetes when not pregnant regardless of hemoglobin A1C status; d. relative risk ratios reported comparing the relative risk of category a-c relative to being normoglycemic; e. Pregnant women excluded from analyses; f. * p<0.05, ** p<0.01, *** p<0.001

Table 3.3- Race-Stratified Final Model Results Predicting Diabetes Risk Relative to being Normoglycemic

	Non-Hispanic Black (N=3124)			Non-Hispanic White (N=7,599)		
	Pre-Diabetes	Undiagnosed Diabetes	Diagnosed Diabetes	Pre-Diabetes	Undiagnosed Diabetes	Diagnosed Diabetes
<i>Social Stressors</i>						
Rarely Experiences Discrimination	0.89	0.91	0.61	1.14	1.27	1.52
Sometimes Experiences Discrimination	1.00	0.74	0.60	1.29 *	1.09	1.60
Often Experiences Discrimination	0.94	0.20 *	0.92	0.93	0.55	2.55 *
<i>Risky Coping Strategies</i>						
Fast Food Consumption	0.99	0.93	1.05	1.02	0.91	1.05
Sugary Drink Consumption	1.00	1.01	0.96 *	1.01 **	0.99	1.00
Daily Drinker	1.41	1.02	0.10 *	0.54 *	0.68	0.35
Regular Smoker	0.72 *	0.71	0.59	1.19	0.92	1.26
<i>Personal Resources</i>						
Mastery Factor Score	0.97	1.22	1.40	1.09	0.73	0.85
<i>Interaction Effects</i>						
Rare Discrimination x Mastery Score	1.12	0.78	0.83	0.88	0.87	0.74
Some Discrimination x Mastery Score	1.16	0.83	0.61	1.08	1.41	1.13
Often Discrimination x Mastery Score	1.07	0.40 *	0.53	0.91	0.99	1.55
<i>Control Variables</i>						
Male	1.43 **	1.36 *	1.26	1.53 ***	1.29	0.80
Adult Income 2009	1.02	1.04	1.01	1.02	1.06	0.97
High School or Less	1.01	1.11	1.84	1.03	1.37	1.61 *
College Degree	0.86	0.85	0.38	0.63 ***	0.78	0.64
Advanced Degree	0.80	0.94	0.48	0.68 *	0.76	0.93
Age in 2009	1.02	1.08	0.93	1.06 **	1.12 *	1.16 *
Immigrant	0.62	0.38	0.00 ***	2.48	0.15	0.00 ***
Doesn't Walk for Exercise	1.25	1.07	1.06	1.08	1.36	0.96
Currently Obese	1.80 ***	2.49 ***	2.95 **	2.32 ***	3.30 ***	4.73 ***
Microsomic	1.26	0.89	0.98	0.99	1.80	1.14
Macrosomic	1.09	0.74	2.23	0.79	0.57	1.21
Parent Diabetic	1.05	1.11	1.66	1.27	2.36 *	1.89 **

Notes: a. Pre-diabetic refers to having a hemoglobin A1C level of 5.7-6.49 and no diagnostic history of diabetes when the respondent was not pregnant; b. Undiagnosed diabetic refers to having a hemoglobin A1C level 6.5+ and no prior diabetes diagnosis when the respondent was not pregnant; c. Diagnosed diabetic refers to the respondent ever being diagnosed with diabetes when not pregnant regardless of hemoglobin A1C status; d. relative risk ratios reported comparing the relative risk of category a-c relative to being normoglycemic; e. Pregnant women excluded from analyses; f. * p<0.05, ** p<0.01, *** p<0.001

Table 3.4 Interaction Effects for Full and Race-Stratified Final Models

Pre-Diabetes	Full Sample	Non-Hispanic Black	Non-Hispanic White
<i>Interaction Effects</i>			
Rare Discrimination x African American	0.77		
Some Discrimination x African American	0.76		
Often Discrimination x African American	0.87		
Rare Discrimination x Mastery Score	0.96	1.12	0.88
Some Discrimination x Mastery Score	1.14	1.16	1.08
Often Discrimination x Mastery Score	0.99	1.07	0.91
<hr/>			
Undiagnosed Diabetes	Full Sample	Non-Hispanic Black	Non-Hispanic White
<i>Interaction Effects</i>			
Rare Discrimination x African American	0.67		
Some Discrimination x African American	0.64		
Often Discrimination x African American	0.53		
Rare Discrimination x Mastery Score	0.76	0.78	0.87
Some Discrimination x Mastery Score	1.01	0.83	1.41
Often Discrimination x Mastery Score	0.59	0.40 *	0.99
<hr/>			
Diagnosed Diabetes	Full Sample	Non-Hispanic Black	Non-Hispanic White
<i>Interaction Effects</i>			
Rare Discrimination x African American	0.35 *		
Some Discrimination x African American	0.32 *		
Often Discrimination x African American	0.40		
Rare Discrimination x Mastery Score	0.69	0.83	0.74
Some Discrimination x Mastery Score	0.88	0.61	1.13
Often Discrimination x Mastery Score	1.11	0.53	1.55

Notes: a. Pre-diabetic refers to having a hemoglobin A1C level of 5.7-6.49 and no diagnostic history of diabetes when the respondent was not pregnant; b. Undiagnosed diabetic refers to having a hemoglobin A1C level 6.5+ and no prior diabetes diagnosis when the respondent was not pregnant; c. Diagnosed diabetic refers to the respondent ever being diagnosed with diabetes when not pregnant regardless of hemoglobin A1C status; d. relative risk ratios reported comparing the relative risk of category a-c relative to being normoglycemic; e. Pregnant women excluded from analyses; f. * p<0.05, ** p<0.01, *** p<0.001

3.9 Appendices

Appendix 3.1 Models Predicting Pre-Diabetes Risk-Relative Risk Ratios Reported Full Sample (N=10,723)

Pre-Diabetes	Model 1		Model 2		Model 3	
<i>Race</i>						
Non-Hispanic Black	2.94	***	2.94	***	3.52	***
<i>Sources of Stress</i>						
Rarely Experiences Discrimination			1.08		1.13	
Sometimes Experiences Discrimination			1.17		1.30	*
Often Experiences Discrimination			0.98		0.98	
<i>Risky Coping Strategies</i>						
Fast Food Consumption					1.01	
Sugary Drink Consumption					1.01	**
Daily Drinker					0.72	
Regular Smoker					1.08	
<i>Personal Resources</i>						
Mastery Factor Score					1.03	
<i>Interaction Effects</i>						
Rare Discrimination x African American					0.77	
Some Discrimination x African American					0.76	
Often Discrimination x African American					0.87	
Rare Discrimination x Mastery Score					0.96	
Some Discrimination x Mastery Score					1.14	
Often Discrimination x Mastery Score					0.99	
<i>Control Variables</i>						
Male	1.49	***	1.49	***	1.48	***
Adult Income 2009	1.02		1.02		1.02	
High School or Less	1.04		1.04		1.03	
College Degree	0.63	***	0.63	***	0.66	***
Advanced Degree	0.66	**	0.67	**	0.71	*
Age in 2009	1.05	**	1.05	**	1.05	**
Immigrant	1.28		1.28		1.30	
Microsomic	1.12		1.12		1.11	
Macrosomic	0.84		0.83		0.84	
Currently Obese	2.19	***	2.19	***	2.21	***
Doesn't Walk for Exercise	1.16	*	1.17	*	1.14	
Parent Diabetic	1.21		1.21		1.20	

Notes: a. Pre-diabetic refers to having a hemoglobin A1C level of 5.7-6.49 and no diagnostic history of diabetes when the respondent was not pregnant, b. relative risk ratios reported comparing the relative risk of pre-diabetes relative to being normoglycemic; c. Pregnant women excluded from analyses; d. * p<0.05, ** p<0.01, *** p<0.001

Appendix 3.2 Models Predicting Undiagnosed Diabetes Risk-Relative Risk Ratios Reported Full Sample (N=10,723)

Undiagnosed Diabetes	Model 1		Model 2		Model 3	
<i>Race</i>						
Non-Hispanic Black	7.63	***	7.76	***	11.04	***
<i>Sources of Stress</i>						
Rarely Experiences Discrimination			1.10		1.29	
Sometimes Experiences Discrimination			0.88		1.09	
Often Experiences Discrimination			0.50		0.43	
<i>Risky Coping Strategies</i>						
Fast Food Consumption					0.93	*
Sugary Drink Consumption					1.00	
Daily Drinker					0.73	
Regular Smoker					0.88	
<i>Personal Resources</i>						
Mastery Factor Score					1.02	
<i>Interaction Effects</i>						
Rare Discrimination x African American					0.67	
Some Discrimination x African American					0.64	
Often Discrimination x African American					0.53	
Rare Discrimination x Mastery Score					0.76	
Some Discrimination x Mastery Score					1.01	
Often Discrimination x Mastery Score					0.59	
<i>Control Variables</i>						
Male	1.32		1.31		1.35	*
Adult Income 2009	1.05		1.04		1.05	
High School or Less	1.20		1.23		1.20	
College Degree	0.80		0.79		0.77	
Advanced Degree	0.87		0.85		0.83	
Age in 2009	1.11	**	1.11	**	1.11	**
Immigrant	0.75		0.75		0.70	
Microsomic	1.19		1.19		1.19	
Macrosomic	0.61		0.61		0.61	
Currently Obese	3.03	***	3.06	***	3.01	***
Doesn't Walk for Exercise	1.13		1.13		1.19	
Parent Diabetic	1.62		1.62		1.67	

Notes: a. Undiagnosed diabetic refers to having a hemoglobin A1C level 6.5+ and no prior diabetes diagnosis when the respondent was not pregnant; b. relative risk ratios reported comparing the relative risk of undiagnosed diabetes relative to being normoglycemic; c. Pregnant women excluded from analyses; d. * p<0.05, ** p<0.01, *** p<0.001

Appendix 3.3 Models Predicting Diagnosed Diabetes Risk-Relative Risk Ratios Reported Full Sample (N=10,723)

Diagnosed Diabetes	Model 1	Model 2	Model 3
<i>Race</i>			
Non-Hispanic Black	1.94 ***	1.95 ***	3.99 ***
<i>Sources of Stress</i>			
Rarely Experiences Discrimination		1.19	1.55
Sometimes Experiences Discrimination		1.16	1.60
Often Experiences Discrimination		1.71	2.47 *
<i>Risky Coping Strategies</i>			
Fast Food Consumption			1.05 *
Sugary Drink Consumption			0.99
Daily Drinker			0.27
Regular Smoker			1.12
<i>Personal Resources</i>			
Mastery Factor Score			1.06
<i>Interaction Effects</i>			
Rare Discrimination x African American			0.35 *
Some Discrimination x African American			0.32 *
Often Discrimination x African American			0.40
Rare Discrimination x Mastery Score			0.69
Some Discrimination x Mastery Score			0.88
Often Discrimination x Mastery Score			1.11
<i>Control Variables</i>			
Male	0.89	0.89	0.90
Adult Income 2009	0.98	0.98	0.98
High School or Less	1.71 **	1.70 **	1.65 **
College Degree	0.52 *	0.53 *	0.55 *
Advanced Degree	0.73	0.74	0.78
Age in 2009	1.10 *	1.10 *	1.10 *
Immigrant	0.00 ***	0.00 ***	0.00 ***
Microsomic	1.10	1.10	1.12
Macrosomic	1.30	1.32	1.36
Currently Obese	4.19 ***	4.15 ***	4.15 ***
Doesn't Walk for Exercise	0.98	0.98	0.98
Parent Diabetic	1.83 **	1.80 **	1.85 **

Notes: a. Diagnosed diabetic refers to the respondent ever being diagnosed with diabetes when not pregnant regardless of hemoglobin A1C status; b. relative risk ratios reported comparing the relative risk of diagnosed diabetes relative to being normoglycemic; c. Pregnant women excluded from analyses; d. * p<0.05, ** p<0.01, *** p<0.001

Appendix 3.4 Race Stratified Models Predicting Pre-Diabetes Risk-Relative Risk Ratios Reported

Pre-Diabetes	Non-Hispanic Black (N=3,124)				Non-Hispanic White (N=7,599)			
	Model 1A	Model 2A	Model 3A	Model 4A	Model 1A	Model 2A	Model 3A	Model 4A
Social Stressors								
Rarely Experiences Discrimination	0.87		0.89	0.89	1.14		1.13	1.14
Sometimes Experiences Discrimination	0.96		0.99	1.00	1.24		1.24	1.29 *
Often Experiences Discrimination	0.91		0.97	0.94	0.99		0.97	0.93
Risky Coping Strategies								
Fast Food Consumption		0.99	0.99	0.99		1.02	1.02	1.02
Sugary Drink Consumption		1.00	1.00	1.00		1.01 **	1.01 **	1.01 **
Daily Drinker		1.43	1.42	1.41		0.54 *	0.54 *	0.54 *
Regular Smoker		0.71	0.71	0.72 *		1.19 *	1.19	1.19
Personal Resources								
Mastery Factor Score			1.05	0.97			1.05	1.09
Interaction Effects								
Rare Discrimination x Mastery Score				1.12				0.88
Some Discrimination x Mastery Score				1.16				1.08
Often Discrimination x Mastery Score				1.07				0.91
Control Variables								
Male	1.37 **	1.41 **	1.42 **	1.43 **	1.55 ***	1.53 ***	1.53 ***	1.53 ***
Adult Income 2009	1.02	1.02	1.02	1.02	1.02	1.02	1.02	1.02
High School or Less	0.97	0.99	1.01	1.01	1.07	1.02	1.03	1.03
College Degree	0.89	0.86	0.85	0.86	0.58 ***	0.63 ***	0.63 ***	0.63 ***
Advanced Degree	0.85	0.81	0.80	0.80	0.61 **	0.67 *	0.67 *	0.68 *
Age in 2009	1.03	1.03	1.02	1.02	1.06 *	1.06 **	1.06 **	1.06 **
Immigrant	0.67	0.63	0.63	0.62	2.40	2.51	2.45	2.48
Doesn't Walk for Exercise	1.24	1.25	1.25	1.25	1.12	1.07	1.07	1.08
Currently Obese	1.84 ***	1.82 ***	1.81 ***	1.80 ***	2.27 ***	2.31 ***	2.31 ***	2.32 ***
Microsomic	1.29	1.26	1.26	1.26	1.00	0.98	0.98	0.99
Macrosomic	1.11	1.10	1.11	1.09	0.79	0.79	0.79	0.79
Parent Diabetic	1.04	1.04	1.05	1.05	1.29	1.28	1.28	1.27

Notes: a. Pre-diabetic refers to having a hemoglobin A1C level of 5.7-6.49 and no diagnostic history of diabetes when the respondent was not pregnant, b. relative risk ratios reported comparing the relative risk of pre-diabetes relative to being normoglycemic; c. Pregnant women excluded from analyses; d. * p<0.05, ** p<0.01, *** p<0.001

Appendix 3.5 Race Stratified Models Predicting Undiagnosed Diabetes Risk-Relative Risk Ratios Reported

Undiagnosed Diabetes	Non-Hispanic Black (N=3,124)				Non-Hispanic White (N=7,599)			
	Model 1A	Model 2A	Model 3A	Model 4A	Model 1A	Model 2A	Model 3A	Model 4A
Social Stressors								
Rarely Experiences Discrimination	0.87		0.88	0.91	1.28		1.28	1.27
Sometimes Experiences Discrimination	0.71		0.72	0.74	1.03		0.93	1.09
Often Experiences Discrimination	0.37 *		0.39	0.20 *	0.72		0.58	0.55
Risky Coping Strategies								
Fast Food Consumption		0.92	0.93	0.93		0.92	0.91	0.91
Sugary Drink Consumption		1.01	1.01	1.01		0.99	0.99	0.99
Daily Drinker		1.03	1.03	1.02		0.71	0.68	0.68
Regular Smoker		0.70	0.71	0.71		0.92	0.92	0.92
Personal Resources								
Mastery Factor Score			1.02	1.22			0.75	0.73
Interaction Effects								
Rare Discrimination x Mastery Score				0.78				0.87
Some Discrimination x Mastery Score				0.83				1.41
Often Discrimination x Mastery Score				0.40 *				0.99
Control Variables								
Male	1.30	1.38 *	1.37 *	1.36 *	1.23	1.34	1.29	1.29
Adult Income 2009	1.04	1.05	1.04	1.04	1.05	1.05	1.06	1.06
High School or Less	1.05	1.07	1.09	1.11	1.37	1.45	1.37	1.37
College Degree	0.91	0.87	0.86	0.85	0.81	0.75	0.78	0.78
Advanced Degree	1.00	0.94	0.94	0.94	0.80	0.71	0.74	0.76
Age in 2009	1.09	1.08	1.09	1.08	1.12 *	1.12 *	1.12 *	1.12 *
Immigrant	0.42	0.39	0.36	0.38	0.15	0.14	0.15	0.15
Doesn't Walk for Exercise	1.03	1.07	1.07	1.07	1.26	1.35	1.34	1.36
Currently Obese	2.53 ***	2.44 ***	2.45 ***	2.49 ***	3.36 ***	3.26 ***	3.29 ***	3.30 ***
Microsomic	0.91	0.87	0.88	0.89	1.77	1.79	1.78	1.80
Macrosomic	0.74	0.71	0.71	0.74	0.53	0.55	0.56	0.57
Parent Diabetic	1.08	1.10	1.11	1.11	2.32 *	2.36 *	2.38 *	2.36 *

Notes: a. Undiagnosed diabetic refers to having a hemoglobin A1C level 6.5+ and no prior diabetes diagnosis when the respondent was not pregnant; b. relative risk ratios reported comparing the relative risk of undiagnosed diabetes relative to being normoglycemic; c. Pregnant women excluded from analyses; d. * p<0.05, ** p<0.01, *** p<0.001

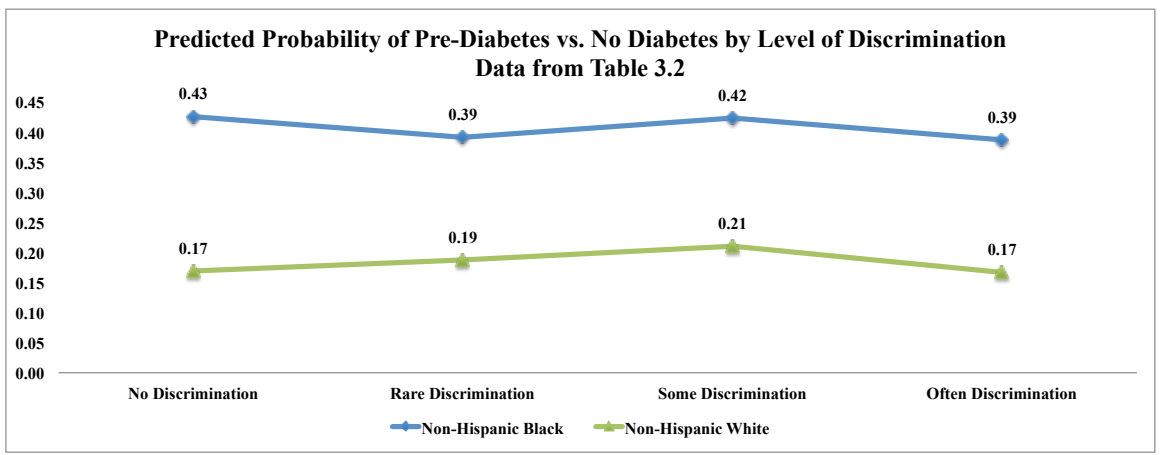
Appendix 3.6 Race Stratified Models Predicting Diagnosed Diabetes Risk-Relative Risk Ratios Reported

Diagnosed Diabetes	Non-Hispanic Black (N=3,124)				Non-Hispanic White (N=7,599)			
	Model 1A	Model 2A	Model 3A	Model 4A	Model 1A	Model 2A	Model 3A	Model 4A
Social Stressors								
Rarely Experiences Discrimination	0.53		0.59	0.61	1.61 *		1.57 *	1.52
Sometimes Experiences Discrimination	0.54		0.64	0.60	1.64		1.50	1.60
Often Experiences Discrimination	1.04		1.18	0.92	2.20		1.85	2.55 *
Risky Coping Strategies								
Fast Food Consumption		1.06	1.05	1.05		1.06 *	1.05 *	1.05
Sugary Drink Consumption		0.96 *	0.96 *	0.96 *		1.00	1.00	1.00
Daily Drinker		0.11	0.11	0.10 *		0.36	0.35	0.35
Regular Smoker		0.57	0.61	0.59		1.30	1.29	1.26
Personal Resources								
Mastery Factor Score			1.12	1.40			0.85	0.85
Interaction Effects								
Rare Discrimination x Mastery Score				0.83				0.74
Some Discrimination x Mastery Score				0.61				1.13
Often Discrimination x Mastery Score				0.53				1.55
Control Variables								
Male	1.17	1.24	1.26	1.26	0.80	0.81	0.79	0.80
Adult Income 2009	1.02	1.01	1.01	1.01	0.97	0.97	0.98	0.97
High School or Less	1.60	1.87 *	1.84	1.84	1.71 *	1.68 *	1.61 *	1.61 *
College Degree	0.43	0.40	0.38	0.38	0.57	0.61	0.63	0.64
Advanced Degree	0.57	0.48	0.48	0.48	0.82	0.85	0.91	0.93
Age in 2009	0.95	0.94	0.94	0.93	1.16 *	1.17 **	1.17 **	1.16 *
Immigrant	0.00 ***	0.00 ***	0.00 ***	0.00 ***	0.00 ***	0.00 ***	0.00 ***	0.00 ***
Doesn't Walk for Exercise	1.03	1.11	1.07	1.06	0.96	0.93	0.94	0.96
Currently Obese	2.91 **	2.91 **	2.85 **	2.95 **	4.66 ***	4.76 ***	4.67 ***	4.73 ***
Microsomic	0.98	0.94	0.96	0.98	1.15	1.15	1.15	1.14
Macrosomic	2.13	2.06	2.14	2.23	1.18	1.19	1.21	1.21
Parent Diabetic	1.72	1.59	1.65	1.66	1.85 **	1.91 **	1.89 **	1.89 **

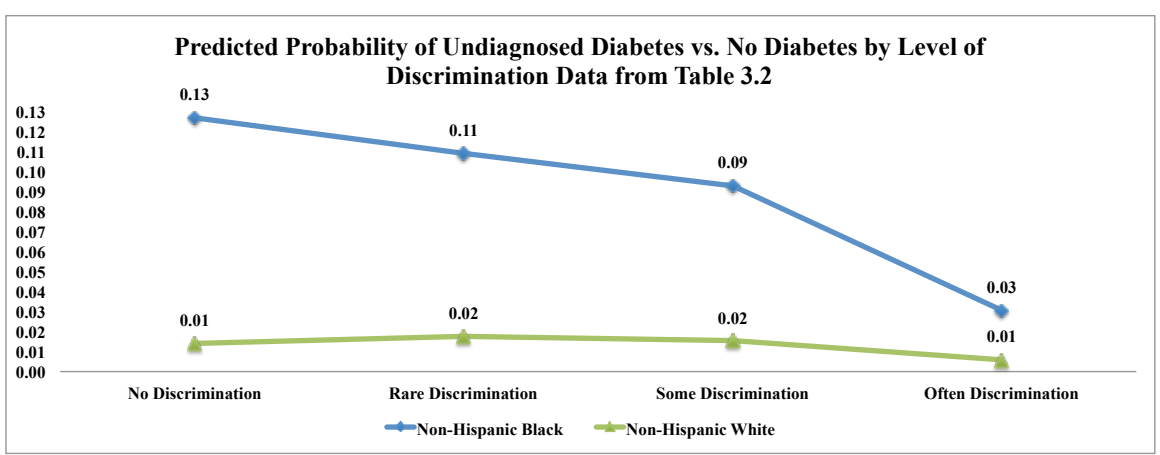
Notes: a. Diagnosed diabetic refers to the respondent ever being diagnosed with diabetes when not pregnant regardless of hemoglobin A1C status; b. relative risk ratios reported comparing the relative risk of diagnosed diabetes relative to being normoglycemic; c. Pregnant women excluded from analyses; d. * p<0.05, ** p<0.01, *** p<0.001

Appendices 3.7-3.9 Predicted Probabilities of Diabetes Risk by Level of Discrimination (Table 3.2)

Appendix 3.7

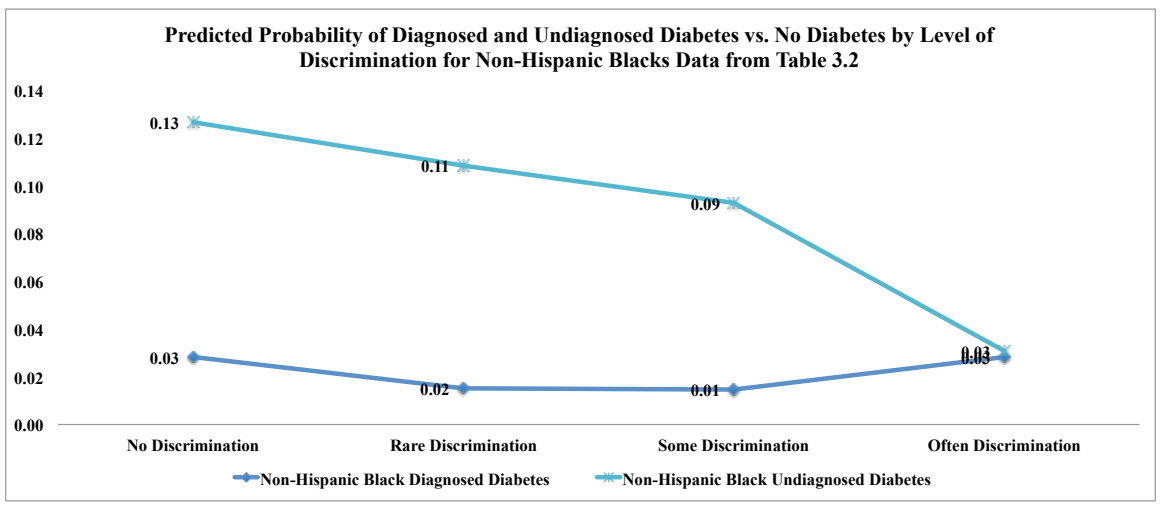


Appendix 3.8

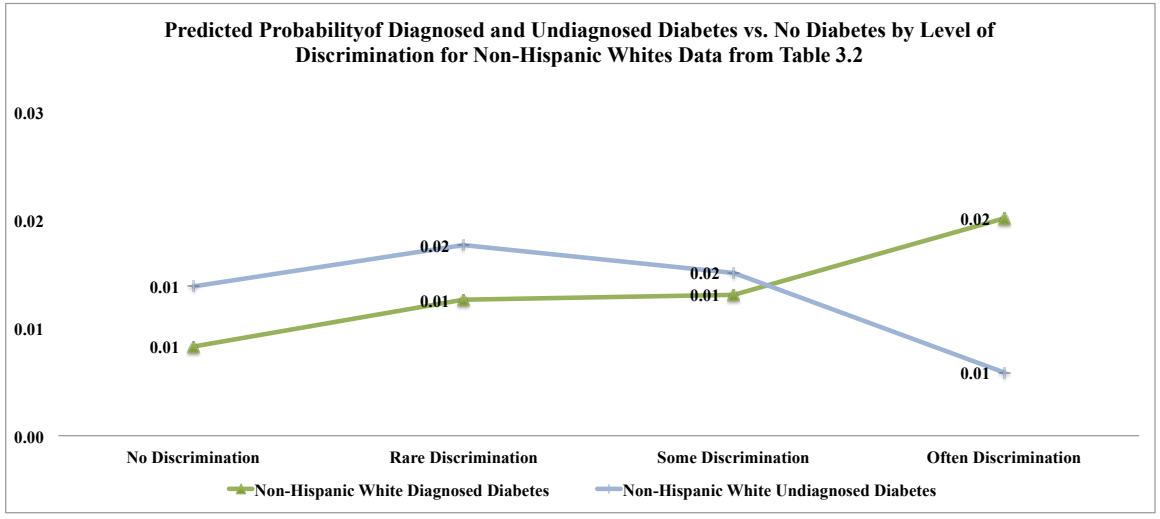


Appendices 3.9-3.10 Predicted Diagnosed and Undiagnosed Diabetes Relative Risk by Level of Discrimination by Race (Table 3.2)

Appendix 3.9

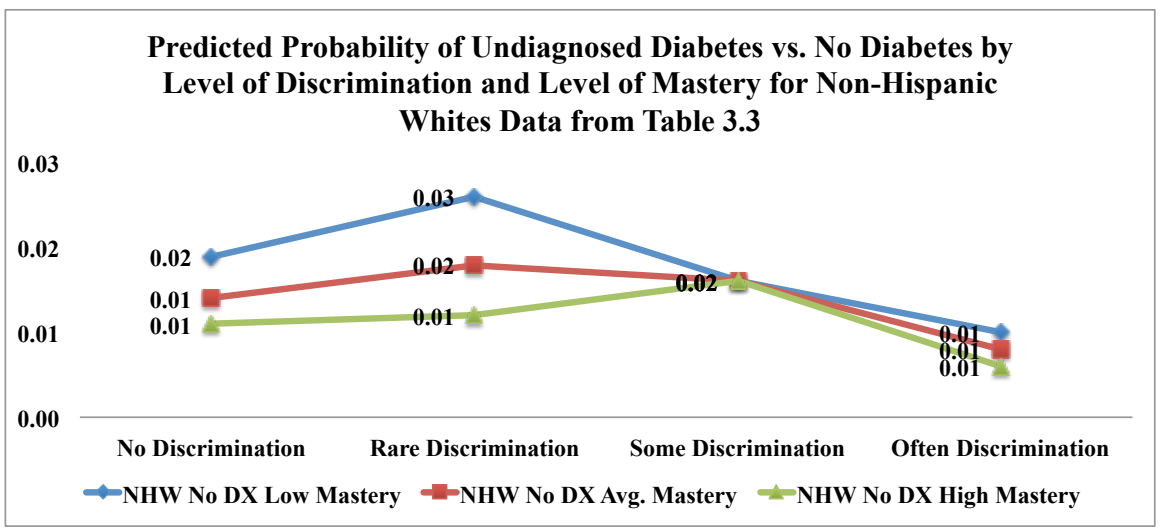


Appendix 3.10



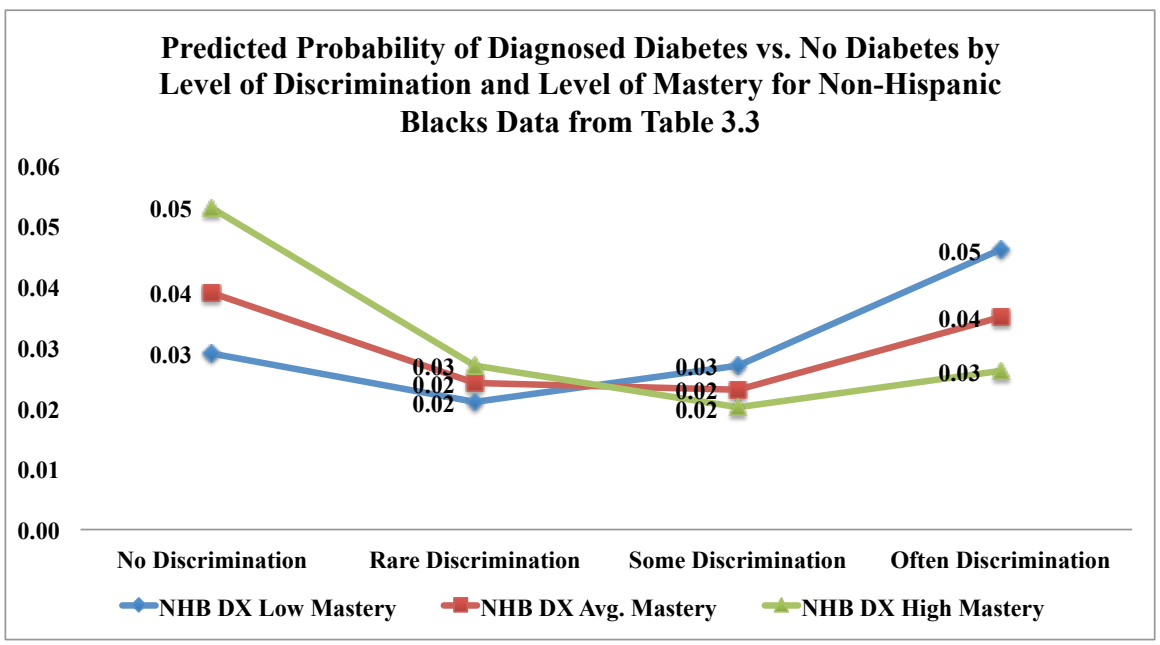
Appendices 3.11 Predicted Probabilities of Undiagnosed Diabetes Risk by Level of Discrimination and Level of Mastery by Race (Table 3.3)

Appendix 3.11

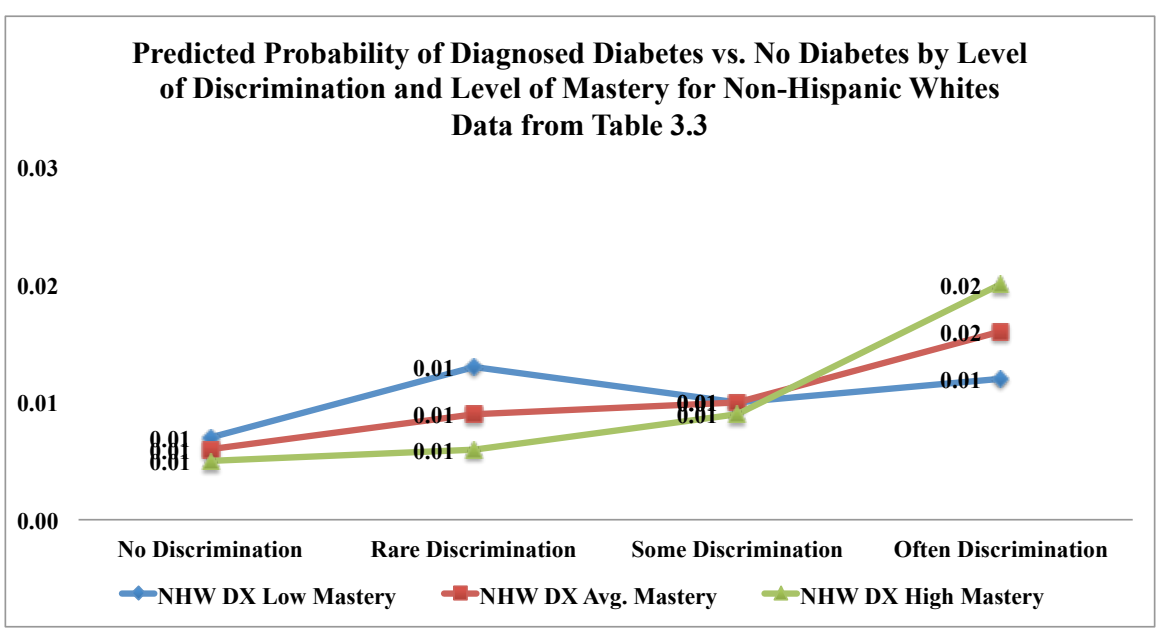


Appendices 3.12-3.13 Predicted Probabilities of Diagnosed Diabetes Risk by Level of Discrimination and Level of Mastery by Race (Table 3.3)

Appendix 3.12



Appendix 3.13



CHAPTER 4**Testing the Behavioral Model of Health Services Use: Are Disparities in Diabetes Diagnoses for Young Adults Due to Differences in Help Seeking or Diagnosis Allocation?****Abstract**

Both early detection and continued monitoring of diabetes are important for proper health maintenance among diabetics. As diabetes increases among young adults, whether diabetes risk is being diagnosed accordingly across demographic groups remains unclear. Andersen's Behavioral Model of Health Services Use (1995) provides a theoretical framework to assess whether diabetes diagnostic disparities are due to differences in help seeking or differences in diagnostic testing among young adults with diabetes. Tests of Andersen's model with young adult diabetics from the National Longitudinal Study of Adolescent Health (N=915) reveal no difference in help seeking across race/ethnic groups. However, although all race/ethnic groups are equally likely to seek care, large diagnostic disparities persist particularly for non-Hispanic blacks. As a result, young adult non-Hispanic black diabetics are more than four times less likely to receive a diagnosis for diabetes even when they sought care in the previous three months. Future research is necessary to determine what it is about doctor visits that contribute to this diagnostic disparity.

Key words: Diabetes, health disparities, and Add Health

Abstract Word count: 163

Word Count: 5,903

Acknowledgement: This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

4.1 Introduction

Diabetes is a growing national health problem for young adults (Mokdad et al. 2001). The national prevalence estimates of diabetes among American adults have risen dramatically since the 1980s (Cowie et al. 2006; Cowie et al. 2010). The extent of racial health disparities in diabetes diagnosis allocation remain unclear for young adults because many studies lack adequate sample sizes of both diagnosed and undiagnosed cases in younger cohorts to allow for comparisons across groups. According to the 2011 National Diabetes Fact Sheet issued by the Centers for Disease Control and Prevention (CDC), the most recent estimates for the prevalence of diabetes indicate racial disparities for non-Hispanic blacks (18.7% of all adults over age 20) when compared to non-Hispanic whites (10.2% of all adults over age 20) (<http://www.cdc.gov/diabetes/pubs/estimates11.htm#4>), but do not provide more information about race-age-diagnosis breakdowns to indicate how racial health disparities affect young adults specifically.

To be diagnosed with diabetes, one must have the condition, seek care, and receive testing to reveal the condition. Delays in seeking care could impede timely diagnosis and increase the risk of long-term morbidity from complications of prolonged undiagnosed diabetes (Sima 2000). Andersen's Behavioral Model of Health Services Use (BMHSU) provides a theoretical framework to model behaviors that may influence the likelihood of seeking care, which should influence diagnosis rates (Andersen 1995; 2008). People with fewer diabetes symptoms may be less likely to seek care if they do not perceive a need for treatment, which may reduce the likelihood of receiving a diagnosis early. Although people with more or worse somatic complaints may be more

likely to receive diagnoses when they seek care they may be in worse health when they are diagnosed, which could lead to increased risk of irreversible complications of diabetes later (Chakrabarti 2000; Koopman et al. 2006).

Receiving a diabetes diagnosis requires more than mere interaction with a doctor, it also requires that a doctor perceive diabetes as a critical concern for an individual based on his or her constellation of symptoms resulting in appropriate testing and issuing the diagnosis. Therefore increased diabetes symptoms and risk factors should increase the likelihood that someone will be diagnosed if they seek care and the doctor thinks diabetes could be the underlying cause. Prior research has demonstrated differences in health care utilization and perceptions of health care efficacy among young adults by race (Bogart et al. 2004; Fiscella et al. 2002; Smedly, Stith, and Nelson 2003), but little attention has been paid to differences in diagnosis allocation for those who seek care by race. If doctors test patients at different rates, this could indicate bias on the part of medical professionals resulting in long-term health disparities for those with delayed diagnoses not from differences in help seeking, but from delayed testing.

Taken together, it is important to assess whether there are disparities in diagnosis rates for young adult diabetics who seek care. Moreover, if differences are found, it is important to discern if these differences arise from differential patterns in help seeking or diagnosis allocation. Andersen's (1995) Behavioral Model for Health Services Use (BMHSU) provides a theoretical framework to address this complex problem. Applying this framework, I ask the following questions 1.) Do young adults with diabetes utilize healthcare equally by race? If not, 2.) Do differential patterns in help seeking explain diagnostic disparities for young adult diabetes? Conversely, if help seeking patterns are

equivalent across demographic groups, 3.) Is diabetes diagnosis allocation equivalent among diabetics who seek care by race?

4.2 Literature Review and Theoretical Model

Diabetes and the Diagnostic Process

Diabetes is a chronic disease that arises from an inability of the pancreas to regulate the balance of glucose and insulin production, which leads to excessive levels of glucose in the blood (American Diabetes Association 2013; World Health Organization 2012). Diabetes can be detected by the presence of excessive glucose in the bloodstream. The most common clinical biomarkers used to determine the presence of diabetes are *fasting glucose*, or a measure of the amount of glucose remaining in the blood after a period of fasting of at least eight hours, and *hemoglobin A1C*, which is an indicator of the proportion of hemoglobin molecules in red blood cells that have become glycated or glucose-containing (Olson et al. 2010). Diabetes is indicated if fasting glucose levels exceed 125 milligrams per deciliter or hemoglobin A1C levels exceed 6.5% of hemoglobin molecules (Olson et al. 2010).

Frequently, glucose testing is indicated when patients report a history of relevant somatic complaints such as frequent urination or increased thirst, have a family history of the condition, or have a history of other conditions shown to co-occur with diabetes (Trull et al. 2002). Clinical presentations of diabetes can vary including symptoms such as excessive thirst, frequent urination, excessive hunger, fatigue, blurry vision, weight loss, and poor overall health (Trull et al. 2002; Borchard 1995). Several conditions may co-occur with diabetes like high blood pressure (Lackland et al. 1992) or high cholesterol (Zoratti et al. 2000) or complex conditions like *metabolic syndrome* where all three

conditions, diabetes, hypertension, and high cholesterol, co-occur (Grundy et al. 2005). Other conditions frequently co-occurring with diabetes include *android obesity*, where patients are clinically obese having a body mass index greater than 30.0, but carry the bulk of their excess weight around the midsection. This condition can be a clinical risk factor for diabetes due to the increased risk of accumulated visceral fat (fat around the organs) particularly fat around the liver or pancreas (Reaven 1988).

Diabetes rarely occurs without symptoms, but failure to diagnose diabetes can happen if the symptoms are subtle, doctors do not suggest testing, or other circumstances lead patients to avoid seeking care, which can delay diagnosis. Delayed diagnosis, or prolonged undiagnosed diabetes, increases the likelihood that a person will suffer long-term negative health effects of diabetes. These effects include increased risk of dangerous and frequently irreversible complications such as slow wound healing, diabetic neuropathy (nerve damage), limb loss, retinopathy (vision condition leading to blindness), and kidney disorders (Borchard 1995; Chakrabarti 2000; Koopman et al. 2006; Sharma and Richards 2000; Trull et al. 2002). Disparities in timing of diagnosis can be cause for concern if timing discrepancies lead to morbidity and mortality differences for those who seek care.

Help Seeking vs. Avoidance of Care

In order to be diagnosed with diabetes one must have access to care, desire for treatment, and be tested by a medical professional. Access to care is more likely to be determined by socioeconomic status or geographic location than choice, but desire for treatment may be related to choices based on health beliefs regarding health maintenance and the efficacy of healthcare. Koszegi (2003) noted how the stigma of disease may be a

deterrent from seeking care among some individuals just as others have cited the cost of care as a major reason for avoiding necessary care specifically related to diabetes (Nichols, Arondekar, and Herman 2008; Nichols and Brown 2005; Zhang et al. 2009). Although there is some literature on the deterrents of seeking care, relatively little research has focused on identifying the factors that motivate diabetics specifically to seek or avoid care.

Both financial and non-financial barriers to access to care may also factor into delaying diagnosis (Kullgren et al. 2012). Prior to the implementation of the Patient Protection and Affordable Care Act, young adults could stay on parent health plans until age 25 usually with the caveat that the adult child needed to be a full-time student (Hall 2011; DeVoe 2008). As a result, several young adults without health care benefits or full-time student status have foregone health insurance to save money (Guy 2010). Those without insurance may seek more transient care from urgent care or specialty clinics, which provide lower quality care and less follow up than primary care physicians (Lutfey and Freese 2005).

Although financial constraints and fear of the stigma of disease are realistic concerns regarding healthcare utilization, the perception of maltreatment by the medical community is another noteworthy contributor to reduced healthcare utilization among minority groups. These perceptions are not unfounded as historically minority groups in general, and African Americans specifically, have a history of maltreatment by the medical community (Corbie-Smith et al. 1999; Kennedy, Mathis, and Woods 2007). Both social science and medical literature include accounts of racialized medical mistreatment from the Tuskegee Syphilis Study (Brandt 1978), to the appropriation of Henrietta Lacks'

cancer cells (Skloot 2010), and numerous other historical accounts of racialized medical exploitation in the United States (Washington 2006). Moreover, the views of racial minorities regarding medical research has been linked with perceptions of lower quality healthcare and negative interactions with medical professionals (Friemuth et al. 2001; Gamble 1997; Heisler et al. 2005).

Research exists suggesting that some racial minorities, particularly African Americans, avoid seeking care due to preconceived notions regarding discriminatory interactions with doctors (Hammond 2010; Lyles et al. 2011; Vaccaro & Huffman 2012). There is evidence, however, that perceptions of lower quality care for race/ethnic minorities continue to be a valid concern in the United States. Recently, Stepanikova (2012) found that when doctors are under time pressure, they are less likely to refer African American patients for advanced testing than white patients with the same symptoms, which may imply racial bias in the allocation of treatment even when patients make it into the office and present with the same symptoms. Taken together, there is reason to suspect that if there are differences in health care utilization by race this may be due to perceptions of lower quality care. However, there is also the possibility that diagnosis allocation may differ by race, which would reinforce perceptions of lower quality care.

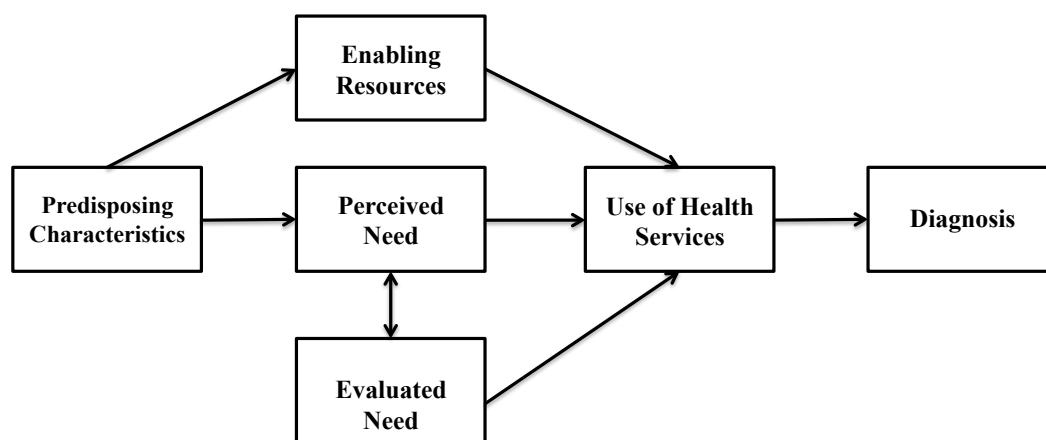
The Behavioral Model

One of the main sociological help seeking models used to predict health care utilization is Andersen's Behavioral Model for Health Services Use (1968; 1995), which may provide a useful framework for understanding diabetes diagnosis disparities. This model focuses on the role of individuals' *predisposing characteristics*, *need* (both

perceived and evaluated) for care, and *enabling resources* to aid in seeking care and predicting health outcomes (Andersen 1995; 2008). Under this framework, demographic characteristics such as race, social class, and gender are considered predisposing characteristics (Andersen 2008).

4.2.1 Figure 4.1 Conceptual Map for the Behavioral Model of Health Services Use

Figure 4.1- Conceptual Map for the Behavioral Model of Health Services Use



In the context of help seeking for diabetes, *perceived need* could come from assessing somatic complaints as indicators of illness that would encourage someone to seek care (Becker 1974; Hopton and Dlugolecka 1995). Sociological research explains the understanding of perceived need in many ways including the internalization of the “sick role” where the presence of illness leads to behavioral differences between those who are “sick” and those who are “well” because illness is seen as a type of deviant status with the obligation of striving for wellness (Becker, Drachman, and Kirscht 1974; Parsons 1951; Segall 1976; Twaddle 1969). Conversely, *evaluated need* could be assessed by decisions of medical professionals based on clinical criteria and tests revealing illness, perhaps as a result of testing for other related conditions (Little et al.

2004). In the context of medical care help seeking, *enabling resources* at the individual level could include any factors that make care easier to obtain such as health insurance, increased availability of providers, or increased ability to navigate the healthcare system; perhaps through increased education or income (Andersen 2008; Dunlop, Coyote, & Mc Isaac 2000).

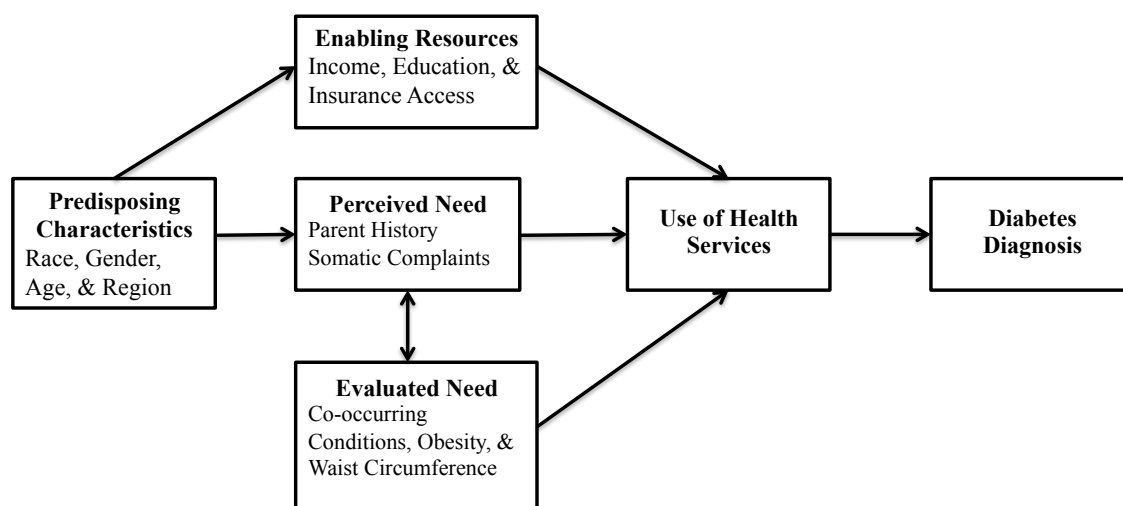
Choices and life experiences can alter how people perceive their need for care and their desire to seek interactions with doctors (Fiscella et al. 2002). In this context, beliefs regarding the efficacy of the health care system and past interactions with doctors could fall under predisposing characteristics. Perceived need would require someone to believe that his or her symptoms warrant consultation with a doctor, which plausibly would involve either increased symptom severity or a perception of increased risk. Failure to recognize symptoms or be aware of risk factors for diabetes may be higher among young adults who do not think they are susceptible to chronic illness this early in the life course (Walker et al. 2003; Van Osch, van den Hout, and Stiggelbout 2006; Vernon 1999). However, some people may recognize their risk of diabetes due to familial risk, but avoid testing out of fear of diagnosis. Fear of diagnosis in this manner has been associated with avoiding diagnostic cancer screenings by people with parent histories of cancer (Lim et al 2011; Benyamini et al. 2003; Kash et al. 1992; Kenen et al. 2003), but has not yet been seen with diabetes.

Enabling resources such as the aforementioned access to care and ability to pay could also be factors related to opting in or out of seeking care. The expense of care following a diabetes diagnosis can be high particularly for people without insurance. However, research has shown that pre-diabetes and undiagnosed diabetes can cost more

in the long run than diabetes that is diagnosed and treated early (Nichols, Arondekar, and Herman 2008; Nichols and Brown 2005; Zhang et al. 2009). Consequently, the initial costs of seeking care may put individuals with lower socioeconomic statuses at higher risk of delaying diagnosis and higher risk of more expensive long-term treatment for diabetes complications.

4.2.2 Figure 4.2 Conceptual Map for the Behavioral Model of Health Services Use for Diabetes Diagnosis

Figure 4.2- Conceptual Map for the Behavioral Model of Health Services Use for Diabetes Diagnosis



4.3 Hypotheses

Hypothesis 1: Health care use will be higher among non-Hispanic whites than racial minorities.

Hypothesis 2: Differences in help seeking by race will correlate with diabetes diagnosis allocation.

Hypothesis 3: Diabetes diagnosis allocation should be equivalent for respondents who utilize health care.

4.4 Methods

Data and Sample

The data for this study come from Wave IV of the National Longitudinal Study of Adolescent Health (Add Health) (Harris 2009). The Add Health study was designed to be a nationally representative sample of students in schools in grades 7-12 in 1994-1995 (c.f. Chantala and Tabor 1999). During the first wave of data collection, both respondents and their parents or legal guardians were interviewed. The focal respondents have been re-interviewed in 1996, 2001-2002, and 2008-2009. Both biological markers and survey data were collected on the focal respondents from in person interviews in the most recent wave of data collection in 2008-2009.

There were 15,701 respondents who participated in Wave IV. Of those respondents, 1,101 people were deemed diabetic from either past diagnoses or meeting current clinical diabetes criteria (Whitsel et al. 2012). The analytic sample for this study only includes the subset of individuals in the Add Health study who were either diagnosed or undiagnosed diabetics in Wave IV who were not pregnant at the time of interview. The sample was further reduced to exclude individuals who racially identified as Asian or Pacific Islander, “other race” individuals, or were missing a racial identification due to too few cases for multivariate analyses. The final analytic sample was comprised of 915 diabetic individuals after dropping cases for the aforementioned reasons.

The Add Health study intentionally oversampled race/ethnic minorities, twins, sibling pairs, and persons with limb deformities. Due to these oversamples it is not possible to generalize to the young adult population without applying complex survey

design weights (<http://www.cpc.unc.edu/projects/addhealth/design/wave4>; Chantala and Tabor 1999). Cross-sectional survey design weights from Wave IV were used because the biological markers used to create the dependent variable were only available in Wave IV, which reduced the need for longitudinal weights. Use of cross-sectional weights allow for meaningful inferences from this sample to the American young adult diabetics of non-Hispanic white, non-Hispanic black, or Hispanic descent in 2008-2009.

Dependent Variables

There are two dependent variables used in these analyses. The first dependent variable is *recent doctor visit* as a measure of health services use. Recent doctor visits were defined such that any respondent in the diabetic subsample who had seen a doctor in the previous three months was given a value of one and a zero otherwise. This coding was selected because the diabetes marker *hemoglobin A1C (A1C)*, which was used to determine diabetes status, is a valid measure of diabetes risk over the preceding 1-3 months over the course of the 120-day red blood cell life cycle (Olson et al. 2010). This coding allows for meaningful analysis of help seeking patterns among diabetics who would have met the clinical criteria for diabetes at the time of their most recent doctor visit.

The second dependent variable used in these analyses is *diabetes diagnosis status*. Diabetes diagnosis status was determined by cross-referencing measured A1C values (Krolewski et al. 1995; Olson et al. 2010) and stated diagnostic history. Diabetes is clinically indicated if A1C levels exceed 6.5% of hemoglobin molecules (Olson et al. 2010). As such, *diagnosis status* was determined by having A1C values greater than 6.5 and a prior diagnostic history of diabetes when the respondent was not pregnant. Any

respondent who indicated that they had a prior diabetes diagnosis when they were not pregnant was classified as “diagnosed diabetic” regardless of their current A1C level in order to differentiate diagnosed and undiagnosed diabetics and identify diabetics with glucose levels currently under control. A1C defined diabetics who lacked prior diagnoses were classified as “undiagnosed diabetics.” All other respondents with normal or pre-diabetic A1C values and no prior diagnoses were dropped from the sample.

Independent Variables

Predisposing Characteristics

Several demographic measures were included as predisposing characteristics to predict health services use. In this analysis, predisposing demographic characteristics of interest included race, sex, age, nativity status, and region of residence. Race/ethnic groups included in this analysis included non-Hispanic white, non-Hispanic black, and Hispanic of any background. For the purposes of these analyses, non-Hispanic white was treated as the reference category. *Age* was measured continuously in years. Both nativity status (*immigrant*=1 and *U.S. born*=0) and sex (*male*=1 and *female*=0) were dichotomously measured. *Region of residence* was included with options for West, Midwest, South and Northeast due to documented regional differences in the prevalence of diabetes and diabetes related complications in the United States (Wrobel, Mayfield, and Reiber 2001) that could reflect cultural predisposition toward diabetes related poorer health. Northeast was omitted as the reference category.

Enabling Resources

Three measures were included to address the conceptual measure of enabling resources for health services use. These measures included household income, education

level, and health insurance coverage. *Household income* was measured in 2009 dollars. This measure was included and natural logarithm (ln) adjusted to account for the skew in income. *Education level* was collapsed into four dummied categories: high school or less education, some college or vocational training, college graduate, and advanced degree. Some college or vocational training was omitted as the reference category. *Health insurance coverage* was included as a dichotomous measure. Respondents were deemed to have insurance coverage if they indicated that they had private, government, or military health care coverage or coverage through the employment of themselves, their spouses, or parents (if applicable). Since lack of access has been shown to reduce medical help seeking (Card, Dobkin, and Maestras 2008; Schoen and Des Roches 2000), having insurance was omitted as the reference category in these analyses.

Need

Both *perceived need* and *evaluated need* are key conceptual components to the Behavioral Model (Andersen 1995). One measure of parent health and four measures of somatic complaints were included in the analyses in order to conceptualize into perceived need. The parent health measure was parent-reported *parent history of diabetes* (1=diabetic parent(s), 0=no diabetic parents). The four somatic measures, *frequent urination*, *having an A1C measure above 10*, *having undiagnosed high blood pressure*, and reporting *fair or poor self-rated health*, were included as dichotomous indicators. Frequent urination is a known symptom of diabetes that is unusual in otherwise healthy individuals (Konen, Curtis, & Summerson 1996), which could elevate the perception that a doctor visit is necessary. Both extremely elevated A1C levels and undiagnosed high blood pressure could indicate the presence of other unmeasured symptoms (Van der Does

et al. 1996) that could alter how well one feels and influence the likelihood that he or she would seek care (Shi et al. 2002).

Evaluated need was conceptualized to include both visible correlates of diabetes risk and prior diagnostic history of known diabetes correlates that might influence doctors to test for diabetes regardless of patient reported symptoms (American Diabetes Association 2008). These measures included visible risk factors such as *current obesity* and *waist circumference above 35 inches* (Reaven 1988) and prior diagnoses of *high cholesterol* or *high blood pressure*, which are conditions that frequently co-occur with diabetes as risk factors for metabolic syndrome (Grundy et al. 2005). All four measures were dichotomized where the measure of interest was coded with a one and not possessing the characteristic of interest was coded with a zero. Dichotomous measures for waist circumference and obesity were used over continuous measures of waist circumference and body mass index due to the high correlation between the measures (Continuous correlation = 0.84, $p=0.000$; dichotomous correlation= 0.50, $p=0.000$).

Health Behavior Controls

Five health behaviors associated with increased diabetes risk were included in the analysis to control for negative health behaviors as a possible deterrent for health services use to avoid negative interactions with doctors (Ashmore et al. 2008; Hansen & Nelson 2011; Wilson et al. 1986). The behaviors controlled for in these models included *fast food* (Feldstein & Tucker 2007) and *sugary drink* (Hallfrisch 1990) consumption in the previous week as continuous measures to control for poor diet. Not exercising has been associated with poor glucose control (Lake and Townsend 2006). Respondents who indicated that they *did not take at least one walk* in the previous week were given a value

of one and those who did were given a zero for this measure. *Regular smoking* was assessed using an indicator variable for the number of days in the previous month a respondent smoked (1= 15-30 days; 0 otherwise). *Regular drinking* was assessed using a question that asked, “During the past 12 months, on how many days did you drink alcohol?” Respondents who indicated that they drank at least 3 days per week were coded as regular drinkers.

Analytic Plan and Missing Data

In order to test the theoretical model, logistic regression was used to determine whether variables of interest predicted health services use and altered the likelihood of being diagnosed with diabetes. Three sets of models were tested. The first model series had six models predicting recent doctor visits among diabetics to test help seeking. The second model series had seven models predicting diabetes diagnoses among diabetics to test diagnosis allocation. The final model sequence had six models predicting no diagnosis among help seeking diabetics to test missed diagnoses among help seekers.

The model sequences used to test each hypothesis followed the same order for the first six models. A seventh model was included for the model sequence used to test diagnosis allocation. For all models, Model 1 included predisposing characteristics. Model 2 included predisposing characteristics and enabling resources. Model 3 included predisposing characteristics and perceived need. Model 4 included predisposing characteristics and evaluated need. Model 5 included predisposing characteristics and both perceived and evaluated need. Model 6 included all prior variables in a fully adjusted model; however in Model 6 of the series predicting diabetes diagnoses also included having a recent doctor visit. Model 7, of the series predicting diabetes

diagnoses, included a race by recent doctor visit interaction in the fully adjusted model. All model sequences in each model series control for negative health behaviors.

Missing values were addressed through multiple imputation using the “ice” command in Stata 11.2 (Royston 2005). Due to the importance of correctly classifying diagnosis status, cases with missing values on either measured hemoglobin A1C or diagnostic history were deleted (Von Hippel 2007). Missing values on all independent variables were imputed to provide complete analytic data (Ragunathan 2004). Analyses were conducted on ten imputed data sets that were combined and analyzed using Rubin’s Combining Rules (Little and Rubin 2002).

4.5 Results

Descriptive Results

Table 4.1 displays the weighted means or proportions for the sample included in the study. The first column displays the means or proportions for the full sample. The second and third columns are stratified by recent doctor visit with the fourth column indicating significant differences in means between those with and without recent doctor visits. The fifth and sixth columns are stratified by diagnosis status with the final column indicating significant differences in means between those with and without diabetes diagnoses.

****Table 4.1 About Here ****

Only two variables significantly differed by recent doctor visit. Interestingly, none of the race/ethnic variables significantly varied by recent doctor visit. The two variables that differed by recent doctor visit included lacking health insurance coverage (12% with

recent doctor visit vs. 30% without) and diagnosis of high blood pressure (37% with recent visit vs. 22% without).

Eight variables or categories of variables significantly differed by diagnosis status among diabetics. The race variables were the most striking with non-Hispanic whites overrepresented among diagnosed diabetics (62% diagnosed vs. 37% undiagnosed) and non-Hispanic blacks overrepresented among undiagnosed diabetics (22% diagnosed vs. 48% undiagnosed). Hispanics were equally represented as 16% of both groups and the overall sample. Immigrants were overrepresented among undiagnosed diabetics (1% diagnosed vs. 4% undiagnosed). Diagnosed diabetics were more likely to have A1C levels above 10 (17% diagnosed vs. 5% undiagnosed), more likely to report fair or poor health (35% diagnosed vs. 17% diagnosed), and more likely to report both diagnosed high blood pressure (35% diagnosed vs. 20% undiagnosed), and diagnosed high cholesterol (29% diagnosed vs. 10% undiagnosed). However, undiagnosed diabetics were more likely to have undiagnosed high blood pressure (9% diagnosed vs. 20% undiagnosed).

Multivariate Results

Help Seeking among Diabetics

Table 4.2 displays the results for the first model series predicting the odds of a recent doctor visit among diabetics. Predisposing characteristics yielded the largest number of predictors of recent doctor visits. Male diabetics were less likely to seek care than women (OR=0.59 95% CI 0.38, 0.90). As age increases, the odds of a recent doctor visit also increases among diabetics (OR=1.14 95% CI 1.02, 1.28). Residing in the South (OR=2.60 95% CI 1.22, 5.54), but not the West (OR=0.83 95% CI 0.33, 2.08) or

Midwest (OR=1.92 95% CI 0.91, 4.08) relative to residing in the Northeast increased the odds of a recent doctor visit among diabetics. Interestingly, race or ethnicity did not significantly predict recent doctor visits (non-Hispanic black OR=1.05 95% CI 0.62, 1.77; Hispanic OR=1.45 95% CI 0.64, 3.29), which does not support hypothesis 1.

****Table 4.2 About Here ****

One variable included as an indicator of enabling resources decreased the odds of having a recent doctor visit. Those without health insurance coverage had significantly lower odds of a recent visit (OR=0.27 95% CI 0.15, 0.49). Unexpectedly, none of the variables included as indicators of perceived need significantly predicted recent doctor visits. One variable included as an indicator of evaluated need for health care, prior diagnosis of high blood pressure (OR=2.15 95% CI 1.14, 4.06), increased the odds of a recent doctor visit.

Diabetes Diagnoses among Diabetics

Table 4.3 displays the results for predicting diabetes diagnoses among diabetics. One variable included as an indicator of predisposing characteristics altered the odds of having a diabetes diagnosis. Non-Hispanic blacks that were clinically diabetic had lower odds of having a diabetes diagnosis (OR=0.19 95% CI 0.11, 0.34) relative to non-Hispanic whites. Odds of diagnosis did not significantly vary by sex, age, nativity status, or region of residence.

****Table 4.3 About Here ****

One variable included as an indicator of enabling resources altered the odds of having a diabetes diagnosis. Having high school or less education (OR=1.79 95% CI 1.03, 3.10) increased the odds of having a diabetes diagnosis relative to those with some

college or vocational training; no effect was observed for increased education. Neither household income nor health insurance coverage influenced the odds of diagnosis among diabetics.

Two variables included as indicators of perceived need increased the odds of diagnosis. Having an A1C level above 10 greatly increased the odds of being diagnosed (OR=5.37 95% CI 2.11, 13.65), as did reporting fair or poor self-rated health (OR=1.71, 95% CI 1.01, 2.90). Parent diagnostic history and other measures of somatic complaints did not significantly alter odds of diagnosis. One indicator of evaluated need increased the odds of diagnosis. Diabetic individuals with a prior diagnosis history of high cholesterol were more likely to have a diabetes diagnosis (OR=2.88 95% CI 1.50, 5.53). Obesity, waist circumference, and prior diagnosis of high blood pressure did not mediate the odds of diabetes diagnosis for non-Hispanic blacks.

Neither seeing a doctor in the previous three months nor the health services use by race interactions were significant predictors of diabetes diagnosis allocation. These findings do not provide support for hypothesis 2, which proposed that the association between race and diabetes diagnoses would be moderated by health services use.

Missed Diagnoses among Diabetic Help Seekers

Table 4.4 displays the results for predicting missed diagnoses among diabetics who have seen a doctor in the previous three months. The results for model sequences predicting the odds of a missed diagnosis among help seeking diabetics follow similar patterns as seen with the models predicting the odds of diagnosis in the previous section; but in reverse. Non-Hispanic black diabetics that saw a doctor in the previous three months had elevated risk of not being diagnosed with diabetes (OR=4.30 95% CI 1.43,

12.89). This finding does not provide support for hypothesis 3, which proposed diabetes diagnosis allocation would be equivalent for respondents who utilized health care.

Conversely, this finding provides support for the opposite conclusion that racial differences in diabetes diagnosis disparities persist despite help seeking.

****Table 4.4 About Here ****

Missed diagnoses were less likely among diabetic help seekers if they had an A1C level above 10 (OR=0.03 95% CI 0.00, 0.57), had fair or poor self-rated health (OR=0.26 95% CI 0.09, 0.77), or had a prior diagnosis of high cholesterol (OR=0.21 95% CI 0.07, 0.63). Missed diagnoses were also less likely among diabetic help seekers if they had an education level of high school or less (OR=0.23 95% CI 0.07, 0.74) relative to those with some college or vocational training.

4.6 Discussion

All three initial hypotheses tested here were not supported. Neither the descriptive statistics nor the multivariate models tested here support the proposition that diabetes race/ethnic minorities seek health care less frequently than non-Hispanic whites. Prior literature suggested that non-Hispanic blacks would seek care at lower rates (Bogart et al. 2004; Fiscella et al. 2002; Smedly, Stith, and Nelson 2003), but an exhaustive literature search failed to find relevant studies examining help seeking with young adult diabetics. Moreover, help seeking did not have an impact on diabetes diagnosis allocation. Neither the tests of the main effect of help seeking nor the interaction effects examining help seeking by race significantly predicted diabetes diagnoses allocation. However, diabetic non-Hispanic blacks were consistently less likely to receive diabetes diagnoses by a wide margin. Perhaps the most disturbing finding of this study is the revelation that diabetic

non-Hispanic blacks that had gone to the doctor while they would have met the clinical criteria for diagnosis of diabetes failed to receive a diagnosis.

The results presented here provide foundational evidence that the diabetes diagnosis disparity for non-Hispanic black young adults is not due to lack of help seeking. Conversely, these findings suggest that there is something about doctor-patient interactions when non-Hispanic black diabetics go to the doctor that does not result in diabetes testing at the same rate as non-Hispanic white diabetics. If these findings reflect implicit biases on the part of doctors, this would provide support for Stepanikova's (2012) findings regarding black-white disparities with cardiac testing in a different medical setting (Stepanikova 2012; Stepanikova, Triplett, and Simpson 2011). However, these findings may also reflect differences in communication patterns between doctors and patients that may reflect doctors spending less time with minority patients or minority patients presenting their symptoms differently than non-Hispanic white patients. The current data makes it impossible to know what specifically has led to these dramatic race differences in diagnosis patterns, but it certainly warrants future research.

Despite the lingering questions about why there appears to be a racial bias in diabetes diagnosis allocation, this study does provide meaningful evidence that such a bias exists. Future studies should aim to address the reasons behind the diagnostic disparity from structural, interpersonal, and historical perspectives. Structural issues may regulate opportunities for access to care (Card, Dobkin, and Maestras 2008; Schoen and Des Roches 2000), but the quality of available care may differ particularly for those who live in racially segregated environments experiencing multifaceted effects of concentrated poverty (Acevedo-Garcia et al. 2003; Collins and Williams 1999; Massey

2004) or for those who rely on irregular or transient care settings over primary care with one regular provider (Lutfey and Freese 2005). Interpersonal issues regarding the importance of doctor-patient interaction and the role of implicit doctor bias could be one component of under diagnosis and under testing of non-Hispanic black diabetics (Stepanikova 2012). Moreover, the United States has an uncomfortable history regarding race and medicine. Historical maltreatment in both research and medical settings for non-Hispanic blacks in the United States (Brant 1978; Skloot 2010; Washington 2006) may interject an additional layer of discomfort between doctor and patient interactions independent of the reason for a particular visit (Friemuth et al. 2001; Gamble 1997; Heisler et al. 2005). Social discomfort must be addressed if it leads doctors to spend less time with minority patients because delayed diagnoses can have irreversible consequences (Borchard 1995; Chakrabarti 2000; Koopman et al. 2006; Sharma and Richards 2000; Trull et al. 2002) and patients who feel dismissed may eventually become less likely to seek care, which could further delay diagnoses.

Limitations

Although this study has several strengths, it is important to acknowledge its limitations. Respondents were asked when they had last seen a medical provider for a “regular check-up,” but there was not additional context about why the visit took place or in what setting. Some of the differences in diagnosis allocation may have come from differences in care settings (e.g.- primary care vs. emergency care) or differences in the chief complaint of the respondent at the time of the visit (e.g.- frequent urination vs. a broken arm). Unfortunately, it is not possible to tease out whether the race-based diagnostic disparities are observed due to irregular help seeking or poor collection of

health histories. Future studies should examine the nuances of doctor patient interactions regarding symptom presentation and decision-making processes by doctors.

Although the reason behind the diagnostic disparities could not be ascertained from this data, this study does add to the help seeking literature by documenting equivalent help seeking by race for young adult diabetics. This finding provides a foundation for future research to explore why diagnoses are not allocated accordingly by race. Moreover, this is the first known study to focus on the help seeking patterns of young adult diabetics with and without diagnoses. As such, the findings presented here may reflect similarities in help seeking patterns for those with undiagnosed health conditions, but may not reflect overall help seeking patterns among young adults. Future research regarding help seeking patterns of young adults should also focus on differences in help seeking patterns for preventative care among young adults.

Conclusions

This study is the first known study to document significant racial diagnostic disparities for diabetes among young adult diabetics who seek care. The findings presented here indicate significant cause for concern as rates of diabetes continue to rise among American young adults (Mokdad et al. 2001). If diabetes continues to be under diagnosed in non-Hispanic black diabetics, this could lead to further health disparities in morbidity and mortality as they age. It is expected that those who were identified as undiagnosed diabetics in this study would have eventually received diagnoses. However, the timing of diagnoses is critical for starting treatments for glucose control that can minimize the future risk of neuropathy, kidney damage, or limb loss. If the findings presented here indicate greater underlying disparities in diagnostic testing of racial

minorities, as is suggested by Stepanikova's (2012) research, policies should be set in place to attempt to catch those who fall through the cracks and train doctors to better identify diabetes risk in non-Hispanic black young adult diabetics.

References

- Acevedo-Garcia D, Lochner KA, Osypuk TL, Subramanian SV. 2003. Future Directions in Residential Segregation and Health Research: A Multilevel Approach. *American Journal of Public Health*, 93(2):215-221.
- American Diabetes Association. 2008. "Diagnosis and Classification of Diabetes Mellitus." *Diabetes Care*. 31(S1):S55-S60.
- American Diabetes Association .2013 <<http://professional.diabetes.org/Admin/UserFiles/0%20-%20Sean/FastFacts%20March%202013.pdf>> Accessed March 20, 2013
- Andersen, Ronald M. 1968. *Behavioral Model of Families' Use of Health Services*. Research Series No. 25 Chicago, IL Center for Health Administration Studies, University of Chicago.
- Andersen, Ronald. 1995. "Revisiting the Behavioral Model and Access to Medical Care: Does it Matter?" *Journal of Health and Social Behavior*. 36(1) 1-10.
- Andersen, Ronald Max. 2008. "National Health Surveys and the Behavioral Model of Health Services Use." *Medical Care* 46(7):647-653.
- Ashmore, Jamile A., Kelli E. Friedman, Simona K. Reichman, & Gerard J. Musante. 2008. "Weight-based Stigmatization, Psychological Distress, & Binge Eating Behavior among Obese Treatment-Seeking Adults." *Eating Behaviors*. 9(2): 203-209.
- Becker, Marshall H. 1974. "The Health Belief Model and Sick Role Behavior." *Health Education & Behavior*. 2(4):409-419.
- Becker, Marshall H., Robert H. Drachman, and John P. Kirscht. 1974. "A New Approach to Explaining Sick-Role Behavior in Low-Income Populations" *American Journal of Public Health*. 64(3). 205-216.
- Benyamini, Yael, Colleen S. McClain, Elaine A. Leventhal, and Howard Leventhal. 2003. "Living with the Worry of Cancer: Health Perceptions and Behaviors of Elderly People with Sel, Vicarious, or No History of Cancer." *Psycho-Oncology*. 12:161-172.
- Bogart, Laura M., Sheryl Thorburn Bird, Lisa C. Walt, Douglas L. Delahanty, and Jacqueline L. Figler. 2004. "Association of Stereotypes about Physicians to Health Care Satisfaction, Help-Seeking Behavior, and Adherence to Treatment." *Social Science and Medicine*. 58(6):1049-1058.
- Bouchard, G. 1995. "Genetics and the Metabolic Syndrome." *International Journal of*

Obesity 19: 52–59.

- Brandt, Allan M. 1978. "Racism and Research: The Case of the Tuskegee Syphilis Study." *The Hastings Center Report*. 8(6):21-29.
- Card, David, Carlos Dobkin, and Nicole Maestras. 2008. "The Impact of Nearly Universal Insurance Coverage on Health Care Utilization: Evidence from Medicare." *American Economic Review*. 98(5):2242-2258.
- Chakrabarti, Subrata. 2000. "Diabetic Retinopathy in Experimental Animal Models and their Feasibility for Understanding the Human Disease." in Anders A.F. Sima and Eleazar Shafir (Eds.) *Chronic Complications in Diabetes: Animal Models and Chronic Complications*. Amsterdam, Netherlands. Pp.229-250.
- Chantala, Kim and Joyce Tabor. 1999. "National Longitudinal Study of Adolescent Health: Strategies to Perform a Design-Based Analysis Using the Add Health Data." Chapel Hill, NC: Carolina Population Center.
- Centers for Disease Control and Prevention 2011
<<http://www.cdc.gov/diabetes/pubs/estimates11.htm>> Accessed October 18, 2013
- Corbie-Smith, Giselle, Stephen B. Thomas, Mark V. Williams, & Sandra Moody-Ayers. 1999. "Attitudes and Beliefs of African Americans Toward Participation in Medical Research." *J Gen Intern Med*. 14:537-546.
- Collins CA, Williams DR. 1999. Segregation and Mortality: The Deadly Effects of Racism? *Sociological Forum*, 1999; 14:495-523.
- Cowie C. 2006. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health and Nutrition Examination Survey 1999-2002." *Diabetes Care*. 29(6):1263–1268.
- (<http://www.cpc.unc.edu/projects/addhealth/design/wave4>)
- De Voe, Jennifer. 2008. "The Unsustainable US Health Care System: A Blueprint for Change." *Annals of Family Medicine*. 6(3): 263-266.
- Dunlop, Sheryl, Peter C. Coyote, & Warren McIsaac. 2000. "Socio-economic Status and the Utilisation of Physicians' Services: Results from the Canadian National Population Health Survey." *Social Science and Medicine*. 51:123-133.
- Feldeisen SE, Tucker KL (2007). "Nutritional strategies in the prevention and treatment of metabolic syndrome". *Appl Physiol Nutr Metab* 32 (1): 46–60.
- Fiscella, Kevin, Peter Franks, Mark P. Doescher, and Barry G. Saver. 2002. "Disparities

- in Health Care by Race, Ethnicity, and Language among the Insured: Findings from a National Sample.” *Medical Care*. 40(1):52-59.
- Ford, ES, WH Giles, and WH Dietz. 2002. “Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey.” *JAMA*. 287(3):356-359.
- Friemuth, Vicki S., Sandra Crouse Quinn, Stephen B. Thomas, Galen Cole, Eric Zook, and Ted Duncan. 2001. “African Americans Views on Research and the Tuskegee Syphilis Study.” *Social Science and Medicine*. 52:797-808.
- Gamble, Vanessa Northington. 1997. “Under the Shadow of Tuskegee: African Americans and Health Care.” *The American Journal of Public Health*. 87(11):1773-1778.
- Grundy SM, Cleeman JI, Daniels SR, *et al.* (October 2005). "Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement". *Circulation* **112** (17): 2735–52.
- Guy, Jr., Gery P. 2010. “The Effects of Cost Sharing on Access to Care Among Childless Adults Effects Cost Sharing on Access to Care.” *Health Services Research*. 45(6):1720-1739.
- Hall, Mark. 2011. “The Mission of Safety Net Organizations Following National Insurance Reform.” *Journal of General Internal Medicine*. 26(7): 802-805. Hallfrisch 1990
- Hallfrisch J. 1990. "Metabolic effects of dietary fructose". *FASEB J* **4** (9): 2652–2660.
- Hammond, Wisdom Powell. 2010. “Psychosocial Correlates of Medical Mistrust Among African American Men.” *American Journal of Community Psychology*. 45(1/2):87-106.
- Harris, Kathleen Mullan. 2009. *The National Longitudinal Study of Adolescent Health (Add Health), Waves I & II, 1994–1996; Wave III, 2001–2002; Wave IV, 2007–2009* [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill. DOI: 10.3886/ICPSR27021.v9
- Hansen, EC, and MR Nelson. 2011. “How Cardiac Patients Describe the Role of Their Doctors in Smoking Cessation: A Qualitative Study.” *Australian Journal of Primary Health*. 17(3):268-273.
- Heisler, Michael B., George Rust, Roland Pattillo, and An Dubois. 2005. “Improving Health, Eliminating Health Disparities: Finding Solutions for Better Health Care for All Populations.” *Ethnicity and Disease*. 15:S2-1-S2-4.

- Hopton, Jane L. and Maria Dlugolecka. 1995. "Patients' Perceptions of Need for Primary Health Care Services: Useful for Priority Setting?" *BMJ*. 310:137-1240.
- Kash K.M., Holland J.C., Halper M.S. and Miller D.G. 1992. "Psychological Distress and Surveillance Behaviors of Women with a Family History of Breast Cancer." *Journal of the National Cancer Institute*, 84(1), 24-30.
- Kenen, Regina, Audrey Arden-Jones, and Rosalind Eeles. 2003. "Living with Chronic Risk: Healthy Women with a Family History of Breast/Ovarian Cancer." *Health, Risk, and Society*. 5(3):315-331.
- Kennedy, Bernice, Christopher Clomus Mathis, and Angela K. Woods. 2007. "African Americans and Their Distrust of the Health Care System: Healthcare for Diverse Populations." *Journal of Cultural Diversity*. 14(2):56-60.
- Koopman RJ, Mainous AG, 3 rd, Liszka HA, Colwell JA, Slate EH, Carnemolla MA, et al. 2006. "Evidence of nephropathy and peripheral neuropathy in US adults with undiagnosed diabetes." *Annals of Family Medicine*. 2006;4(5):427-432.
- Köszegi B. .2003. "Health Anxiety and Patient Behavior." *Journal of Health Economics*, 22(6), 1073-1084.
- Konen, JC, LG Curtis, and JH Summerson. 1996. "Symptoms and Complications of Adult Diabetic Patients in a Family Practice." *Archives of Family Medicine*. 5(3):135-145.
- Krolewski AS, Laffel LMB, Krowlewski M, et al. 1995. "Glycosylated hemoglobin and the risk of microalbuminuria in patients with insulin dependent diabetes mellitus." *New England Journal of Medicine*;332:1251-5
- Kullgren, Jeffrey, Catherine G. McLaughlin, Nandita Mitra, and Katrina Armstrong. 2012. "Nonfinancial Barriers and Access to Care for U.S. Adults." *Health Services Research*. 47(1): 462-485.
- Lake, Amelia and Tim Townshend. 2006. "Obesogenic Environments: Exploring the Built and Food Environments." *Journal of the Royal Society for the Promotion of Health*. 126(6):262-267.
- Lackland DT, Orchard TJ, Keil JE, Saunders DE Jr, Wheeler FC, Adams-Campbell LL, et al. 1992. "Are race differences in the prevalence of hypertension explained by body mass and fat distribution? A survey in a biracial population." *International Journal of Epidemiology*. 21(2):236-245.
- Lim, Jennifer N. W., Jenny Hewison, Carol E. Chu, and Hamdan Al-Habsi. 2011.

“Factors Influencing Consultation to Discuss Family History of Cancer by Asymptomatic Patients in Primary Care.” *Journal of Community Genetics*. 2:19-26.

- Little, Paul, Martina Doward, Greg Warner, Katharine Stephens, Jane Senior, and Michael Moore. 2004. “Importance of Patient Pressure and Perceived Medical Need for Investigations, Referral, and Prescribing in Primary Care: Nested Observational Study.” *BMJ*. 328-444.
- Little, Roderick and Donald Rubin. 2002. *Statistical Analysis with Missing Data*. Hoboken, NJ: Wiley Interscience.
- Lutfey, Karen and Jeremy Freese. 2005. “Toward Some Fundamentals of Fundamental Causality: Socioeconomic Status and Health in the Routine Clinic; Visit for Diabetes.” *American Journal of Sociology* 110:1326–72
- Lyles, Courtney, Andrew Karter, Bessie Young, Clarence Spingner, David Grembowski, Dean Schillinger, and Nancy Adler. 2011. “Patient-Reported Racial/Ethnic Healthcare Provider Discrimination and Medication Intensification in the Diabetes Study of Northern California (Distance).” *Journal of General Internal Medicine*. 26(10):1138-1144.
- Massey, DS. 2004. Segregation and Stratification: A Biosocial Perspective. DuBois Review: Social Science Research on Race. 1: 7-25.
- Mokdad , Ali H, Barbara A. Bowman, Earl S. Ford, Frank Vinicor, James S. Marks, and Jeffrey P. Koplan. 2001. “The Continuing Epidemics of Obesity and Diabetes in the United States.” *JAMA* 286(10):1195-1200.
- Nichols GA, Arondekar B, Herman WH. 2008. “Medical care costs one year after identification of hyperglycemia below the threshold for diabetes.” *Medical Care*. 2008;46(3):287–292.
- Nichols GA, Brown JB. 2005. “Higher medical care costs accompany impaired fasting glucose.” *Diabetes Care*. 28(9):2223–2229.
- Olson DE, Rhee MK, Herrick K, Ziemer DC, Twombly JG, Phillips LS. 2010. “Screening for diabetes and pre-diabetes with proposed A1C-based diagnostic criteria.” *Diabetes Care*. 33(10):2184–2189.
- Parsons, Talcott. 1951. *The Social System*. London: Routledge & Kegan Paul Ltd.
- Raghunathan, T. E. 2004. “What do we do with missing data? Some options for analysis of incomplete data.” *Annual Review of Public Health*, 25, 99–117.
- Reaven, Gearld M. 1988. "Role of Insulin Resistance in Human

- Disease.” *Diabetes* 37 (12): 1595–1607.
- Royston, Patrick. 2005. "Multiple Imputation of Missing Values: Update of Ice." *The Stata Journal*. 5(4):527-536.
- Schoen, Cathy and Catherine Des Roches. 2000. “Uninsured and Unstably Insured: The Importance of Continuous Insurance Coverage.” *HSR: Health Services Research* 35(1):187-206.
- Segall, Alexander. 1976. “The Sick Role Concept: Understanding Illness Behavior.” *Journal of Health and Social Behavior*. 17(2):162-169.
- Sharma, Ashutosh and Penelope A. Richards. 2000. “Diabetic Neuropathy in Various Animal Models.” in Anders A.F. Sima and Eleazar Shafir (Eds.) *Chronic Complications in Diabetes: Animal Models and Chronic Complications*. Amsterdam, Netherlands. Pp.229-250.
- Shi, Leiyu, Barbara Starfield, Robert Politzer, and Jerri Ryan. 2002. “Primary Care, Self-Rated Health, and Reductions in Social Disparities in Health.” *HSR: Health Services Research* 37(3):529-550.
- Skloot, Rebecca. 2010. *The Immortal Life of Henrietta Lacks*. New York, NY: Crown Publishers.
- Smedly, Brian D., Adrienne Y. Stith, and Alan R. Nelson. 2003. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. Washington, D.C.:The National Academies Press.
- Stepanakova, Irena. 2012. “Racial-Ethnic Biases, Time Pressure, and Medical Decisions.” *Journal of Health and Social Behavior*. 53(3)329-343.
- Stepanikova, Irena, Jennifer Triplett, and Brent Simpson. 2011. "Implicit Racial Bias and Prosocial Behavior." *Social Science Research* 40:1186-1195.
- Trull, Andrew, Lawrence M. Demers, David W. Holt, Atholl Johnston, J. Michael Tredger, and Christopher P. Price. 2002. *Biomarkers of Disease: An Evidence Based Approach*. Cambridge University Press: New York, New York.
- Twaddle, Andrew C. 1969. “Health Decisions and Sick Role Variations: An Exploration” *Journal of Health and Social Behavior*, 10(2): 105-115.
- Vaccaro, Joan A. and Fatma G. Huffman. 2012. “Reducing Health Disparities: Medical Advice Received for Minorities with Diabetes.” *Journal of Health and Human Services*. 34(4):391-417.
- Van Osch S., van den Hout W. and Stiggelbout A.M. 2006. “Exploring the Reference

- Point in Prospect Theory: Gambles for Length of Life.” *Medical Decision Making*. 26, 338-346.
- Van der Does FERDINAND J. Nic:o D. DE NEF.LING, FRANK J. SNOEK, PIETERJ. KOSTENSE, PETER A. GROOTENHUIS, LEX M. BOUTER, and ROBERT J. HEINE. 1996. “Symptoms and Weil-Being in Relation to Glycemic Control in Type II Diabetes” *DIABETES CARE*, 19(3):204-210.
- Vernon S. 1999. “Risk Perception and Risk Communication for Cancer Screening Behaviors: a review.” *Journal of the National Cancer Institute. Monographs*, 25, 101-119.
- Von Hippel, Paul T. 2007. “Regression with Missing Y’s: An Improved Strategy for Analyzing Multiply Imputed Data.” *Sociological Methodology*, 37:83-117.
- Walker, Elizabeth A., Maria R. Kalten, C.K. Mertz, and James Flynn. 2003. “Risk Perception for Developing Diabetes: Comparative Risk Judgments by Physicians.” *Diabetes Care*. 26: 2543-2548.
- Washington, Harriet A. 2006.*Medical Apartheid:The Dark History of Medical Experimentation on Black Americans from Colonial Times to the Present*. New York, NY:Doubleday.
- Whitsel, Eric A., Joyce W. Tabor, Quynh C. Nguyen, Carmen C. Cuthbertson, Mark H. Wener, Alan J. Potter, Ley A. Killeya-Jones, and Kathleen Mullan Harris. 2012. *Add Health Wave IV Documentation Report: Measures of Glucose Homeostasis*.Chapel Hill, NC: UNC Chapel Hill: Carolina Population Center
- Wilson, W., DVArY, A Biglan, RE Glasgow, DJ Toobert, and DR Campbell. 1986. “Psychosocial Predictors of Self-Care Behaviors (Compliance) and Glycemic Control in Non-Insulin Dependent Diabetes Mellitus.” *Diabetes Care*. 9(6): 614-622.
- World Health Organization. 2012. “Diabetes Fact Sheet.” <http://www.who.int/mediacentre/factsheets/fs312/en/index.html>. Accessed 11/4/2012.
- Wrobel, James S., Jennifer A. Mayfield, and Gayle E. Reiber 2001. “Geographic Variation of Lower-Extremity Major Amputation in Individuals With and Without Diabetes in the Medicare Population” *Diabetes Care* 24:860–864.
- Zhang Y, Dall TM, Mann SE, Chen Y, Martin J, Moore V, et al.2009. “The economic costs of undiagnosed diabetes.” *Population Health Management*. 12(2):95–101.
- Zoratti R, Godsland IF, Chaturvedi N, Crook D, Stevenson JC, McKeigue PM.2000.

“Relation of plasma lipids to insulin resistance, nonesterified fatty acid levels, and body fat in men from three ethnic groups: relevance to variation in risk of diabetes and coronary disease. *Metabolism*.49(2):245–252.

4.8 Tables

Table 4.1: Weighted Means or Proportions by Diagnosis Status and Recent Doctor Visit

	All Diabetics	Recent Dr. Visit (<3m)	No Recent Dr. Visit (3+m)	Significant Difference	Diagnosed	Undiagnosed	Significant Difference
<i>Recent Doctor Visit</i>							
Less than 3 Months Since Dr. Visit	0.25				0.32	0.21	
<i>Diagnosis Status</i>							
Undiagnosed Diabetic	0.61	0.50	0.63				
Diagnosed Diabetic	0.39	0.50	0.37				
<i>Predisposing Characteristics</i>							
Non-Hispanic White	0.47	0.46	0.47		0.62	0.37	*
Non-Hispanic Black	0.38	0.39	0.37		0.22	0.48	*
Hispanic	0.16	0.15	0.16		0.16	0.16	
Male	0.52	0.43	0.56		0.47	0.56	
Female	0.48	0.57	0.44		0.53	0.44	
Age in 2009	29.31	29.60	29.20		29.35	29.28	
Immigrant	0.03	0.02	0.03		0.01	0.04	*
West	0.15	0.10	0.16		0.16	0.14	
Midwest	0.25	0.25	0.25		0.28	0.22	
South	0.50	0.59	0.47		0.47	0.52	
Northeast	0.11	0.06	0.12		0.09	0.12	
<i>Enabling Resources</i>							
Adult Income 2009	9.13	8.77	9.28		8.68	9.43	
High School or Less	0.36	0.28	0.39		0.42	0.33	
Some College	0.45	0.45	0.45		0.42	0.47	
College Degree	0.11	0.17	0.09		0.09	0.12	
Advanced Degree	0.08	0.10	0.08		0.07	0.09	
No Health Insurance	0.26	0.12	0.30	*	0.24	0.27	
<i>Perceived Need</i>							
Diabetic Parent	0.18	0.16	0.18		0.19	0.17	
Frequent Urination	0.07	0.08	0.07		0.10	0.05	
A1C Above 10	0.10	0.10	0.10		0.17	0.05	*
Undiagnosed High Blood Pressure	0.16	0.12	0.17		0.09	0.20	*
Fair or poor Self-Rated Health	0.24	0.26	0.23		0.35	0.17	*
<i>Evaluated Need</i>							
Diagnosed High Cholesterol	0.18	0.25	0.16		0.29	0.10	*
Diagnosed High Blood Pressure	0.26	0.37	0.22	*	0.35	0.20	*
Waist Circumference 35+ Inches	0.85	0.86	0.85		0.88	0.84	
Currently Obese	0.63	0.64	0.63		0.69	0.59	
<i>Health Behavior Controls</i>							
Fast Food Consumption	2.67	2.69	2.66		3.10	2.39	
Sugary Drink Consumption	11.83	9.58	12.62		11.50	12.05	
Doesn't Walk for Exercise	0.44	0.34	0.47		0.40	0.46	
Regular Drinker	0.10	0.10	0.10		0.08	0.11	
Regular Smoker	0.27	0.26	0.28		0.32	0.24	
N	915	248	667		360	555	
Proportion	1.00	0.27	0.73		0.39	0.61	

Table 4.2: Logistic Regression Models Predicting Recent Doctor Visit in the Past 3 Months for All Diabetics (N=915)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
<i>Predisposing Characteristics</i>						
Non-Hispanic Black	0.92	0.96	0.95	1.02	1.03	1.05
Hispanic	1.25	1.37	1.28	1.29	1.36	1.45
Male	0.58 **	0.64 *	0.59 **	0.54 **	0.53 **	0.59 *
Age in 2009	1.17 **	1.15 *	1.18 **	1.16 *	1.16 *	1.14 *
Immigrant	0.51	0.43	0.52	0.52	0.48	0.42
West	1.04	0.88	1.01	1.02	1.01	0.83
Midwest	2.09	2.08	2.11	1.92	1.93	1.92
South	2.65 **	2.64 *	2.74 **	2.58 *	2.59 **	2.6 *
<i>Enabling Resources</i>						
Adult Income 2009		0.93				0.94
High School or Less		0.88				0.89
College Degree		1.79				1.71
Advanced Degree		1.28				1.31
No Health Insurance		0.29 ***				0.27 ***
<i>Perceived Need</i>						
Diabetic Parent			0.75		0.73	0.74
Frequent Urination			1.19		1	1.17
A1C Above 10			1.1		1.03	1.05
Undiagnosed High Blood Pressure			0.8		1.03	0.99
Fair or poor Self-Rated Health			1.21		1.03	1.01
<i>Evaluated Need</i>						
Diagnosed High Cholesterol				1.5	1.51	1.26
Diagnosed High Blood Pressure				1.95 *	1.95 *	2.15 *
Waist Circumference 35+ Inches				1.08	1.09	1.05
Currently Obese				0.85	0.87	0.94
<i>Health Behavior Controls</i>						
Fast Food Consumption	1.02	1.03	1.02	1.02	1.02	1.03
Sugary Drink Consumption	0.98 *	0.98 *	0.98 *	0.98 *	0.98 *	0.98
Doesn't Walk for Exercise	0.61	0.64	0.62	0.65	0.65	0.68
Regular Drinker	1.04	1.07	0.99	1.16	1.13	1.19
Regular Smoker	1.01	1.28	0.97	0.99	0.99	1.26

Notes: a. Diagnosis refers to a prior diabetes diagnosis when the respondent was not pregnant; b. Odds ratios reported; c. Pregnant women excluded from analyses; d. Some college or vocational training is the reference category for achieved education; e. * p<0.05, ** p<0.01, *** p<0.001

Table 4.3 Logistic Regression Models Predicting Diabetes Diagnosis Odds Ratios Reported (N=915)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
<i>Predisposing Characteristics</i>							
Non-Hispanic Black	0.23 ***	0.21 ***	0.21 ***	0.25 ***	0.23 ***	0.20 ***	0.19 ***
Hispanic	0.63	0.58	0.49 *	0.65	0.53	0.49 *	0.50
Male	0.73	0.76	0.73	0.69 *	0.69	0.74	0.74
Age in 2009	1.07	1.06	1.08	1.04	1.06	1.04	1.04
Immigrant	0.31	0.26	0.43	0.32	0.40	0.35	0.35
West	1.33	1.25	1.32	1.43	1.46	1.44	1.42
Midwest	1.44	1.36	1.45	1.42	1.47	1.40	1.41
South	1.43	1.49	1.65	1.49	1.69	1.77	1.77
<i>Enabling Resources</i>							
Adult Income 2009		0.92 *				0.94	0.94
High School or Less		1.68 *				1.80 *	1.79 *
College Degree		0.88				0.64	0.64
Advanced Degree		0.96				1.03	1.02
No Health Insurance		0.67				0.67	0.67
<i>Perceived Need</i>							
Diabetic Parent			0.99		0.97	0.97	0.97
Frequent Urination			1.02		0.89	0.90	0.90
A1C Above 10			5.12 ***		5.05 **	5.43 ***	5.37 ***
Undiagnosed High Blood Pressure			0.54		0.55	0.57	0.57
Fair or poor Self-Rated Health			2.24 **		1.94 *	1.72 *	1.71 *
<i>Evaluated Need</i>							
Diagnosed High Cholesterol				2.62 **	2.67 **	2.87 **	2.88 **
Diagnosed High Blood Pressure				1.63	1.17	1.13	1.14
Waist Circumference 35+ Inches				0.84	0.92	0.89	0.89
Currently Obese				1.27	1.14	1.21	1.20
<i>Health Services Use</i>							
Less than 3 Months Since Dr. Visit						1.48	1.41
<i>Interactions</i>							
African American x Recent Doctor Visit							1.20
Hispanic x Recent Doctor Visit							0.90
<i>Health Behavior Controls</i>							
Fast Food Consumption	1.16 ***	1.16 ***	1.15 ***	1.16 ***	1.15 ***	1.16 ***	1.16 ***
Sugary Drink Consumption	1.00	0.99	1.00	1.00	1.00	0.99	0.99
Doesn't Walk for Exercise	0.66 *	0.70	0.68 *	0.71	0.71	0.75	0.75
Regular Drinker	0.63	0.72	0.55	0.74	0.63	0.74	0.74
Regular Smoker	1.34	1.23	1.12	1.36	1.16	1.06	1.06

Notes: a. Diagnosis refers to a prior diabetes diagnosis when the respondent was not pregnant; b. Odds ratios reported; c. Pregnant women excluded from analyses; d. Some college or vocational training is the reference category for achieved education; e. * p<0.05, ** p<0.01, *** p<0.001

Table 4.4: Models Predicting No Diagnosis among Help-Seeking Diabetics- Odds Ratios Reported (N=244)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
<i>Predisposing Characteristics</i>						
Non-Hispanic Black	3.56 **	4.90 **	4.72 **	2.97 *	3.71 **	4.30 **
Hispanic	2.63	3.36	2.61	2.21	1.91	2.03
Male	0.44	0.45	0.40	0.49	0.39	0.42
Age in 2009	1.09	1.11	1.09	1.13	1.09	1.08
West	0.47	0.46	0.43	0.51	0.49	0.50
Midwest	1.95	1.87	1.84	2.71	1.91	1.87
South	1.56	2.00	1.33	1.78	1.24	1.98
<i>Enabling Resources</i>						
Adult Income 2009		1.04				1.01
High School or Less		0.23 **				0.23 *
College Degree		0.77				0.80
Advanced Degree		0.63				0.45
No Health Insurance		0.97				0.42
<i>Perceived Need</i>						
Diabetic Parent			1.21		1.07	1.46
Frequent Urination			1.80		1.79	1.89
A1C Above 10			0.05 *		0.03 *	0.03 *
Undiagnosed High Blood Pressure			1.99		2.55	2.55
Fair or poor Self-Rated Health			0.35		0.30 *	0.26 *
<i>Evaluated Need</i>						
Diagnosed High Cholesterol				0.37 *	0.32 *	0.21 **
Diagnosed High Blood Pressure				0.99	2.04	2.54
Waist Circumference 35+ Inches				0.64	0.69	0.71
Currently Obese				1.19	1.52	1.54
<i>Health Behavior Controls</i>						
Fast Food Consumption	0.82 **	0.78 **	0.80 **	0.83 **	0.81 **	0.77 **
Sugary Drink Consumption	1.03	1.03	1.04	1.04	1.05 *	1.04 *
Doesn't Walk for Exercise	1.30	1.55	1.37	1.13	1.40	1.72
Regular Drinker	1.98	1.69	2.42	1.68	2.08	1.62
Regular Smoker	0.42 *	0.54	0.52	0.39 *	0.45	0.62

Notes: a. Undiagnosed diabetic refers to having a hemoglobin A1C level 6.5+ and no prior diabetes diagnosis when the respondent was not pregnant; b. Odds ratios reported; c. Pregnant women excluded from analyses; d. Some college or vocational training is the reference category for achieved education; e. 4 Immigrant cases were dropped due to too few cases for analysis; f. * p<0.05, ** p<0.01, *** p<0.001

CHAPTER 5

5.1 DISCUSSION

This dissertation has added to the sociological literature in several important ways. Moreover, the relevance this research may have on policy implications could be substantial. The racial health disparities revealed by this work suggest that undiagnosed diabetes may not be a just health problem, but rather it may serve as an indicator of larger social problems surrounding racial minority status and health in the United States for young adults. Racial health disparities were observed for both the prevalence of diabetes risk and the allocation of diabetes diagnoses. It is particularly troubling that non-Hispanic blacks are simultaneously at increased risk of diabetes and have reduced odds of diagnosis even when they seek care.

The findings presented here are the first step towards finding better ways to measure diabetes risk. The first analytic chapter demonstrates that sample composition matters greatly when discerning the prevalence of diabetes risk. The Add Health sample was a larger and more racially diverse sample that yielded higher prevalence estimates of all three diabetes risk categories than the NHANES study. Using the Add Health data made it possible to explore possible connections between social factors, like perceived discrimination, and diabetes risk severity by race that would not be possible with the NHANES data. Further, because the Add Health sample had such a large discrepancy between those with and without diagnoses, it was possible to isolate whether risk of undiagnosed diabetes was more strongly associated with failure to seek care or differences in diagnosis allocation.

Taken together the foundational studies presented here provide documentation that undiagnosed diabetes is a significant problem for American young adults. Moreover, this dissertation demonstrates that diabetes risk and racial minority status are linked in ways that cannot be explained strictly by biological or behavioral pathways. The association between racial minority status and diabetes risk warrants significant further research as preliminary results from the second analytic chapter suggest that the stress of discrimination may shed light on the remaining racial disparity in diabetes severity that is not explained by biological predisposition or risky coping strategies. However, the final analytic chapter provides support an additional avenue for discrimination to affect the impact of diabetes by race in the doctor's office.

Population Inferences of Racial Disparities in Diabetes Risk

Although my findings did not identify nonresponse bias in either the NHANES or Add Health studies, this research provides insight into the differences between the studies and confirms that undiagnosed diabetes is of particular risk for non-Hispanic blacks. However, the persistence of large differences in prevalence estimates for undiagnosed pre-diabetes and undiagnosed diabetes that cannot be explained by nonresponse suggests that either the studies sample different populations or the testing strategies used to generate the hemoglobin A1C values contribute to the differences in prevalence estimates. The noted sample composition differences between the two studies provide a more reasonable explanation than to conclude that the different methods used to test for hemoglobin A1C shifted the prevalence estimates so dramatically as to observe these results. However, future research is warranted to definitively conclude that the methods

used to test the blood samples had no effect on the differences in the prevalence estimates across studies.

Regardless of the impact of the sample composition differences on the prevalence estimates, it is clear that non-Hispanic black young adults are a particular group at heightened risk for diabetes. This finding suggests that further research is needed to better measure the magnitude of diabetes risk for racial minority groups. In absence of actual population parameters for diabetes prevalence in young adults, repeated studies among multiple samples of the young adult population are the best way to approximate the depth of the racial disparities related to diabetes severity.

Perceived Discrimination and Race-Stratified Diabetes Risk

The results of the second analytic chapter demonstrate complex relationships between racial differences in perceived discrimination and diabetes risk severity. The descriptive findings illustrate greater disparities for young adult diabetes risk than those observed for all adults over age 20 from the CDC (American Diabetes Association Fact Sheet 2013; CDC 2011). However, my findings also document disparities in pre-diabetes rates as well, which were not observed when looking at all adults over age 20 (CDC 2011). These discrepancies may provide foundational evidence of racial health disparities in diabetes risk for young adults. However, it remains unclear how early these trends emerge. If both pre-diabetes and diabetes are indeed increasing for non-Hispanic black young adults at rates significantly higher than non-Hispanic white young adults at ages earlier than expected, this could contribute to later racial health disparities in diabetes-related morbidity and mortality for those who remain undiagnosed for longer than

necessary. Identifying how early these patterns emerge could be critical in reducing later health disparities unnecessary morbidity and mortality for these conditions.

Although these studies document the noteworthy differences in sample composition across studies, potentially calling into question the true prevalence of diabetes risk in American young adults, the Add Health sample's larger number of participants allowed for meaningful analysis regarding the factors for those with elevated risk of multiple levels of diabetes severity. In doing so, it was possible to identify preliminary evidence that perceived discrimination is experienced differently by race and those differences affect diabetes risk severity for young adults in measurable ways. Perceived discrimination appears to increase diabetes risk for both non-Hispanic blacks and non-Hispanic whites. However, the lack of specificity in the discrimination measure suggests that further research is necessary to determine if these findings reflect the impact of general discrimination and unfair treatment or if there are different effects for more specific racial discrimination. Although these findings leave the role of racial discrimination and diabetes risk severity unclear, the race-specific findings for the effects of more general discrimination warrant further research in the area.

Although the focus of this chapter was not to determine why undiagnosed diabetics lack diagnoses, these findings demonstrate that this is a serious problem. Further study is needed to determine if additional structural or behavioral patterns contribute to the dramatic under diagnosis of young non-Hispanic black diabetics. If diagnoses are allocated differently by race, this could create the conditions for future health disparities as people age that may contribute to increased morbidity and mortality. Moreover, if diabetes diagnoses are allocated differently by race this could be an

additional indicator for how systemic discrimination is for young adult non-Hispanic blacks.

Racial Disparities in Diabetes Diagnoses

The revelation that racial differences in rates of undiagnosed diabetes cannot be explained by differences in help seeking patterns and instead are influenced by differences in diagnosis allocation—even for those who seek care—is one of the most disturbing findings of this entire dissertation. The final analytic chapter of this dissertation supports the conclusion that the difference in rates of undiagnosed diabetes are influenced by differences in diagnosis allocation that again disadvantage non-Hispanic blacks compared to non-Hispanic whites.

These findings suggest that there is something about doctor-patient interactions when non-Hispanic black diabetics go to the doctor that does not result in diabetes testing at the same rate as non-Hispanic white diabetics. If these findings reflect implicit biases on the part of doctors, this would provide support for findings regarding black-white disparities in diagnostic testing in a different medical setting (Stepanikova 2012; Stepanikova, Triplett, and Simpson 2011). Alternatively, these findings may reflect differences in communication patterns between doctors and patients that may reflect doctors spending less time with minority patients or minority patients presenting their symptoms differently than non-Hispanic white patients. Although the current data made it impossible to know what specifically led to these dramatic race differences in diagnosis patterns, it certainly warrants future research.

Conclusions

The purpose of this dissertation was to identify the prevalence, predictors, and pathways to diagnosis of diabetes in young adults. At the end of this dissertation, both more and less is known about racial disparities in undiagnosed diabetes for young adults. While documenting the lack of discernable nonresponse bias in the NHANES study for estimates of undiagnosed diabetes suggests that the delay in collecting biological data may not reduce the estimates of undiagnosed diabetes risk, it does not provide clarity as to which estimates better reflect the true prevalence of undiagnosed diabetes in young adults compared to national benchmarks. Documenting that perceived discrimination impacts diabetes risk severity is a step toward understanding the biopsychosocial impact of minority status on diabetes, if only in documenting a foundational association for future research. Establishing that diagnosis disparities exist for non-Hispanic black diabetics in the Add Health sample are more closely associated with missed opportunities for diagnoses than failure to seek care is a substantial finding, but it does not identify why this is happening. Although each of these studies contributes to the sociological literature in different ways, they share the result of presenting multiple new questions for future research. Racial disparities in undiagnosed diabetes have now been documented for American young adults. Future research must focus on addressing this problem, crafting policies, and targeting interventions to stop it for future generations.

5.2 References

- American Diabetes Association .2013 <<http://professional.diabetes.org/Admin/UserFiles/0%20-%20Sean/FastFacts%20March%202013.pdf>> Accessed March 20, 2014
- CDC 2011 < <http://non.cdc.gov/diabetes/pubs/estimates11.htm>> Accessed July 30, 2013
- Stepanakova, Irena. 2012. "Racial-Ethnic Biases, Time Pressure, and Medical Decisions." *Journal of Health and Social Behavior*. 53(3)329-343.
- Stepanikova, Irena, Jennifer Triplett, and Brent Simpson. 2011. "Implicit Racial Bias and Prosocial Behavior." *Social Science Research* 40:1186-1195.