

Kim, Hyuncheol Bryant; Lee, Suejin; Lim, Wilfredo

Working Paper

Knowing Is Not Half the Battle: Impacts of the National Health Screening Program in Korea

IZA Discussion Papers, No. 10650

Provided in Cooperation with:

IZA – Institute of Labor Economics

Suggested Citation: Kim, Hyuncheol Bryant; Lee, Suejin; Lim, Wilfredo (2017) : Knowing Is Not Half the Battle: Impacts of the National Health Screening Program in Korea, IZA Discussion Papers, No. 10650, Institute of Labor Economics (IZA), Bonn

This Version is available at:

<https://hdl.handle.net/10419/161273>

Standard-Nutzungsbedingungen:

Die Dokumente auf EconStor dürfen zu eigenen wissenschaftlichen Zwecken und zum Privatgebrauch gespeichert und kopiert werden.

Sie dürfen die Dokumente nicht für öffentliche oder kommerzielle Zwecke vervielfältigen, öffentlich ausstellen, öffentlich zugänglich machen, vertreiben oder anderweitig nutzen.

Sofern die Verfasser die Dokumente unter Open-Content-Lizenzen (insbesondere CC-Lizenzen) zur Verfügung gestellt haben sollten, gelten abweichend von diesen Nutzungsbedingungen die in der dort genannten Lizenz gewährten Nutzungsrechte.

Terms of use:

Documents in EconStor may be saved and copied for your personal and scholarly purposes.

You are not to copy documents for public or commercial purposes, to exhibit the documents publicly, to make them publicly available on the internet, or to distribute or otherwise use the documents in public.

If the documents have been made available under an Open Content Licence (especially Creative Commons Licences), you may exercise further usage rights as specified in the indicated licence.

DISCUSSION PAPER SERIES

IZA DP No. 10650

**Knowing Is Not Half the Battle: Impacts of the
National Health Screening Program in Korea**

Hyuncheol Bryant Kim
Suejin A. Lee
Wilfredo Lim

MARCH 2017

DISCUSSION PAPER SERIES

IZA DP No. 10650

Knowing Is Not Half the Battle: Impacts of the National Health Screening Program in Korea

Hyuncheol Bryant Kim

Cornell University and IZA

Suejin A. Lee

Cornell University

Wilfredo Lim

Mathematica Policy Research

MARCH 2017

Any opinions expressed in this paper are those of the author(s) and not those of IZA. Research published in this series may include views on policy, but IZA takes no institutional policy positions. The IZA research network is committed to the IZA Guiding Principles of Research Integrity.

The IZA Institute of Labor Economics is an independent economic research institute that conducts research in labor economics and offers evidence-based policy advice on labor market issues. Supported by the Deutsche Post Foundation, IZA runs the world's largest network of economists, whose research aims to provide answers to the global labor market challenges of our time. Our key objective is to build bridges between academic research, policymakers and society.

IZA Discussion Papers often represent preliminary work and are circulated to encourage discussion. Citation of such a paper should account for its provisional character. A revised version may be available directly from the author.

ABSTRACT

Knowing Is Not Half the Battle: Impacts of the National Health Screening Program in Korea*

Health screening provides information on disease risk and diagnosis, but whether this promotes health is unclear. We estimate the impacts of the National Health Screening Program in Korea for diabetes, obesity, and hyperlipidemia. In this setting, information on disease risk and prompting for a secondary examination vary at different biomarker thresholds. We find evidence for increased diabetes medication and weight loss around the high risk threshold for diabetes, where information is combined with prompting for a secondary examination and subsequent medical treatment. However, we find no differences around other thresholds, where information is not combined with further intervention.

JEL Classification: I12, I18

Keywords: health screening, information, obesity, diabetes, hyperlipidemia, health behavior

Corresponding author:

Hyuncheol Bryant Kim
Department of Policy Analysis and Management
Cornell University
Ithaca, NY 14835
USA
E-mail: hk788@cornell.edu

* We thank Douglas Almond, Matias Cattaneo, John Cawley, Karen N Eggleston, Tal Gross, Ayako Kondo, Soonman Kwon, Ajin Lee, Yong Suk Lee, Zhuan Pei, Cristian Pop-Eleches, Yasuyuki Sawada, Hitoshi Shigeoka, Miguel Urquiola, and seminar participants at Columbia University, Cornell University, University of Tokyo, Stanford University, KDI School of Public Policy, and ASHEcon 2016 for helpful suggestions.

1 Introduction

Health behavior is an important determinant for health, especially in industrialized countries where morbidity and mortality are primarily related to chronic or lifestyle diseases (Cawley and Ruhm, 2011).¹ However, people often resist engaging in healthy behaviors that have positive future health outcomes and economists have tried to explain why this is the case (e.g., Thaler and Shefrin, 1981; Becker and Murphy, 1988; Suranovic et al., 1999).² In addition, many health policies aim to encourage healthy behaviors by addressing the lack of information about the benefits of healthy behaviors or about individual health status. For example, many developed countries have provided public health screening.³ The motivation for providing information in screening is that such knowledge would promote desirable health behaviors and early treatment that would prevent disease or reduce complications.

In this paper, we investigate whether individuals modify their behavior as a result of the information on disease risk provided from health screening. We also investigate the impact of when information on disease risk is combined with further medical intervention. We provide comprehensive evidence on the impacts of health screening by studying one of the world’s largest general screening programs, the National Health Screening Program (NHSP) in Korea, analyzing longitudinal administrative data that includes

¹For instance, the World Health Organization (2009) identifies that the leading causes of mortality and morbidity in high income countries are modifiable risk factors (i.e. tobacco use, overweight and obesity, physical inactivity, high blood pressure, high blood sugar, high cholesterol, low fruit and vegetable intake, and alcohol use).

²For instance, Becker and Murphy (1988) suggest that people initiate addictive behaviors involving forward-looking maximization with time-consistent preferences. Thaler and Shefrin (1981) suggest that time-inconsistent preferences prevent individuals from adopting healthy behaviors – individuals may face internal competition between the farsighted desire to obtain better health and the nearsighted desire for immediate pleasure. Cognitive limitation or bounded rationality is also suggested as an alternative model to explain myopic behaviors (e.g., Suranovic et al., 1999).

³Under the Affordable Care Act, the U.S. requires almost all health plans to cover a set of preventive services including health screening at no cost and Medicare started covering annual wellness visits, including a wide range of health screening and counseling benefits, in 2011 (Chung et al., 2015). In 2011, the United Kingdom also implemented national screening for cardiovascular disease in adults ages 40 to 74 (Dalton and Soljak, 2012). Asian countries including South Korea (Lee and Lee, 2010) and Japan (Kohro et al., 2008) and other European countries such as Austria (Hackl et al., 2015) also have national health screening programs for adults.

medical claims, biomarkers, and a survey of health behavior, and considering three common lifestyle diseases in the same setting.

We apply a regression discontinuity design that takes advantage of the fact that information on disease risk and prompting for a secondary examination for diabetes, obesity, and hyperlipidemia⁴ vary discontinuously with levels of fasting blood sugar, body mass index (BMI)⁵, and low-density lipoprotein (LDL) cholesterol⁶, respectively—at cutoffs that are arbitrary in the sense that individuals just below and just above a cutoff share otherwise similar characteristics.^{7,8} While risk classification varies at all thresholds we study, a secondary examination is only offered to those with the high risk classification for diabetes, which is a further medical intervention which consists of confirmatory tests, counseling with a physician, and opportunity for medical treatment. Specifically, we evaluate the impacts on biomarkers such as BMI, waist circumference, blood sugar level, and serum LDL cholesterol; health-related behaviors including future screening participation, exercise, drinking, and smoking; and health care utilization such as outpatient visits and disease-specific prescription medications.

In our study setting, the NHSP provides free general health screening every two years to the entire population aged 40 and over. The NHSP includes a variety of tests for health screening including diabetes, obesity, and

⁴Hyperlipidemia is a condition in which there are abnormally high levels of fats or lipids in the blood. Examples of lipids include cholesterol and triglycerides. These substances can deposit in blood vessel walls and restrict blood flow which can lead to heart attack or stroke. In this study, the relative risk of hyperlipidemia is determined by the level of LDL cholesterol.

⁵BMI is calculated from height and weight measurements. Specifically, BMI is weight in kilograms divided by height in meters, squared ($BMI = \text{weight}/\text{height}^2$).

⁶LDL cholesterol is often termed the “bad” cholesterol. Medical evidence suggests that elevated levels of LDL cholesterol in the blood are associated with increased risk of cardio-cerebrovascular diseases.

⁷For example, high risk for diabetes is defined as having a fasting blood sugar level of 126 mg/dL, meaning that an individual with a fasting blood sugar level of 126 mg/dL would be considered to have high risk for diabetes and would be prompted to take the secondary examination, while an individual with a fasting blood sugar level of 125 mg/dL would be considered to have medium risk for diabetes and would not be prompted to take the secondary examination.

⁸Compared to the margin of screening versus not screening, studying impacts of different information on disease risk and further intervention conditional on screening is important because it provides insight into the mechanisms underlying the effectiveness of screening.

hyperlipidemia. It is combined with a survey that collects information on health-related behaviors. We use data on a 2% random sample of the population from administrative data provided by the National Health Insurance Service (NHIS) in Korea, which includes more than 350,000 baseline screening participants observed from 2009 to 2013. The size of the dataset allows us to implement a regression discontinuity design with enough precision to estimate null impacts or rule out even small impacts. Also, the richness of the dataset allows us to test the effect of screening on a range of behaviors and health outcomes, including biomarkers.

We find evidence that those just above the 126 mg/dL fasting blood sugar threshold, where individuals are classified as high risk for diabetes and also prompted to take a secondary examination, take more medication for diabetes and exhibit reduced BMI and waist circumference in the subsequent screening. On the other hand, we find little to no differences around the medium risk threshold for diabetes, as well as risk classification thresholds of obesity and hyperlipidemia which are not combined with additional intervention. These findings suggest that information should be combined with further medical intervention to increase the marginal benefit of screening.

This paper complements the literature on how information or disease diagnosis affects health behaviors and health outcomes. Our study is closest to papers that employ a regression discontinuity design based on diagnosis or risk classification thresholds. Zhao et al. (2013) examine the effect of hypertension diagnosis on food consumption in China by applying a regression discontinuity design to the systolic blood pressure threshold for hypertension diagnosis. They find that the hypertension diagnosis causes individuals to reduce fat intake on average and that richer individuals reduce more. Similarly, Almond et al. (2010) exploit the very low birth weight (VLBW) classification threshold for newborns. They find discontinuous increases in medical spending and decreases in mortality risk for newborns with birth weights just below the threshold than those just above. Almond et al. (2016) study the BMI cutoffs for overweight categorization in New York City public school students. They find a small but significant increase in BMI and weight for teenage girls in the following academic year. Dahlberg et al. (2016) exploit the aorta size cutoffs for abdominal aortic aneurysm risk classifications in the Swedish screening program and study their effects on individuals' subjective physical and psychological well-being. Lastly, Iizuka et al. (2017), using the fasting blood sugar cutoffs for determining the relative risk for diabetes, show that those just above the 110 fasting blood sugar cutoff (those classified borderline

type) increases diabetes related physician visit and medical expenditure.⁹

One benefit of our study setting compared to Zhao et al. (2013), Almond et al. (2010), and Dahlberg et al. (2016) is that the running variables used in our analysis are not prone to measurement errors such as heaping and manipulation. For example, blood pressure readings may suffer from heaping at multiples of five and ten.¹⁰ Also, birth weight could be subject to manipulation around the reference points, so as to lower health insurance premia or to receive more preferable treatment (Barreca et al., 2011). Dahlberg et al. (2016) also find bunching around the aorta size cutoffs. Such manipulation of the running variable could result in non-random heaping in the distribution and can lead to bias in a regression discontinuity design (Barreca et al., 2016; Lee and Lemieux, 2010). The running variables in this study are difficult to precisely manipulate because BMI is a continuous function of the two measurements that are observed, and fasting blood sugar and LDL cholesterol are based on laboratory measurements. In addition, this setting provides a large sample size, which is necessary to be able to precisely measure impacts using a regression discontinuity design (Deke and Dragoset, 2012).

Our study makes three important contributions. First, the quasi-experimental regression discontinuity design that we implement allows us to control for confounding factors that would make it difficult to disentangle the endogenous relationship between information on disease risk and outcomes. In fact, the use of suitable methods to control for endogeneity is particularly important to understand the behavior changes after a disease diagnosis because the relative risk of developing a disease is often correlated with past behaviors of individuals. However, some papers use a suboptimal control group to study the effect of a disease diagnosis. For example, to study the effects of diabetes diagnosis on health-related behavior changes among elderly, Slade

⁹In addition, some papers study the screening participation effects using randomized controlled trials or an instrumental variables approach (e.g., Deutekom et al., 2011; Prina and Royer, 2014; Hackl et al., 2015). The focus of these studies is different from our study in the sense that our study focuses on how individuals who received different risk classifications respond differently to the information provided while these studies focus on the impact of screening participation versus not. On the other hand, rather than examining the screening participation effect or diagnosis effect, Darden (Forthcoming) examines whether repeated exposure to one’s biomarker values promotes smoking cessation by developing a theoretical model of lifetime smoking behavior.

¹⁰Not shown here, we find significant heaping at multiples of five and ten in systolic and diastolic blood pressure in our dataset. As a result, baseline observable characteristics are not balanced across the cutoffs.

(2012) constructs a control group of non-diagnosed individuals at risk, which may represent a population with lower interest in health, who undertake less health check-ups compared to the diagnosed group.¹¹ In addition, Oster (2015) uses shows an improved food purchase pattern after diabetes diagnosis based on high frequency household scanner data, using an event study approach to compare behavior changes before and after diabetes diagnosis, implicitly assuming that behaviors before the diagnosis remain the same in the counterfactual scenario—if the person had not been diagnosed. Lastly, many papers in the medical literature use healthy individuals as a control group (e.g., Kersaw et al., 2004; Keenan, 2009; Newsom et al., 2011; Wu et al., 2016).¹²

Second, this study provides evidence of the impact of screening on outcomes at a population level, based on a national health screening program. As Kim and Lee (2017) discuss, effects in the population-based screening setting might differ from those provided by clinical RCTs due to selection and crowd out. Thus, our setting provides rare practical evidence to inform national-level screening initiatives in other countries.

Lastly, our unique setting and comprehensive administrative data allows us to abstract away from some of the differences in results that might be driven by different study settings and designs. This is important because earlier papers have found mixed results depending on the disease of interest, types of outcome variables, length of study period, and the empirical strategy used.¹³ In our setting, we are able to study multiple biomarkers with multiple treatment cutoffs which allow us to examine the impacts of different combinations of information on disease risk and further intervention for three

¹¹Slade (2012) defines individuals at risk for developing diabetes or pre-diabetes by calculating a predicted probability (propensity score) for high blood sugar using observable characteristics of individuals.

¹²Earlier studies conducted a one-time survey and depend solely on patients’ memory to measure the behavior changes (e.g., Blanchard et al. (2003) and Patterson et al. (2003) study patients with cancer diagnosis; Fortenberry et al. (2002) study patients diagnosed with sexually transmitted diseases).

¹³For instance, large impacts are found in individuals with confirmed HD, which is a life threatening disease with no cure (Oster et al., 2013) and in newborns with VLBW classification, which is combined with medical treatment (Almond et al., 2010). Modest behavior changes are found in diagnosis of diabetes (Slade, 2012; Oster, 2015), HIV (Thornton, 2008), hypertension (Zhao et al., 2013), while no effects are found in overweight classification (Almond et al., 2016) and diabetes genetic risk group (Wu et al., 2016).

major lifestyle diseases including diabetes, obesity, and hyperlipidemia.¹⁴ In addition, a wide range of outcomes observed multiple years after the baseline screening allows us to examine the full picture of the dynamics in behavior changes and health outcomes.

The remainder of the paper is organized as follows. Section 2 describes the institutional context and the screening program which creates the setting for our analysis. Section 3 describes the data and Section 4 presents the empirical framework. Section 5 describes and discusses the results. Section 6 concludes.

2 Institutional Details

The National Health Screening Program (NHSP) in Korea has provided free general health screening since 1995.¹⁵ The NHSP consists of various tests and measurements including systolic and diastolic blood pressure, fasting blood sugar, cholesterol, hemoglobin, height, weight, waist circumference, and many others. In addition, before the screening, a survey is conducted of the screening participants regarding health behavior such as exercise, alcohol consumption, and cigarette smoking. Screening through the NHSP is available every other year.¹⁶

During 2008-2009, the NHSP implemented a range of reforms to improve the precision and understanding of the screening results. For instance, waist

¹⁴In addition, we are able to study the medium risk cutoff (or pre-diagnosis cutoff) in addition to the high risk cutoff (or diagnosis cutoff). While many studies only focus on the major health shocks or disease diagnosis, medium risk classification is also important to study if it results in larger life expectancy gains by inducing preventive behaviors before experiencing major health shocks, which is one of the purposes of periodic health screening.

¹⁵Korea provides universal coverage of health insurance through National Health Insurance (NHI) and Medical Care Assistance (MCA), which are both administered by the NHIS. NHI, which covers about 97% of the population, consists of two types of health insurance—employee health insurance and self-employed health insurance. The MCA covers the remaining 3% of people living under the national poverty line. For those enrolled in the MCA, the NHSP was offered since 2012.

¹⁶Those born in odd-numbered (even-numbered) years are encouraged to undergo screening in odd-numbered (even-numbered) years. Age restrictions vary across insurance type as shown in Appendix Table A.1. Blue-collar workers with employee insurance are an exception in that they are eligible for screening every year. In addition to the free screening offered every one or two years, people are allowed to participate in the screening program through out-of-pocket expenditures anytime.

circumference measurement was added to the obesity screening, which was based solely on BMI prior to the reform. Measurement of HDL cholesterol, LDL cholesterol, and triglycerides were also added. Lastly, they introduced a “Health Risk Evaluation” section to the screening report with health traffic light indicators that we describe in further detail below.

A screening report is sent to the household by regular mail within two weeks after screening. The screening report consists of two pages. Figure 1 shows a sample of the screening report of 2009, our baseline year. Variations in information on disease risk come from the general screening results in the first page (Figure 1a) and health traffic light indicators in the second page (Figure 1b). Table 1a summarizes the study sample, cutoffs, and interventions (e.g. information obtained from the screening report) which change discretely around the cutoffs for each physiologic measurement (i.e., running variable).

In the first page of the screening report card, reference ranges for classifying Normal A (Satisfactory) and Normal B (Warning; need preventive care, but no problem in health) are provided for each physiologic measurement (Figure 1a). If a measurement is outside the range of Normal A and Normal B, it is classified as Disease Suspected. Individuals are notified of which measurements are determined as Normal B or Disease Suspected in the bottom of the first page (bottom red box).¹⁷ Note that the determination of Normal A, Normal B and Disease Suspected varies discontinuously with the levels of physiological measurements at different reference range cutoffs.

Screening participants are also notified of their “Health Risk Evaluation (HRE)” in the second page of the screening report card (Figure 1b). The HRE is designed to help individuals better understand and control their risk factors by visualizing the degree of risk. We focus on blood sugar, obesity, and LDL cholesterol in the “Knowing your health risk factors” section (within the red box, in the middle of the page) where the level of risk indicated by different “health traffic light” colors (low risk (green), medium risk (yellow), and high risk (red)) also changes discontinuously by the level of BMI, waist circumference, blood sugar, and LDL cholesterol at the cutoffs. The cutoffs in the health traffic lights of obesity, blood sugar, and LDL cholesterol are determined using the same or additional reference range cutoffs as those reported in the first page. We report the risk classification rules for these

¹⁷For example, the red boxes in Figure 1a indicate that a person has a fasting blood sugar level of 120 mg/dL, and thus determined as “Normal B: Manage diabetes.”

health traffic lights in Table 1b.

Those with a fasting blood sugar or blood pressure level outside the range of Normal A and B are offered a secondary examination at no cost to confirm a diagnosis of diabetes or hypertension.¹⁸ Specifically, the bottom of the screening report card for these individuals will indicate “If you are determined as hypertension or diabetes suspected within, it is recommended to take a secondary examination within 30 days (until next January) from the date of this notification.”¹⁹ In addition, they are contacted by a hospital to undergo a secondary examination to confirm the diagnosis and/or receive counseling. For other diseases, individuals outside the range of Normal A and B are recommended in the screening report to seek further evaluation, but it is not covered by the NHSP.

In this paper, we consider three physiologic measurements, fasting blood sugar, BMI, and LDL cholesterol which respectively determine the relative risk of diabetes, obesity, and hyperlipidemia (Table 1a).²⁰ First, we study diabetes screening by exploring the cutoffs 100 and 126 of fasting blood sugar. The cutoffs 100 and 126 are used as the critical values of the reference ranges of the general screening results (Normal A or B) as well as the health traffic lights. For example, those at or above 126 are informed as “Diabetes Suspected” and “high risk of diabetes (red)”, while those in between 100 and 125 are informed as “Normal B” and “medium risk of diabetes (yellow).” Individuals below 100 are informed as “Normal A” and “low risk of diabetes (green).” As noted above, those above the 126 cutoff are also prompted to undergo free secondary examination. This also coincides with the clinical guideline to prescribe diabetes medications if upon secondary examination, the blood sugar level of 126 and above is confirmed and thus diagnosed

¹⁸They are determined “Diabetes Suspected” (i.e., fasting blood sugar ≥ 126) or “Hypertension Suspected” (i.e. systolic blood pressure ≥ 140 or diastolic blood pressure ≥ 90). They are also classified as “high risk (red)” in the health traffic lights.

¹⁹Unfortunately, our data do not support individual level information on the secondary examination. According to the statistical yearbook of the NHIS, the participation rate for the secondary examination among eligible individuals was 37% in 2009 and 39% in 2010 (NHIS, 2009; 2010). The secondary examination report card is shown at the Figure A.1, Individuals are informed about diet control, exercise, and need of medical treatment.

²⁰We considered studying other disease risk classifications such as hypertension. However, blood pressure readings suffer from clustering at multiples of five and ten, and hence are not suitable for a regression discontinuity analysis which requires smooth density around the cutoffs. Also, behavior measures such as smoking, exercise, and drinking are not used because they are discrete choice variables.

as diabetes (Korean Diabetes Association, 2007; 2015; American Diabetes Association, 2015).

Second, we examine obesity screening by exploring the cutoffs 23 and 25 of BMI.^{21,22} The risk classification for obesity depends upon BMI and abdominal obesity (measured by waist circumference). As reported in Table 1b, for the health traffic light, the cutoff 23 divides “low risk” and “medium risk” for those who are not abdominal obese (below the waist circumference cutoff), while the cutoff 25 divides “medium risk” and “high risk” for those who are abdominal obese (at or above the waist circumference cutoff).²³ Therefore, we restrict our sample to people with or without abdominal obesity depending on the BMI cutoffs we are investigating. Note that all individuals around the cutoff 23 fall into the same reference range of Normal A in the first page of general screening results. On the other hand, individuals at or above the cutoff 25 are outside the reference range and informed as Normal B in the first page, while those below 25 remain inside the Normal A range. Diagnosis cutoffs for determining obesity based on BMI and abdominal obesity based on waist circumference corresponds to the clinical guidelines. The basic treatment for obesity is healthy diet and regular physical exercise. Drug treatment or weight loss surgery for obesity is only recommended for special cases including those with cardiovascular complications, extreme obese, or sleep apnoea (Korean Endocrine Society and Korean Society for the Study of Obesity, 2010).

Lastly, we examine the cutoff 160 of LDL cholesterol. In the analysis, we restrict our sample to people without a previous history of hyperlipidemia and people who don’t have diabetes.²⁴ We focus on examining the highest cutoff

²¹The World Health Organization (2000) introduced the classification of obesity based on BMI thresholds of 18.5, 25, 30, 35 and 40 in 2000. Modifications of the WHO definitions are used across different countries, especially in Asia. In Korea, BMI over 25 with waist circumference greater than 85cm (for females) and 90cm (for males) results in a diagnosis of obesity.

²²We do not study the underweight classification—and thus the BMI threshold of 18.5—in this paper because a classification of underweight is fundamentally different from a classification of overweight or obese. We do not assess the BMI threshold of 30 for individuals without abdominal obesity because the sample size around this threshold is small and represents people at the very tail of the distribution (Appendix Figure A.5(c)).

²³Waist circumference cutoffs are 85cm for female and 90cm for male.

²⁴As reported in Table 1b, those who have diabetes use a lower LDL cholesterol cutoff to determine the relative risk of hyperlipidemia in the health traffic lights. In order to focus on a single cutoff, we drop individuals with previous diabetes diagnosis and with

for LDL cholesterol because the other cutoffs for dividing Normal A and B were not consistent throughout our baseline period (100 in 2009 and 130 in 2010). People 160 or above are informed as Hyperlipidemia Suspected and high risk of hyperlipidemia, while those below 160 are informed as Normal B and medium risk in our baseline period 2009-2010. Medical treatment is recommended at the 160 cutoff for people with confirmed diagnosis²⁵ and with zero or one condition among the following five conditions: currently smoking, hypertension, low HDL (≤ 40 mg/dL), family history of coronary artery diseases, and age (45 or older if men and 55 or older if women) (Korean Society of Lipidology and Atherosclerosis, 2009).²⁶

3 NHIS Data

Our analysis uses the NHIS’s National Sample Cohort (NHIS-NSC) data that is a 2% random sample of the population (Lee et al., 2015). Our empirical analysis requires data on take-up and the results from general screening, and future health behaviors and health outcomes. The NHIS database consists of three parts—eligibility information, medical claims, and screening information. Information from eligibility such as gender, age group, income bracket, and type of insurance as well as disease-specific medical claims data are available regardless of screening participation.²⁷ However, information from screening, such as self-reported health behaviors, self-reported previously diagnosed diseases, and biomarkers, is available only for the screening participants.²⁸

fasting blood sugar level ≥ 126 mg/dL.

²⁵Hyperlipidemia is diagnosed if LDL cholesterol of 160 and above is confirmed on two different days. In addition to the 160 cutoff of LDL cholesterol, different cutoffs in total cholesterol, HDL cholesterol, and triglycerides can also be used to diagnose hyperlipidemia.

²⁶The recommended treatment cutoffs of LDL cholesterol are 160, 130, or 100 mg/dL for those with 0-1 risk factor, 2+ risk factors, or coronary artery disease, respectively (Lorenzo et al., 2007; Korean Society of Lipidology and Atherosclerosis, 2009). Using the baseline screening information, we can estimate that 63.1% of those classified as high risk for LDL cholesterol fall into the 0-1 risk factor category, and hence, their treatment for hyperlipidemia is determined by the 160 cutoff.

²⁷Medical claims data are recorded based on the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10), which allows us to measure disease-specific medical treatments.

²⁸In addition, we do not observe the actual risk classifications that individuals receive in their screening report card. However, we are able to reconstruct the risk classifications

Our study sample consists of those who participated in the NHSP in 2009 or 2010. We define 2009-2010 screening as the baseline or round 1 screening as most people are eligible for screening every other year. We use a stacked sample of 2009 and 2010 for our main analysis to increase statistical power.²⁹ In our data, 352,896 individuals participated in the baseline screening. Among eligible individuals, the baseline screening participation rate was about 66% in 2009 and 68% in 2010 (NHIS, 2009; 2010).

Table 2 reports the baseline summary statistics of variables for individuals who participated in the baseline screening in 2009 or 2010. Of those baseline screening participants, about 77% and 53% participated in round 2 (after one or two years) and round 3 (after three or four years).³⁰ We define outcomes in the second and third round as short- and long-run outcomes, respectively.³¹

Even though our analysis is based on voluntary screening participants, we are able to measure some of the characteristics of the screening non-participants. To shed light on external validity, we compare the characteristics of screening participants and non-participants (Table A.2). We find that screening participants are older, more likely to have employee insurance, have higher income, and have lower total medical expenditure compared to screening non-participants.

Outcome variables are 1) future screening take-up,³² 2) biomarkers in-

(i.e. Normal A, Normal B, and Disease Suspected in the first page of screening report; and low risk, medium risk, and high risk in health traffic lights) for each individual following the NHSP rules and using biomarker information.

²⁹For those who took screening in both years, however, we only use 2009 observation to avoid duplication. As mentioned in Section 2, blue-collar workers with employee insurance are eligible for screening every year.

³⁰The observed participation rate in round 3 screening is lower than in round 2 screening because for the 2010 cohort we do not have data on screening participation after 4 years (i.e., no data in 2014).

³¹Short-run outcomes are measured in round 2 screening—one or two years after the baseline, taking the earlier observation if an individual participated in both years. Similarly, long-run outcomes are measured in round 3 screening—three or four years after the baseline, taking the earlier observation if an individual participated in both years.

³²Future screening take-up is an important outcome variable for two reasons. First, future screening take-up is an important health behavior in itself. Second, since information on biomarkers and health behaviors are observed only for screening participants, any change in screening take-up is related with selection into screening. Therefore, it is necessary to test whether screening results in the baseline have an impact on future screening participation. As shown in Column 4 of Table 3, Column 3 of Table 4, and Column 4 of Table 5, we do not find evidence that those below the cutoff in the baseline (i.e., control

cluding fasting blood sugar, BMI, waist circumference, and LDL cholesterol, 3) health behaviors such as physical exercise,³³ number of drinks per week, and number of cigarettes per week, and 4) health care utilization such as outpatient visits and prescription medications for diabetes, obesity, and hyperlipidemia.³⁴

4 Empirical Framework

4.1 Setup of the empirical analysis

We implement a regression discontinuity design by taking advantage of the fact that disease-specific information on disease risk and prompting for a secondary examination vary discontinuously with fasting blood sugar, BMI, and LDL cholesterol. The arbitrary thresholds of risk classification enable us to estimate causal effect of screening. Running variables are baseline fasting blood sugar, BMI, and LDL cholesterol.³⁵ For each running variable, we

group) are more or less likely to participate in future screening compared to those above the cutoff in most specifications.

³³Exercise is measured as an indicator function of engagement in “basic exercise.” An individual is engaging in “basic exercise” if, in the survey, one answered as engaging in 3 or more days of vigorous exercise, or 5 or more days of moderate exercise, or 5 or more days of mild exercise during the last week. Vigorous exercise is defined from the question “During the last week, how many days did you exercise vigorously for more than 20 minutes until you were almost out of breath? (example: running, aerobics, cycling in high speed, mountain hiking, etc.)”; moderate exercise is defined from the question “During the last week, how many days did you exercise in a moderate level for more than 30 minutes until you had to breathe a little faster than usual? (example: fast walking, tennis, bicycle riding, cleaning, etc.)”; and mild exercise is defined from the question “During the last week, how many days did you walk for a total of more than 30 minutes, including separate 10-minutes walks? (example: light exercise, walking to work, walking for leisure, etc.).”

³⁴Short- and long-run health care utilization is defined as follows. For outpatient visits, we use the total number of outpatient visits for a disease of interest during the year and one year after the baseline screening take-up (short-run outcome) and two and three years after the baseline screening take-up (long-run). For prescription medications, we examine the sum of the prescribed number of days for each medication in the short- and long-run.

³⁵In the data, fasting blood sugar and LDL cholesterol are available as an integer which is the unit used in the report card, while we calculate BMI using data on weight and height. In the report card, BMI is shown rounded to the nearest tenth, but in our analysis, we use the calculated continuous BMI measure without rounding. Hence, technically speaking, for our continuous BMI measure, we use 22.95 and 24.95 as the cutoff. For the sake of brevity, however, we use 23 and 25 when reporting.

estimate the following equation:

$$Y_{ict} = \beta \cdot 1\{M_{it} \geq \tau\} + f(M_{ic}) + \theta_c + \psi_t + \varepsilon_{ict}$$

where Y_{ict} is the outcome of interest for individual i , screening cohort c (=2009 or 2010), t years after screening. $1\{M_{ic}\}$ is an indicator function of individual i 's baseline running variable (M_{ic}) being greater than or equal to the relevant cutoff τ , $f(M_{ic})$ is a flexible function of the running variable, θ_c is the cohort-fixed effect, ψ_t is the year-fixed effect, and ε_{ict} is the idiosyncratic error term.³⁶ Errors are clustered by the unique value of the running variable as suggested by Lee and Card (2008).

Modeling $f(M_{ic})$ and bandwidth selection are important decisions in a regression discontinuity design. We use a non-parametric approach to model the function $f(M_{ic})$ by approximating it via a polynomial function of M_{ic} over a narrow range of data. In the main analysis, we estimate the discontinuity parameter β using a local linear regression with uniform kernel. Our preferred bandwidth is 10 mg/dL for blood sugar and LDL cholesterol, and 1 kg/m² for BMI because we believe these are narrow enough to compare observations below and above the cutoff and wide enough to be precise.³⁷ However, we report a series of robustness checks with different polynomial degrees and bandwidths to show how sensitive our findings are to these parameters (reported in Appendix Figures A.6 to A.15).³⁸ We also estimate our main outcomes after controlling for a standard set of variables including gender, age, residential area, insurance type, and baseline amount of medical expenditure (reported in Appendix Tables A.4 to A.6).

³⁶Year-fixed effect is not included in the regression for future screening participation, outpatient visits, and medication because we aggregate these outcomes for two consecutive years (e.g., indicator for taking medication for 0-1 years (or 2-3 years) after the baseline screening, number of days of taking medication for 0-1 years (or 2-3 years) after the baseline screening, number of outpatient visits for 0-1 years (or 2-3 years) after the baseline screening, and indicator for taking screening 1 or 2 years (3 or 4 years) after the baseline screening).

³⁷We fix our bandwidth choice in our main analysis because the MSE optimal bandwidth choices suggested by Calonico et al. (2014) or Imbens and Kalyanaraman (2012) are often too large in our case to evaluate two different cutoffs per running variable.

³⁸We report sensitivity tests using different bandwidths and polynomial degrees (linear, quadratic, and cubic). For fasting blood sugar and LDL cholesterol, we use bandwidths from 3 to 17, in increments of 1 mg/dL and for BMI, we use bandwidths from 0.3 to 0.7 in increments of 0.1 kg/m².

4.2 Validity of regression discontinuity design

A critical assumption to our identification strategy is that individuals just below a threshold are indeed comparable to individuals just above a threshold. Specifically, no one (screening participants, physicians, and hospitals) should be able to precisely manipulate the running variables around the cutoff. One way to test the validity of our model is testing the smoothness of the baseline observable characteristics around the cutoffs. As reported in Appendix Figures A.2 to A.4 and Table A.3, with very few exceptions, we do not find differences between individuals just below and just above the cutoff in their observable characteristics, including basic demographics, baseline health outcomes and health behaviors. Two of the 55, or 3.6%, of the discontinuity estimates at the cutoffs are statistically significant at the 5% level of significance, suggesting that our sample is balanced around the cutoffs (Table A.3). Moreover, we estimate regressions that control for these baseline variables and find that the results from the regressions including the control variables are similar to those from the regressions without them (Tables A.4-A.6).

Lastly, we examine whether the density function of the running variables is smooth around the threshold (Lee 2008; Barreca et al. 2016). Appendix Figure A.5 illustrates the density of three running variables. To the naked eye, densities of fasting blood sugar in Panel (a) and LDL cholesterol in Panel (b) seem very smooth at the treatment cutoffs.³⁹ Panels (c) and (d) respectively present the densities of BMI for the no abdominal obesity and abdominal obesity samples, and we do not observe extraordinary spikes around the BMI thresholds in either case. We conduct the McCrary test to formally test for the smoothness of the densities at the treatment cutoffs (McCrary, 2008). The test results with various bandwidths at the cutoff as well as falsified cutoffs are reported in Appendix Table A.7. The results show that some cutoffs with several bandwidths (i.e. BMI 23 with most bandwidths, BMI 25

³⁹For fasting blood sugar, we restrict our sample to individuals who took screening in the general hospital where density of the blood sugar level is very smooth to prevent potential manipulation around the threshold. Among the baseline screening participants, 30% took screening in general hospital. Indeed, we find a small heaping around the 126 cutoff for the people who took screening in private clinics and public health centers. Screening participants and physician are unlikely to manipulate the measurement in the larger size hospital where laboratory test results are automatically recorded. However, we do not restrict sample by hospital type for BMI and LDL cholesterol in the analysis because we do not observe such a heaping.

with bandwidths greater than 1.6, and blood glucose 126 with bandwidths 18 and 20) are not smooth. However, we do not find evidence for manipulation given that the t-statistics at the treatment cutoffs (23 for no abdominal obesity sample and 25 for abdominal obesity sample) are not large compared to the ones at the placebo cutoffs. In sum, we do not think that there exists stacking, and even if it exists, it is not correlated with baseline characteristics that are related to the outcome variables.

5 Estimation Results

In this section, we present evidence on the impact of and behavioral responses to diabetes, obesity, and hyperlipidemia screening in Sections 5.1, 5.2, and 5.3, respectively. We discuss the results in Section 5.4.

5.1 Results from diabetes screening

We first examine the responses to information on diabetes risk obtained from diabetes screening by comparing individuals just below and just above the fasting blood sugar cutoffs of 100 and 126 (mg/dL). As shown in Table 1a, cutoff 100 divides low risk and Normal A vs. medium risk and Normal B, while cutoff 126 divides medium risk and Normal B vs. high risk and Diabetes Suspected. In addition, those above the 126 cutoff are offered a secondary examination which may induce medical treatment if the diagnosis is confirmed.

Figures 2 and 3 illustrate short-run and long-run outcomes against baseline fasting blood sugar level, respectively. In the figures, the scatter plot indicates the mean of the outcome variable within 1 point bins. The solid lines represent the fitted values from equation (1) using a local linear regression with a bandwidth of 10 mg/dL and a uniform kernel, which are plotted separately below and above the cutoff. Its regression analog is presented in Table 3. It is worth noting again that the outcome variables shown in Columns 1 to 4 are available regardless of screening participation, but variables shown in Columns 5 to 12 are available only for screening takers.

Figure 2 shows that at the 126 cutoff there are clear deviations from the overall trend in both the short-run probability of taking diabetes medication (Panel (b)) and the number of prescribed days of diabetes medication (Panel (c)), which implies that individuals who are classified as high risk for diabetes

are more likely to take diabetes medications during the year of or the year after the baseline screening, compared to those who are classified as medium risk. The discontinuity estimates in Table 3 indicate that high risk individuals are 5.6 percentage points (a 50.9% increase) more likely to take diabetes medication (Panel A.1, Column 2) and are prescribed diabetes medication about 20.9 more days (a 73.3% increase) (Panel A.1, Column 3) than medium risk individuals.^{40,41} These results are not surprising because a secondary examination to confirm diabetes diagnosis was given only for those with blood sugar level above 126. Those whose diabetes was confirmed at the secondary examination were recommended to receive medical treatment as shown in Figure A.1.⁴²

In addition, we find a decrease in BMI of 0.16 kg/m² (a 0.6% change) and waist circumference of 1 cm (a 1.1% change) in the short-run (Panel A.1, Columns 6 and 7).⁴³ We provide evidence that the changes in BMI and waist circumference are driven by behavioral improvement and not by diabetes medication. There are certain types of diabetes medication that may lead to weight reduction.⁴⁴ However, as shown in Table A.8, a decrease

⁴⁰Even though we see a short-run impact on medications whose action is to control blood sugar level, we do not observe a corresponding short-run impact on fasting blood sugar level (Panel (e)) or future diabetes risk classification determined in round 2 (Panels (j), (k), and (l)). It is because diabetes medication is not recommended on the day of the screening and the half-life of diabetes medications is short. Therefore, effects of the medications will not be reflected in the blood sugar results. Specifically, the half-lives of biguanide and sulfonylurea, which are the most common types of diabetes medications, are 4-8.7 hours (Dunn and Peters, 1995) and 2-10 hours (Prendergast, 1984), respectively.

⁴¹We also find an increase in the number of outpatient visits for diabetes in the short run around the 126 cutoff (Panel (a) in Figure 2 and Column 1 of Panel A in Table 3). Note that the secondary examination in the NHSP is not counted as an outpatient visit.

⁴²According to the statistical yearbook of the NHIS, among the participants of the secondary examination, 51% and 40% were diagnosed as having diabetes in 2009 and 2010, respectively (NHIS, 2009; 2010). Considering the fact that the participation rate of the secondary examination was 37% in 2009 and 39% in 2010, we estimate that 18.9% (=37%*51%) and 15.6% (=39%*40%) of those above the 126 cutoff were finally diagnosed with diabetes in 2009 and 2010, respectively.

⁴³A decrease in BMI of 0.16 kg/m² is equivalent to a reduction of 0.4 kg (=0.16*(1.63)²) for a person with the average height of 163 cm

⁴⁴We do not think that the decrease of BMI and waist circumference is driven by diabetes medication because of the following reasons. First, there are a small number of patients who take only Metformin (24.3% under diabetes medication), which is one of the first-line pharmacologic treatments for Type 2 diabetes patients, leading to a weight loss (Diabetes Prevention Program Research Group, 2012). If the treatment is combined with a second-

of BMI and waist circumference is driven by those who do not take diabetes medication (Panel A), not those who take medication (Panel B). A possible explanation is that individuals who were just above the 126 cutoff and who did not take medication either were associated with doctors who did not strictly follow medication treatment guidelines and who instead recommended lifestyle changes, or the individuals themselves were driven to substitute lifestyle changes for medication.⁴⁵

If the reductions in BMI and waist circumference are not driven by diabetes medication, they should be due to changes in eating patterns or physical activity. Since we find that high risk classification does not lead to changes in exercise (Panel A.1, Column 8) and alcohol consumption (Panel A.1, Column 9), we speculate that the change is driven by changes in diet. Assuming that the weight loss for individuals classified as high risk for diabetes is fully due to changes in diet, a decrease in BMI of 0.16 kg/m² translates to an overall caloric reduction 6.8 calories per day.⁴⁶

In the long-run, the observed short-run impacts described above are attenuated as shown in Figure 3 and Panel A.2 of Table 3.⁴⁷ Specifically, we still observe decreases in BMI and waist circumference, but they are smaller and no longer statistically significant in the long-run. However, we find suggestive evidence that the decrease of BMI and waist circumference as well

line drug, impacts of diabetes treatment on weight are mixed (Phung et al., 2010; Gross et al., 2011). Second, short-run reductions in BMI and waist circumference are driven by people without diabetes medication.

⁴⁵Although lifestyle modifications are clinically recommended to all people who are at risk of or diagnosed of diabetes, we do not find behavioral (or weight) changes among those who are taking medication. Analogous to the explanation for individuals who did not take medication, individuals who are less likely to change their behavior may be more likely to take medication (driven either by the doctor or the patient). In addition, individuals may not change their behavior if they rely on the medication treatment.

⁴⁶This is about a 0.3% decrease of overall calories per day from the baseline of 2,012 calories (2,290 for men and 1,734 for women), calculated using medical estimates of the caloric intake required to maintain weight, and using the average height and weight in our sample (estimates are drawn from the Baylor College of Medicine: <https://www.bcm.edu/cnrc-apps/caloriesneed.cfm>).

⁴⁷It is worth noting that the long-run estimates could be affected by the screening results in round 2 if information in the baseline influences risk classification in the second round of screening as discussed by Cellini et al. (2010). However, as discussed above, we find only minimal impacts of baseline information on future diabetes risk classifications in round 2 screening. This implies that our long-run estimates are solely driven by the treatment in the baseline.

as early medical treatment in the short-run may lead to long-term health improvement. For example, for those classified as high risk at baseline, the long-run blood sugar level decreases by 2.6 mg/dL (about a 2.3% change from the mean) although statistically insignificant (Panel A.2, Column 5 of Table 3), and the probability of being classified as “high risk” in round 3 screening decreases by 9.2 percentage points as shown in Panel A.2, Column 12 of Table 3. This result is consistent with findings that lifestyle modification or diabetes treatment decreases diabetes incidence (Diabetes Prevention Program Research Group, 2015).

Around the 100 cutoff, we do not find a meaningful impact on all outcomes. Even though the estimated coefficients on BMI and waist circumference in our main specification are statistically significant, these appear to be spurious because they are not statistically significant in other specifications as shown in Panels (f) and (g) of Figures A.8 and A.9.⁴⁸

5.2 Results from obesity screening

In this subsection, we examine the responses to information on obesity risk obtained from obesity screening by comparing individuals just below and just above the BMI cutoffs of 23 and 25. As previously explained, 25 is the high risk and Normal B threshold for individuals with abdominal obesity, and 23 is the medium risk threshold for individuals without abdominal obesity (Table 1a). Figures 4 and 5 respectively present graphical illustrations of short-run and long-run outcomes against baseline BMI, and Table 4 reports the corresponding discontinuity estimates at the cutoffs.

We first investigate the BMI threshold of 25 among people with abdominal obesity. We find that information on obesity risk has no or little impacts in general. For example, we do not observe changes in outpatient visits, medication, waist circumference, health behaviors, other biomarkers, and future obesity risk classifications (Panel A of Table 4 and Figures 4 and 5). It seems that there is a decrease of BMI both in the short- and long-term, but these results are small in magnitude and not robust to other specifications

⁴⁸Also, even though those just below the 100 cutoff are more likely to be classified to “medium risk” for diabetes in future screenings (both round 2 and round 3) by about 2 to 3 percentage points (Panels B.1 and B.2, Columns 11 of Table 3), we do not find corresponding results in blood sugar level as shown in Panels B.1 and B.2, Column 5 of Table 3.

(Panel (d) of Appendix Figures A.10 and A.11).⁴⁹

Next, we study the 23 cutoff among people without abdominal obesity. As shown in Panel B.1 of Table 4, the additional information from risk classification at the 23 cutoff increases waist circumference for medium risk individuals just above the BMI cutoff 23 by 0.21 cm (a 0.27% change) as shown in Column 5. In addition, those just above the cutoff are more likely to be classified as medium risk (rather than low risk) by five and three percentage points in round 2 and 3, respectively (Panel B, Columns 10-12 of Table 4).⁵⁰ If the increase in waist circumference and probability of medium risk classification is true, our finding is consistent with Almond et al. (2016) who study the impact of overweight classification and find a small but statistically significant increase in future weight among teenage girls in New York City.⁵¹ In addition, Hunger and Tomiyama (2014) show an association that being labeled “too fat” at age 10 years remained a significant predictor of obesity at age 19 years.

Some possible explanations for this unexpected but consistent finding is that individuals close to this threshold who receive a medium-risk classification are discouraged by the result and lose motivation to pursue healthy behaviors, those who received a low-risk classification reacted positively to continue maintaining reduced weight, or a combination of both these explanations. However, our results should be interpreted with caution. First, we do not find a similar negative impact around the 25 cutoff. Second, we do not find a corresponding change in BMI. Third, we do not find meaningful corresponding changes in health behaviors including exercise, drinking, smoking, obesity medication, and blood sugar around the 23 cutoff (Panel B of Table 4 and Figures 4 and 5).⁵² However, the consistency of this finding

⁴⁹Specifically, the estimated differences in BMI of 0.085 (short-run) and 0.114 (long-run) are small—these are 0.4% and 0.5% of the mean short-run and long-run BMI measured at the threshold, respectively.

⁵⁰Regression results are robust in other specifications as shown in Appendix Figure A.12.

⁵¹The BMI cutoff for determining overweight is defined as the 85th percentile for 1970s US children of the same age and sex in Almond et al. (2016), while the cutoff for determining medium risk is 23 kg/m² in our context.

⁵²Standard errors for these estimates are small enough to rule out small changes in the outcome variables. For example, we are able to rule out more than a 0.015 percentage point change ($=0.023 \times 1.96$) (or a 4% change at the mean) in the probability of engaging in basic exercise in the short-run. Similarly, for short-run estimates, we can rule out a change of 0.36 alcohol drinks per week, 0.19 cigarettes per week, and 0.58 mg/dL in blood sugar level. Long-run estimates also have similar magnitudes of precision.

across studies warrants future research into the reasons for these patterns.

5.3 Results from hyperlipidemia screening

We examine the cutoff 160 of LDL cholesterol to understand the responses to information on hyperlipidemia risk from the hyperlipidemia screening. LDL cholesterol 160 is the cutoff for dividing high risk and Hyperlipidemia Suspected vs. medium risk and Normal B individuals (Table 1a). The discontinuity estimates are reported in Table 5, and the corresponding graphical illustrations are presented in Figures 6 and 7.

As shown in Panel (a) of Figure 6 and Panel A.1, Column 1 of Table 5, we find an increase in the number of outpatient visits around the 160 cutoff by 0.17 days (a 18.5% increase). The increase in outpatient visits for hyperlipidemia may imply that at least some individuals classified as high risk underwent follow-up examinations to confirm disease on their own, not through the NHSP secondary examination.

However, the increase in outpatient visits is relatively small compared to the high risk classification for diabetes and did not translate into medical treatment. This could be because doctors or patients do not strictly follow treatment guidelines and focus only on lifestyle modifications when blood LDL cholesterol is just above the 160 cutoff. In addition, even though information is provided based solely on blood LDL cholesterol level, medication treatment is not necessarily combined with the 160 cutoff. For example, the 160 cutoff for treatment applies only for people with low risk.⁵³ Lastly, we do not find evidence on changes in health behaviors.

5.4 Discussion

In previous subsections, we described that we find evidence of weight loss around the high risk threshold for diabetes, while we find little to no differences around other risk classification thresholds. In this subsection, we discuss several possible explanations for why people change behaviors only at the blood sugar 126 cutoff.

⁵³The cutoff for treatment of high LDL cholesterol is determined by the number of other risk factors including smoking, hypertension, low HDL (≤ 40 mg/dL), family history of coronary artery diseases, and age (men over 45 and women over 55). The cutoffs are 160, 130, and 100 for those with 0-1 risk factors, 2+ risk factors, and coronary artery disease, respectively (Lorenzo et al., 2007; Korean Society of Lipidology and Atherosclerosis, 2009).

First, one of the most striking differences between the high risk classification for diabetes compared to the other classifications we study (including the medium risk classification for diabetes) is that those classified as high risk for diabetes receive further intervention in addition to the information on disease risk.⁵⁴ Those above the 126 cutoff were invited to undergo a secondary examination where they had an opportunity to talk to a physician about their risk factors and treatment plans. This also translated into an increase in diabetes medication use. The fact that we don't find behavioral changes around other cutoffs suggests that information is not a strong enough tool in itself to encourage people to engage in healthy behaviors.

Second, it is possible that individuals who understand that they are just above the risk classification cutoffs from the first page of the screening report may not take the information on disease risk seriously. In other words, individuals who are just above that cutoff may understand that their actual risk is not different from that for those just below the cutoff. If it is true, we might expect this effect to be lower for those with lower levels of education who might be less capable of inferring from the report how close they are to the boundary of a given risk classification. To test this, we conduct a subsample analysis by income quintiles (a proxy for education level) but do not find any patterns of behavioral responses across different levels of household income (Appendix Figures A.16 to 18).

Third, one may reason that people consider diabetes a more serious disease than obesity and hyperlipidemia.⁵⁵ The limited impact in information on obesity and hyperlipidemia risk may suggest that patients do not perceive obesity and hyperlipidemia to be a long-run threat to their health or quality

⁵⁴While we find an increase in outpatient visits among people just above the cutoff 160 of LDL cholesterol, the magnitude of this increase is small in magnitude (0.17 more outpatient visits) relative to that for the cutoff 126 of blood sugar (approximately 38 percentage point increase in secondary examination and 0.64 more outpatient visit), which may explain the lack of subsequent changes in behaviors around the LDL cholesterol cutoff.

⁵⁵It seems plausible that people respond more to diseases where perceived threat is greater. For example, Oster et al. (2013) explains a large change in behaviors for those who are confirmed of Huntington disease (HD) could be due to the fact that people with HD have a relatively short life expectancy and HD has no cure.

of life.^{56,57} We do not think it is likely because we do not find behavioral changes around the medium risk threshold for diabetes, where we would expect to find an observable impact if individuals take into account diabetes as a serious disease. Moreover, we do find a small but significant increase in outpatient visits around the cutoff 160 of LDL cholesterol, implying individuals are taking the hyperlipidemia risk seriously.

In summary, our results are consistent with the explanation that secondary examination and subsequent medical treatment in addition to information on disease risk is a vital complement to health screening. These results shed light on the mixed findings in previous literature that moderate behavioral changes are observed for diabetes (Slade, 2012; Oster, 2015) and hypertension (Zhao et al., 2013) diagnosis which is often combined with further medical intervention, while no effects are found for overweight classification (Almond et al., 2016), which is unlikely to be combined with further medical intervention. While there could be other explanations that we have not accounted for, this suggests that a next step for future research would be to understand the extent to which different interventions complement information.

6 Conclusion

Using administrative data from the population based health screening program in Korea that includes more than 350,000 baseline screening takers and observations over four years after screening, this paper provides comprehensive evidence on short- and long-term impacts of information obtained from health screening on a rich set of biomarkers, behavior measures, and health outcomes. Specifically, we apply a regression discontinuity design that takes advantage of the fact that risk classifications for diabetes, obesity, and hyperlipidemia vary discontinuously at various thresholds of fasting blood sugar, BMI, and LDL cholesterol, respectively.

⁵⁶In fact the World Health Organization (2009) identifies that overweight and obesity as the third leading cause of mortality and burden of disease in high income countries, while high blood sugar and high cholesterol are identified as the fifth and sixth leading causes, respectively.

⁵⁷It is also possible that the fact that only those who are high risk for diabetes are offered the free secondary examination makes individuals to understand that diabetes is a serious disease.

We find that those who are informed of high risk for diabetes are more likely to take diabetes medications and more likely to experience significant reductions in BMI and waist circumference at least in the short-run. On the other hand, we find little to no differences around the medium risk threshold for diabetes, as well as risk classification thresholds of obesity and hyperlipidemia with respect to individual behaviors, the demand for health care, and future health outcomes. These results are consistent with the fact that the NHSP provides a secondary examination only to those classified as high risk for diabetes, while at other thresholds information is not combined with prompting for a secondary examination and individuals only receive screening results by mail. Furthermore, even though such information promotes outpatient visits (i.e., for those who exceed the 160 LDL cholesterol cutoff), the impact is relatively small and it does not translate to increased medical treatment, nor to behavior modifications.

There are some limitations to this study. First, while we have a rich source of data covering several years, including as soon as one year after screening, it is possible that our estimates of null impacts could be missing very short-term behavior changes that happen within as soon as the first few months after receipt of screening results. However, to the extent that long-term behavior changes are important for the lifestyle diseases that are relevant to our study, the time period we study may be more relevant than the period immediately after screening. Second, an important component of the lifestyle diseases relevant to our study is food intake, which we do not observe. However, our administrative and self-reported data provide strong and relevant correlates to food consumption (e.g., BMI) that combined with our other variables like exercise, help us to gain a thorough picture of relevant behavior changes.

Although the findings of this study specifically reflected the behavioral responses and health outcomes to the NHSP in Korea, this analysis still provides a number of implications for other health and social programs that provide information on disease risk. Our results suggest that information itself might not be sufficient to lead to behavioral changes, and that further interventions in addition to the information may increase the marginal benefits of screening.

References

- Almond, Douglas, Ajin Lee, and Amy Ellen Schwartz**, “Impacts of classifying New York City students as overweight,” *Proceedings of the National Academy of Sciences*, 2016, *113* (13), 3488–3491.
- , **Joseph J. Doyle, Amanda E. Kowalski, and Heidi Williams**, “Estimating Marginal Returns to Medical Care: Evidence from At-risk Newborns,” *The Quarterly Journal of Economics*, 2010, *125* (2), 591–634.
- American Diabetes Association**, “Standards of Medical Care in Diabetes,” *Diabetes Care*, 2015, *38* (Supplement 1).
- Barreca, Alan I., Jason M. Lindo, and Glen R. Waddell**, “Heaping-induced Bias in Regression Discontinuity Designs,” *Economic Inquiry*, 2016, *54* (1), 268–293.
- , **Melanie Guldi, Jason M. Lindo, and Glen R. Waddell**, “Saving Babies? Revisiting the Effect of Very Low Birth Weight Classification,” *The Quarterly Journal of Economics*, 2011, *126* (4), 2117.
- Becker, Gary S. and Kevin M. Murphy**, “A Theory of Rational Addiction,” *Journal of Political Economy*, 1988, *96* (4), 675–700.
- Blanchard, Chris M., Maxine M. Denniston, Frank Baker, Stuart R. Ainsworth, Kerry S. Courneya, Danette M. Hann, Dean H. Gesme, Douglas Reding, Thomas Flynn, and John S. Kennedy**, “Do Adults Change Their Lifestyle Behaviors After a Cancer Diagnosis?,” *American Journal of Health Behavior*, 2003, *27* (3), 246–256.
- Calonico, Sebastian, Matias D. Cattaneo, and Rocio Titiunik**, “Robust Nonparametric Confidence Intervals for Regression-Discontinuity Designs,” *Econometrica*, 2014, *82* (6), 2295–2326.
- Cawley, John and Christopher J. Ruhm**, “Chapter Three - The Economics of Risky Health Behaviors,” in Thomas G. McGuire Mark V. Pauly and Pedro P. Barros, eds., *Handbook of Health Economics*, Vol. 2 of *Handbook of Health Economics*, Elsevier, 2011, pp. 95–199.

- Cellini, Stephanie Riegg, Fernando Ferreira, and Jesse Rothstein**, “The Value of School Facility Investments: Evidence from a Dynamic Regression Discontinuity Design,” *The Quarterly Journal of Economics*, 2010, *125* (1), 215–261.
- Chung, Sukyung, Lenard I. Lesser, Diane S. Lauderdale, Nicole E. Johns, Latha P. Palaniappan, and Harold S. Luft**, “Medicare Annual Preventive Care Visits: Use Increased Among Fee-For-Service Patients, But Many Do Not Participate,” *Health Affairs*, 2015, *34* (1), 11–20.
- Dahlberg, Matz, Kevin Mani, Mattias Öhman, and Anders Wanhainen**, “Health Information and Well-Being: Evidence from an Asymptomatic Disease,” 2016. Working Paper Series No 2016:2, Department of Economics, Uppsala University.
- Dalton, A.R. and M. Soljak**, “The Nationwide Systematic Prevention of Cardiovascular Disease: the UK’s Health Check Programme,” *The Journal of Ambulatory Care Management*, 2012, *35* (3), 206–215.
- Darden, Michael**, “Smoking, Expectations, and Health: A Dynamic Stochastic Model of Lifetime Smoking Behavior,” *Journal of Political Economy*, Forthcoming.
- Deke, John and Lisa Dragoset**, “Statistical Power for Regression Discontinuity Designs in Education Empirical Estimates of Design Effects Relative to Randomized Controlled Trials,” 2012. Working Paper 8, Mathematica Policy Research.
- Deutekom, Marije, Fleur Vansenne, Kirsten McCaffery, Marie-Louise Essink-Bot, Karien Stronks, and Patrick M.M. Bossuyt**, “The effects of screening on health behaviour: a summary of the results of randomized controlled trials,” *Journal of Public Health*, 2011, *33* (1), 71–79.
- Diabetes Prevention Program Research Group**, “Long-Term Safety, Tolerability, and Weight Loss Associated With Metformin in the Diabetes Prevention Program Outcomes Study,” *Diabetes Care*, 2012, *35*, 731–737.
- , “Long-Term Effects of Lifestyle Intervention or Metformin on Diabetes Development and Microvascular Complications over 15-Year Follow-Up:

- the Diabetes Prevention Program Outcomes Study,” *Lancet Diabetes Endocrinol*, 2015, *3*, 866–875.
- Dunn, D.J. and D.H. Peters**, “Metformin. A Review of its Pharmacological Properties and Therapeutic Use in Non-Insulin-Dependent Diabetes Mellitus,” *Drugs*, 1995, *49* (5), 721–749.
- Fortenberry, J.D., E.J. Brizendine, B.P. Katz, and D.P. Orr**, “Post-treatment Sexual and Prevention Behaviours of Adolescents with Sexually Transmitted Infections,” *Sexually Transmitted Infections*, 2002, *78*, 365–368.
- Gross, J.L., C.K. Kramer, C.B. Leita, N. Hawkins, L.V. Viana, B.D. Schaan, L.C. Pinto, T.C. Rodrigues, and M.J. Azevedo**, “Effect of Antihyperglycemic Agents Added to Metformin and a Sulfonylurea on Glycemic Control and Weight Gain in Type 2 Diabetes: A Network Meta-analysis,” *Annals of Internal Medicine*, 2011, *154*, 672–679.
- Hackl, Franz, Martin Halla, Michael Hummer, and Gerald J. Pruckner**, “The Effectiveness of Health Screening,” *Health Economics*, 2015, *24* (8), 913–935.
- Hunger, J.M. and A. Tomiyama**, “Weight Labeling and Obesity: A Longitudinal Study of Girls Aged 10 to 19 Years,” *JAMA Pediatrics*, 2014, *168* (6), 579–580.
- Iizuka, Toshiaki, Katsuhiko Nishiyama, Brian Chen, and Karen Eggleston**, “Is Preventive Care Worth the Cost? Evidence from Mandatory Checkups in Japan,” 2017. Working paper.
- Imbens, Guido and Karthik Kalyanaraman**, “Optimal Bandwidth Choice for the Regression Discontinuity Estimator,” *The Review of Economic Studies*, 2012, *79* (3), 933–959.
- Keenan, P.S.**, “Smoking and Weight Change After New Health Diagnoses in Older Adults,” *Archives of Internal Medicine*, 2009, *169* (3), 237–242.
- Kersaw, T.S., J.R. Ickovics, J.B. Lewis, L.M. Niccolai, S. Milan, and K.A. Ethier**, “Sexual Risk Following a Sexually Transmitted Disease Diagnosis: The More Things Change the More They Stay the Same,” *Journal of Behavioral Medicine*, 2004, *27* (5), 445–461.

- Kim, Hyuncheol Bryant and Sun-mi Lee**, “When Public Health Intervention Is Not Successful: Cost-sharing, Crowd-out, and Selection in Korea’s National Cancer Screening Program,” *Journal of Health Economics*, 2017, 53, 100 – 116.
- Kohro, T., Y. Furui, N. Mitsutake, R. Fujii, H. Morita, S. Oku, K. Ohe, and R. Nagai**, “The Japanese National Health Screening and Intervention Program Aimed at Preventing Worsening of the Metabolic Syndrome,” *International Heart Journal*, 2008, 49 (2), 196–203.
- Korean Diabetes Association**, *2007 Treatment Guidelines for Diabetes*, Seoul: Korean Diabetes Association, 2007. In Korean.
- , *2015 Treatment Guidelines for Diabetes*, Seoul: Korean Diabetes Association, 2015. In Korean.
- Korean Endocrine Society and Korean Society for the Study of Obesity**, “Management of Obesity, 2010 Recommendation,” *Endocrinology and Metabolism*, 2010, 25 (4), 301–304. In Korean.
- Korean Society of Lipidology and Atherosclerosis**, *Treatment Guidelines for Dyslipidemia*, Seoul: Korean Society of Lipidology and Atherosclerosis, 2009. In Korean.
- Lee, David S.**, “Randomized experiments from non-random selection in U.S. House elections,” *Journal of Econometrics*, 2008, 142 (2), 675 – 697. The regression discontinuity design: Theory and applications.
- **and David Card**, “Regression Discontinuity Inference with Specification Error,” *Journal of Econometrics*, 2008, 142 (2), 655 – 674. The Regression Discontinuity Design: Theory and Applications.
- **and Thomas Lemieux**, “Regression Discontinuity Designs in Economics,” *Journal of Economic Literature*, 2010, 48 (2), 281–355.
- Lee, Juneyoung, Ji Sung Lee, Sook-Hee Park, Soon Ae Shin, and KeeWhan Kim**, “Cohort Profile: The National Health Insurance Service-National Sample Cohort (NHIS-NSC), South Korea,” *International Journal of Epidemiology*, 2015, pp. 1–8.

- Lee, W.-C. and S.-Y. Lee**, “National Health Screening Program of Korea,” *Journal of the Korean Medical Association*, 2010, *53* (5), 363–370.
- Lorenzo, C., K. Williams, K.J. Hunt, and S.M. Haffner**, “The National Cholesterol Education Program: Adult Treatment Panel III, International Diabetes Federation, and World Health Organization Definitions of the Metabolic Syndrome as Predictors of Incident Cardiovascular Disease and Diabetes,” *Diabetes Care*, 2007, *30* (1), 8–13.
- McCrary, Justin**, “Manipulation of the Running Variable in the Regression Discontinuity Design: A Density Test,” *Journal of Econometrics*, 2008, *142* (2), 698–714.
- National Health Insurance Service**, *2009 National Health Screening Statistical Yearbook*, Seoul: National Health Insurance Service, 2009.
- , *2010 National Health Screening Statistical Yearbook*, Seoul: National Health Insurance Service, 2010.
- Newsom, J.T., N. Huguet, M.J. McCarthy, P. Ramage-Morin, M.S. Kaplan, J. Bernier, B.H. McFarland, and J. Oderkirk**, “Health Behavior Change Following Chronic Illness in Middle and Later Life,” *The Journal of Gerontology, Series B: Psychological Sciences and Social Sciences*, 2011, *67* (3), 279–288.
- Oster, Emily**, “Diabetes and Diet: Behavioral Response and the Value of Health,” 2015. NBER Working Paper No. 21600, National Bureau of Economic Research.
- , **Ira Shoulson, and E. Ray Dorsey**, “Optimal Expectations and Limited Medical Testing: Evidence from Huntington Disease,” *American Economic Review*, April 2013, *103* (2), 804–30.
- Patterson, R.E., M.L. Newhouser, M.M. Hedderson, S.M. Schwartz, L.J. Standish, and D.J. Bowen**, “Changes in Diet, Physical Activity, and Supplement Use Among Adults Diagnosed with Cancer,” *Journal of the American Dietetic Association*, 2003, *103* (3), 323–328.
- Phung, O.J., J.M. Scholle, M. Talwar, and C.I. Coleman**, “Effect of Noninsulin Antidiabetic Drugs Added to Metformin Therapy on Glycemic

- Control, Weight Gain, and Hypoglycemia in Type 2 Diabetes,” *Journal of the American Medical Association*, 2010, *303* (14), 1410–1418.
- Prendergast, B.D.**, “Glyburide and Glipizide, Second-Generation Oral Sulfonylurea Hypoglycemic Agents,” *Clin Pharm*, 1984, *3* (5), 473–485.
- Prina, Silvia and Heather Royer**, “The Importance of Parental Knowledge: Evidence from Weight Report Cards in Mexico,” *Journal of Health Economics*, 2014, *37*, 232 – 247.
- Slade, Alexander N.**, “Health investment decisions in response to diabetes information in older Americans,” *Journal of Health Economics*, 2012, *31* (3), 502 – 520.
- Suranovic, Steven M., Robert S. Goldfarb, and Thomas C. Leonard**, “An Economic Theory of Cigarette Addiction,” *Journal of Health Economics*, 1999, *18* (1), 1–29.
- Thaler, Richard H. and H. M. Shefrin**, “An Economic Theory of Self-Control,” *Journal of Political Economy*, 1981, *89* (2), 392–406.
- Thornton, Rebecca L.**, “The Demand for, and Impact of, Learning HIV Status,” *American Economic Review*, December 2008, *98* (5), 1829–63.
- World Health Organization**, “Obesity: Preventing and Managing the Global Epidemic,” 2000. WHO Technical Report Series 894.
- , *Global Health Risks: Mortality and Burden of Disease Attributable to Selected Major Risks*, Geneva: WHO Press, 2009.
- Wu, R.R., R.A. Myers, E.R. Hauser, A. Vorderstrasse, A. Cho, G.S. Ginsburg, and L.A. Orlando**, “Impact of Genetic Testing and Family Health History Based Risk Counseling on Behavior Change and Cognitive Precursors for Type 2 Diabetes,” *Journal of Genetic Counseling*, 2016, pp. 1–8.
- Zhao, Meng, Yoshifumi Konishi, and Paul Glewwe**, “Does information on health status lead to a healthier lifestyle? Evidence from China on the effect of hypertension diagnosis on food consumption,” *Journal of Health Economics*, 2013, *32* (2), 367 – 385.

Figures and Tables

Figure 1a: NHSP report card, page 1

Results of Regular Medical Checkup (1st)

Name	XXXXXX	Resident registration number	XXXXXX	
Date of examination	July 14, 2009	Health checkup institution	<input checked="" type="checkbox"/> Visit, <input type="checkbox"/> On-site checkup	

Test type	Medical history	Diagnosis	N/A		External wound or Sequela	N/A
		Medication	N/A			
	Lifestyle				General status	Good
Section	Targeting diseases	Examination item	Examination result	Reference range		
				Normal A (Satisfactory)	Normal B (Warning) (need preventive care, but no problem in health)	
Measuring examination	Obesity	Height	178 cm			
		Weight	75 kg			
		Waist circumference	84 cm	Male: under 90 Female: under 85	-	
		Body Mass Index (BMI)	23.6 kg/m ²	18.5-24.9	-	
	Optic acuity	Eyesight (left/right)	1.2 / 1.2			
	Auditory acuity	Hearing ability (left/right)	Normal / Normal			
	Hypertension	Blood pressure (Systolic/ diastolic)	114 / 65 mmHg	under 120 / under 80	120-139 / 80-89	
Urine test	Kidney disease	Albuminuria	Negative (-)	Negative	Weak benign ±	
Blood test	Anemia, etc.	Hemoglobin	14.5 g/dL	Male: 13-16.5 Female: 12-15.5	Male: 12-12.9 / 16.6-17.5 Female: 10-11.9 / 15.6-16.5	
	Diabetes	Fasting blood sugar	120 mg/dL	under 100	100-125	
	Hyperlipidemia, Hypertension, Arteriosclerosis	Total Cholesterol	152 mg/dL	under 200	200-239	
		HDL-Cholesterol	52 mg/dL	60 and over	40-59	
		Triglyceride	32 mg/dL	under 100-150	150-199	
	LDL-Cholesterol	94 mg/dL	under 100	100-159		
		Chronic kidney disease	Creatinine	0.8 mg/dL	1.5 and below	-
	Liver disease	Glomerular filtration rate	114 mL/min	60 and over	-	
		AST (SGOT)	24 U/L	40 and below	41-50	
		ALT (SGPT)	20 U/L	35 and below	36-45	
Gamma-GTP	30 U/L	Male: 11-63 Female: 8-35	Male: 64-77 Female: 36-45			
	Radio-exam	TB, chest disease	Chest radiology examination	Normal	Normal, unactive	-
Results and Recommendations						
Determination	Normal B: Manage diabetes			Date of determination	July 18, 2009	
				Examined Doctor	License number: XXXXXX Name: XXXXXX (signature)	

※ If you are determined as hypertension or diabetes suspected within, it is recommended to take the 2nd medical checkup within **30 days** (until next January) from the date of this notification.

※ Because not all diseases are screened in this medical checkup, please talk to your doctor if you have any suspicious health problems (e.g., excessive weight loss).

※ Reference range may differ by health checkup institutions.

We are notifying you of these medical examination results as above. Date July 21, 2009

Health checkup institution registration number (XXXXX)	Health checkup institution name (XXXXX)
---	--

Figure 1b: NHSP report card, page 2

Health Risk Evaluation Result

Name	XXXXX	Resident registration number	XXXXX
------	-------	------------------------------	-------

Health risk evaluation by disease

Below graph presents your health risk evaluation by disease, compared to a counterfactual person with no health risk.

Diseases	Degree of Risk			Your corresponding risk factors
	Low-risk	Medium-risk	High-risk	
Stroke (Cerebral Infarction)				smoking, insufficient exercise
Angina/ Myocardial Infarction				smoking, insufficient exercise, blood sugar
Vascular Dementia				smoking, insufficient exercise

The above evaluation is estimated by your screening results. The evaluation may change by genetic and environmental factors which are not included in the screening. If you had a corresponding disease previously, your actual degree of risk will be higher than the suggested evaluation above. If you have not answered some of the questions in the behavioral survey, the evaluation may differ from your actual degree of risk.

Knowing your health risk factors

	Current Status	Targeting Status	Health traffic light		
			Low-risk	Medium-risk	High-risk
Obesity (Weight/ Waist circumference)	Obese/ Abdominal obese (75kg/ 84cm)	Normal weight/ waist circum. (53-66kg/ 90cm)			
Alcohol consumption	Healthy drinking	Less than 2 glasses per day			
Smoking	Smoker	Quit smoking			
Exercise	Insufficient	Sufficient exercise			
Blood pressure (mmHg)	114/65	Less than 120/80			
Blood sugar level (mg/dL)	120	Less than 100			
LDL cholesterol (mmHg)	94	Less than 130 (Less than 100 if diabetes)			

Above evaluation presents both your current status and the targeting status based on your behavioral survey and screening results.

If you received medium-risk or high-risk appraisal, much effort is needed to improve your status.

Controlling your health risk factors

Your highest risk factor is smoking.

If you quit smoking, you can reduce the risk of stroke, angina/ myocardial infarction, and lung cancer.

cardio-cerebrovascular diseases

※ Larger area indicates more crucial risk factors

Please make an effort to reduce your degree of risk (quit smoking)

It is recommended to talk to your doctor about your risk factors. Professional counseling with a doctor can effectively improve your risk factors.

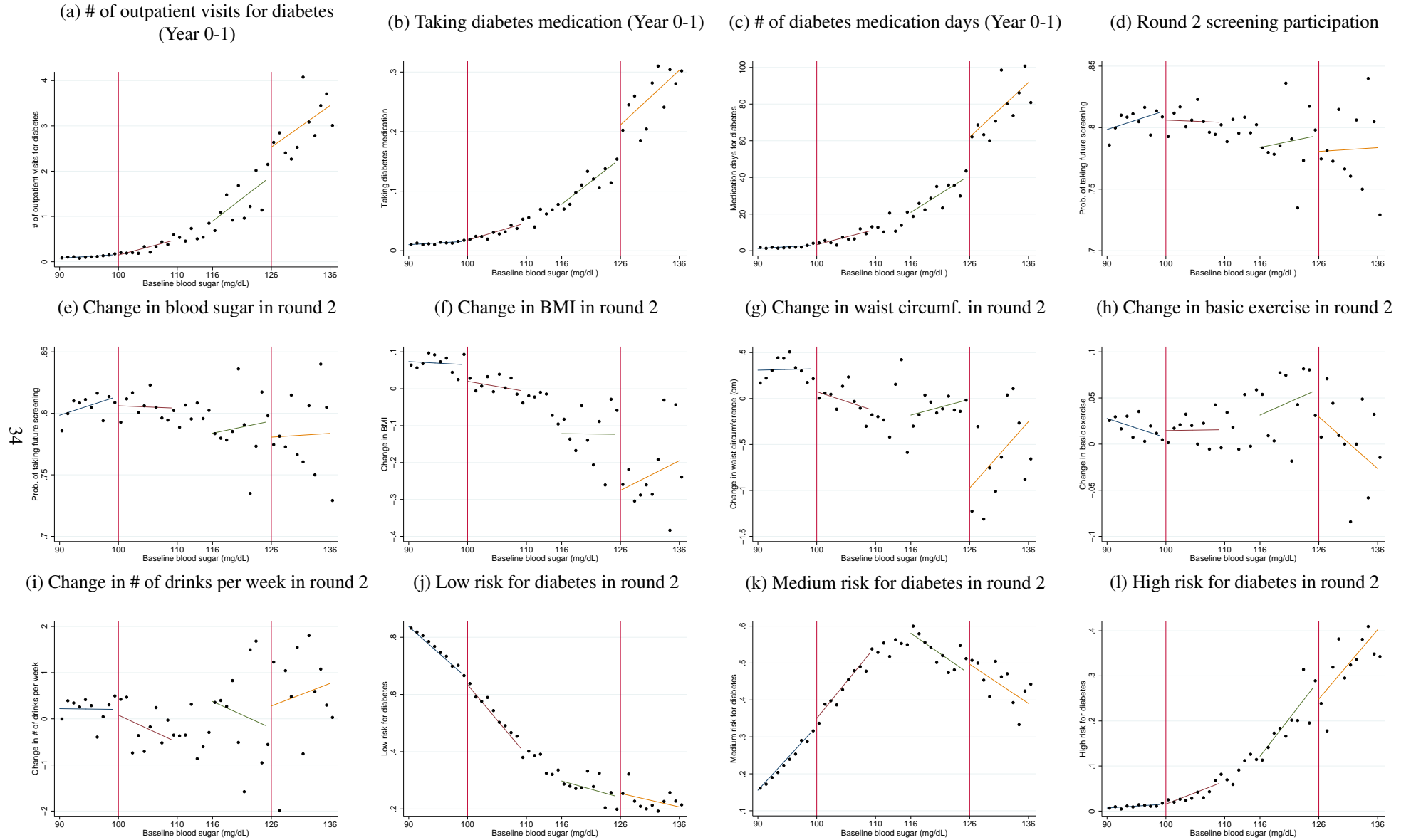
Tests for cognitive impairments (70 and 74 years old only)

- ☐ Normal (Score: 0~3)
☐ Need additional tests and counseling in the 2nd medical checkup (Score: 4~10)

※ A health risk evaluation is carried out to induce improved behavior toward health from subjects to reduce health risks by predicting possible diseases they may have and their future health status through the analysis of the subjects' health risk factors based on the questionnaire completed by the subjects and their checkup result

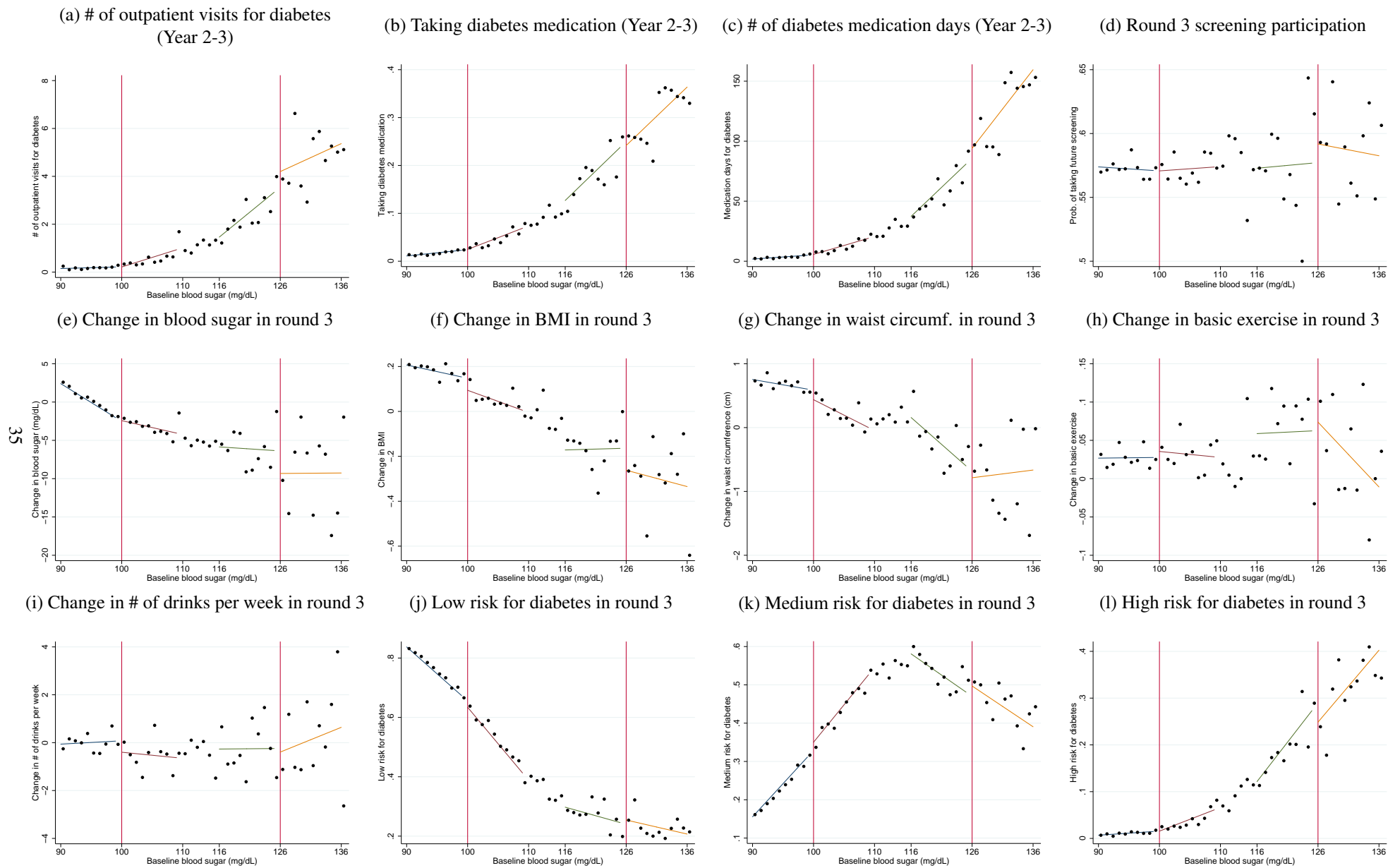
Notes: The report card is based on the 2009 standards. In the 2010 revision, BMI between "25.0-29.9" is classified as Normal B; and Normal A range for LDL cholesterol is "under 130" and Normal B range is "130-159."

Figure 2: Short-run impact of diabetes screening



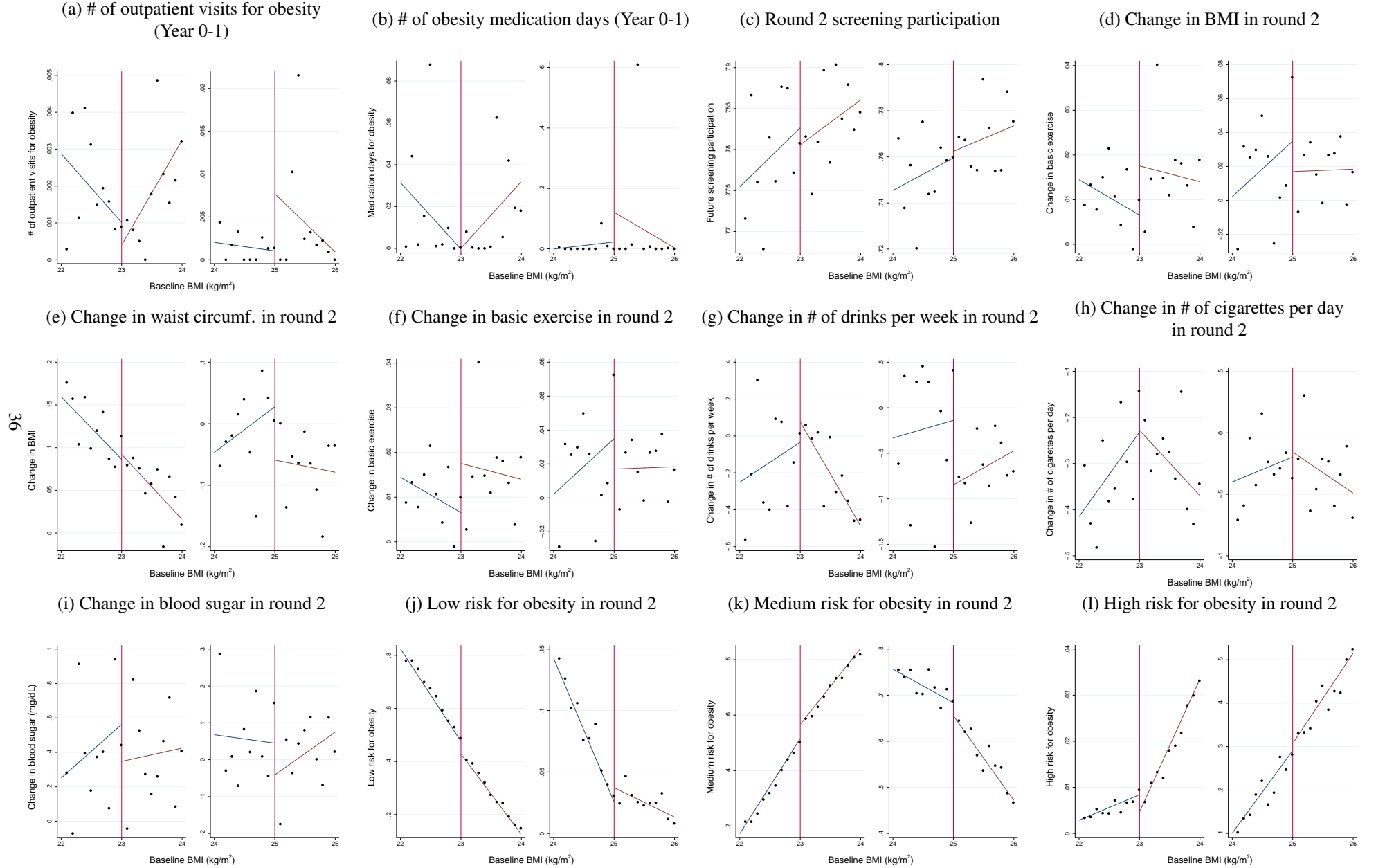
Notes: The running variable is baseline fasting blood sugar. The scatter plot indicates the mean of the dependent variable within 1 point bins. Around two cutoffs, 100 and 126, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 10 mg/dL. These are estimated separately on each side of the cutoff.

Figure 3: Long-run impact of diabetes screening



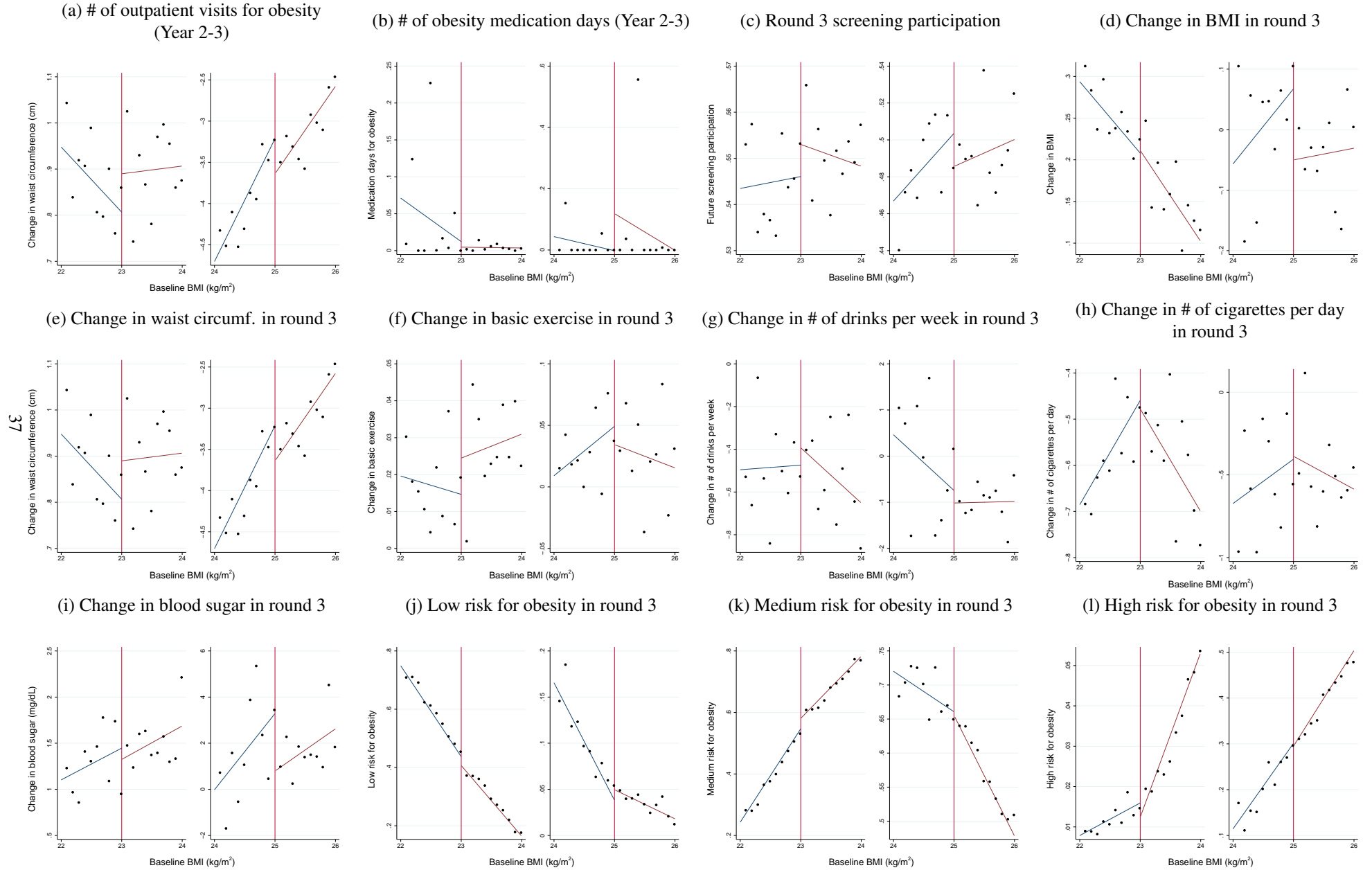
Notes: The running variable is fasting blood sugar. The scatter plot indicates the mean of the dependent variable within 1 point bins. Around two cutoffs, 100 and 126, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 10 mg/dL. These are estimated separately on each side of the cutoff.

Figure 4: Short-run impact of obesity screening



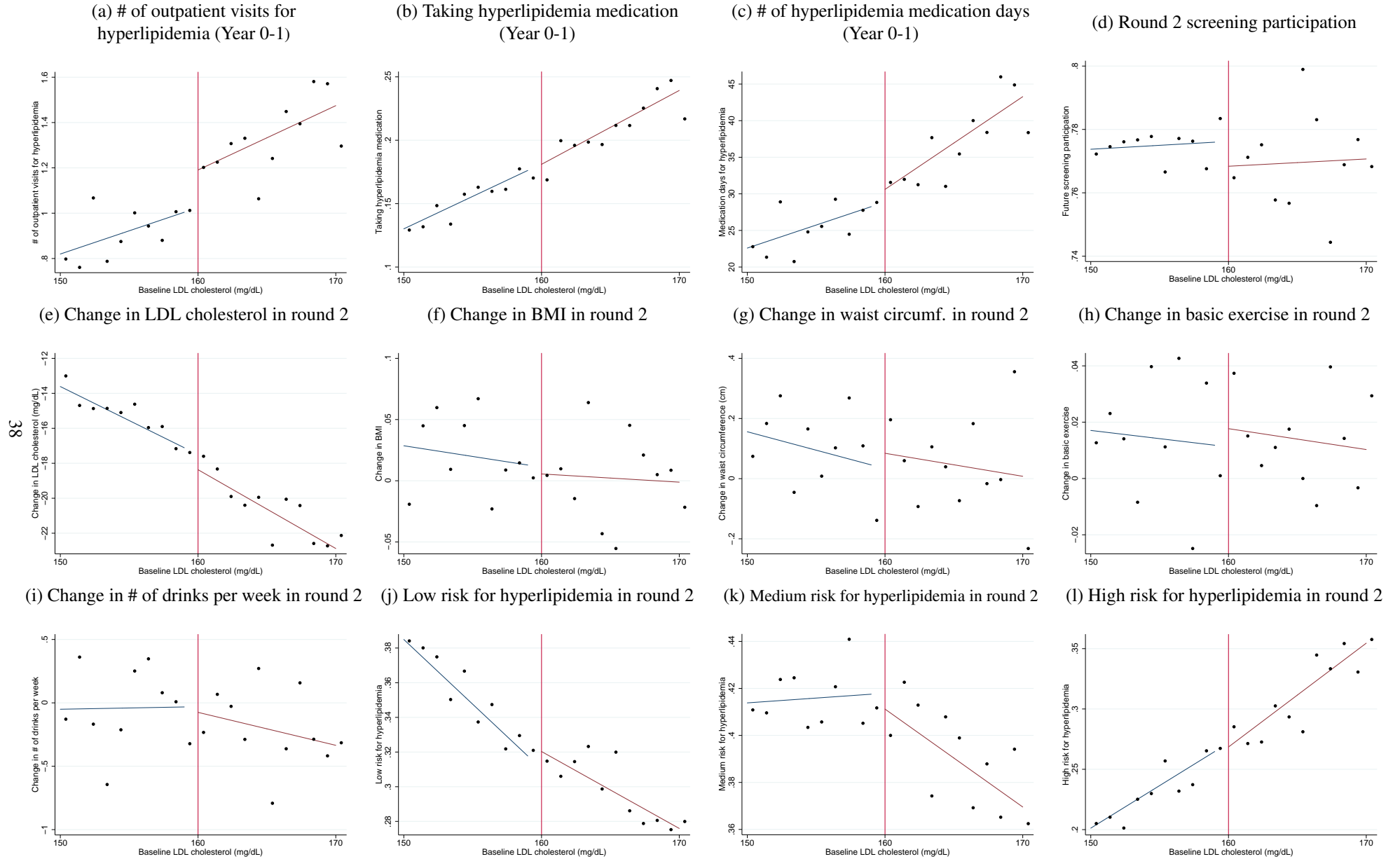
Notes: The running variable is baseline BMI. The scatter plot indicates the mean of the dependent variable within 0.05 point bins. Around two cutoffs, 23 and 25, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 1kg/m^2 . For each panel, the left figure with cutoff 23 is based on the sample without abdominal obesity and the right figure with cutoff 25 is based on the sample with abdominal obesity.

Figure 5: Long-run impact of obesity screening



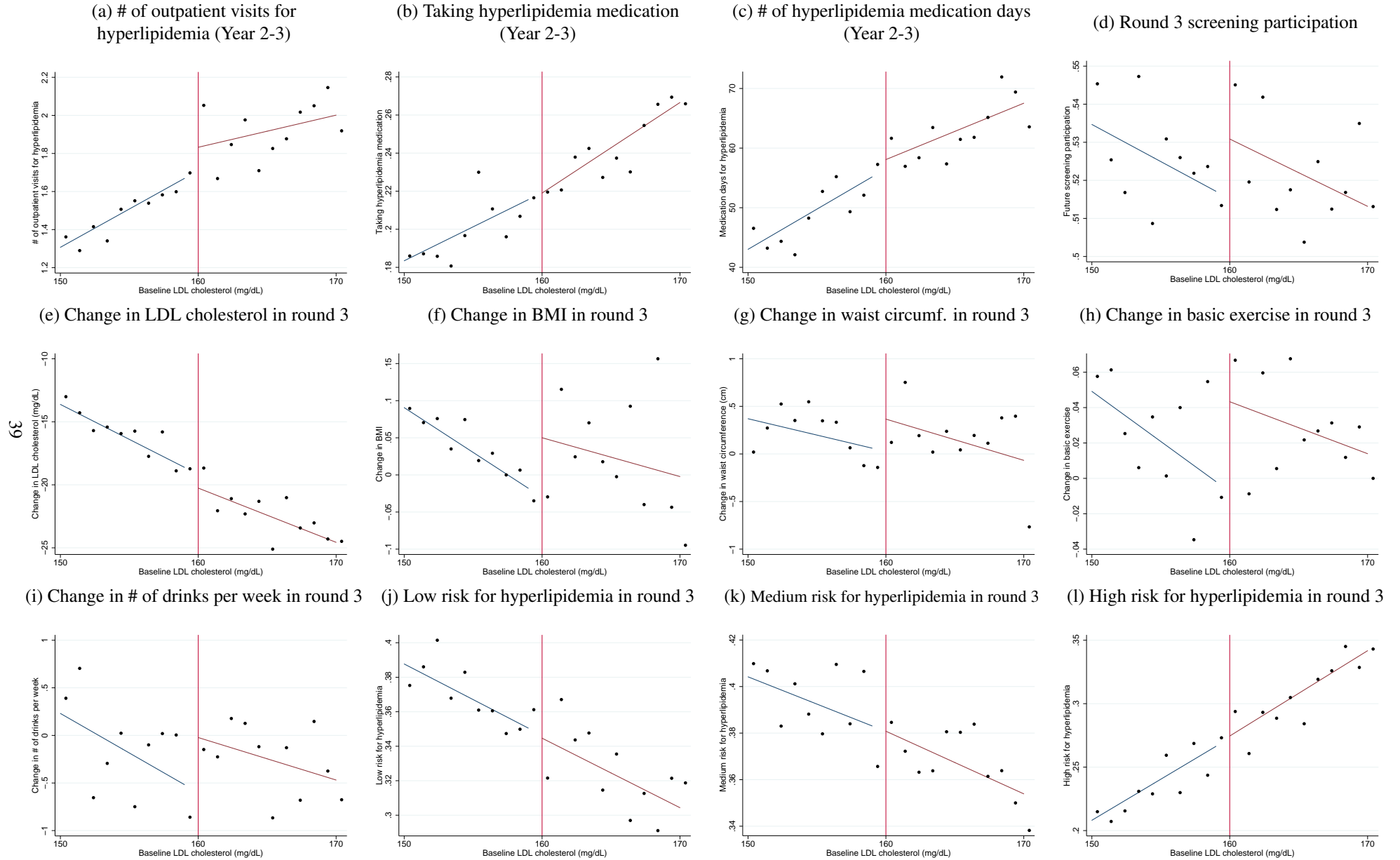
Notes: The running variable is baseline BMI. The scatter plot indicates the mean of the dependent variable within 0.05 point bins. Around two cutoffs, 23 and 25, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 1kg/m^2 . For each panel, the left figure with cutoff 23 is based on the sample without abdominal obesity and the right figure with cutoff 25 is based on the sample with abdominal obesity.

Figure 6: Short-run impact of hyperlipidemia screening



Notes: The running variable is baseline LDL cholesterol. The scatter plot indicates the mean of the dependent variable within 1 point bins. Around cutoff 160, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 10 mg/dL.

Figure 7: Long-run impact of hyperlipidemia screening



Notes: The running variable is baseline LDL cholesterol. The scatter plot indicates the mean of the dependent variable within 1 point bins. Around cutoff 160, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 10 mg/dL.

Table 1a: Study samples and treatments around the cutoff

Running variables	Samples	Cutoffs	Treatments					
					Treatment group (at or just above cutoff)		Comparison group (just below cutoff)	
Fasting blood	No previous diagnosis	126	Information	1st page	Diabetes suspected		Normal B	
				2nd page	High risk	■	Medium risk	■
			Secondary examination		Yes		No	
		100	Information	1st page	Normal B		Normal A	
				2nd page	Medium risk	■	Low risk	■
			Secondary examination		No		No	
BMI (kg/m ²)	Abdominal obesity	25	Information	1st page	Normal B		Normal A	
				2nd page	High risk	■	Medium risk	■
			Secondary examination		No		No	
	No abdominal obesity	23	Information	1st page	Normal A		Normal A	
				2nd page	Medium risk	■	Low risk	■
			Secondary examination		No		No	
LDL cholesterol (mg/dL)	No previous hyperlipidemia diagnosis and no diabetes	160	Information	1st page	Hyperlipidemia suspected		Normal B	
				2nd page	High risk	■	Medium risk	■
			Secondary examination		No		No	

Notes:

1. “Normal A,” “Normal B,” and “Disease suspected” refer to the results reported in the first page of the screening report. “Low risk,” “Medium risk,” and “High risk” refer to the risk classifications used in health traffic lights in the second page.
2. In the case of fasting blood sugar, the analysis sample is further restricted to those who took baseline screening in general hospitals (excluding those took screening in private clinics and public health centers) in order to avoid potential manipulation around the threshold. See footnote 43 for further details.
3. In the case of LDL cholesterol, the analysis sample is restricted to those with triglycerides less than or equal to 400 mg/dL and those without diabetes because LDL cholesterol is not reported in the screening results if the triglycerides are abnormally high (i.e. > 400 mg/dL) and cutoffs for risk classification are different for those with diabetes (Table 1b).
4. “Abdominal obesity” is defined as waist circumference being 85 cm or above for females and 90 cm or above for males.
5. “No diabetes” is defined as having no previous diabetes diagnosis or fasting blood sugar level greater or equal to 126 in the baseline screening result.

Table 1b: Risk classification rules of the health traffic lights

		■ Low risk	■ Medium risk	■ High risk
BMI (kg/m ²)	No abdominal obesity	18.5-22.9	23.0-29.9 or <18.5	≥30.0
	Abdominal obesity		<25.0	≥25.0
Fasting blood sugar (mg/dL)	Taking medication		<130	≥130
	No medication	<100	100-125	≥126
LDL cholesterol (mg/dL)	Taking medication		<130	≥130
			if diabetes, <100	if diabetes, ≥100
	No medication	<130	130-159	≥160
		if diabetes, <100		if diabetes, ≥100

Notes: Abdominal obesity is defined as waist circumference being 85 cm or above for females and 90 cm or above for males. Risk classification for LDL cholesterol varies by one’s diabetes status (recorded as diabetes if previously diagnosed as diabetes in the survey or if fasting blood sugar level is greater or equal to 126 in the screening result). Risk classifications for blood sugar and LDL cholesterol vary by self-reported medication information from survey (self-reported medication information is not available in our data).

Table 2: Baseline summary statistics

Variables (N=352,896)	Mean	Std. Dev.
Male	0.51	0.50
Age group		
Below 50	0.54	0.50
50 and older	0.46	0.50
Insurance type		
Self-employed insurance	0.23	0.42
Employee insurance	0.76	0.42
Medical care assistance	0.003	0.06
BMI (kg/m ²)	23.71	3.24
Height (cm)	163.29	9.23
Weight (kg)	63.46	11.58
Waist circumference (cm)	80.21	9.17
Blood sugar (mg/dL)	97.79	24.18
LDL Cholesterol (mg/dL)	114.19	39.01
Basic exercise	0.36	0.48
Number of drinks per week	6.90	13.85
Number of cigarettes per day	3.80	7.74
Self-reported diabetes diagnosis	0.09	0.28
Self-reported hyperlipidemia diagnosis	0.05	0.22
Previous year diabetes medication days	13.44	64.74
Previous year obesity medication days	0.01	1.41
Previous year hyperlipidemia medication days	11.06	52.60
Previous year total medical expenditure (USD)	521.83	1639.68
Round 2 screening participation	0.77	0.42
Round 3 screening participation	0.53	0.50

Notes: This table reports the baseline characteristics of the study sample. In the analysis, we restrict our sample as summarized in Table 1a. Baseline (or round 1) is defined as the screening in 2009 and 2010, taking the observation in 2009 if one took screening in both years. Round 2 screening is defined as the screening one or two years after the baseline, taking the earlier observation if an individual participated in both years. Similarly, round 3 screening is defined as the screening three or four years after the baseline, taking the earlier observation if an individual participated in both years.

Table 3: Impact of diabetes screening

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	# of outpatient visits for diabetes	Taking diabetes medication	# of diabetes medication days	Future screening participation	Change in blood sugar	Change in BMI	Change in waist circumference	Change in basic exercise	Change in # of drinks per week	Low risk in future screening	Medium risk in future screening	High risk in future screening
<i>Panel A. High risk cutoff at fasting blood sugar of 126</i>												
<i>Panel A.1 Short-run impact</i>												
RD estimate	0.635+ (0.325)	0.056** (0.018)	20.853** (4.587)	-0.012 (0.019)	-0.367 (2.197)	-0.157* (0.062)	-0.951** (0.247)	-0.027 (0.029)	0.403 (1.088)	0.007 (0.034)	0.030 (0.028)	-0.037 (0.042)
Mean of the Dep. Var. in levels at [116, 126)	1.27	0.11	28.46	0.79	112.12	24.68	83.89	0.40	9.72	0.28	0.54	0.18
Observations	4,642	4,642	4,642	4,642	3,648	3,643	3,644	3,543	3,555	3,648	3,648	3,648
<i>Panel A.2 Long-run impact</i>												
RD estimate	0.652 (0.720)	-0.009 (0.028)	8.252 (10.271)	0.020 (0.026)	-2.625 (3.214)	-0.110 (0.092)	-0.102 (0.301)	0.012 (0.047)	-0.200 (1.037)	-0.003 (0.040)	0.095* (0.033)	-0.092+ (0.051)
Mean of the Dep. Var. in levels at [116, 126)	2.24	0.17	55.45	0.57	113.69	24.59	83.67	0.41	9.72	0.30	0.47	0.23
Observations	4,601	4,601	4,601	4,601	2,662	2,658	2,656	2,562	2,585	2,662	2,662	2,662
<i>Panel B. Medium risk cutoff at fasting blood sugar of 100</i>												
<i>Panel B.1 Short-run impact</i>												
RD estimate	-0.013 (0.030)	0.001 (0.002)	0.164 (0.922)	-0.007 (0.009)	0.183 (0.296)	-0.044* (0.021)	-0.243* (0.090)	0.006 (0.008)	-0.143 (0.326)	-0.023* (0.010)	0.023* (0.010)	-0.0003 (0.005)
Mean of the Dep. Var. in levels at [90, 100)	0.11	0.01	2.01	0.81	93.81	23.72	80.18	0.38	6.61	0.76	0.23	0.01
Observations	51,666	51,666	51,666	51,666	41,578	41,558	41,567	40,440	40,584	41,578	41,578	41,578
<i>Panel B.2 Long-run impact</i>												
RD estimate	0.004 (0.086)	0.0004 (0.003)	0.376 (1.202)	-0.001 (0.004)	0.150 (0.250)	-0.052+ (0.027)	-0.162* (0.069)	0.008 (0.011)	-0.495 (0.369)	-0.025** (0.007)	0.029** (0.007)	-0.004 (0.004)
Mean of the Dep. Var. in levels at [90, 100)	0.18	0.02	3.28	0.57	94.55	23.83	80.66	0.39	6.94	0.74	0.25	0.02
Observations	51,429	51,429	51,429	51,429	29,436	29,417	29,428	28,374	28,570	29,436	29,436	29,436

Notes: This table reports estimates of β from local linear regression of Equation (1) with bandwidth 10 mg/dL and uniform kernel. The running variable is baseline fasting blood sugar. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

Table 4: Impact of obesity screening

	(1) # of outpatient visits for obesity	(2) # of obesity medication days	(3) Future screening participation	(4) Change in BMI	(5) Change in waist circumference	(6) Change in basic exercise	(7) Change in # of drinks per week	(8) Change in # of cigarettes per day	(9) Change in blood sugar	(10) Low risk in future screening	(11) Medium risk in future screening	(12) High risk in future screening
<i>Panel A. High risk cutoff at BMI of 25</i>												
<i>Panel A.1 Short-run impact</i>												
RD estimate	0.007 (0.005)	0.097 (0.117)	0.004 (0.015)	-0.085* (0.041)	-0.352 (0.235)	-0.018 (0.023)	-0.702 (0.478)	0.039 (0.209)	-0.858 (0.953)	0.011 (0.007)	-0.028+ (0.016)	0.016 (0.016)
Mean of the Dep. Var. in levels at [24, 25)	0.001	0.01	0.75	24.49	86.33	0.37	6.61	3.28	104.03	0.08	0.72	0.20
Observations	15,403	15,403	15,403	11,737	11,736	11,574	11,525	11,472	11,737	11,736	11,736	11,736
<i>Panel A.2 Long-run impact</i>												
RD estimate	0.003 (0.003)	0.119 (0.108)	-0.002 (0.017)	-0.114 (0.070)	-0.425 (0.267)	-0.015 (0.027)	-0.248 (0.731)	0.018 (0.264)	-2.501+ (1.345)	0.011 (0.011)	-0.005 (0.018)	-0.006 (0.019)
Mean of the Dep. Var. in levels at [24, 25)	0.002	0.02	0.49	24.51	86.23	0.38	7.20	3.49	104.70	0.10	0.69	0.22
Observations	15,216	15,216	15,216	7,466	7,465	7,324	7,310	7,269	7,466	7,465	7,465	7,465
<i>Panel B. Medium risk cutoff at BMI of 23</i>												
<i>Panel B.1 Short-run impact</i>												
RD estimate	-0.001 (0.001)	0.0004 (0.009)	-0.002 (0.008)	0.005 (0.025)	0.212** (0.079)	0.011 (0.008)	0.108 (0.183)	0.001 (0.097)	-0.209 (0.294)	-0.047** (0.012)	0.051** (0.012)	-0.004* (0.002)
Mean of the Dep. Var. in levels at [22, 23)	0.002	0.02	0.78	22.58	77.80	0.38	6.42	3.47	96.09	0.65	0.35	0.01
Observations	82,059	82,059	82,059	64,103	64,094	62,926	62,950	62,702	64,095	64,093	64,093	64,093
<i>Panel B.2 Long-run impact</i>												
RD estimate	0.0004 (0.001)	-0.008 (0.027)	0.006 (0.009)	0.003 (0.039)	0.083 (0.125)	0.010 (0.013)	0.081 (0.233)	-0.018 (0.146)	-0.121 (0.342)	-0.030* (0.013)	0.034* (0.013)	-0.004 (0.003)
Mean of the Dep. Var. in levels at [22, 23)	0.002	0.04	0.54	22.71	78.24	0.39	6.52	3.55	96.61	0.59	0.40	0.01
Observations	81,533	81,533	81,533	44,654	44,652	43,635	43,746	43,583	44,649	44,651	44,651	44,651

Notes: This table reports estimates of β from local linear regression of Equation (1) with bandwidth 1kg/m^2 and uniform kernel. The running variable is baseline BMI. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

Table 5: Impact of hyperlipidemia screening

	(1) # of outpatient visits for hyperlipidemia	(2) Taking hyperlipidemia medication	(3) # of hyperlipidemia medication days	(4) Future screening participation	(5) Change in LDL cholesterol	(6) Change in BMI	(7) Change in waist circumference	(8) Change in basic exercise	(9) Change in # of drinks per week	(10) Low risk in future screening	(11) Medium risk in future screening	(12) High risk in future screening
<i>Panel A. High risk cutoff at LDL cholesterol of 160</i>												
<i>Panel A.1 Short-run impact</i>												
RD estimate	0.168** (0.054)	0.00003 (0.007)	1.798 (1.580)	-0.008 (0.006)	-0.872+ (0.502)	-0.007 (0.022)	0.050 (0.118)	0.007 (0.017)	-0.053 (0.200)	0.010 (0.006)	-0.007 (0.012)	-0.003 (0.010)
Mean Dep. Var. in levels at [150, 160)	0.91	0.15	25.25	0.77	138.98	24.37	81.87	0.38	5.52	0.35	0.42	0.23
Observations	25,910	25,910	25,910	25,910	19,898	20,010	20,000	19,681	19,657	19,898	19,898	19,898
<i>Panel A.2 Long-run impact</i>												
RD estimate	0.126 (0.103)	0.0001 (0.007)	1.668 (2.537)	0.010 (0.006)	-1.061 (0.915)	0.081+ (0.041)	0.340 (0.231)	0.051+ (0.029)	0.580+ (0.286)	-0.002 (0.013)	0.0003 (0.012)	0.002 (0.013)
Mean of the Dep. Var. in levels at [150, 160)	1.48	0.20	48.73	0.53	138.25	24.44	82.16	0.38	5.87	0.37	0.39	0.24
Observations	25,747	25,747	25,747	25,747	13,470	13,511	13,510	13,224	13,227	13,470	13,470	13,470

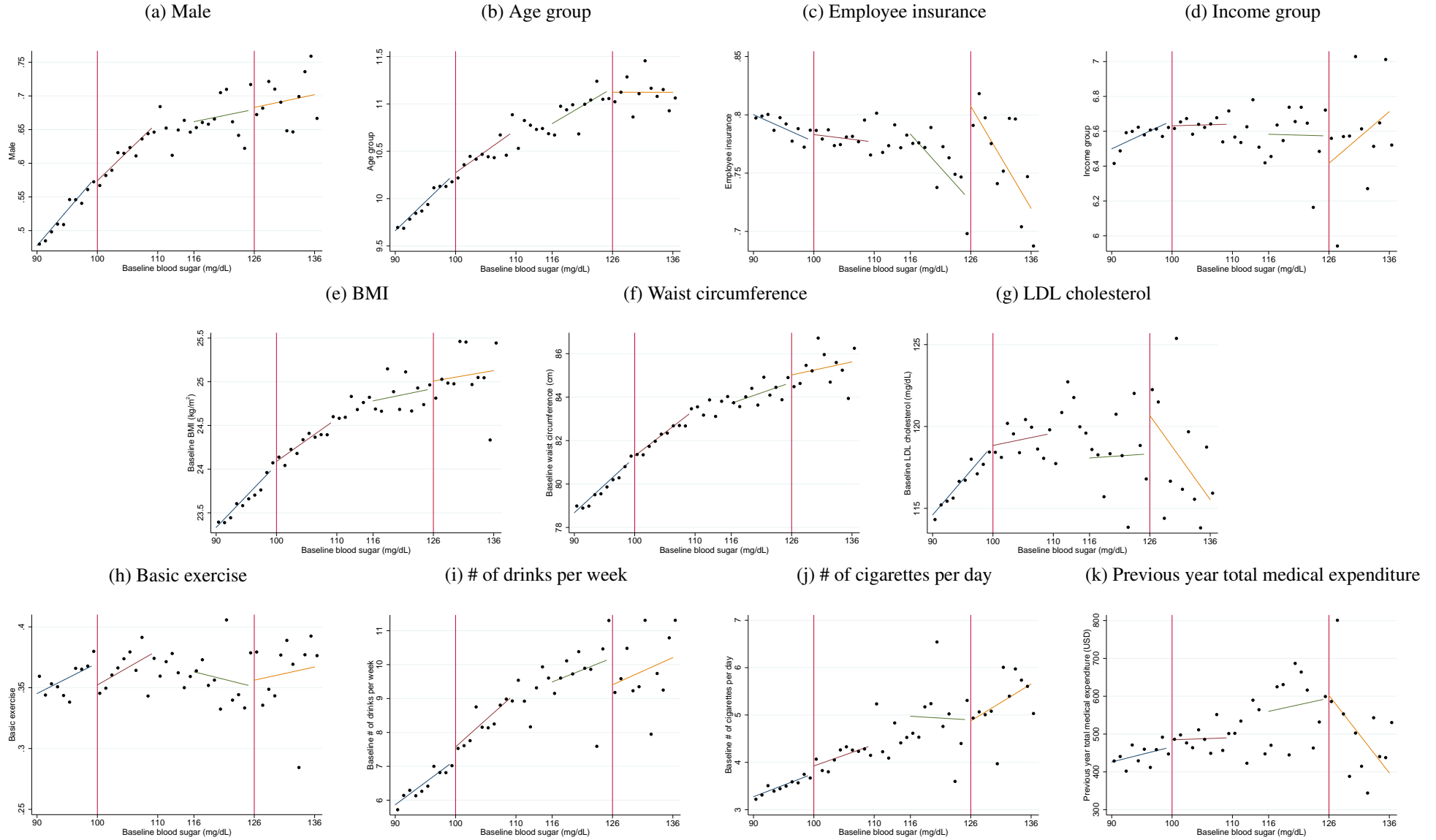
Notes: This table reports estimates of β from local linear regression of Equation (1) with bandwidth 10 mg/dL and uniform kernel. The running variable is baseline LDL cholesterol. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

Appendix Figures and Tables

Figure A.1: Secondary examination report card

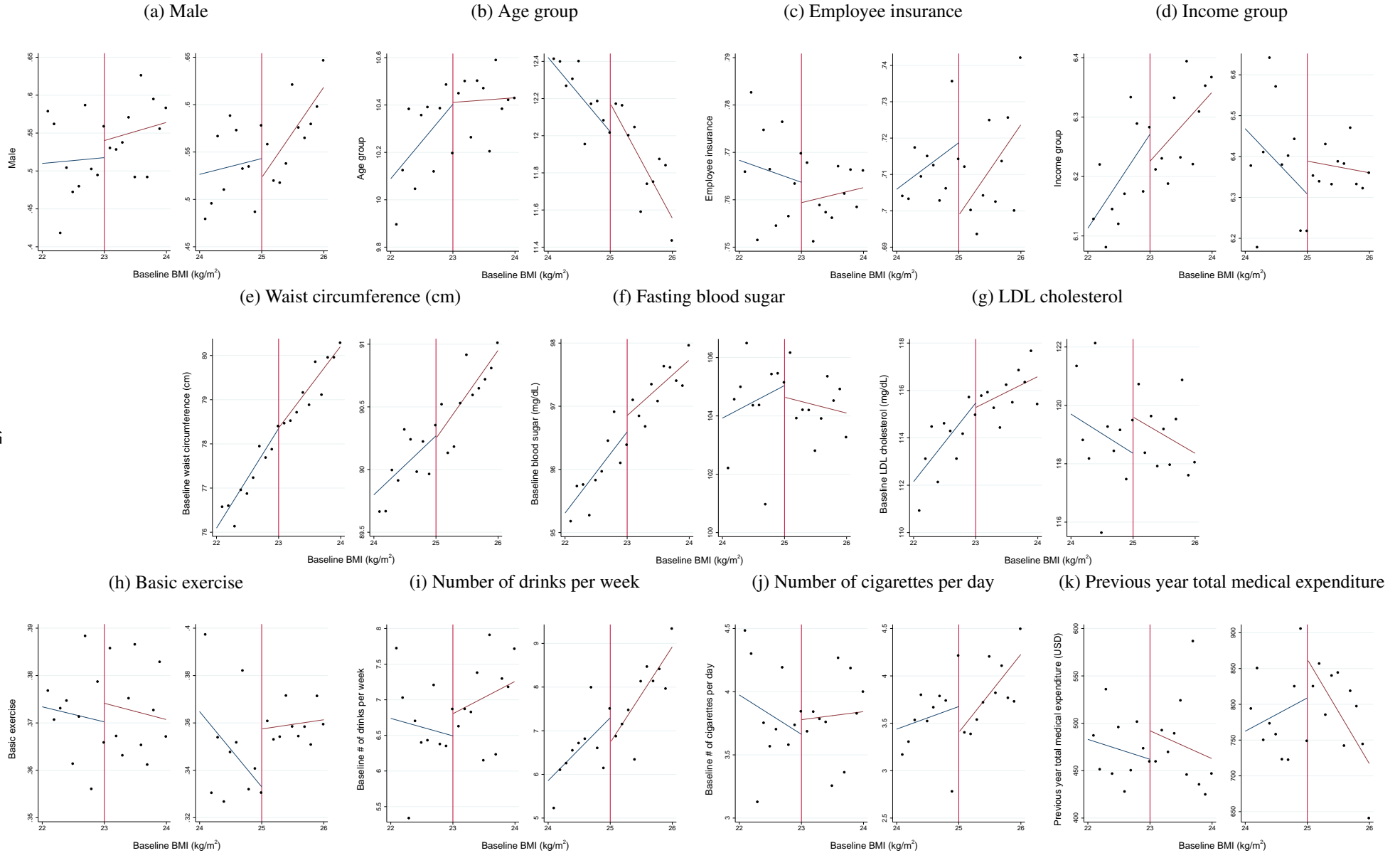
Results of Regular Medical Checkup (2nd)				
Name		Resident registration number		
Date of examination		Health checkup institution	<input type="checkbox"/> Visit, <input type="checkbox"/> on-site checkup	
The 2nd test results of diabetes, hypertension, and cognitive function difficulty				
Section	<input type="checkbox"/> Diabetes		<input type="checkbox"/> Hypertension	
Results	Fasting blood sugar mg/dL		Blood pressure mmHg	
	<input type="checkbox"/> Normal <input type="checkbox"/> Pre-diabetes <input type="checkbox"/> Diabetes		<input type="checkbox"/> Normal <input type="checkbox"/> Pre-hypertension <input type="checkbox"/> Hypertension	
Treatment Plan	<input type="checkbox"/> Checkup at the next medical exam <input type="checkbox"/> Control diet and exercise, then checkup again a couple of month later <input type="checkbox"/> Needs medical treatment		<input type="checkbox"/> Checkup at the next medical exam <input type="checkbox"/> Control diet and exercise, then checkup again a couple of month later <input type="checkbox"/> Needs medical treatment	
Cognitive function difficulty (70 or 74 years old only)	<input type="checkbox"/> Nothing specific (0-5 points) <input type="checkbox"/> Declined cognitive function (6-30 points; needs additional medical examination and counseling in Neurology or Psychiatric Department)			
Final prescription				
Diabetes result	<input type="checkbox"/> Normal <input type="checkbox"/> Pre-diabetes <input type="checkbox"/> Diabetes (<input type="checkbox"/> with diabetes history)		Date of determination	
Hypertension result	<input type="checkbox"/> Normal <input type="checkbox"/> Pre-hypertension <input type="checkbox"/> Hypertension (<input type="checkbox"/> with hypertension history)		Examined doctor	License number Name <div style="text-align: right;">(signature)</div>
※ This notification letter of health checkup can serve as a medical care referral (treatment referral) if it has an impression note that states that the subject is required to be treated at a more advanced hospital. ※ Because not all diseases are examined, if you have any specific problems (e.g., excessive weight loss), please talk to a physician.				
We are notifying you of these medical examination results as above.			Date	
Health checkup institution registration number ()		Health checkup institution name ()		

Figure A.2: Covariate balance test: fasting blood sugar



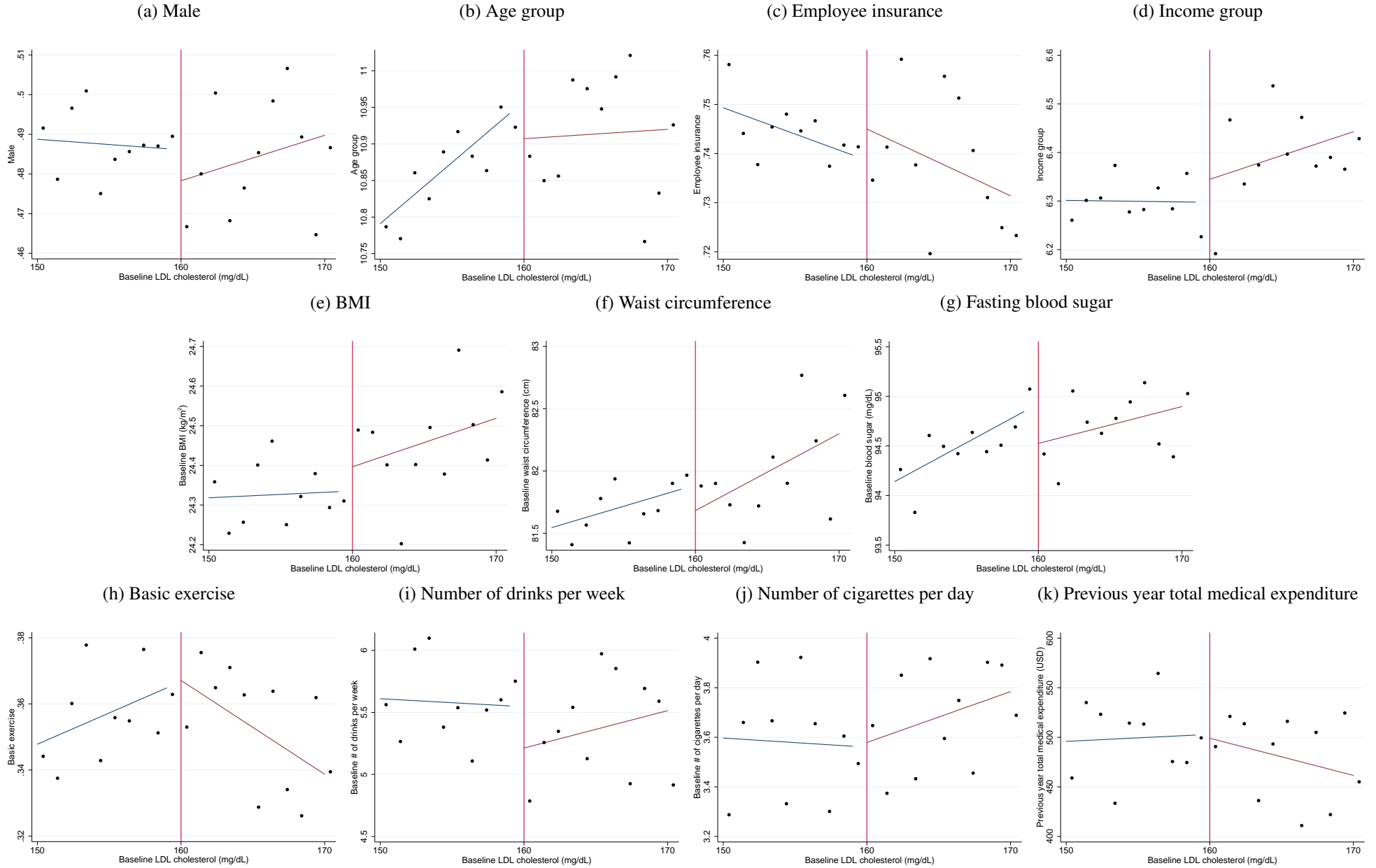
Notes: The running variable is baseline fasting blood sugar. The scatter plot indicates the mean of the dependent variable within 1 point bins. Around two cutoffs, 100 and 126, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 10 mg/dL. These are estimated separately on each side of the cutoff.

Figure A.3: Covariate balance test: BMI



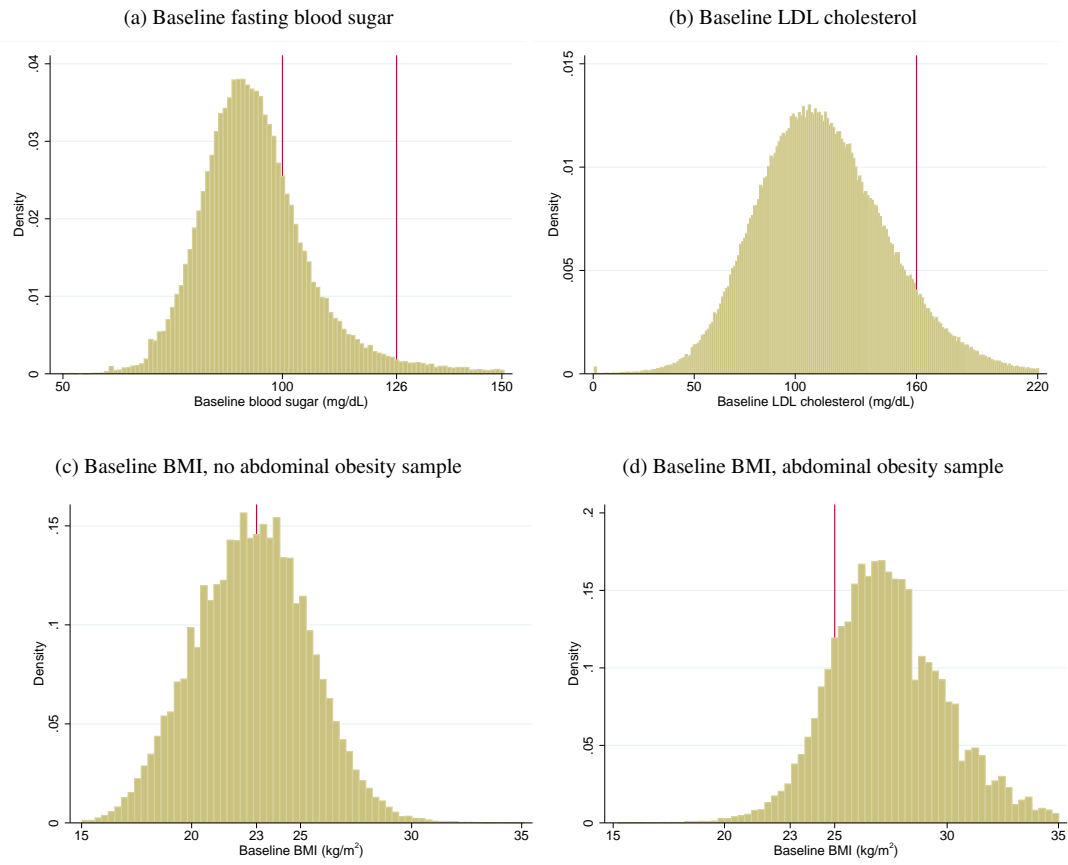
Notes: The running variable is baseline BMI. The scatter plot indicates the mean of the dependent variable within 0.05 point bins. Around two cutoffs, 23 and 25, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 1kg/m². For each panel, the left figure with cutoff 23 is based on the sample without abdominal obesity sample and the right figure with cutoff 25 is based on the sample with abdominal obesity.

Figure A.4: Covariate balance test: LDL cholesterol



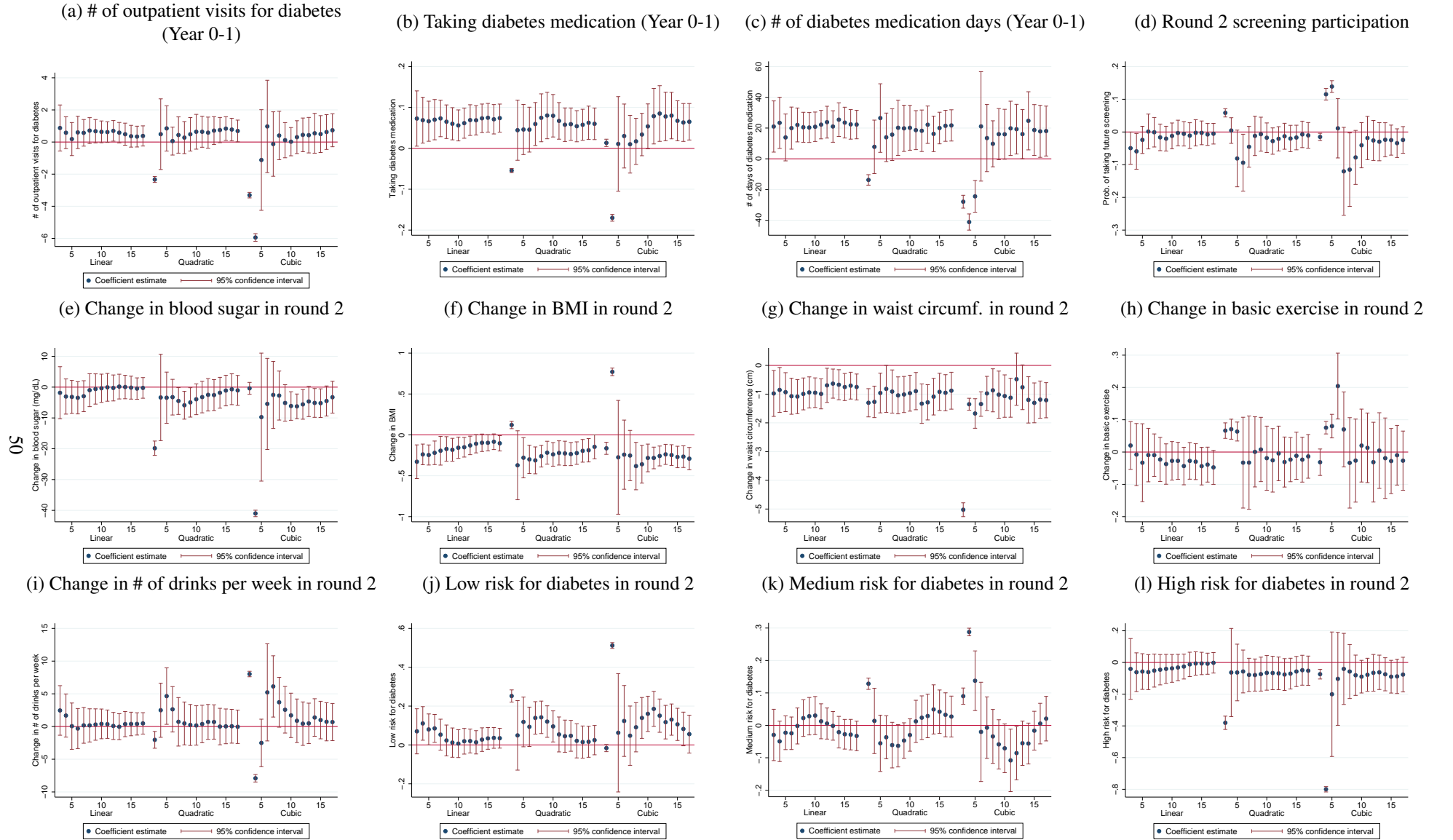
Notes: The running variable is baseline LDL cholesterol. The scatter plot indicates the mean of the dependent variable within 1 point bins. Around cutoff 160, the solid lines are the fitted values from the local linear regression of the dependent variable using a uniform kernel with a bandwidth of 10 mg/dL.

Figure A.5: Density of running variables



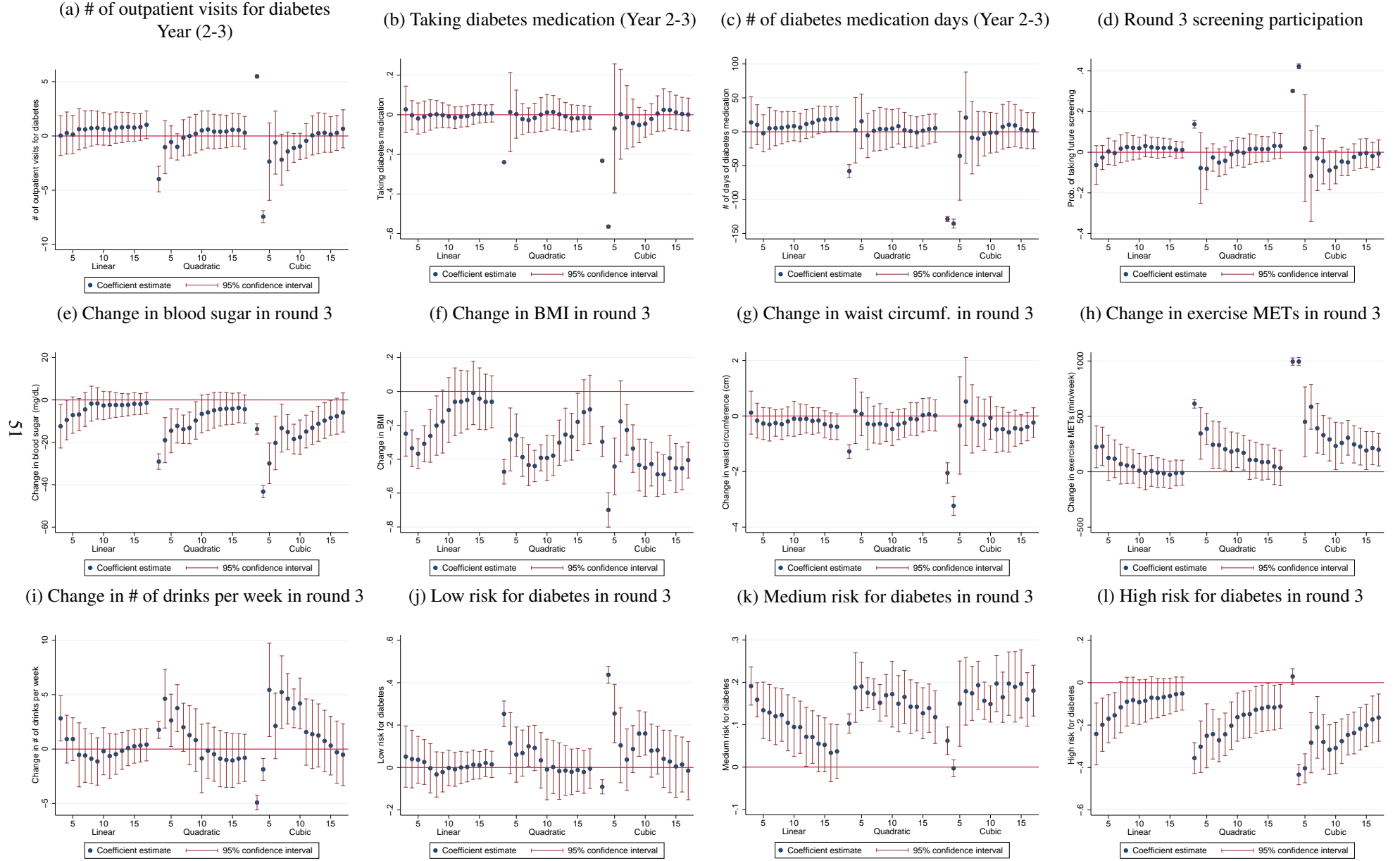
Notes: Bin size of 1 is used for baseline fasting blood sugar and LDL cholesterol, and bin size of 0.3 is used for baseline BMI. Solid vertical lines indicate the treatment cutoffs.

Figure A.6: Sensitivity to bandwidth and polynomial degree: fasting blood sugar, cutoff 126, short-run outcomes



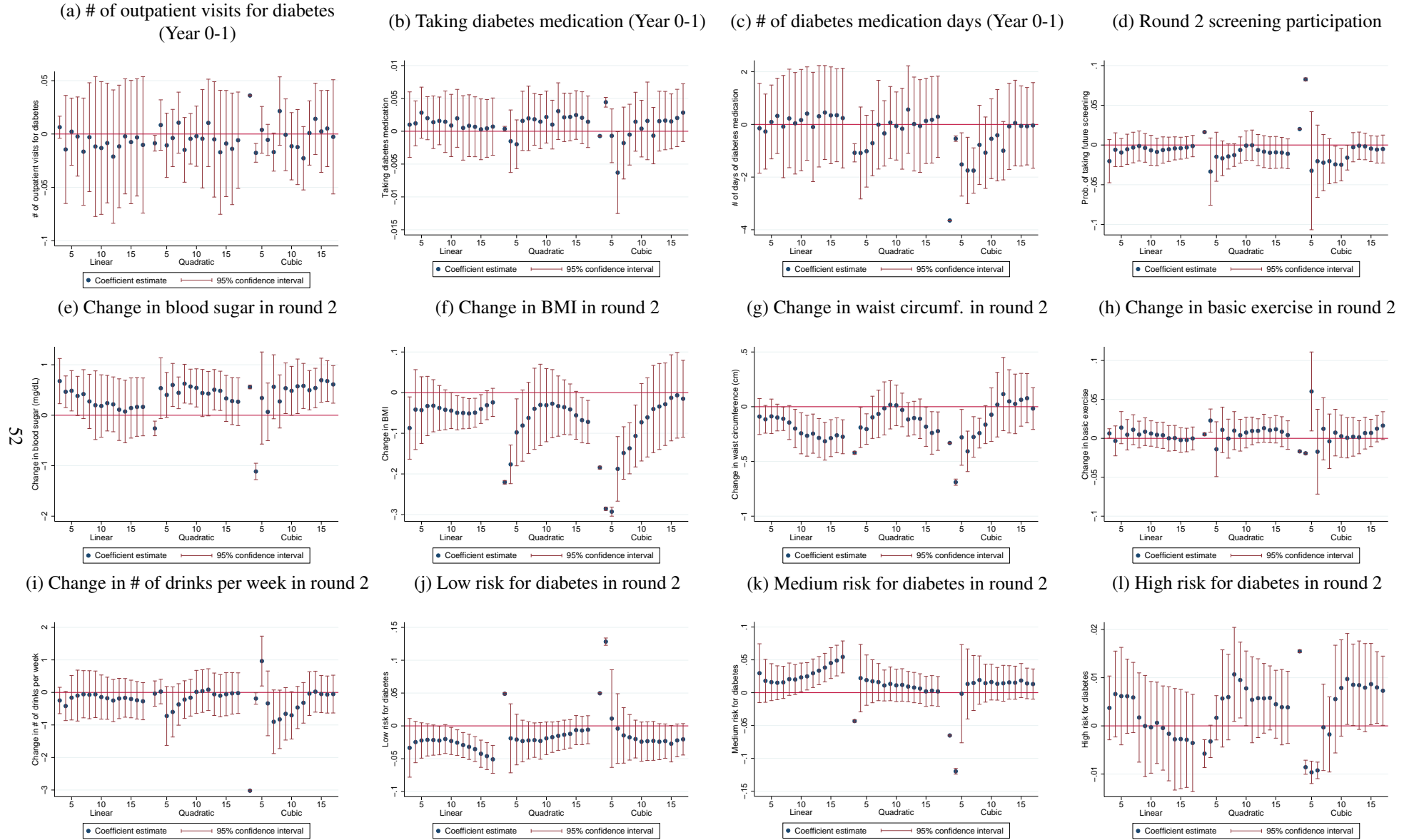
Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 3 to 17 in increments of 1 mg/dL, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline fasting blood sugar. Standard errors are clustered at the unique value of the running variable.

Figure A.7: Sensitivity to bandwidth and polynomial degree: fasting blood sugar, cutoff 126, long-run outcomes



Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 3 to 17 in increments of 1 mg/dL, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline fasting blood sugar. Cohort-fixed effects are controlled. Standard errors are clustered at the unique value of the running variable.

Figure A.8: Sensitivity to bandwidth and polynomial degree: fasting blood sugar, cutoff 100, short-run outcomes



Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 3 to 17 in increments of 1 mg/dL, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline fasting blood sugar. Cohort-fixed effects are controlled. Standard errors are clustered at the unique value of the running variable.

Figure A.9: Sensitivity to bandwidth and polynomial degree: fasting blood sugar, cutoff 100, long-run outcomes

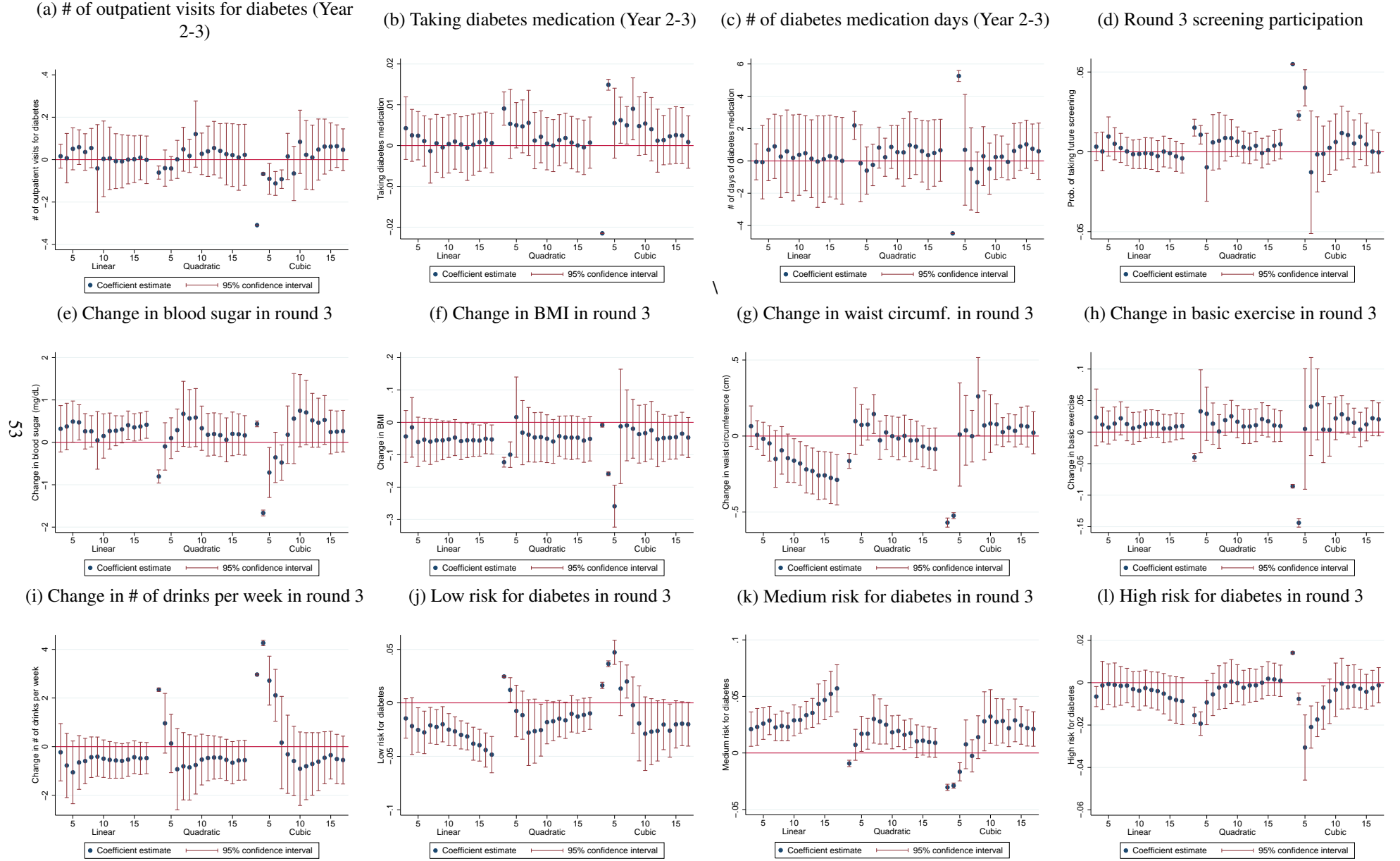
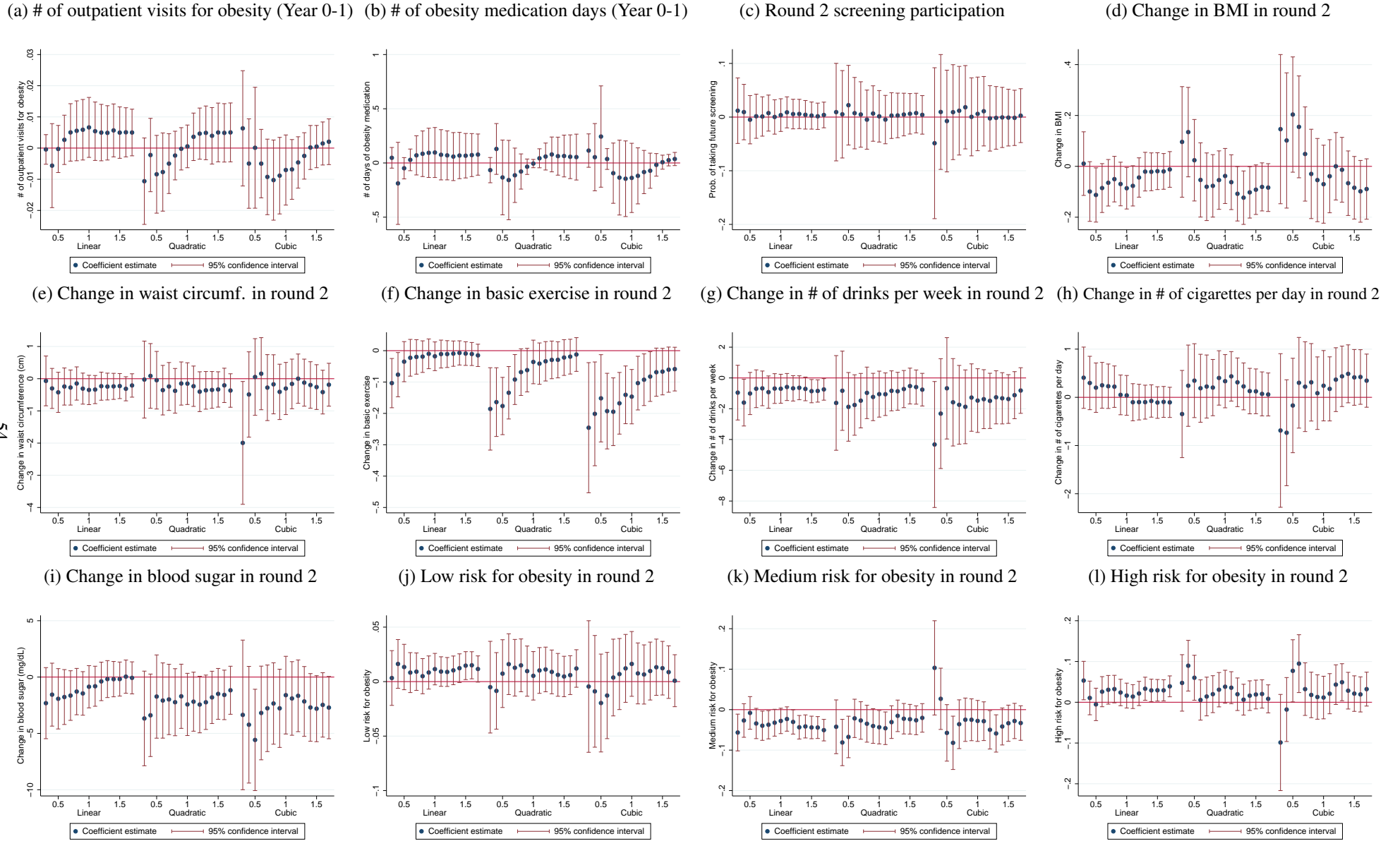
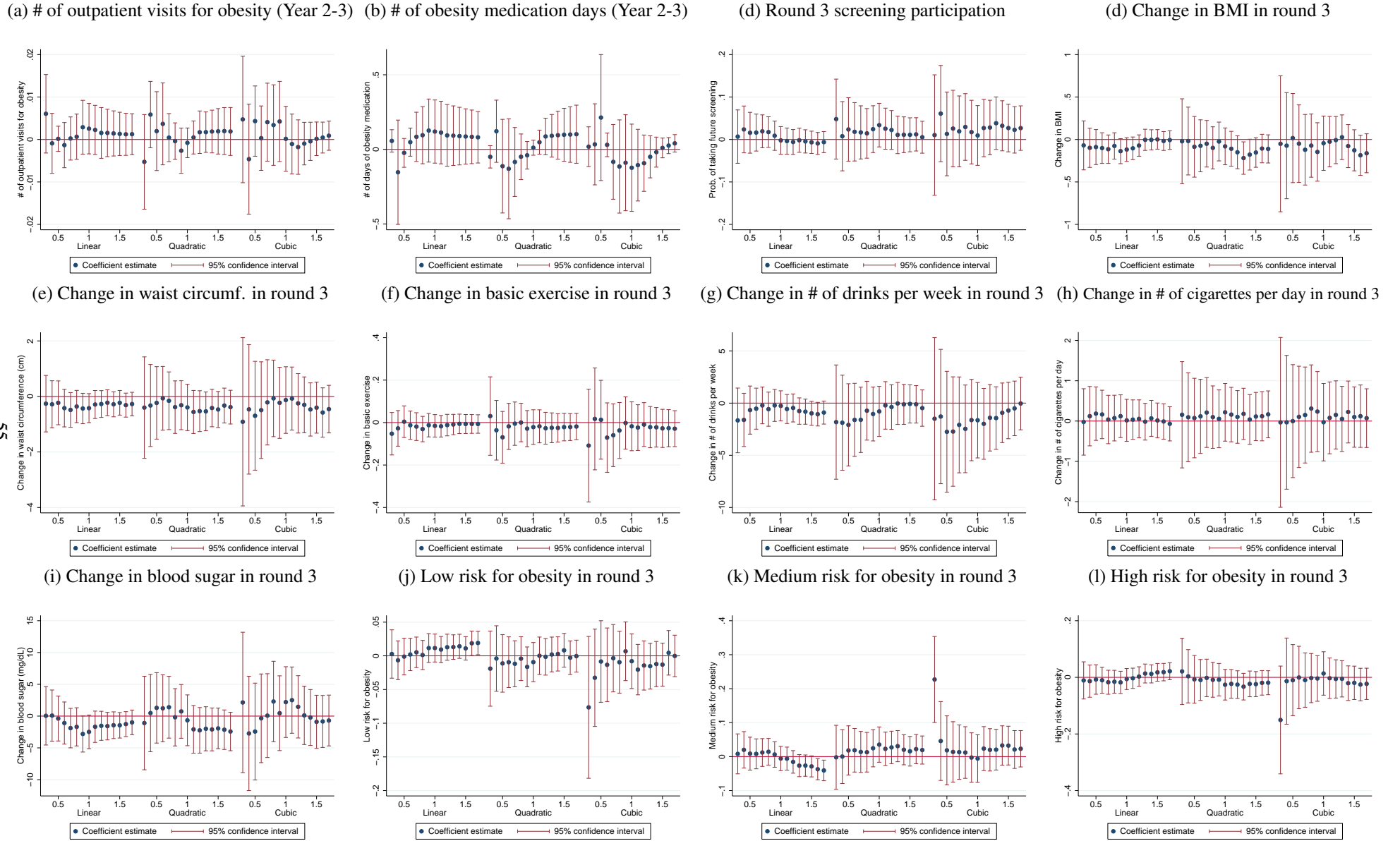


Figure A.10: Sensitivity to bandwidth and polynomial degree: BMI, cutoff 25, short-run outcomes



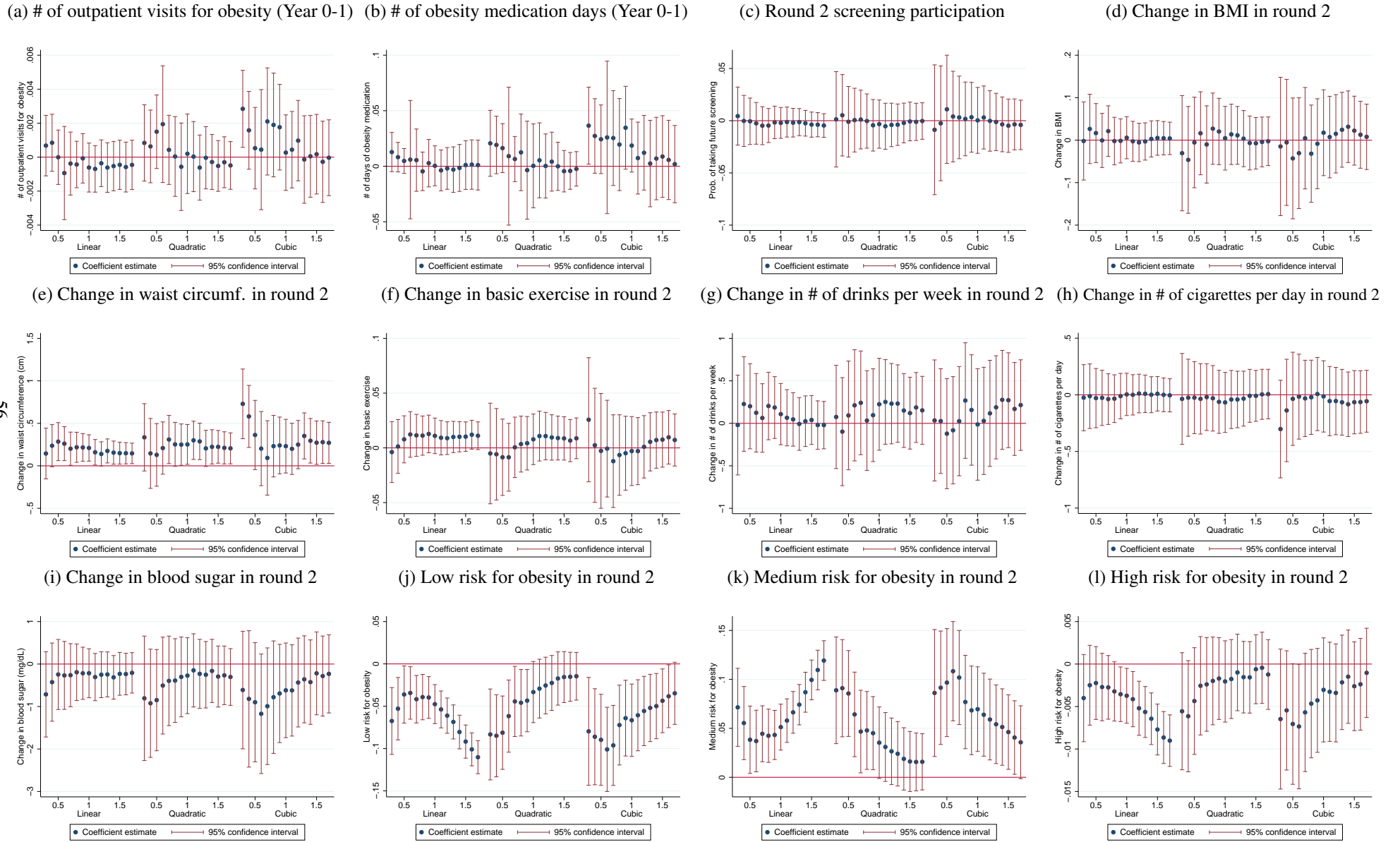
Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 0.3 to 1.7 in increments of 0.1, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline BMI. Cohort-fixed effects are controlled. Standard errors are clustered at the unique value of the running variable.

Figure A.11: Sensitivity to bandwidth and polynomial degree: BMI, cutoff 25, long-run outcomes



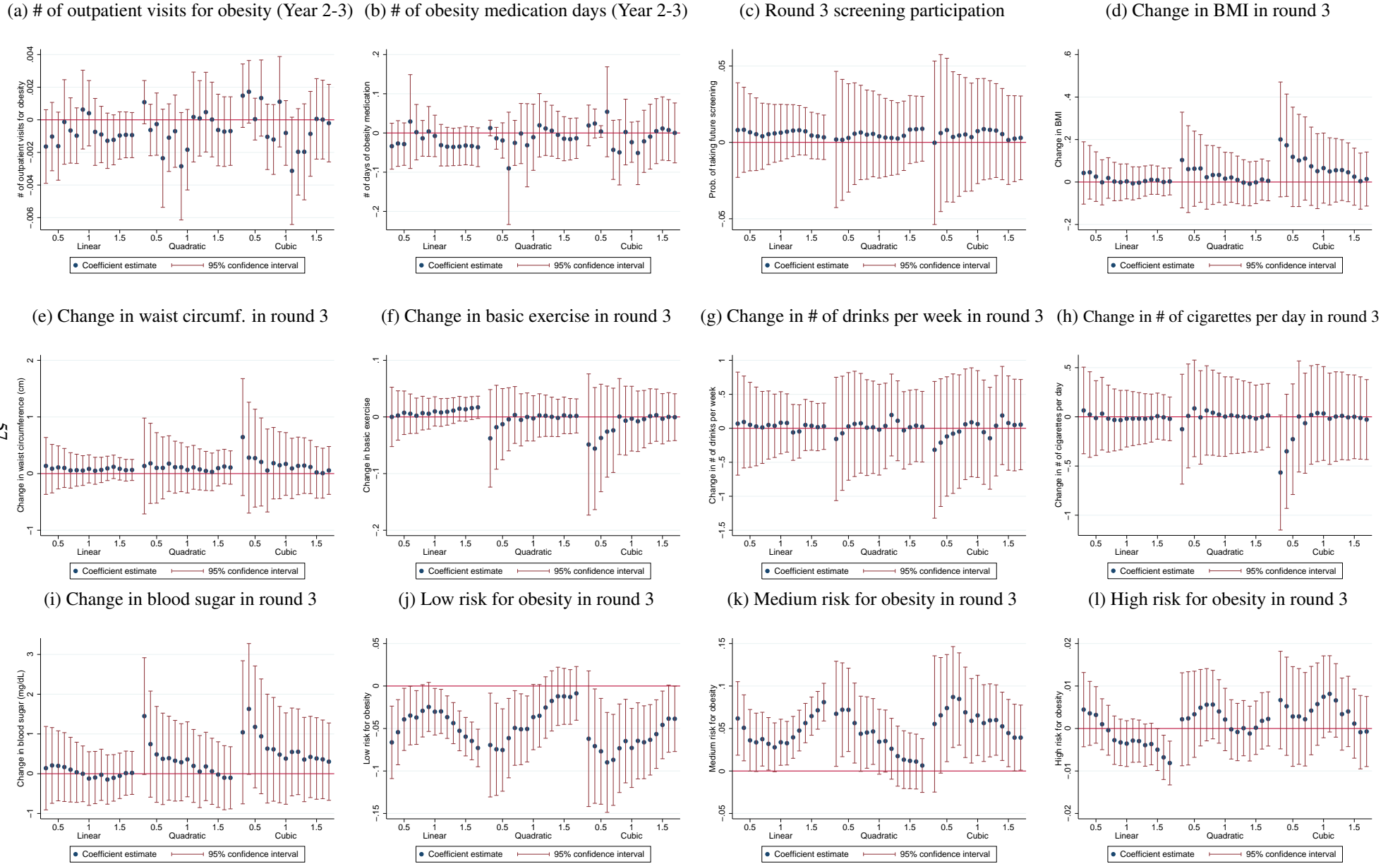
Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 0.3 to 1.7 in increments of 0.1, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline BMI. Cohort-fixed effects are controlled. Standard errors are clustered at the unique value of the running variable.

Figure A.12: Sensitivity to bandwidth and polynomial degree: BMI, cutoff 23, short-run outcomes



Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 0.3 to 1.7 in increments of 0.1, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline BMI. Cohort-fixed effects are controlled. Standard errors are clustered at the unique value of the running variable.

Figure A.13: Sensitivity to bandwidth and polynomial degree: BMI, cutoff 23, long-run outcomes



Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 0.3 to 1.7 in increments of 0.1, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline BMI. Cohort-fixed effects are controlled. Standard errors are clustered at the unique value of the running variable.

Figure A.14: Sensitivity to bandwidth and polynomial degree: LDL cholesterol, cutoff 160, short-run outcomes

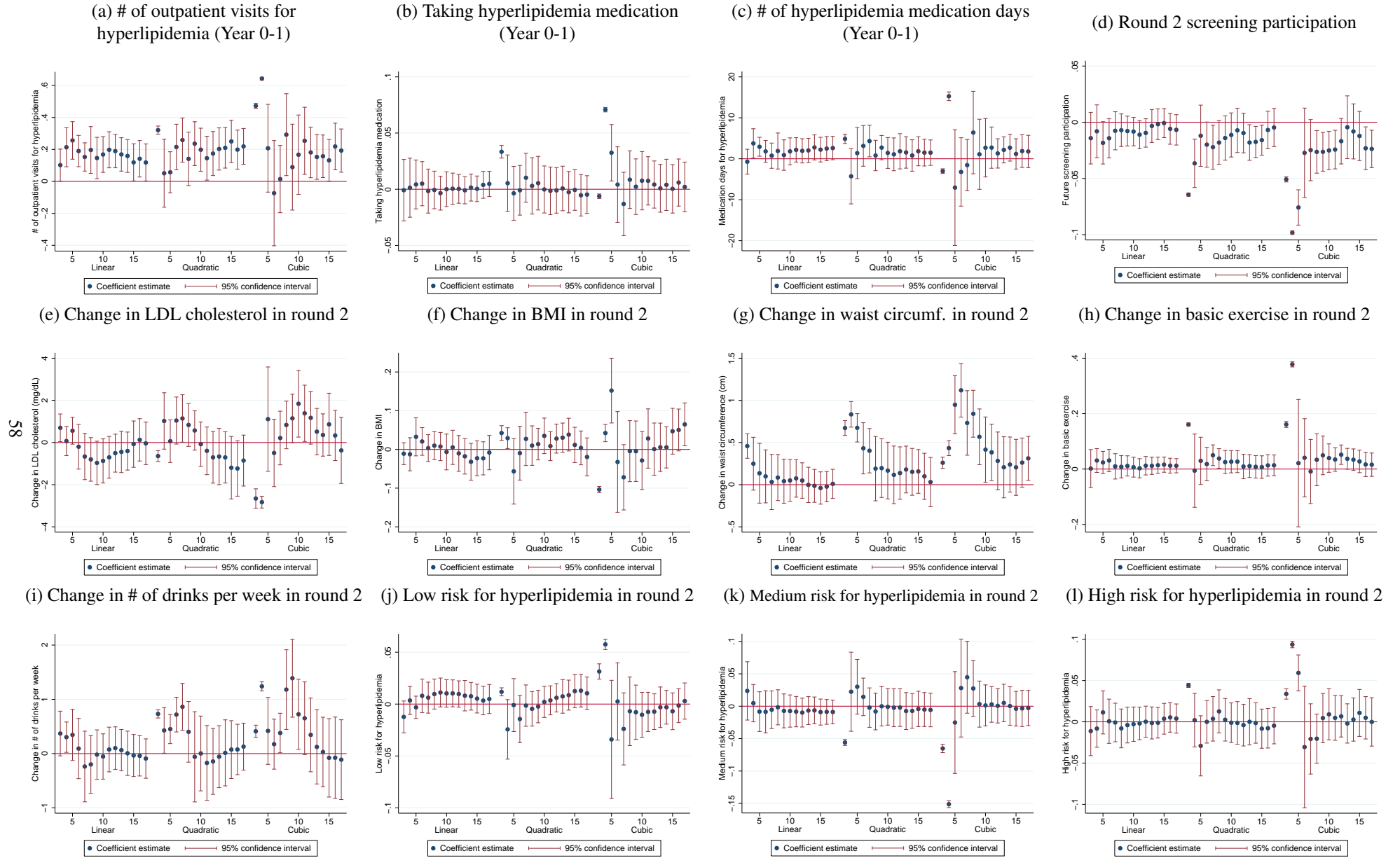
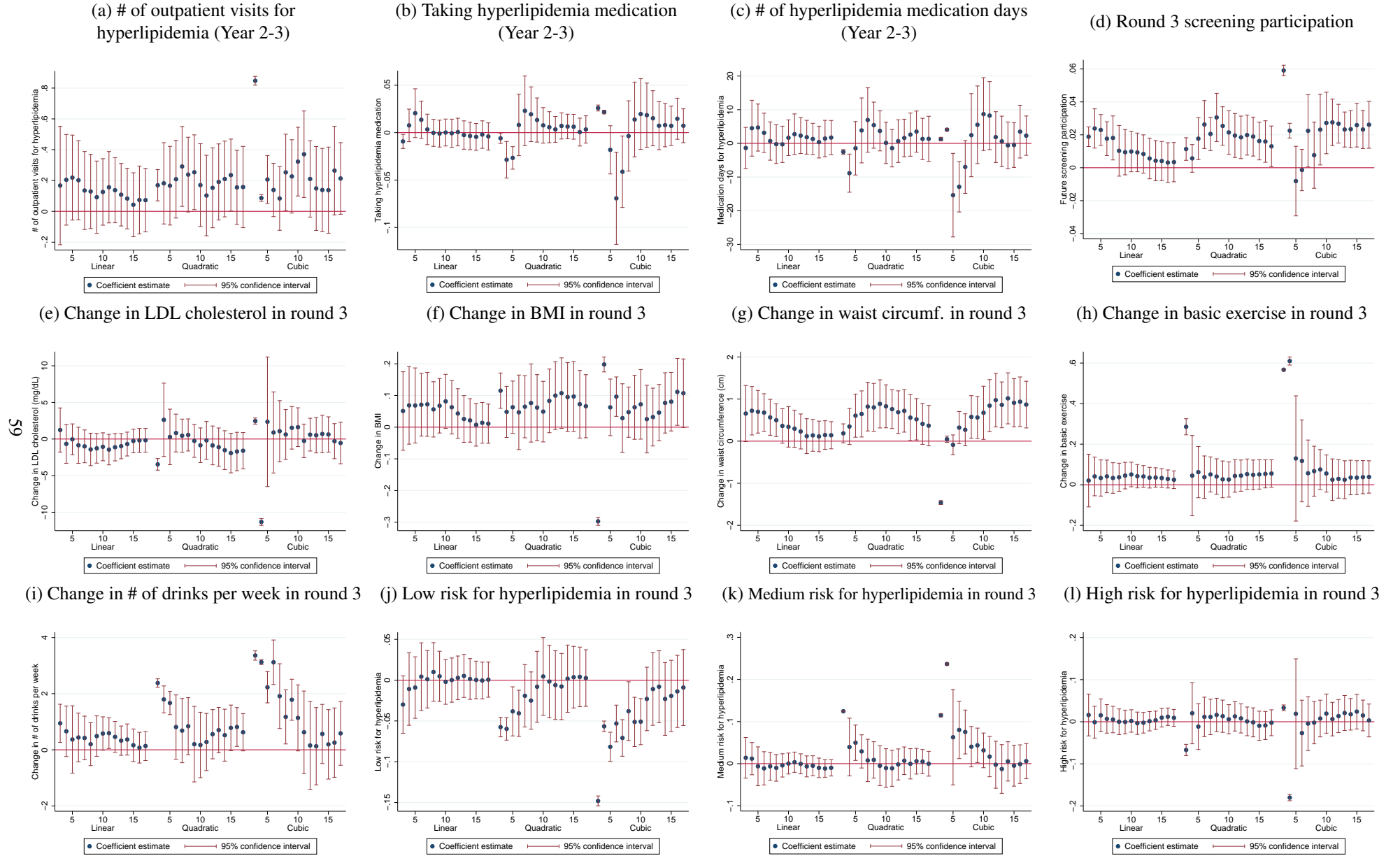
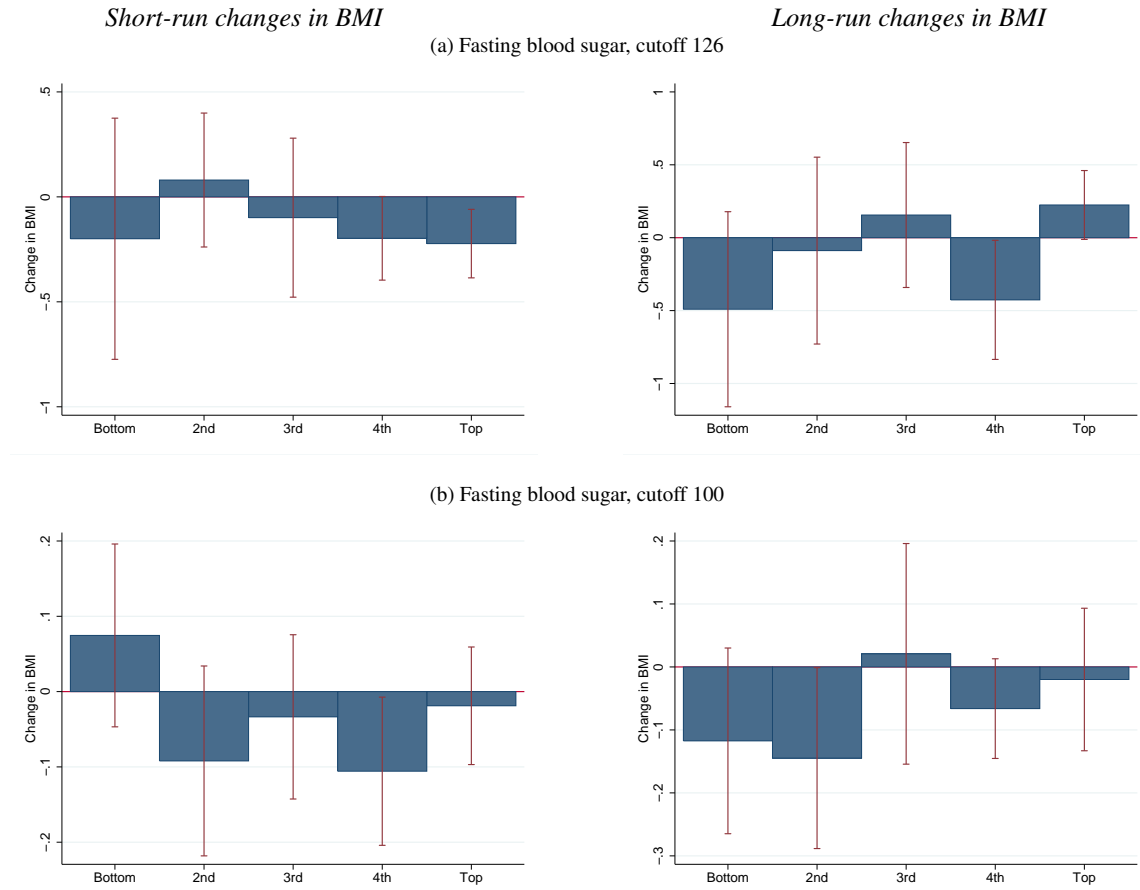


Figure A.15: Sensitivity to bandwidth and polynomial degree: LDL cholesterol, cutoff 160, long-run outcomes



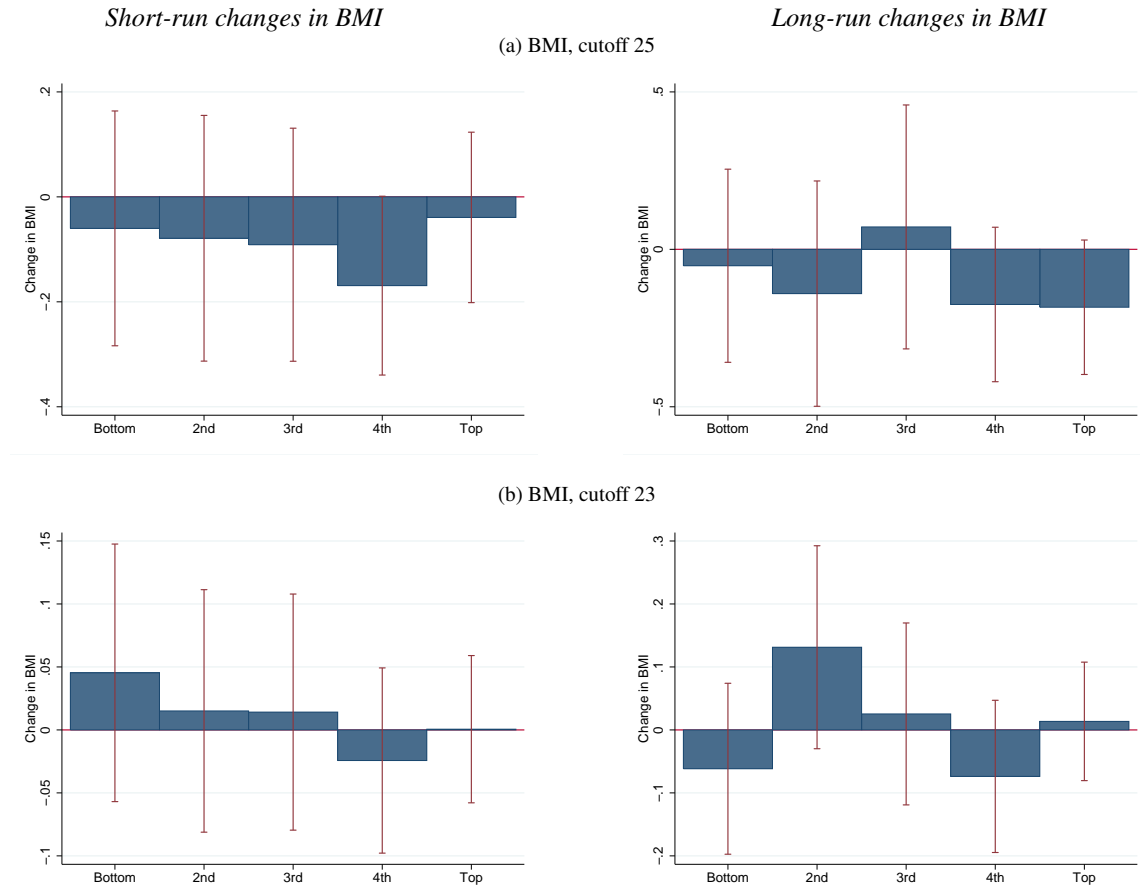
Notes: Each figure displays estimates of β from local polynomial regression of Equation (1) at different bandwidths from 3 to 17 in increments of 1 mg/dL, for linear, quadratic and cubic polynomial specifications. Degree of $f(\cdot)$ and the bandwidth size are on the x-axis. Coefficient estimates and the lower and upper 95% confidence bounds are on the y-axis. The running variable is baseline LDL cholesterol. Cohort-fixed effects are controlled. Standard errors are clustered at the unique value of the running variable.

Figure A.16: Subgroup analysis by income: Fasting blood sugar



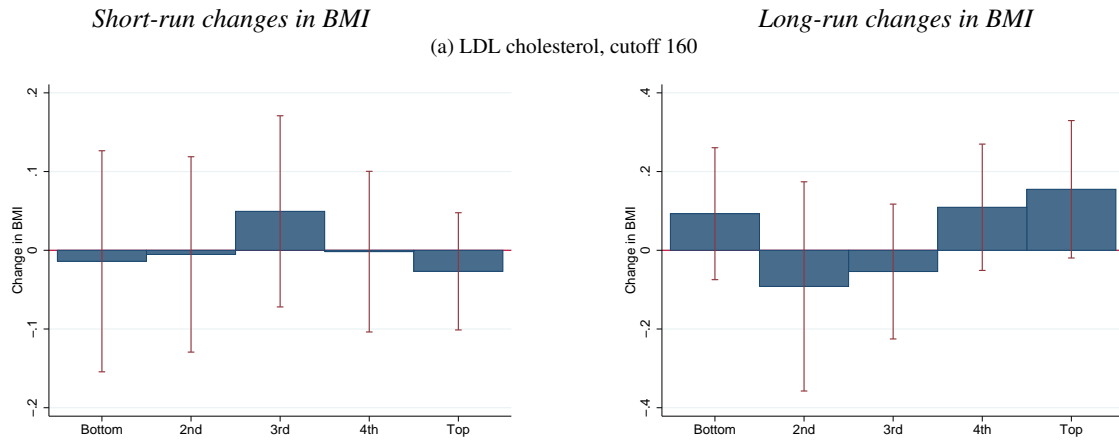
Notes: The dependent variables are the short-run changes in BMI (left column) and the long-run changes in BMI (right column). Each bar displays estimates of β from local linear regression of Equation (1) using a uniform kernel with a bandwidth of 10 mg/dL. Vertical lines indicate 95 percent confidence intervals.

Figure A.17: Subgroup analysis by income: BMI



Notes: The dependent variables are the short-run changes in BMI (left column) and the long-run changes in BMI (right column). Each bar displays estimates of β from local linear regression of Equation (1) using a uniform kernel with a bandwidth of 1 kg/m². Vertical lines indicate 95 percent confidence intervals.

Figure A.18: Subgroup analysis by income: LDL cholesterol



Notes: The dependent variables are the short-run changes in BMI (left column) and the long-run changes in BMI (right column). Each bar displays estimates of β from local linear regression of Equation (1) using a uniform kernel with a bandwidth of 10 mg/dL. Vertical lines indicate 95 percent confidence intervals.

Table A.1: Eligible ages in the National Health Screening Program by insurance type

Insurance Type	Classification	Eligible ages
Self-employed insurance	Household head	All ages
	Dependent	Ages 40 and older
Employee insurance	Employee	All ages
	Dependent	Ages 40 and older
Medical care assistance	Household head	Ages 19 and older
	Dependent	Ages 40 and older

Notes: All individuals are eligible to participate in the NHSP every two years, except blue-collar workers with employee insurance who are eligible once a year. Insurance types are mutually exclusive. Individuals enrolled in Medical Care Assistance (MCA) became eligible for the NHSP since 2012.

Table A.2: Characteristics of baseline screening participants and eligible non-participants

Variables	(1) Eligible Screening non-participants	(2) Screening participants	(3) t-statistics for mean differences (non-participants vs. participants)
Male	0.51 (0.50)	0.51 (0.50)	0.05
Age group			
Below 50	0.59 (0.49)	0.55 (0.50)	30.22
50 and older	0.41 (0.49)	0.45 (0.50)	-30.22
Insurance type			
Self-employed insurance	0.50 (0.50)	0.24 (0.43)	215.01
Employee insurance	0.50 (0.50)	0.75 (0.43)	-212.02
Average income decile	5.87 (2.92)	6.18 (2.82)	-42.05
2008 Taking diabetes medication	0.06 (0.23)	0.05 (0.22)	8.87
2008 Taking obesity medication	0.0005 (0.02)	0.0004 (0.02)	0.65
2008 Taking hyperlipidemia medication	0.05 (0.22)	0.06 (0.25)	-21.72
2008 Total medical expenditure (USD)	801.94 (3648.44)	492.50 (1521.89)	45.21
Observations	289,294	352,896	

Notes: Columns (1) and (2) report group-specific means and standard deviations in parentheses. Column (3) presents t-stats to test the difference between screening participants and non-participants. The statistics are calculated based on 2009 information, except for medication and medical expenditure variables. In order to compare characteristics of participants and non-participants, we restrict the non-participant sample to those who are eligible to take screening (i.e. aged 40 or older if classified as a dependent in self-employed insurance or employee insurance, no restrictions otherwise).

Table A.3: Covariate balance test

	(1) Male	(2) Age group	(3) Employee insurance	(4) Income group	(5) BMI	(6) Waist circumference	(7) Fasting blood sugar	(8) LDL cholesterol	(9) Basic exercise	(10) # of drinks per day	(11) # of cigarettes per day	(12) Previous year total medical expenditure
<i>Panel A.1 Fasting blood sugar, cutoff 126</i>												
RD estimate	0.004 (0.028)	-0.051 (0.109)	0.083** (0.018)	-0.156 (0.217)	0.085 (0.147)	0.338 (0.425)		2.332 (2.355)	0.005 (0.019)	-0.793 (0.861)	-0.009 (0.525)	7.289 (87.924)
Observations	4,717	4,717	4,717	4,717	4,713	4,712		4,663	4,599	4,613	4,627	4,717
<i>Panel A.2 Fasting blood sugar, cutoff 100</i>												
RD estimate	-0.011+ (0.006)	0.019 (0.049)	0.004 (0.006)	-0.023 (0.035)	0.038 (0.062)	0.047 (0.158)		0.129 (0.556)	-0.016+ (0.008)	0.347* (0.153)	0.083 (0.123)	17.825 (16.597)
Observations	52,426	52,426	52,426	52,426	52,398	52,413		51,988	51,110	51,224	51,434	52,426
<i>Panel B.1 BMI, cutoff 25</i>												
RD estimate	-0.019 (0.115)	0.140 (0.299)	-0.018 (0.018)	0.077 (0.122)		-0.017 (0.499)	-0.429 (0.927)	1.222 (1.784)	0.024 (0.015)	-0.544 (1.415)	-0.255 (0.822)	41.517 (86.202)
Observations	15,616	15,616	15,616	15,616		15,616	15,613	15,487	15,410	15,364	15,299	15,616
<i>Panel B.2 BMI, cutoff 23</i>												
RD estimate	0.022 (0.111)	0.009 (0.244)	-0.004 (0.017)	-0.045 (0.112)		0.010 (0.687)	0.260 (0.491)	-0.187 (1.055)	0.004 (0.007)	0.309 (1.156)	0.114 (0.795)	29.682 (35.170)
Observations	83,379	83,379	83,379	83,379		83,379	83,365	82,887	82,062	82,086	81,834	83,379
<i>Panel C LDL cholesterol, cutoff 160</i>												
RD estimate	-0.003 (0.012)	-0.046 (0.060)	0.002 (0.010)	0.063 (0.068)	0.051 (0.071)	-0.174 (0.199)	-0.772 (0.546)		0.001 (0.009)	-0.083 (0.286)	0.068 (0.181)	-13.265 (35.762)
Observations	29,059	29,059	29,059	29,059	29,054	29,045	29,059		28,655	28,617	28,544	29,059

Notes: This table reports estimates of β from local linear regression of Equation (1). The running variables are baseline blood sugar in Panel A, baseline BMI in Panel B and baseline LDL cholesterol in Panel C. Uniform kernel with bandwidth 10 mg/dL is used in Panel A and C, and bandwidth 1kg/m² is used in Panel B. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

Table A.4: Impact of diabetes screening: with control variables

	(1) # of outpatient visits for diabetes	(2) Taking diabetes medication	(3) # of diabetes medication days	(4) Future screening participation	(5) Change in blood sugar	(6) Change in BMI	(7) Change in waist circumference	(8) Change in basic exercise	(9) Change in # of drinks per week	(10) Low risk in future screening	(11) Medium risk in future screening	(12) High risk in future screening
<i>Panel A. High risk cutoff at fasting blood sugar of 126</i>												
<i>Panel A.1 Short-run impact</i>												
RD estimate	0.612 (0.356)	0.058** (0.017)	20.170** (5.743)	-0.029 (0.019)	-0.566 (1.860)	-0.155** (0.052)	-0.968** (0.311)	-0.028 (0.030)	0.261 (1.132)	0.012 (0.036)	0.029 (0.025)	-0.041 (0.039)
Mean of the Dep. Var. in levels at [116, 126)	1.27	0.11	28.46	0.79	112.12	24.68	83.89	0.40	9.72	0.28	0.54	0.18
Observations	4,552	4,552	4,552	4,552	3,578	3,573	3,574	3,475	3,488	3,578	3,578	3,578
<i>Panel A.2 Long-run impact</i>												
RD estimate	0.688 (0.658)	-0.007 (0.028)	6.870 (11.069)	0.004 (0.026)	-2.734 (3.186)	-0.111 (0.091)	-0.122 (0.284)	0.011 (0.050)	-0.245 (1.008)	0.007 (0.040)	0.080* (0.035)	-0.087 (0.051)
Mean of the Dep. Var. in levels at [116, 126)	2.24	0.17	55.45	0.57	113.69	24.59	83.67	0.41	9.72	0.30	0.47	0.23
Observations	4,513	4,513	4,513	4,513	2,618	2,614	2,612	2,520	2,543	2,618	2,618	2,618
<i>Panel B. Medium risk cutoff at fasting blood sugar of 100</i>												
<i>Panel B.1 Short-run impact</i>												
RD estimate	-0.017 (0.040)	0.001 (0.002)	0.049 (1.006)	-0.008 (0.009)	0.213 (0.299)	-0.042+ (0.023)	-0.231* (0.089)	0.006 (0.008)	-0.235 (0.329)	-0.023* (0.009)	0.023* (0.010)	-0.00004 (0.004)
Mean of the Dep. Var. in levels at [90, 100)	0.11	0.01	2.01	0.81	93.81	23.72	80.18	0.38	6.61	0.76	0.23	0.01
Observations	50,495	50,495	50,495	50,495	40,673	40,652	40,661	39,546	39,692	40,673	40,673	40,673
<i>Panel B.2 Long-run impact</i>												
RD estimate	0.002 (0.091)	0.001 (0.004)	0.419 (1.372)	-0.003 (0.004)	0.161 (0.258)	-0.048 (0.029)	-0.177* (0.073)	0.006 (0.012)	-0.529 (0.363)	-0.025** (0.007)	0.029** (0.006)	-0.004 (0.005)
Mean of the Dep. Var. in levels at [90, 100)	0.11	0.01	3.28	0.57	94.55	23.83	80.66	0.39	6.94	0.74	0.25	0.02
Observations	50,264	50,264	50,264	50,264	28,865	28,846	28,857	27,815	28,012	28,865	28,865	28,865

Notes: This table reports estimates of β from local linear regression of Equation (1) with bandwidth 10 mg/dL and uniform kernel. The running variable is baseline fasting blood sugar. Cohort-fixed effects are controlled. We also control for gender, age group dummies, residential area dummies, insurance type dummies, income deciles dummies, and previous year total medical expenditure. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

Table A.5: Impact of obesity screening: with control variables

	(1) # of outpatient visits for obesity	(2) # of obesity medication days	(3) Future screening participation	(4) Change in BMI	(5) Change in waist circumference	(6) Change in basic exercise	(7) Change in # of drinks per week	(8) Change in # of cigarettes per day	(9) Change in blood sugar	(10) Low risk in future screening	(11) Medium risk in future screening	(12) High risk in future screening
<i>Panel A. High risk cutoff at BMI of 25</i>												
<i>Panel A.1 Short-run impact</i>												
RD estimate	0.007 (0.005)	0.100 (0.119)	0.007 (0.013)	-0.078+ (0.041)	-0.327 (0.209)	-0.017 (0.024)	-0.745 (0.476)	-0.009 (0.198)	-0.736 (0.982)	0.011+ (0.006)	-0.026 (0.016)	0.015 (0.015)
Mean of the Dep. Var. in levels at [24, 25)	0.001	0.01	0.75	24.49	86.33	0.37	6.61	3.28	104.03	0.08	0.72	0.20
Observations	15,034	15,034	15,034	11,454	11,453	11,295	11,250	11,194	11,454	11,453	11,453	11,453
<i>Panel A.2 Long-run impact</i>												
RD estimate	0.003 (0.003)	0.121 (0.109)	0.002 (0.013)	-0.103 (0.068)	-0.423 (0.261)	-0.009 (0.026)	-0.411 (0.731)	-0.009 (0.245)	-2.682* (1.342)	0.011 (0.011)	-0.004 (0.018)	-0.007 (0.019)
Mean of the Dep. Var. in levels at [24, 25)	0.002	0.02	0.49	24.51	86.23	0.38	7.20	3.49	104.70	0.10	0.69	0.22
Observations	14,852	14,852	14,852	7,298	7,297	7,158	7,145	7,103	7,298	7,297	7,297	7,297
<i>Panel B. Medium risk cutoff at BMI of 23</i>												
<i>Panel B.1 Short-run impact</i>												
RD estimate	-0.001 (0.001)	-0.002 (0.009)	-0.001 (0.006)	0.010 (0.019)	0.223** (0.076)	0.012 (0.008)	0.113 (0.185)	0.027 (0.083)	-0.254 (0.297)	-0.047** (0.010)	0.051** (0.010)	-0.004* (0.002)
Mean of the Dep. Var. in levels at [22, 23)	0.002	0.02	0.78	22.58	77.80	0.38	6.42	3.47	96.09	0.65	0.35	0.01
Observations	80,068	80,068	80,068	62,608	62,599	61,449	61,474	61,228	62,600	62,598	62,598	62,598
<i>Panel B.2 Long-run impact</i>												
RD estimate	0.0004 (0.001)	-0.008 (0.027)	0.006 (0.005)	-0.001 (0.028)	0.102 (0.116)	0.009 (0.013)	0.073 (0.217)	-0.025 (0.115)	-0.188 (0.357)	-0.029** (0.011)	0.034** (0.011)	-0.004 (0.003)
Mean of the Dep. Var. in levels at [22, 23)	0.002	0.04	0.54	22.71	78.24	0.39	6.52	3.55	96.61	0.59	0.40	0.01
Observations	79,554	79,554	79,554	43,684	43,682	42,680	42,790	42,627	43,679	43,681	43,681	43,681

Notes: This table reports estimates of β from local linear regression of Equation (1) with bandwidth 1kg/m^2 and uniform kernel. The running variable is baseline BMI. Cohort-fixed effects are controlled. We also control for gender, age group dummies, residential area dummies, insurance type dummies, income deciles dummies, and previous year total medical expenditure. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

Table A.6: Impact of hyperlipidemia screening: with control variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	# of outpatient visits for hyperlipidemia	Taking high cholesterol medication	# of hyperlipidemia medication days	Future screening participation	Change in LDL cholesterol	Change in BMI	Change in waist circumference	Change in basic exercise	Change in # of drinks per week	Low risk in future screening	Medium risk in future screening	High risk in future screening
<i>Panel A. High risk cutoff at LDL cholesterol of 160</i>												
<i>Panel A.1 Short-run impact</i>												
RD estimate	0.181** (0.054)	0.002 (0.007)	1.852 (1.604)	-0.005 (0.005)	-0.824 (0.532)	-0.012 (0.021)	0.052 (0.122)	0.010 (0.018)	-0.042 (0.211)	0.010 (0.007)	-0.007 (0.012)	-0.003 (0.010)
Mean of the Dep. Var. in levels at [150, 160)	0.91	0.15	25.25	0.77	138.98	24.37	81.87	0.38	5.52	0.35	0.42	0.23
Observations	25,322	25,322	25,322	25,322	19,462	19,571	19,562	19,247	19,223	19,462	19,462	19,462
<i>Panel A.2 Long-run impact</i>												
RD estimate	0.159 (0.109)	0.002 (0.007)	2.352 (2.619)	0.012* (0.005)	-1.293 (0.858)	0.078+ (0.044)	0.340 (0.223)	0.043 (0.029)	0.622+ (0.313)	0.003 (0.013)	-0.004 (0.012)	0.001 (0.012)
Mean of the Dep. Var. in levels at [150, 160)	1.48	0.20	48.73	0.53	138.25	24.44	82.16	0.38	5.87	0.37	0.39	0.24
Observations	25,164	25,164	25,164	25,164	13,197	13,238	13,237	12,955	12,956	13,197	13,197	13,197

Notes: This table reports estimates of β from local linear regression of Equation (1) with bandwidth 10 mg/dL and uniform kernel. The running variable is baseline LDL cholesterol. Cohort fixed effects are controlled. We also control for gender, age group dummies, residential area dummies, insurance type dummies, income deciles dummies, and previous year total medical expenditure. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

Table A.7: McCrary test

(a) Fasting blood sugar										
Cutoff	Bandwidth									
	2	4	6	8	10	12	14	16	18	20
95	0.48	0.31	0.57	0.07	-0.99	<u>-2.64</u>	<u>-4.81</u>	<u>-7.39</u>	<u>-10.30</u>	<u>-13.37</u>
100	1.04	0.01	-0.55	-1.89	-3.63	-6.07	-9.37	-13.41	-18.22	-23.62
110	2.17	1.95	2.28	2.48	2.89	<u>3.27</u>	<u>3.78</u>	<u>3.96</u>	<u>3.37</u>	<u>1.78</u>
115	-0.18	-0.48	0.10	0.19	0.86	<u>1.99</u>	<u>3.83</u>	<u>6.10</u>	<u>8.87</u>	<u>11.84</u>
120	2.44	2.54	2.13	2.35	2.87	<u>3.74</u>	<u>4.80</u>	<u>6.79</u>	<u>9.79</u>	<u>14.00</u>
125	0.54	0.17	0.12	-0.29	-0.23	<u>0.19</u>	<u>1.08</u>	<u>2.70</u>	<u>4.88</u>	<u>8.14</u>
126	-0.55	-1.00	-1.18	-1.31	-1.08	-0.56	0.36	1.79	3.96	6.97
130	0.32	0.58	1.31	1.96	2.45	<u>2.97</u>	<u>3.60</u>	<u>4.61</u>	<u>6.14</u>	<u>8.32</u>

(b) BMI										
Cutoff	Bandwidth									
	0.2	0.4	0.6	0.8	1.0	1.2	1.4	1.6	1.8	2.0
<i>Panel A. No abdominal obesity sample</i>										
21	20.50	14.24	10.95	9.21	7.66	6.86	6.25	5.89	5.67	5.77
22	-9.80	-7.07	-6.23	-4.22	-3.53	<u>-2.66</u>	<u>-2.10</u>	<u>-1.46</u>	<u>-0.89</u>	<u>-0.46</u>
23	-14.05	-11.77	-9.98	-8.31	-7.08	-6.20	-5.78	-5.50	-4.96	-4.60
24	-13.73	-10.54	-8.90	-7.44	-6.73	<u>-6.19</u>	<u>-5.83</u>	<u>-5.67</u>	<u>-5.57</u>	<u>-5.49</u>
25	9.72	6.66	5.62	5.37	4.92	<u>4.27</u>	<u>3.66</u>	<u>3.06</u>	<u>2.29</u>	<u>1.26</u>
26	15.20	10.19	8.28	6.60	5.66	<u>4.67</u>	<u>3.75</u>	<u>2.70</u>	<u>1.86</u>	<u>0.84</u>
27	7.39	8.85	6.78	5.73	5.18	<u>5.07</u>	<u>4.94</u>	<u>4.61</u>	<u>4.29</u>	<u>3.90</u>
<i>Panel B. Abdominal obesity sample</i>										
21	1.36	1.26	1.15	1.07	0.84	0.36	-0.31	-1.00	-1.71	-2.37
22	-2.73	-1.55	-0.69	-0.14	0.33	0.49	0.29	-0.22	-0.98	-1.82
23	-2.52	-1.64	-1.16	-0.96	-0.54	-0.21	-0.16	-0.30	-0.34	-0.47
24	-4.02	-2.23	-1.95	-1.67	-1.68	<u>-1.55</u>	<u>-1.36</u>	<u>-1.00</u>	<u>-0.55</u>	<u>0.07</u>
25	4.38	2.10	2.13	1.95	1.75	1.84	1.94	2.37	2.91	3.42
26	11.23	6.69	5.71	5.06	4.37	<u>4.24</u>	<u>4.11</u>	<u>3.95</u>	<u>4.25</u>	<u>4.32</u>
27	4.87	6.03	4.18	3.98	3.33	<u>2.71</u>	<u>2.55</u>	<u>1.87</u>	<u>1.75</u>	<u>1.36</u>

(c) LDL cholesterol										
Cutoff	Bandwidth									
	2	4	6	8	10	12	14	16	18	20
130	0.41	-0.83	-1.77	-2.49	-3.11	-3.75	-4.18	-4.42	-4.71	-4.99
140	0.74	0.68	0.39	0.34	0.41	0.69	1.08	1.30	1.28	1.03
150	0.90	1.45	1.76	1.51	1.20	<u>1.10</u>	<u>0.93</u>	<u>0.63</u>	<u>0.32</u>	<u>0.04</u>
160	-0.23	-0.97	-1.46	-1.51	-1.22	-1.24	-1.27	-1.24	-1.29	-1.31
170	1.28	1.42	1.43	1.46	1.44	<u>1.52</u>	<u>1.57</u>	<u>1.38</u>	<u>1.13</u>	<u>0.99</u>
180	-2.23	-1.14	-0.62	-0.47	-0.29	-0.05	0.14	0.36	0.67	1.01
190	0.85	0.42	0.01	-0.24	-0.32	-0.48	-0.55	-0.55	-0.57	-0.46

Notes: These tables present the t-statistics of the McCrary test for the smoothness of frequency density at different cutoffs and bandwidths for three running variables, fasting blood sugar, BMI, and LDL cholesterol (McCrary, 2008). Cutoffs that are printed in bold represent the treatment cutoffs for each sample and others are placebo cutoffs. For fasting blood sugar and LDL cholesterol, which are only available as integers, bin size 1 is used. For BMI, we used the default bin size calculations in the McCrary test (bin size = 0.01). If there are manipulations at the treatment cutoffs, placebo tests which includes the treatment cutoff within bandwidth (underlined cells) could be problematic and thus should be interpreted with caution.

Table A.8: Impact of diabetes screening by diabetes medication

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Future screening participation	Change in blood sugar	Change in BMI	Change in waist circumference	Change in basic exercise	Change in # of drinks per week	Low risk in future screening	Medium risk in future screening	High risk in future screening
Panel A. No diabetes medication during or one year after baseline									
<i>Panel A.1 Short-run impact of high risk cutoff at fasting blood sugar of 126</i>									
RD estimate	-0.018 (0.024)	-0.062 (2.117)	-0.220** (0.062)	-1.346** (0.300)	0.037 (0.035)	0.924 (1.380)	-0.010 (0.038)	0.051 (0.031)	-0.041 (0.039)
Mean of the Dep. Var. in levels at [116, 126)	0.79	110.53	24.65	83.79	0.39	9.96	0.29	0.55	0.16
Observations	3,938	3,086	3,081	3,083	2,989	3,005	3,086	3,086	3,086
<i>Panel A.2 Long-run impact of high risk cutoff at fasting blood sugar of 126</i>									
RD estimate	0.012 (0.027)	-1.995 (3.553)	-0.125 (0.118)	-0.046 (0.381)	-0.020 (0.053)	0.477 (1.050)	0.004 (0.040)	0.083* (0.032)	-0.087 (0.053)
Mean of the Dep. Var. in levels at [116, 126)	0.58	112.46	24.59	83.60	0.40	10.00	0.32	0.47	0.21
Observations	3,912	2,293	2,289	2,288	2,203	2,224	2,293	2,293	2,293
Panel B. Took diabetes medication during or one year after baseline									
<i>Panel B.1 Short-run impact of high risk cutoff at fasting blood sugar of 126</i>									
RD estimate	-0.039 (0.046)	-6.827 (4.671)	0.126 (0.267)	0.583 (0.468)	-0.290** (0.068)	-2.863 (3.391)	0.106* (0.046)	-0.007 (0.045)	-0.099 (0.075)
Mean of the Dep. Var. in levels at [116, 126)	0.80	125.24	24.95	84.74	0.43	7.70	0.15	0.45	0.40
Observations	704	562	562	561	554	550	562	562	562
<i>Panel B.2 Long-run impact of high risk cutoff at fasting blood sugar of 126</i>									
RD estimate	0.075 (0.044)	-11.696+ (6.231)	0.096 (0.213)	-0.040 (0.867)	0.212** (0.063)	-3.922 (2.830)	-0.008 (0.081)	0.200** (0.066)	-0.192* (0.075)
Mean of the Dep. Var. in levels at [116, 126)	0.51	125.52	24.65	84.31	0.46	7.08	0.13	0.45	0.42
Observations	689	369	369	368	359	361	369	369	369

Notes: This table reports estimates of β from local linear regression of Equation (1) with bandwidth 10 mg/dL and uniform kernel. The running variable is baseline blood sugar. Shown in parentheses are standard errors which are clustered at the unique value of the running variable. **, *, and + indicate statistical significance at the 1%, 5%, and 10% levels, respectively.