

SPECIAL ARTICLE

Adolescent Overweight and Future Adult Coronary Heart Disease

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ABSTRACT

BACKGROUND

The effect of adolescent overweight on future adult coronary heart disease (CHD) is not known.

METHODS

We estimated the prevalence of obese 35-year-olds in 2020 on the basis of adolescent overweight in 2000 and historical trends regarding overweight adolescents who become obese adults. We then used the CHD Policy Model, a state-transition computer simulation of U.S. residents who are 35 years of age or older, to project the annual excess incidence and prevalence of CHD, the total number of excess CHD events, and excess deaths from both CHD and other causes attributable to obesity from 2020 to 2035. We also modeled the effect of treating obesity-related increases in blood pressure and dyslipidemia.

RESULTS

Adolescent overweight is projected to increase the prevalence of obese 35-year-olds in 2020 to a range of 30 to 37% in men and 34 to 44% in women. As a consequence of this increased obesity, an increase in the incidence of CHD and in the total number of CHD events and deaths is projected to occur in young adulthood. The increase is projected to continue in both absolute and relative terms as the population reaches middle age. By 2035, it is estimated that the prevalence of CHD will increase by a range of 5 to 16%, with more than 100,000 excess cases of CHD attributable to the increased obesity. Aggressive treatment with currently available therapies to reverse modifiable obesity-related risk factors would reduce, but not eliminate, the projected increase in the number of CHD events.

CONCLUSIONS

Although projections 25 or more years into the future are subject to innumerable uncertainties, extrapolation from current data suggests that adolescent overweight will increase rates of CHD among future young and middle-aged adults, resulting in substantial morbidity and mortality.

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N Engl J Med 2007;357:2371-9.

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OBESITY HAS BECOME A MAJOR PUBLIC health issue in the United States; children and adolescents are substantially affected.

Since 1970, the prevalence of overweight among children between the ages of 2 and 5 years has doubled, and that of children and adolescents between the ages of 6 and 19 years has tripled. More than 9 million children and adolescents (17%) are now considered to be overweight.¹⁻⁴

An elevated body-mass index (BMI, the weight in kilograms divided by the square of the height in meters) is associated with several risk factors for coronary heart disease (CHD), including hypertension, dyslipidemia, and diabetes.⁵ Since overweight adolescents are likely to become obese adults,⁶⁻⁸ the high prevalence of adolescent overweight might be expected to increase future rates of CHD in adults. Using the CHD Policy Model, we estimated the potential effect of adolescent overweight on future adult CHD. We further examined whether the expected increase in the number of CHD events might be reversed by treatment of modifiable obesity-related risk factors for CHD.

METHODS

STRUCTURE OF THE MODEL

The CHD Policy Model is a computer-simulation, state-transition (Markov cohort) model of the incidence, prevalence, mortality, and costs associated with CHD in U.S. residents who are 35 years of age or older.⁹⁻¹¹ The demographic-epidemiologic submodel predicts the incidence of CHD and death from other causes among subjects without CHD, stratified according to age, sex, and as many as six categorized risk factors, including diastolic blood pressure, smoking status, levels of high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol, BMI, and the presence or absence of diabetes mellitus.

After CHD develops, the bridge submodel characterizes the initial CHD event (cardiac arrest, myocardial infarction, or angina) and its sequelae for 30 days. Then, the disease-history submodel predicts the number of subsequent CHD events, revascularization procedures, and deaths from CHD and other causes among subjects with CHD, stratified according to age, sex, and history of events. All population distributions, risk-factor levels, coefficients, event rates, case fatality rates,

costs, and quality-of-life adjustments can be modified for forecasting simulations.

DATA SOURCES

Version 3 of the CHD Policy Model includes data from prior versions⁹⁻¹¹ as well as many updates and upgrades. Data sources include U.S. Census data and projections,^{12,13} National Center for Health Statistics mortality data,¹⁴⁻¹⁶ the Framingham Heart Study data for the incidence of CHD as a function of risk factors,¹⁷⁻²¹ and Olmsted County data for the incidence of myocardial infarction and cardiac arrest.²² The risks associated with smoking and environmental tobacco exposure have been updated to reflect more recent investigations.^{18,23} Rates of myocardial infarction, hospitalization for cardiac arrest, revascularization procedures, and associated case fatalities were estimated from the National Hospital Discharge Survey and related published reports^{24,25}; 30-day survival rates were based on data from Medicare, from California, and from Seattle.²⁶⁻²⁸ The background prevalence of CHD in 2000 was estimated from the National Health Interview Survey.²⁹ Our model closely replicates that showing the reduction in coronary events with statins in primary and secondary prevention trials.³⁰⁻³² (For details, see the Supplementary Appendix, available with the full text of this article at www.nejm.org.)

OBESITY ESTIMATES

We defined adolescent overweight as a weight above the 95th percentile on the growth charts of the Centers for Disease Control and Prevention³³ and defined adult obesity as a BMI of 30 or more. We used data from the National Health and Nutrition Examination Survey (NHANES) I (1971-1974), NHANES II (1976-1980), NHANES III (1988-1994), and NHANES IV (1999-2000) to determine the proportion of adolescents between the ages of 12 and 19 years who were above the 95th percentile for weight and the proportion of obese 35-year-old men and women.^{2,4} Data from previous NHANES cohorts and elsewhere^{3,4,6} provided a linear function that appeared to give the best estimate of the rate at which overweight adolescents become obese adults 20 years later on the basis of previous NHANES cohorts. Separate linear functions for the average trends and for the highest and lowest trends on the basis of

historical data were applied to the proportion of overweight adolescents in NHANES IV to project the proportion of obese 35-year-old men and women in 2020.

We implemented the change of prevalence as a shift in the distribution of BMI, with the magnitude of the shift equal to the projected increase in mean BMI. From age 35 on, we applied the transition probabilities derived from the BMI distribution to simulate the natural increase in BMI that occurs with age. For each historical trend, we modeled separate projections of future obesity, including average, high, and low projections.

On the basis of our analysis of data from the Framingham Heart Study and other epidemiologic data, we did not assign a CHD risk function to obesity but assumed that increased BMI results in higher rates of CHD primarily through its effects on diastolic blood pressure, LDL and HDL cholesterol, and diabetes. We assumed that BMI affects mortality from causes unrelated to CHD primarily through diastolic blood pressure and diabetes. In sensitivity analyses, we also modeled an additional increase in mortality from cancer that has been attributed to obesity in some studies.^{34,35} The distribution of these risk factors was modeled as a shift in their means that reflected their observed relationship to BMI (Table 1). The resulting relative risk of death from any cause attributable to obesity in our model is within 0.5 sigma of the relative risk predicted by Flegal et al.⁴⁰

SIMULATIONS

We modeled multiple, successive cohorts of 35-year-olds from 2020 to 2035 to determine the estimated effect on CHD events under each of the three assumptions about future adult obesity in this age group. We estimated the absolute and relative annual excess in the incidence of CHD (new angina, first myocardial infarction, cardiac arrest, and death from CHD), in the prevalence of CHD, and in the total number of CHD events (myocardial infarctions, revascularization procedures, cardiac arrests, and deaths from CHD) and deaths from CHD and from other causes; these results are reported graphically.

We estimated how the increase in the number of CHD events might be ameliorated by projecting the elimination of obesity-related increases in diastolic blood pressure and LDL cholesterol

Table 1. Projected Changes in Cardiovascular Risk Factors per 1-Point Increase in the Body-Mass Index (BMI).*

Risk Factor	Change per 1-Point Increase in BMI (95% CI)	
	Men	Women
Diastolic blood pressure (mm Hg)	0.90 (0.76 to 1.04)	0.74 (0.63 to 0.84)
Cholesterol (mg/dl)		
Low-density lipoprotein	2.75 (1.44 to 3.67)	2.24 (0.54 to 3.36)
High-density lipoprotein	-1.55 (-1.93 to -1.16)	-0.77 (-1.16 to -0.39)
Relative risk of diabetes†	2.1 (1.2 to 3.6)	1.9 (1.5 to 2.3)

* Data are adapted from Wilsgaard et al.,³⁶ Wilsgaard and Arnesen,³⁷ Koh-Banerjee et al.,³⁸ and Colditz et al.³⁹ Calculations of the BMI are based on average heights for men and women in the United States.

† The relative risks are associated with a weight gain of 7 to 11 kg over 5 years for men and a gain of 5 to 8 kg over 14 years for women, as compared with stable weight.

among all subjects. Although the ability to raise HDL cholesterol safely through lifestyle and pharmacologic means is less clear, we also modeled the additional effect of reversing obesity-related decreases in HDL cholesterol.

STATISTICAL ANALYSIS

For each simulation, we determined the uncertainty associated with our estimates with the use of Monte Carlo simulations. Beta coefficients for the association of diastolic blood pressure, LDL and HDL cholesterol, and diabetes with both CHD events and deaths not associated with CHD were assumed to have a normal probability distribution, with standard errors derived from the fitted regression. We generated covariance matrixes for each of these beta coefficients, and on the basis of evidence for minimal correlation between factors, we assumed effects to be independent. For each simulation, we report the mean (±SE) for 1000 simulations. We also examined the effect of varying our assumptions for the association between increases in BMI and changes in obesity-related risk factors at the extremes of the 95% confidence interval for key estimates (Table 1).

RESULTS

The prevalence of adolescent overweight in 2000 was 16.7% in boys and 15.4% in girls.² By the time these adolescents turn 35 years old in 2020, the proportion of obese 35-year-olds is projected

to be 30 to 37% in men (as compared with 25% now) and 34 to 44% in women (as compared with 32% now). This increase in obesity is predicted to result in a higher prevalence of elevated diastolic blood pressure, elevated LDL cholesterol levels, decreased HDL cholesterol levels, and diabetes (Table 2).

The higher predicted prevalence of obesity among future 35-year-olds is projected to increase the rates of annual CHD events and events not associated with CHD (Fig. 1), with the absolute number of excess events rising with each year from 2020 to 2035. The steepest rise is projected in the total number of CHD events, with 550 absolute excess events in 2020 (an excess of 10%) increasing to 33,000 excess events in 2035 (an excess of 14%). On the basis of historical trends, low projections call for 250 excess CHD events in 2020 (an excess of 4%) increasing to 14,000 in 2035 (an excess of 6%); high projections call for 770 excess events in 2020 (an excess of 13%) increasing to 45,000 in 2035 (an excess of 19%).

The annual excess in the incidence of CHD is projected to rise from 1600 in 2020 to 40,000 in 2035, an excess of 15% over the incidence that would have been expected without the increase in future obesity. Low projections call for a rise in the annual excess in incidence of CHD from 740 in 2020 to 16,000 in 2035 (an excess of 7%); high projections call for a rise in the annual excess in incidence from 2300 in 2020 to 55,000 in 2035 (an excess of 21%).

The number of excess deaths from CHD is

projected to rise from 59 in 2020 (an excess of 9%) to 3600 in 2035 (an excess of 13%). Low projections call for an increase in the number of CHD deaths from 26 in 2020 (an excess of 4%) to 1500 in 2035 (an excess of 6%); high projections call for an increase in the number of CHD deaths from 84 in 2020 (an excess of 13%) to 5000 in 2035 (an excess of 19%).

Deaths that are associated with obesity but not with CHD are projected to increase by 4% each year, with the absolute number rising from an excess of 250 in 2020 to 6200 in 2035. Low projections call for an increase of 2%, from an excess of 120 events in 2020 to an excess of 2600 events in 2035; high projections call for an increase of 6%, from an excess of 380 events in 2020 to an excess of 8900 events in 2035. An additional increase in obesity-related deaths from cancer would result in an additional 0.4% increase in deaths that are not related to CHD, without substantially affecting the rate of death from CHD.

In addition to varying projections about future adult obesity, varying assumptions about the relationship between obesity and obesity-related risk factors would affect the projections of future events related to CHD and to other causes. For example, the use of the highest boundary of the 95% confidence intervals for all the risk factors for the highest estimate of the projected rate of obesity would result in a 43% increase in the number of excess events. At the other extreme, the use of the lowest boundary of the 95% confidence intervals for this projection would

Table 2. Estimated Prevalence of Cardiovascular Risk Factors among 35-Year-Old Men and Women in 2020, According to the Presence or Absence of a Projected Increase in Adult Obesity Associated with Adolescent Overweight.

Variable	Proportion with Risk Factor			
	No Projected Increase in Future Obesity		Projected Increase in Future Obesity (Low-to-High Projections)	
	Men	Women	Men	Women
	<i>percent</i>			
Body-mass index ≥ 30	25	32	34 (30–37)	38 (34–44)
Diastolic blood pressure ≥ 90 mm Hg	7	3	9 (8–9)	4 (3–4)
Cholesterol				
Low-density lipoprotein ≥ 130 mg/dl	42	27	46 (44–47)	32 (30–34)
High-density lipoprotein < 35 mg/dl	20	6	25 (23–28)	7 (6–8)
Diabetes	3.1	2.5	3.3 (3.2–3.4)	2.8 (2.6–3.0)

decrease the number of excess events by 40%, as compared with that estimated by the lowest projected rate of obesity.

The projected prevalence of CHD increases as this young population ages (Fig. 2). The higher prevalence of obesity among 35-year-olds would be expected to increase the overall prevalence of CHD by 2% in 2021 and by 11% in 2035. During those years, low projections call for an increase of 1% and 5%, respectively; high projections call for an increase of 3% and 16%, respectively. Under

each of the three projections of future obesity, the excess prevalence of CHD that could be attributed to adolescent overweight would be expected to exceed 100,000 events by 2035 or before.

Reversing the obesity-related increase in diastolic blood pressure and LDL cholesterol could potentially blunt the increase in events from both CHD and other causes (Fig. 3). Successful treatment that could raise HDL cholesterol levels safely and provide a corresponding decrease in CHD events would result in a substantially greater

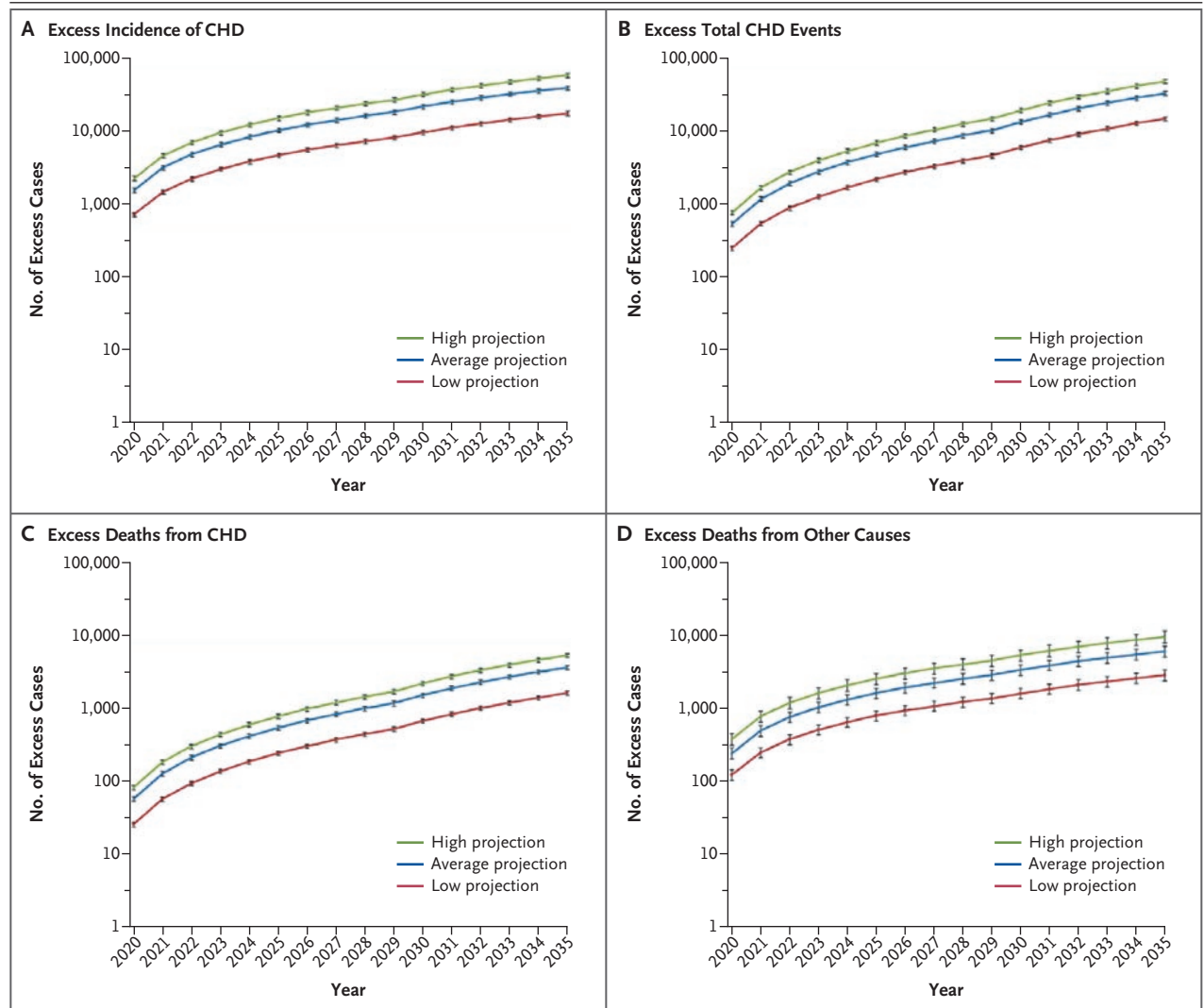


Figure 1. Annual Excess Coronary Heart Disease (CHD) Events from 2020 to 2035 Associated with Three Projections of Future Adult Obesity.

High rates of current adolescent overweight are expected to increase the excess incidence of CHD, including new angina, first myocardial infarction, and death from CHD (Panel A); of the total number of CHD events, including myocardial infarction, cardiac arrest, coronary revascularization procedure, and death from CHD (Panel B); and of death from CHD (Panel C) and from other causes (Panel D). Curves are shown for average projections, low projections, and high projections.

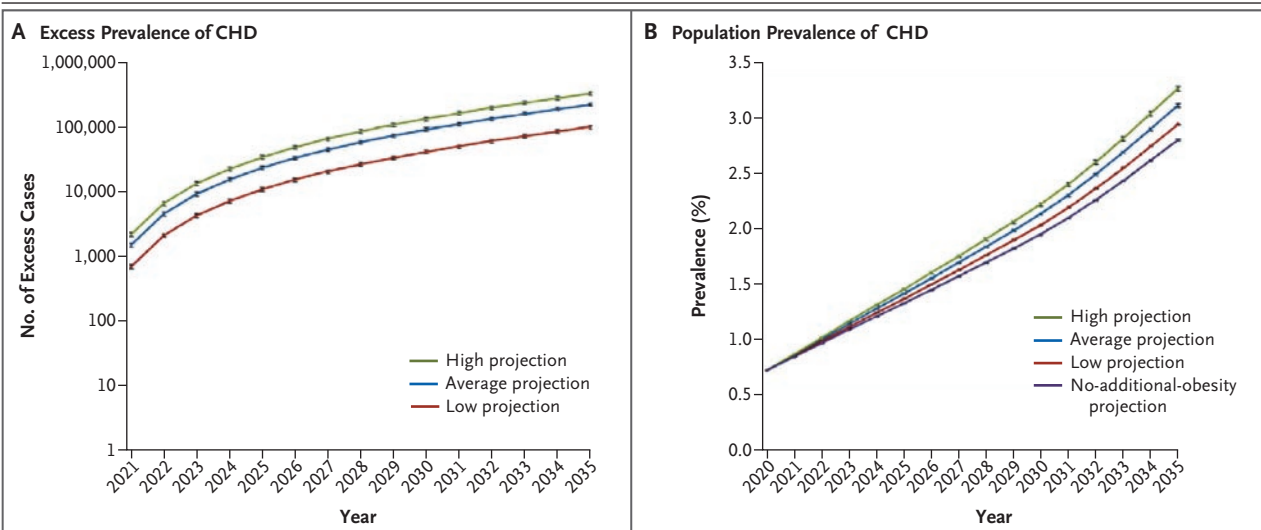


Figure 2. Prevalence of Coronary Heart Disease (CHD) Associated with Three Projections of Future Adult Obesity.

High rates of current adolescent overweight are expected to increase the excess prevalence of CHD (Panel A) and to increase the overall population prevalence of CHD (Panel B).

reduction in morbidity and mortality from CHD, particularly at younger ages. Nevertheless, the projected number of events associated with CHD and other causes would remain elevated because of the persistent risk of diabetes associated with obesity.

DISCUSSION

Projections 25 or more years into the future are notoriously unreliable because many factors that are important to the projection may change in the interim. For example, U.S. population estimates may be affected by trends that we do not model, including future immigration. Our estimates also would differ greatly if new treatments substantially changed obesity trends or the prevention and treatment of CHD. Just as medicine has changed dramatically in the past several decades, new treatments are likely to change it sufficiently in the future to make any current projections speculative.

Nevertheless, on the basis of current treatments, data, and trends, we project that the current epidemic of adolescent overweight will substantially increase future rates of adult CHD unless other changes intervene. Significant morbidity and mortality are projected to begin in young adulthood, resulting in more than 100,000 excess cases of CHD by 2035, even with the most

modest projection of future obesity. Aggressive treatment with currently available therapies to reverse obesity-related risk factors could mitigate, though not eliminate, the increase in CHD events; the remaining increased risk of diabetes associated with obesity is projected to continue to result in higher rates of events from CHD and from other causes. Our projections suggest that barring a major advance in the treatment of either excessive weight gain itself or its associated alterations in blood pressure, lipid levels, and glucose metabolism, current adolescent overweight will have a substantial effect on public health far into the future.

Overweight in adolescence can result in immediate adverse effects on health before adulthood.⁴¹⁻⁴³ In addition, most overweight teenagers continue to have an elevated BMI in young adulthood,^{6,8} with studies estimating that 80% of overweight adolescents become obese adults.⁸ We project that the effect of adolescent overweight on future adult CHD will be substantial even in young adulthood and will continue to rise in middle age. The expected higher rates of hospitalizations, procedures, disability, long-term use of medications, and premature death in a working-age population that would otherwise be at low risk for CHD could be dramatic.

On the basis of analysis of Framingham data and observations from other cohort studies,⁴⁴

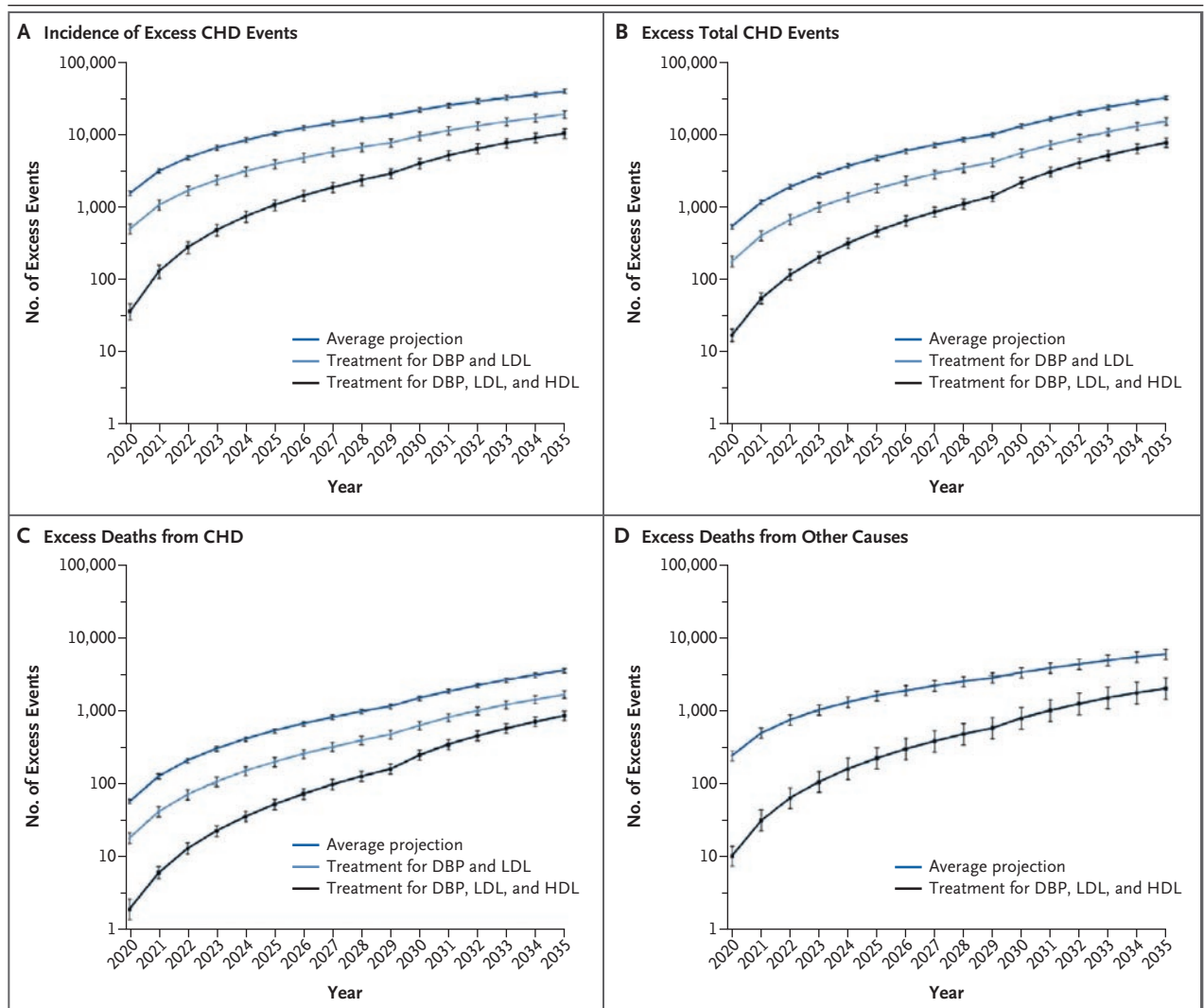


Figure 3. Effect of Treatment for Obesity-Related Risk Factors for Coronary Heart Disease (CHD) on Annual Excess CHD Events from 2020 to 2035 Associated with Future Adult Obesity.

Various treatments to reverse obesity-related changes in diastolic blood pressure (DBP), LDL cholesterol (LDL), and HDL cholesterol (HDL) are projected to lower the annual excess incidence of CHD events, including new angina, first myocardial infarction, and death from CHD (Panel A); of the total number of CHD events, including myocardial infarction, cardiac arrest, revascularization procedure, and death from CHD (Panel B); and of the number of deaths from CHD (Panel C) and from other causes (Panel D). In Panel D, only two lines can be seen because the curve for treatment of DBP and LDL is superimposed over the curve for treatment of both these factors plus HDL.

we assumed that BMI does not directly increase the risk of CHD but, rather, is associated with adverse changes in other risk factors for CHD. We found that reversing the obesity-related effect on diastolic blood pressure and LDL cholesterol could blunt the increase in CHD events associated with obesity. Additional treatment to raise HDL cholesterol levels would have an even greater effect on CHD rates, particularly in young

adults, although current therapies have only limited efficacy in raising HDL cholesterol levels safely. On the basis of therapies that are currently available, we project that aggressive treatment of hypertension and dyslipidemia in young adulthood will be required to lower the risk of CHD that is associated with obesity. A reversal of this risk will require additional measures to address the remaining obesity-related risk of dia-

betes. We do not account for possible future advances that may either reverse weight gain in young adults or effectively treat the obesity-related factors that increase the risk of CHD. Our projections would suggest that the magnitude of effect for such new treatments would need to be substantial, and such treatments would probably need to be initiated early in adulthood to stave off CHD that would otherwise develop in obese young adults.

Many of our modeling assumptions may lead to conservative estimates of the true future effect of adolescent overweight. For example, we did not directly model atherogenesis that may occur in children and adolescents as a result of their overweight. Since elevations in risk factors for CHD and markers of atherosclerotic disease are observed in obese children, our estimates for disease rates in adults as the consequence of these processes in childhood may be underestimated.^{5,45} Also, we did not model any additional increases in BMI in adulthood beyond those expected in an aging population. Obesity is on the rise in adults as well as adolescents,³ and the overall effect of the general obesity epidemic may be greater than our projections of the effect of the adolescent epidemic alone.

Several limitations should be noted in the interpretation of our projections. First, we based our estimates on the effects of risk factors from the Framingham data. These data have been useful for explaining past changes in CHD,¹⁰ but we cannot guarantee similar reliability going forward. Second, we recognize that the current higher prevalence of obesity does not appear to be associated with the expected increase in hypertension and dyslipidemia.⁴⁶ However, we believe that the treatment of these risk factors has

blunted the otherwise expected increase. Our projections suggest that additional aggressive treatment of hypertension and dyslipidemia to levels below those in obese persons would be required to reduce rates of CHD. In addition, the current higher prevalence of obesity has been associated with a rise in diabetes that our projections suggest would still need to be addressed if the effect of obesity on future CHD is to be reversed. Third, our model is designed to estimate average effects across the entire U.S. population. Differences that occur at the extremes or in subgroups of the population may therefore be missed or understated in our analysis. For example, a very high BMI (>40) may be associated with a different pattern of CHD risk factors or may be directly linked to a risk of CHD.⁴⁴ Also, increases in overweight are markedly higher in certain populations, particularly black and Hispanic adolescents.³ Our projections, which represent average increases, will underestimate the effect of adolescent overweight on these populations.

Although projections 25 or more years into the future must be interpreted with great caution, currently available data and trends suggest that overweight among adolescents can be projected to cause substantial increases in the rate and the effect of CHD among future young and middle-aged adults over the next 20 years. A number of interventions might blunt or offset this projection, but reducing overweight among adolescents can be expected to yield considerable benefits in adulthood.

Supported by a grant from the Flight Attendants Medical Research Institute, a grant from the Swanson Family Fund to the University of California, San Francisco (to Dr. Goldman), and grants from the Robert Wood Johnson Foundation (Amos Medical Faculty Development Award) and the National Heart, Lung and Blood Institute (both to Dr. Bibbins-Domingo).

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