

# Behavioral Consequences of Testing for Obesity Risk

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## Abstract

**Objective:** There is considerable hope that genetic susceptibility testing will motivate behavior that can prevent or reduce the risk of complex conditions such as obesity. This study examined potential behavioral consequences of such testing.

**Methods:** Participants ( $n = 249$ ) were randomly assigned to review one of four vignettes that asked them to imagine that they had been tested for their risk of becoming overweight or obese. The experimental factors were test type (genetic versus hormone test) and risk level (increased versus average risk for obesity). Study measures included behavioral intentions and perceived behavioral control related to eating a healthy diet, as well as weight locus of control.

**Results:** Participants assigned to the increased risk conditions indicated greater intentions to eat a healthy diet compared with participants assigned to the average risk

conditions ( $P < 0.02$ ). There were significant interaction effects of risk  $\times$  perceived behavioral control ( $P < 0.02$ ) and risk  $\times$  weight locus of control ( $P < 0.003$ ) on dietary intentions. Individuals with low perceived behavioral control or an external weight locus of control who were told to imagine they were at average risk expressed significantly lower intentions to eat a healthy diet. A three-way interaction of body mass index, family history of overweight/obesity, and assigned risk level found the greatest effects of risk feedback among those who either had a family history or a higher body mass index ( $P < 0.007$ ).

**Conclusion:** This study provides preliminary evidence that testing for susceptibility to obesity may motivate healthier behavior. However, some individuals may be susceptible to a false reassurance effect after receiving test results indicating a lack of increased risk. (Cancer Epidemiol Biomarkers Prev 2005;14(6):1485–9)

## Introduction

Obesity has become an increasingly urgent threat to the health of the U.S. population. Between 1991 and 2001, the prevalence of obesity, defined as a body mass index (BMI) of  $\geq 30$ , increased from 12.1% to 20.9% of the population (1). Although genetic factors undoubtedly play a role in becoming obese, the rapid increase in prevalence suggests that environmental and behavioral factors are a driving force in the current epidemic. Obesity accounts for 14% of all cancer mortality among men over 50 and 20% of all cancer mortality among women over 50 (2). Therefore, reducing and preventing obesity in the population is a critical component of a comprehensive cancer control strategy.

Some scientists have suggested that genomic medicine will play an important role in preventing conditions such as obesity. This perspective is highlighted by the following quote from the testimony of Dr. Francis Collins, Director of the National Human Genome Research Institute, delivered to the House of Representatives in 2003:

"In the next 10 years, I expect that predictive genetic tests will exist for many common conditions in which interventions can alleviate inherited risk, so that each of us can learn of our individual risks for future illness and practice more effective health maintenance and disease prevention (3)."

Whereas a great deal of research has focused on responses of individuals to genetic information regarding disease susceptibility, little is known about whether people will respond to results from genetic tests for common complex conditions, such as obesity, in ways that fulfill this promise (4).

The purpose of the present study was to examine potential behavioral consequences of genetic testing for susceptibility to obesity. Although genetic testing for obesity risk is not yet available, genetic research in obesity may lead to susceptibility testing in the future (5). Consequently, we utilized a common vignette paradigm that asks individuals to consider different hypothetical scenarios for testing (6). We used the Theory of Planned Behavior as our conceptual model (7). Our primary dependent measure was participants' intentions to eat a "healthy diet" in the next 3 months (8). A previous study found that behavioral intentions to eat a healthy diet were a significant positive predictor of healthy dietary behavior measured 6 years later (8). We hypothesized that (a) individuals who were told they were at increased risk of becoming obese would express stronger intentions to eat a healthy diet compared with those told they were at average risk (main effect of risk status); (b) this effect would be most pronounced among those who were told to imagine the feedback was from a genetic test compared with those told that feedback was from a hormone test (risk status by test type interaction). We chose a hormone test as the comparison condition because blood is collected for both tests and the testing process (venipuncture) entails the same degree of immediate physical invasiveness, yet the hormone test does not require analysis of DNA (9). Consistent with our conceptual model, we also measured participants' perceived behavioral control over eating a healthy diet. In light of evidence suggesting the importance of this construct in engaging in healthy dietary behavior, we hypothesized that (c) the effects of risk feedback on intentions to eat a healthy diet would be lower among individuals with a low sense of control over their dietary behavior or body weight (8, 10).

## Materials and Methods

**Experimental Design.** We used a fully crossed  $2 \times 2$  factorial design that randomly assigned participants to review one of four vignettes (see Appendix 1) that asked them to

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imagine that they had been tested for their risk of becoming overweight or obese. The experimental factors were test type (genetic versus hormone test) and risk level (increased versus average risk for obesity).

**Participants and Procedure.** The study protocol was reviewed and approved by the Institutional Review Board of the University of Pennsylvania. We recruited 249 undergraduate students from the University of Pennsylvania campus who self-identified as being "average weight" and responded to flyers advertising a "weight survey." Participants were recruited campus-wide and represented a wide range of majors, including natural (e.g., biology), applied (e.g., engineering) and social sciences (e.g., economics, psychology), humanities (e.g., history), and the arts. We deemed undergraduate students an appropriate target population for this study because data describing the natural history of obesity suggest that the problem develops gradually over several years, and prevention interventions are likely to have the greatest impact when applied early in adult life (11).

Participants provided informed consent and were measured for weight and height. Individuals with a BMI <18.5 (indicating underweight) or >29.9 (indicating obesity) were excluded from participation. Participants were then exposed to a randomly selected experimental vignette and completed the study questionnaire. All participants were debriefed at the end of the study and received \$15 remuneration.

### Measures

**Background Measures.** In addition to demographics (age, gender, race, and BMI), we queried participants about their family history of overweight/obesity by asking them "As far as you know, is your mother/father overweight or obese?"

**Dependent Measures.** Intentions to eat a healthy diet were assessed as the mean of five items (e.g., "I intend to eat a healthy diet in the next 3 months";  $\alpha = 0.93$ ; ref. 8). Consistent with the Theory of Planned Behavior, we also measured participants' attitudes, perceived behavioral control, perceived social norms, and outcome expectancies related to eating a healthy diet (7, 8). Attitudes about eating a healthy diet were assessed as the mean of six semantic differential items (e.g., "My eating a healthy diet in the next 3 months would be: bad/good; foolish/wise," etc.;  $\alpha = 0.76$ ; ref. 8). Perceived behavioral control for eating a healthy diet was assessed as the mean of three items (e.g., "I am confident that if I started eating a healthy diet in the next 3 months I could keep to it";  $\alpha = 0.82$ ; ref. 8). Perceived social norms were assessed with a single item ("People who are important to me think I should eat a healthy diet in the next 3 months"; ref. 8). Outcome expectancies were also assessed with a single item ("Eating a healthy diet in the next 3 months will help me not become overweight or obese"; ref. 8). Questionnaire items were scaled from -3 (unlikely/strongly disagree) to +3 (likely/strongly agree). We measured participants' weight locus of control using a previously validated scale on which lower scores (range 4-24) indicate internality and higher scores indicate externality (12). Internality implies that an individual believes that body weight can be controlled by their own behavior, whereas externality implies that an individual believes that body weight is determined by forces beyond personal control, including chance. In addition to these measures, we assessed perceived risk of becoming overweight/obese using a seven-point Likert scale.

**Statistical Analysis.** We used factorial ANOVA to compare the dependent variables by experimental group (test type and risk level), controlling for family history of overweight/obesity and BMI. Stepwise hierarchical multiple regression analysis was used to test hypotheses concerning the effects of a low sense of control over dietary behavior and/or weight gain on

intentions to eat a healthy diet. Additional exploratory analyses were conducted to examine potential moderating effects of BMI and family history of overweight/obesity on dietary intentions.  $\alpha$  was set at 0.05 for evaluations of our primary hypotheses and 0.01 for exploratory analyses.

### Results

**Descriptive Data.** There were no differences in demographic or background variables between the four experimental groups. On average, participants were 20.5 years old (SD = 1.7) and had an average BMI of 22.8 (SD = 2.3). The majority were female (56.2%), 58.4% were identified as Caucasian, 23.3% as Asian American, 12.7% as African American, and 5.7% were indicated as "Other." Six percent reported Hispanic ethnicity. The majority reported neither parent being overweight or obese, 33.7% reported one parent as overweight or obese, and 12.4% reported both. Individuals who reported having an overweight or obese parent had higher BMI values (mean = 23.31, SD = 2.38) than those with no family history [mean = 22.45, SD = 2.12;  $t(247) = -3.02$ ,  $P < 0.01$ ].

**Experimental Manipulation Check.** Our measure of perceived risk of becoming overweight or obese allowed us to determine whether the different levels of risk for obesity (i.e., average versus increased) factor were understood by participants as we intended. We detected a significant difference, with those in the "Increased" risk conditions rating their perceived risk as higher (mean = 3.5, SD = 2.1) than those in the "Average" risk conditions [mean = 2.9, SD = 1.8;  $F_{(1,248)} = 4.86$ ,  $P < 0.03$ ]. This finding indicates that the experimental risk manipulation succeeded in conveying an increased sense of risk in the corresponding condition. There was no effect of test type (genetic versus hormone) on perceived risk. We did not include a measure to determine whether participants perceived the alternative test types differently.

A precondition to using intentions to eat a healthy diet as a meaningful outcome measure is that participants perceive that such behavior would help them avoid becoming overweight or obese. There were no between-group differences in how participants rated their outcome expectancies. On average, participants indicated a high level of agreement with the statement "Eating a healthy diet in the next 3 months will help me not become overweight or obese" (mean = 2.0, SD = 1.4, range -3 to +3).

**Effects of Risk Status and Test Type on Healthy Diet Intentions.** Controlling for family history of overweight/obesity and BMI, we found a significant effect of risk level on participants' intentions to eat a healthy diet, confirming our primary hypothesis [ $F_{(1,247)} = 5.97$ ,  $P < 0.02$ ]. Those who were told they were at increased risk of becoming overweight/obese indicated stronger intentions to eat a healthy diet (mean = 2.0, SD = 1.0) than those who were told they were at average risk (mean = 1.7, SD = 1.2). Our hypothesis that those who were given genetic test feedback would indicate stronger intentions was not confirmed. There was no effect of test type on intentions to eat a healthy diet and no interaction effect.

**Effects of Risk Status and Test Type on Intermediate Variables.** There were no differences between groups in attitudes toward eating a healthy diet (mean = 1.8, SD = 0.9) and perceived social norms (mean = 1.7, SD = 1.3). We found an interaction effect for perceived behavioral control over eating a healthy diet [ $F_{(1,247)} = 4.17$ ,  $P < 0.05$ ]. Within the genetic test group, those who were told they were at increased risk of becoming overweight/obese indicated lower perceived behavioral control (mean = 1.4, SD = 1.2) than those who were told they were at average risk (mean = 1.6, SD = 1.0). Within the hormone test group, the pattern of results was the opposite.

**Table 1. Regression analysis results for intention to eat a healthy diet**

Variable entered	B	$\Delta R^2$	Statistic
BMI	-0.507	0.009	n.s.
Family history	-0.578	0.010	n.s.
Test type	0.186	0.015	n.s.
Risk level	-0.746	0.020	$F_{(1,240)} = 5.01, P < 0.03$
Weight locus of control	-0.091	0.060	$F_{(1,239)} = 16.32, P < 0.001$
Perceived behavioral control	0.607	0.218	$F_{(1,238)} = 77.89, P < 0.001$
Risk $\times$ weight locus of control	0.102	0.028	$F_{(1,237)} = 10.29, P < 0.003$
Risk $\times$ perceived behavioral control	-0.252	0.015	$F_{(1,236)} = 5.68, P < 0.02$
Risk $\times$ BMI	0.815	0.004	n.s.
Risk $\times$ family history	0.884	0.002	n.s.
BMI $\times$ family history	0.426	0.002	n.s.
Risk $\times$ BMI $\times$ family history	-1.286	0.020	$F_{(1,232)} = 7.56, P < 0.007$

NOTE: Final model statistics,  $F_{(12,232)} = 13.02, P < 0.001; R^2 = 0.402$ .  
Abbreviation: n.s., not significant.

Those who were told they were at increased risk of becoming overweight/obese indicated higher perceived behavioral control (mean = 1.6, SD = 1.0) than those who were told they were at average risk (mean = 1.3, SD = 1.2). There were no effects of the experimental manipulation on weight locus of control. The average weight locus of control score was 9.7 (SD = 3.2). There was a significant but modest correlation between weight locus of control and perceived behavioral control for eating a healthy diet ( $r = -0.24, P < 0.001$ ).

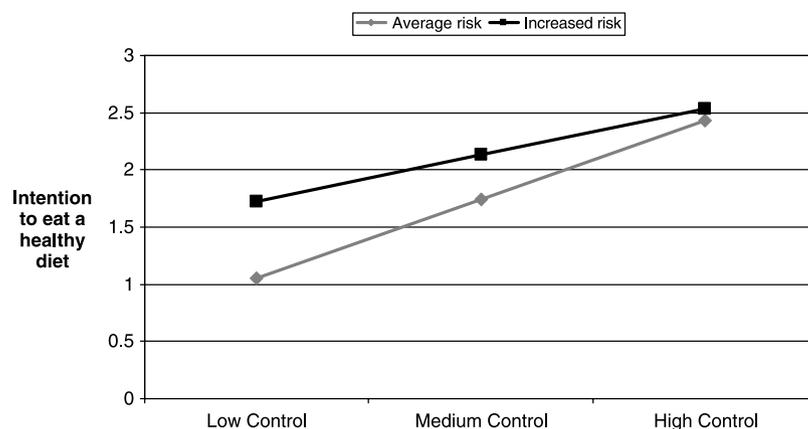
**Moderating Effects of Control on Intentions to Eat a Healthy Diet.** There were significant interactions of risk with both perceived behavioral control and weight locus of control. Detailed statistics for each step of the regression model are shown in Table 1. Results shown in Fig. 1 indicate that among individuals with a high degree of perceived behavioral control, intentions to eat a healthy diet were high regardless of experimental risk level. Among those with a low degree of perceived behavioral control, intentions to eat a healthy diet were significantly lower after receiving test results indicating average risk. Figure 2 shows that, among participants who were told they are at increased risk, intentions to eat a healthy diet did not vary by whether weight locus of control was internal or external. However, individuals who received average risk results had significantly lower intentions to eat a healthy diet if they indicated an external weight locus of control.

**Moderating Effects of BMI and Family History of Overweight/Obesity.** Building on the regression model

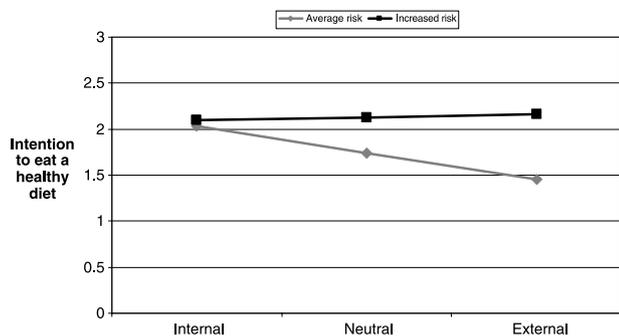
described above, we explored whether BMI and/or family history moderated the effects of our experimental conditions on intentions to eat a healthy diet. As also shown in Table 1, we found a significant three-way interaction effect of BMI, family history, and assigned risk level, after controlling for all other identified effects described above. This effect was below the more conservative  $\alpha$  threshold ( $P < 0.01$ ) set for this exploratory analysis. There were no main effects of BMI and family history on dietary intentions and no effects of test type (genetic versus hormone test) by BMI or family history. Figure 3 shows the predicted means for the three-way interaction. The effects of risk feedback (average versus increased) on intentions to eat a healthy diet were strongest for individuals who had either high BMI or a positive family history. Among individuals who had both high BMI and a positive family history, there was no effect of risk feedback and dietary intentions were lower. Among individuals with low BMI and no family history, there also seemed to be no effect of risk feedback, but intentions were substantially higher.

## Discussion

This experiment was designed to test hypotheses about the behavioral consequences of genetic feedback about obesity risk among normal weight individuals. The results indicated that individuals told to imagine that they were at increased risk (based on a genetic or hormone test) showed higher overall intentions to eat a healthy diet in the next 3 months, compared with those told to imagine they were at average risk. Hence, this study provides preliminary evidence that biological testing for obesity risk may motivate healthier dietary behavior. Participants in the genetic test condition who were told they were at increased risk expressed a lower sense of control than those at average risk, which is consistent with a sense of fatalism that may result from peoples' beliefs that genetics are immutable. This finding contrasts with a recent study that examined self-efficacy for weight loss among a group of obese individuals who tested positive for a genetic variant thought to influence weight gain and energy expenditure. Contrary to expectations, confidence and self-efficacy to lose weight were not undermined by knowledge of positive genetic status (13). However, it is important to note that this study examined self-efficacy in an intervention context among individuals who were already obese. This is substantially different from using genetic risk feedback as part of a prevention strategy. We are unable to explain why participants in the hormone test condition who were told they were at increased risk expressed a higher sense of control. It is possible that individuals perceive hormone levels to be under environmental (e.g., dietary) control, and hence a test indicating an increased risk may increase perceptions of ability to alter susceptibility.



**Figure 1.** Interaction of risk level and perceived behavioral control on intention to eat a healthy diet.



**Figure 2.** Interaction of risk level and weight locus of control on intention to eat a healthy diet.

Our regression analyses suggest that some individuals may engage in less healthy behavior after receiving results indicating average risk for obesity (compared with increased risk). Individuals with low perceived behavioral control over eating a healthy diet or an external weight locus of control had significantly lower predicted intentions to eat a healthy diet than those with high perceived behavioral control or an internal weight locus of control. This finding mirrors previous research indicating the importance of perceived behavioral control or self-efficacy in a variety of health behaviors, including those related to diet (8, 10, 14).

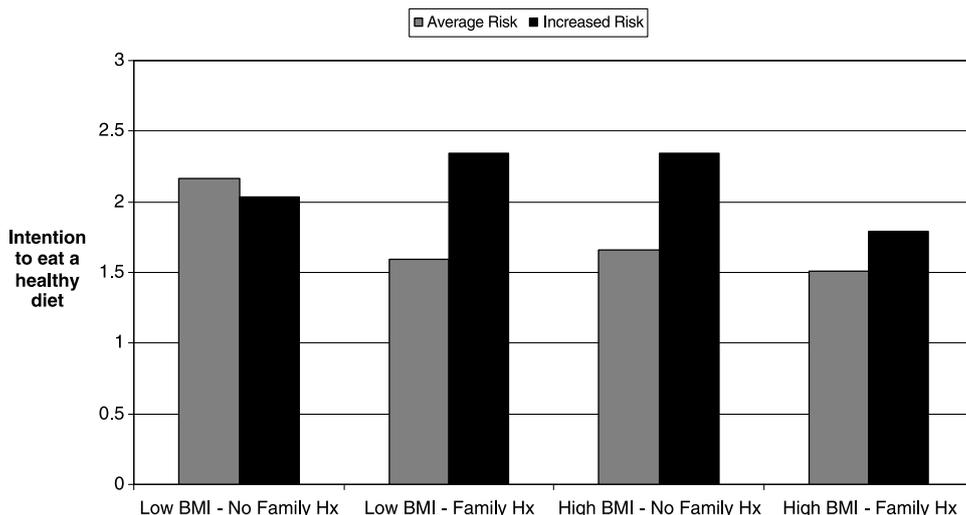
We also found moderating effects of BMI and family history of overweight/obesity on intentions to eat a healthy diet. The effects of increased risk feedback were greatest among those that had one observable risk factor (increased BMI or family history). Among those with both an increased BMI and a family history, there was no effect of risk feedback and dietary intentions were comparatively low. These findings may reflect a sense of fatalism among these individuals. There was also no effect of risk feedback among individuals who had no observable risk factors for obesity. However, intentions to eat a healthy diet were considerably higher. This may reflect that these individuals are already more health conscious and doing everything they can with regard to dietary behavior.

There are several limitations to this study. First, the data we present are in response to hypothetical testing for obesity risk rather than in response to actual test results (because such testing is not yet available). Our vignettes suggested a doubling of risk for obesity among those assigned to the increased risk vignettes. This figure is in the range of possible

effects of individual genetic polymorphisms and/or multigenic testing (15-17). However, if genetic findings are validated and such testing is offered in the future, actual risk levels may be lower or higher. Therefore, future studies should test the effects of different magnitudes of risk, as well as comprehension of genetic risk information. Because we did not include any measures to assess whether participants in the hormone test condition perceived this to be measuring something different than what a genetic test assesses, we cannot be sure that participants made the distinction we intended. Further, our undergraduate sample reflected a broad cross-section of academic majors, but it is unclear if some participants (e.g., biology majors) perceived the test conditions differently than other participants. However, these limitations do not alter our findings regarding participants' assigned risk level. Future qualitative research could explore how participants understand a hormone test, and to what degree this differs from the implications of a genetic test.

Previous research on genetic testing for cancer susceptibility indicated that individuals' intentions to receive a hypothetical genetic test overestimated actual testing behavior, suggesting that vignette studies provide an imperfect measure of actual behavioral responses to genetic testing (4). Whether or not individuals will respond in the same way to real genetic obesity risk testing remains to be seen. However, this concern about external validity is balanced by the high level of internal validity afforded by an experimental design. Moreover, other data support the utility of using behavioral intentions for eating a healthy diet as an outcome measure (8). Due to concerns about sensitizing participants to our outcome measures, we did not use a pre-post-test design. Therefore, we are unable to address to what degree questionnaire responses may have changed as a result of the experimental manipulation. Our undergraduate student sample also limits the generalizability of our findings, as educational levels of these participants may be higher than in the general population and levels of understanding of genetic concepts may differ in a community sample; however, young adults could arguably be considered a primary target of potential future screening campaigns for obesity risk. Finally, we did not examine how our experimental variables affected participants' physical activity intentions. Because obesity often results from a combination of dietary behavior and low levels of physical activity, future studies should examine whether risk testing also affects physical activity intentions and behavior.

There is tremendous enthusiasm for the potential of genomic profiling to personalize health risks and help people practice more effective disease prevention (18, 19). Our



**Figure 3.** Interaction of risk level, BMI, and family history on intention to eat a healthy diet.

findings provide some support for this enthusiasm but include important caveats. Consistent with previous studies in other clinical areas, we found that some individuals, particularly those who have low perceived control over their behavior, may be susceptible to a false reassurance effect after receiving test results indicating a lack of increased risk (20-22). Results from our analyses also suggest that risk testing may have little, if any, effect for individuals who have both higher BMI and a family history of overweight/obesity. Although preliminary, these findings underscore the importance of providing risk feedback in conjunction with motivational and behavioral interventions to provide the necessary skills for behavioral change (23). Several companies market genomic profiling directly to consumers sometimes overstating what test results mean and emphasizing the apparent deterministic nature of the tests (19, 24). Regulatory intervention may be necessary to ensure that consumers who are interested in genomic profiling receive accurate information and counseling that can limit potential unintended negative effects of testing (24).

### Appendix 1. Experimental Vignettes

*Genetic Test/Increased Risk.*<sup>4</sup> You may have heard media coverage about a growing problem with obesity in our society. You learn from these reports that being overweight or obese increases the risk of serious diseases, including diabetes, high blood pressure, heart disease, and cancer. Being overweight or obese is defined by the ratio of your body weight and how tall you are. This ratio is called the body mass index (BMI). A BMI of 25 or greater indicates overweight and 30 or greater indicates obesity. For example, for a person who is 5 ft 8 in. tall, a body weight of 165 lb corresponds to a BMI of 25.1. A body weight of 198 lb for the same person corresponds to a BMI of 30.1. The BMI is valid for both men and women.

Imagine that there is a new test that will provide information about a person's risk of becoming overweight or obese. This test involves giving a blood sample that is tested for different genes that influence how your body processes what you eat. Based on this genetic information, your risk of becoming obese can be determined.

Imagine that you decide to take this test. A nurse takes a sample of blood from you for this purpose. Three days later you receive the test results.

The results of your genetic test are positive. Your genes make it twice more likely that you will gain weight and become overweight or obese compared with a person with a negative result.

*Hormone Test/Average Risk.*<sup>4</sup> You may have heard media coverage about a growing problem with obesity in our society. You learn from these reports that being overweight or obese increases the risk of serious diseases, including diabetes, high blood pressure, heart disease, and cancer. Being overweight or obese is defined by the ratio of your body weight and how tall you are. This ratio is called the body mass index (BMI). A BMI of 25 or greater indicates overweight and 30 or greater indicates obesity. For example, for a person who is 5 ft 8 in. tall, with a body weight of 165 lb, corresponds to a BMI of 25.1. A body weight of 198 lb for the same person corresponds to a BMI of 30.1. The BMI is valid for both men and women.

Imagine that there is a new test that will provide information about a person's risk of becoming overweight or

obese. This test involves giving a blood sample that is measured for levels of certain hormones that influence how your body processes what you eat. Based on this information, your risk of becoming obese can be determined.

Imagine that you decide to take this test. A nurse takes a sample of blood from you for this purpose. Three days later you receive the test results.

The results of your blood test are negative. Your hormone levels show that you have an average risk of becoming overweight or obese. This means that your chances of becoming overweight or obese are no greater than the average person in the population.

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<sup>4</sup> Identical risk levels ("twice more likely" or "no greater than the average") were used in the corresponding genetic and hormone test vignettes.