

THE OBESITY GENE AND THE (MISPLACED) SEARCH FOR A PERSONALIZED APPROACH TO OUR WEIGHT GAIN PROBLEMS

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I. INTRODUCTION

A recent study published in *The Lancet* concluded that 2.1 billion people are now either overweight or obese.¹ The authors of the study also concluded that being overweight or obese was the cause of 3.4 million deaths per year worldwide, and that despite the billions invested in public health campaigns and preventative programs, no national success stories have been reported in the past thirty-three years.² In other words, the world population continues to put on weight and all attempts to stop this unhealthy trend have been unsuccessful.³

Given this reality, it is no surprise that there has been a quest for a range of high-tech and research-informed answers.⁴ Indeed, as the public health and health system ramifications of the

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1. Marie Ng et al., *Global, Regional, and National Prevalence of Overweight and Obesity in Children and Adults During 1980-2013: A Systematic Analysis for the Global Burden of Disease Study 2013*, 384 LANCET 766, 770 (2014).

2. See *id.* at 1; see also Donald G. McNeil, Jr., *No Nation Has Lowered Obesity Rate in 33 Years*, N.Y. TIMES (June 2, 2014), http://www.nytimes.com/2014/06/03/health/no-nation-has-lowered-obesity-rate-in-33-years.html?_r=0.

3. Ng et al., *supra* note 1, at 766. Since 2006, the increase in adult obesity in developed countries has slowed. *Id.* In the aggregate, however, the increase continues. *Id.*

4. For example, in 2011 the National Institutes of Health published its report, *Strategic Plan for NIH Obesity Research: A Report of the NIH Obesity Research Task Force*, with the explicit goal of “accelerating research progress.” NAT’L INSTS. OF HEALTH, No. 11-549 3, STRATEGIC PLAN FOR NIH OBESITY RESEARCH v (2011).

obesity dilemma become ever more apparent,⁵ the search for the biological contributors to obesity has intensified.⁶ It seems that every week there is a media announcement about a new “obesity gene.”⁷ And, almost always, the new discovery is accompanied with a suggestion about how uncovering the molecular basis of some aspect of weight gain—be it in relation to appetite, metabolism, or hunger—will lead to a new drug or preventative strategy that will help to address the obesity problem.⁸

The interest in obesity genes has, as of late, become part of the push toward “personalized medicine.”⁹ Having gained a great deal of traction in both the research and health policy communities, the personalized medicine concept has emerged as one of the most talked about practical applications of the “genetic revolution.”¹⁰ At its core, the concept of personalized medicine is the belief that we can use genetic information to individualize and guide decisions for the prevention, diagnosis, and treatment of disease.¹¹ In the context of obesity, this means using genetic

5. See André Picard, *Obesity Costs Economy Up to \$7-Billion a Year*, GLOBE & MAIL (June 20, 2011, 5:02 PM), <http://www.theglobeandmail.com/life/health-and-fitness/health/conditions/obesity-costs-economy-up-to-7-billion-a-year/article583803>; see also PUB. HEALTH AGENCY OF CAN., *OBESITY IN CANADA*, (2009), available at <http://www.phac-aspc.gc.ca/publicat/2009/oc/index-eng.php#eco>; Franco Sassi, *ORG. FOR ECON. CO-OPERATION AND DEV., OBESITY AND THE ECONOMICS OF PREVENTION: FIT NOT FAT*, (2010), available at <http://www.oecd.org/els/health-systems/46044572.pdf>.

6. See, e.g., Andrew J. Walley et al., *The Genetic Contribution to Non-Syndromic Human Obesity*, 10 NATURE REV. GENETICS 431, 437 (2009) (noting that “[t]he last few years have seen a major push to identify obesity genes. . .”).

7. See Amir Khan, *Health Buzz: Do You Have the Obesity Gene?*, U.S. NEWS (May 28, 2014, 9:49 AM), <http://health.usnews.com/health-news/health-wellness/articles/2014/05/28/do-you-have-the-obesity-gene>; Catherine Paddock, *How Obesity Gene May Be Driving Overeating*, MED. NEWS TODAY (July 16, 2013, 2:00 AM), <http://www.medicalnewstoday.com/articles/263404.php>; Lizzie Parry, *Do YOU have the obesity gene? Scientists Discover Defective DNA Affects Impulse Eating and Food Choices*, DAILY MAIL (May 28, 2014, 6:59 PM), <http://www.dailymail.co.uk/health/article-2641597/Do-YOU-obesity-gene-Scientists-discover-defective-DNA-affects-impulse-eating-food-choices.html>.

8. Amanda Onion, *Can Geneticists Cure Obesity?*, ABC NEWS (Jan. 11, 2006), <http://abcnews.go.com/Technology/Health/story?id=1477757> (“[T]he discovery of obesity susceptibility genes will identify new molecules and pathways that will lead to effective new medications and other interventions.”).

9. See Julia S. El-Sayed Moustafa & Philippe Froguel, *From Obesity Genetics to the Future of Personalized Obesity Therapy*, 9 NATURE REV. ENDOCRINOLOGY. 402 (2013).

10. See Isaac S. Chan & Geoffrey S. Ginsburg, *Personalized Medicine: Progress and Promise*, 12 ANN. REV. GENOMICS & HUM. GENETICS 217, 218 (2011).

11. *Id.*

information to inform diet, exercise, and other weight loss strategies.¹² It is a concept that, as we shall see below, has been embraced by many in the scientific community and, most robustly, the direct-to-consumer (“DTC”) genetic testing industry.¹³

In this paper, I briefly critique both the idea of focusing on obesity genes as a way to address obesity as a public health dilemma, and the suggestion that personalizing our attempts at weight loss is an effective strategy. I draw together evidence from a broad range of disciplines to highlight the folly of moving away from basic health advice (i.e. exercise and maintenance of a balanced diet with an appropriate amount of calories) and suggest that an emphasis on genetics might, in the long run, have an adverse impact on obesity and nutrition policy.

II. THE GENETICS OF OBESITY

It has long been known that family history is a risk factor for both childhood and adult obesity.¹⁴ If your parents are obese, it is more likely that you will also be obese—even if you are not obese as a child.¹⁵ Of course, the relationship is complex. Teasing out how much of this increased risk is associated with genetics as compared to environmental factors is far from easy. However, available evidence suggests that there is a significant hereditary component. Studies conducted among twins, for instance, have consistently shown that genetic predisposition clearly plays a

12. To be fair, the concept has also been related to the use of tailored pharmaceuticals, a point I will return to in the conclusion. See Tanya Agurs-Collins et al., *Public Health Genomics: Translating Obesity Genomics Research into Population Benefits*, 16 OBESITY S85, S86 (2008), available at <http://onlinelibrary.wiley.com/doi/10.1038/oby.2008.517/pdf>.

13. Stuart Hogarth et al., *The Current Landscape for Direct-to-Consumer Genetic Testing: Legal, Ethical and Policy Issues*, 9 ANN. REV. GENOMICS HUMAN GENETICS 161, 168 (2008), available at <http://www.annualreviews.org/doi/abs/10.1146/annurev.genom.9.081307.164319>.

14. See, e.g., Caroline S. Fox et al., *Trends in the Association of Parental History of Obesity over 60 Years*, 22 OBESITY 919 (2014), available at <http://www.ncbi.nlm.nih.gov/pubmed/23836774>.

15. *Id.* at 922 (“Parental history of obesity is an important risk factor for increasing BMI among offspring of affected parents, in a dose-related manner (i.e., stronger association for those with 2 as compared to 1 affected parent).”).

role,¹⁶ though finding a highly predictive obesity gene or even a set of obesity genes has remained elusive.¹⁷ As noted in a report on the genetics of obesity published by the United Kingdom's Public Health Genomics Foundation, "[body mass index] is a highly heritable trait, with heritability estimates of 40% to 70%; however, much remains unknown about the identity and biological mechanisms of the contributing genes."¹⁸ Many of the existing genetic studies have been criticized on methodological grounds¹⁹ and many of the gene associations have not been replicated in subsequent studies.²⁰

Even the most predictive of the obesity related mutations, the fat mass and obesity associated gene ("FTO gene"), seems to be associated with only a relatively modest amount of increased body mass.²¹ While the association of the FTO gene to a range of obesity related characteristics—such as BMI (body mass index) and waist circumference—have been replicated in a variety of studies,²² the presence of the gene is not terribly predictive and

16. See, e.g., J. Naukkarinen et al., *Causes and Consequences of Obesity: The Contribution of Recent Twin Studies*, 36 INT'L J. OBESITY 1017 (2012), available at <http://www.nature.com/ijo/journal/v36/n8/abs/ijo2011192a.html>.

17. Walley et al., *supra* note 6, at 437 (noting that while there has been considerable success at locating genes that may contribute to obesity, this needs to be "tempered by the fact that the contribution of these gene variants to obesity is currently estimated to be small; for example, the strongest association for obesity, that of FTO, is estimated to account for only ~1% of the heritability of obesity").

18. Louise M. Aston & Mark Kroese, PHG FOUND., GENOMICS OF OBESITY: THE APPLICATION OF PUBLIC HEALTH GENOMICS TO THE PREVENTION AND MANAGEMENT OF OBESITY IN THE UK 18, (2013), available at <http://www.phgfoundation.org/file/13532>; see E. K. Speliotes et al., *Association Analyses of 249,796 Individuals Reveal 18 New Loci Associated with Body Mass Index*, 42 NATURE GENETICS 937, 937 (2010).

19. See, e.g., M. J. Müller et al., *Genetic Studies of Common Types of Obesity: A Critique of the Current Use of Phenotypes*, 11 OBESITY REV. 612 (2010) (noting that while studies often use BMI in obesity gene studies, this measure is both faulty and too heterogeneous to be of value in this context). For a general critique of the methods used to find obesity genes, see R. Rosmond, *Association Studies of Genetic Polymorphisms in Central Obesity: A Critical Review*, 27 INT'L J. OBESITY 1141 (2003).

20. Li-Jun Tan et al., *Replication of 6 Obesity Genes in a Meta-Analysis of Genome-Wide Association Studies from Diverse Ancestries*, 9 PLOS ONE 1 (May 30, 2014), <http://www.plosone.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pone.0096149&representation=PDF>.

21. See, e.g., Ruth J.F. Loos, *Genetic Determinants of Common Obesity and Their Value in Prediction*, 26 BEST PRAC. & RES. CLINICAL ENDOCRINOLOGY & METABOLISM 211, 214 (2012) (noting that "each FTO risk allele increas[es] BMI by on average 0.39kg").

22. See Tan et al., *supra* note 20, at 12.

“accounts for only a small fraction of the gene-related susceptibility to obesity.”²³ Indeed, many individuals with the gene are not obese.²⁴ This poor predictive power is similar for virtually all of the genes that have been associated with obesity. As recently summarized by Tan et al. (2014), all genomic markers identified along with their putative genes have only been shown to have very small effects on BMI or the risk of obesity.²⁵

In the context of social policy, testing for obesity related genes would seem to add little to our knowledge of future risk. There are well-known health risks for anyone who is obese.²⁶ Having the ability to disclose that an individual has a slight genetic predisposition for a particular adverse outcome—such as type 2 diabetes²⁷—is not, from a health prevention perspective, informative or actionable. It does not alter preventative approaches or broader health policy strategies. A recent large population study published in the journal *PLoS Medicine* nicely highlights the limited public health value of genetic testing in this context.²⁸ The research explored the cumulative predictive power of the genes associated with type 2 diabetes as compared to the risk associated with being obese. The researchers found that while genetics can provide predictive information, it is, in general, dwarfed by the risks associated with obesity.²⁹ Your genes matter, but not as much as your weight. The authors conclude with a policy recommendation that can only be read as a strong rebuke of personalized approaches to common diseases.³⁰ “The high absolute risk associated with obesity at any level of genetic risk

23. *Genes Are Not Destiny*, HARV. SCH. OF PUB. HEALTH, <http://www.hsph.harvard.edu/obesity-prevention-source/obesity-causes/genes-and-obesity> (last visited July 16, 2014); see also Speliotes et al., *supra* note 18, at 943.

24. *Genes Are Not Destiny*, *supra* note 23 (“FTO[] accounts for only a small fraction of the gene-related susceptibility to obesity.”).

25. Tan et al., *supra* note 20, at 2.

26. WORLD HEALTH ORG., *Obesity: Preventing and Managing the Global Epidemic*, 894 WHO TECH. REP. SERIES 1, 39 (2000).

27. Sarah H. Wild & Christopher D. Byrne, *Risk Factors for Diabetes and Coronary Heart Disease*, 333 *BMJ* 1009 (2006).

28. Claudia Langenberg et al., *Gene-Lifestyle Interaction and Type 2 Diabetes: The EPIC InterAct Case-Cohort Study*, 11 *PLOS MED.* 1 (May 2014), <http://www.plosmedicine.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pmed.1001647&representation=PDF>.

29. *Id.*

30. *Id.* at 11.

highlights the importance of universal rather than targeted approaches to lifestyle intervention.”³¹

In addition, aside from speculation about the possible (and relatively insignificant) effect of selective mating on the percentage of individuals with a genetic predisposition to obesity,³² it seems highly unlikely that we can *blame* the increase in obesity on our genes. Our genes have not changed; our environment has.³³ Thus, it seems misdirected to target our genes, rather than our environment, as a way to dig out of this public health crisis.

The bottom line is that there are countless factors that increase the risk of obesity. A 2010 systematic review, to cite just one example, explored the evidence surrounding early-life influences that might contribute to the chance of becoming obese.³⁴ The authors found a huge number of potentially relevant factors, including maternal diabetes, maternal smoking, rapid infant growth, no or limited breastfeeding, obesity during infancy, short sleep duration, television viewing, less than thirty minutes of daily physical activity, and the consumption of sugar-sweetened beverages.³⁵ Yes, genetics plays a role in the development of obesity, but the complexity of that contribution and the low

31. *Id.* at 1.

32. It has been argued that genetics has contributed to the obesity epidemic through the process of selective mating. Studies have shown that individuals with high BMIs marry and have children with individuals with high BMIs and that these individuals have disproportionately more children. See Krista Casazza et al., *Weighing the Evidence of Common Beliefs in Obesity Research*, CRITICAL REV. FOOD SCI. & NUTRITION (forthcoming 2014) (manuscript at 27) (“[T]he combined effects of assortative mating and differential realized fertility play a small but significant role in the recent rise in obesity prevalence in the U.S.”). Even if this were true, what would be the policy response?

33. Many obesity experts note this reality. For example, obesity expert Dr. David L. Katz, director of the Prevention Research Center at Yale University School of Medicine, has been quoted thus: “Let us by all means study our genes, and their associations with our various shapes and sizes. But, let’s not let it distract us from the fact that our genes have not changed to account for the modern advent of epidemic obesity—our environments and lifestyles have.” Steven Reinberg, *Researchers Pinpoint Array of Obesity Genes*, HEALTHDAY (Oct. 10, 2010), <http://consumer.healthday.com/health-technology-information-18/genetics-news-334/researchers-pinpoint-array-of-obesity-genes-644136.html>.

34. L. Monasta et al., *Early-Life Determinants of Overweight and Obesity: A Review of Systematic Reviews*, 11 OBESITY REV. 695, 696 (2010).

35. *Id.* at 703.

predictive value of the identified genes make this an area of questionable value for the development of public health strategies.

III. HEALTH ADVICE, BEHAVIOR CHANGE AND PERSONALIZED MEDICINE

Even if existing genetic testing technologies are not—at least at the current time—terribly predictive of obesity risk, might genetic testing for obesity related genes still reveal useful information about predispositions? Might we use genetic testing to optimize our approach to weight loss or to target and motivate individuals who might be at risk?³⁶

One of the often-stated reasons for investing in research on the genetics of obesity is to develop personalized strategies for prevention and treatment. As noted in a 2008 review article that explored the role of genetic research in these contexts: “The expectation is that population-based gene–environment interaction obesity studies can provide information on polymorphisms that may predict response to diet and physical activity interventions.”³⁷ In other words, the research will arm us with the information necessary to individualize diets and exercise routines in order to maximize weight loss and weight maintenance. In addition, it is hoped that if individuals know they are at increased risk for either obesity, or for the detrimental health outcomes associated with obesity, they will be more motivated to change their behavior (i.e., eat a healthier diet and fewer calories).³⁸ The provision of genetic information will create a “teachable movement,” as argued by Francis Collins in 1999, which will result in a “lifelong change in health-related behavior.”³⁹

36. See Moustafa & Froguel, *supra* note 9, at 402.

37. Agurs-Collins et al., *supra* note 12, at S89 (2008); see also PERSONALIZED MED. COAL., *THE CASE FOR PERSONALIZED MEDICINE* (4th ed. 2014), at 9, www.personalizedmedicinecoalition.org/userfiles/PMC-Corporate/file/pmc_the_case_for_personalized_medicine.pdf (last visited Sept. 6, 2014) [hereinafter “PMC”] (reflecting the idea that genetic screening can allow physicians to predict responses and select an optimal therapy the first time treatment is given).

38. See PMC, *supra* note 37, at 14 (stating that knowledge of a genetic predisposition provides patients with incentive to make lifestyle changes and manage their condition).

39. Francis S. Collins, *Medical and Societal Consequences of the Human Genome Project*, 341 *NEW ENG. J. MED.* 28, 35 (1999).

This disclose-individual-risks and motivate-behavior-change narrative can be found throughout media reports and scientific literature that support a research investment in personalized medicine more generally.⁴⁰ For example, a 2013 article advocating for the social benefits of genetic testing and personalized medicine argued, “it will also provide the basis for concrete action by consumers to improve their health as they observe the impact of lifestyle decisions.”⁴¹ Moreover, “consumers will be able to reduce the incidence of the complex chronic diseases that currently account for seventy-five percent of disease-care costs in the USA.”⁴² This behavior change rhetoric can also be found in statements from government and research-funding entities that are aimed at promoting personalized medicine.⁴³

The idea that we can personalize our lifestyles to avoid or combat weight gain is a core part of the marketing strategy for many DTC genetic testing companies.⁴⁴ To cite just a few examples, the website for the United Kingdom company, DNAFit, tells consumers that “[b]y analysing the relationship between genes, nutrition and lifestyle our gene tests provide a valuable tool

40. See Dominic Basulto, *A Changing Battlefield in the Fight Against Fat*, WASH. POST (June 20, 2013), <http://www.washingtonpost.com/blogs/innovations/wp/2013/06/20/a-changing-battlefield-in-the-fight-against-fat/>; Shelina Begum, *Salford Firm Launches DNA Test to Help Kids Battling Obesity*, MANCHESTER EVENING NEWS, (Jan. 8, 2014, 7:30 AM), <http://www.manchestereveningnews.co.uk/news/greater-manchester-news/salford-firm-bioclinics-dna-test-6477877>; Tamara Cohen, *DNA Diet Tailored to Your Genes Can Help You Lose 11lbs Over Four Months*, MAIL ONLINE (Jan. 12, 2012, 7:25 PM), <http://www.dailymail.co.uk/health/article-2085972/DNA-diet-tailored-genes-help-lose-11lbs-months.html>.

41. Mauricio Flores et al., *P4 Medicine: How Systems Medicine Will Transform the Healthcare Sector and Society*, 10 PERSONALIZED MED. 565, 565 (2013).

42. *Id.*

43. See, e.g., *Personalized Health*, GENOME QUÉBEC, <http://www.genomequebec.com/en/home.html> (last visited July 16, 2014) (explaining that the value of personalized medicine as an approach that “includes health care as well as our lifestyle” and “empowers patients to take more responsibility for their health and care”); see also *Clinical Utility of Personalised Medicine*, NAT’L HEALTH & MED. RES. COUNCIL 1, 7 (2011) (Austl.), http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/ps0001_clinical_utility_personalised_medicine_feb_2011.pdf (“Genetic knowledge has the potential to influence lifestyle choices and decisions about preventative measures . . .”).

44. This has been a consistent part of the marketing message from the most high profile of DTC companies, 23andMe—a point driven home during a March 2014 speech by CEO Anne Wojcicki, where she suggested that genetic testing can help people to “make better decisions and lead healthier lives.” Tanya Lewis, *Genetic Testing Controversy Takes Center Stage at SXSW*, LIVE SCI. (Mar. 12, 2014, 10:27 AM), <http://www.livescience.com/44042-genetic-testing-controversy-takes-stage-at-sxsw.html>.

for you to manage your health and wellness.”⁴⁵ The website for International Bioscience outlines the social harms caused by our “increasingly obese society” and states that “[g]enetic predisposition DNA testing for obesity is useful in order to identify whether the condition can be caused by genetic factors” in order to allow you to make “lifestyle changes such as diet and exercise.”⁴⁶ Furthermore, the website ThinnerGene suggests we should all test our DNA so we can “lose weight the easy way.”⁴⁷ As with all of these gene-based programs, the idea is to use genetic information to customize weight loss recommendations and motivate a healthy behavior change.

But, as noted, there is no evidence to support the approach promoted by these companies, particularly since the information on the DTC websites invariably ignores both the complexity of the relevant genetics and the relatively weak predictive power of the genes that are tested.⁴⁸ In general, the material available on the DTC websites provides little to explain how, exactly, their service resolves these issues or why, despite the state of the science, their services will be effective.⁴⁹ The impression that is given by these DTC companies is that the relationship between genes and obesity is clear, strong and actionable.⁵⁰ These companies leverage the

45. *Our Vision*, DNAFIT, <http://www.dnafit.com/who-we-are> (last visited Aug. 21, 2014).

46. *Genetic Predisposition DNA Testing for Obesity*, INT’L BIOSCIENCES, <http://www.ibdn.com/regions/UK/EN/?page=genetic-predisposition-obesity> (last visited Aug. 21, 2014).

47. *Thinnergene*, INDIEGOGO, <https://www.indiegogo.com/projects/get-thin-with-thinnergene> (last visited Aug. 21, 2014). This website frames their activity as being a citizen-based, crowd-funded research project that will allow you to “get advance access to a DNA test that will show you what weight loss program will make it easier for you to lose weight.” *Id.*

48. See HELEN WALLACE, GENEWATCH UK, YOUR DIET TAILORED TO YOUR GENES: PREVENTING DISEASES OR MISLEADING MARKETING? 65–66 (2006), available at <http://www.genewatch.org/uploads/f03c6d66a9b354535738483c1c3d49e4/Nutrigenomics.pdf>, for a critique of the nutritional claims.

49. I had my genes tested by the company 23andMe. 23ANDME, <https://www.23andme.com> (last visited Aug. 21, 2014). The company tested me for eight obesity related mutations. Three of these mutations indicate I am at lower risk for obesity and weight gain. Four suggest I am at average risk. One suggests I am at increased risk. So, am I to average all this genetic information? See TIMOTHY CAULFIELD, THE CURE FOR EVERYTHING (2012), for a detailed outline of this experience.

50. A good example of this oversimplification can be found on the website of Genetic Performance, a genetic testing company that sells a product called DNA Slim. *DNA Slim—The Scientific Approach to Weight Loss: Overweight? Maybe You Really Can Blame Your Genes*, GENETIC PERFORMANCE, <http://www.geneticperformance.com/dna-slim-%E2>

hype around genetics and the growing concern about obesity to market services—like nutritional supplements⁵¹—that are unlikely to provide much, if any, added health value.⁵²

More importantly, the idea that personalized advice is necessary or that it will motivate the adoption of health improving behavior seems entirely misplaced. First, for the vast majority of individuals, there is no need for a personalized approach. As noted by Veerman, “testing for genetic traits that are associated with obesity makes no difference in the advice to overweight persons: increased physical activity and a healthy diet are indicated regardless of the genes.”⁵³ Most individuals in North America are

[%80%93-the-scientific-approach-to-weight-loss](#) (last visited Aug. 21, 2014) (“Finally, science may help explain why some people put on weight easily while others eat all they want and seem never to gain an ounce.”). See also *Diet and Fitness Optimization Testing*, DNA TESTING CENTRES OF CAN., <http://dnatestingcanada.com/medica-services/diet-and-fitness-optimization-testing> (last visited Aug. 21, 2014); *XRPredict+*, XR Genomics, <http://www.xrgenomics.com/xrpredictplus> (last visited Aug. 21, 2014).

51. The brochure for the DTC company, Gonidio, tells consumers they can “identify your genetic needs to manage and prevent obesity” and suggests that “[y]our genetic profile can be used to provide you with personalized nutritional supplements and a personalized line of cosmetics which are one hundred percent organic, based on the results of your DNA test.” *Weight Control*, GONIDIO, http://www.gonidio.com/weightcontr_0LBROCHURE_english.pdf (last visited Sept. 6, 2014).

52. Even the advocates within the scientific community of personalized approaches to nutrition have noted that the science is in its early stages and is not yet ready for clinical application. See, e.g., Valentini Konstantinidou et al., *Personalized Nutrition and Cardiovascular Disease Prevention: From Framingham to PREDIMED*, 5 *ADVANCES NUTRITION* 368S, 368S, 370S (2014) (noting that the research progress has been slow and that “the current evidence level of applying genomic information to tailoring [diets] is at its early stages”). This paper provides yet another example of the flawed thinking associated with much of the push toward personalized medicine. It highlights that certain individuals with an increased risk for cardiovascular disease would benefit from following the Mediterranean diet. As such, their argument suggests that, in the future, we should test individuals to identify those who would benefit from this diet. But we *all* would benefit from an adherence to the Mediterranean diet. We don’t need personalized advice to point us in that direction. See also, Dolores Corella & Jose M. Ordovas, *Nutrigenomics in Cardiovascular Medicine*, 2 *CIRCULATION CARDIOVASCULAR GENETICS* 637, 639, 648 (2009) (noting that due to methodological problems—such as a “lack of replication of the initially reported gene-diet interactions . . . nutrigenomics cannot be rigorously applied to cardiovascular prevention and treatment at this time.”). Though Corella and Ordovas remain hopeful that the science will improve, they conclude that at present, “it is premature to recommend the use of nutrigenomics in the prevention of [cardiovascular disease] at the population level.” *Id.* at 648.

53. J. Lennert Veerman, *On the Futility of Screening for Genes That Make You Fat*, 8 *PLOS MED.* 1 (Nov. 1, 2011), <http://www.plosmedicine.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pmed.1001114&representation=PDF>.

not engaging in the basic elements of a healthy lifestyle.⁵⁴ Few individuals eat enough fruits and vegetables,⁵⁵ even less exercises enough⁵⁶ and a ridiculously small proportion of the population (perhaps as few as one in one thousand⁵⁷ embrace all of the basic steps that are considered part of a healthy lifestyle. Given these profound lifestyle deficits, it seems absurd to worry about maximizing the details of our diet based on genetic characteristics that are weakly, if at all, associated with a variety of obesity related traits.

Yes, there is emerging evidence that genetic predispositions may have an impact on how people respond to certain foods and exercise. For example, research has found that exercise can help negate the impact of the FTO gene on weight gain.⁵⁸ Other studies have suggested there are genes that create variation in how we break down carbohydrates (a process that may

54. A recent study by the Centers for Disease Control and Prevention (“CDC”) estimates that fifty percent of Americans have a chronic disease that is largely the result of a poor lifestyle. See Ursula E. Bauer et al., *Prevention of Chronic Disease in the 21st Century: Elimination of the Leading Preventable Causes of Premature Death and Disability in the USA*, 384 THE LANCET 45, 45 (2014) (“The chronic disease burden in the USA largely results from a short list of risk factors—including tobacco use, poor diet and physical inactivity (both strongly associated with obesity), excessive alcohol consumption, uncontrolled high blood pressure, and hyperlipidaemia—that can be effectively addressed for individuals and populations.”).

55. See, e.g., *Americans Don’t Consume Enough Fruits and Vegetables*, JOHN HOPKINS BLOOMBERG SCH. OF PUB. HEALTH (Mar. 19, 2007), <http://www.jhsph.edu/news/stories/2007/gary-ajpm.html>; see also *State-Specific Trends in Fruit and Vegetable Consumption Among Adults—United States, 2000–2009*, 59 CDC MORBIDITY & MORTALITY WKLY REP., no. 35 (Sept. 10, 2010), <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5935a1.htm#tab1>.

56. *Physical Activity*, HEALTHYPEOPLE.GOV, <http://www.healthypeople.gov/2020/topicobjectives2020/overview.aspx?topicid=33> (last visited July 16, 2014); see also *Physical Activity Needs for Health Payoffs Often Underestimated*, CBC NEWS (July 16, 2014), <http://www.cbc.ca/news/health/physical-activity-needs-for-health-payoffs-often-underestimated-1.2707897> (estimating that fewer than fifteen percent of the Canadian population get the minimal recommended amount of exercise).

57. A study by the American Heart Association concluded that only one in one thousand Americans were taking the steps necessary to maximize health. Stephen Adams, *Only One in 1,000 ‘Heart Healthy’*, THE TELEGRAPH (Mar. 18, 2013, 8:00 PM), <http://www.telegraph.co.uk/health/healthnews/9937832/Only-one-in-1000-heart-healthy.html>.

58. See generally Tuomas O. Kilpeläinen et al., *Physical Activity Attenuates the Influence of FTO Variants on Obesity Risk: A Meta-Analysis of 218,166 Adults and 19,268 Children*, PLOS MED. (Nov. 1, 2011), <http://www.plosmedicine.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pmed.1001116&representation=PDF>.

contribute to obesity)⁵⁹ and metabolize fried food.⁶⁰ While leveraging this kind of data in order to customize an individual's lifestyle may seem theoretically promising, there is no evidence that tailoring diet and lifestyle advice based on this kind of information will have a long-term beneficial impact on weight loss, weight maintenance or, more broadly, health and wellbeing. We should all exercise and eat a healthy, balanced diet (including the avoidance of fried foods!), regardless of our genetic predisposition.⁶¹

Moreover, given the complexity of the biological and social contributors to weight gain, it seems unlikely that, for most individuals, small lifestyle tweaks based on information regarding a relatively small set of genes could result in a behavioral response that would provide a benefit over and above the benefits derived from following general lifestyle advice. In other words, there is no reason to believe that a genetics-informed lifestyle would result in better outcomes than basic lifestyle advice. Once again, we should all exercise and eat a healthy, balanced diet.

Second, and more fundamentally, there is no evidence that the provision of genetic risk information will motivate the healthy behavior change so often predicted and desired by the advocates of personalized medicine. In fact, the available information tells us that people do *not* change their behavior based on genetic risk information.⁶² A comprehensive systematic review of all the

59. Mario Falchi et al., *Low Copy Number of the Salivary Amylase Gene Predisposes to Obesity*, 46 NATURE GENETICS 492, 493–94 (2014); see also Susan Scutti, *Obesity Linked to Genetics: People with Higher Number of 'Carb Breakdown' Genes Less Likely To Be Overweight*, MED. DAILY (Mar. 31, 2014, 2:31 PM), <http://www.medicaldaily.com/obesity-linked-genetics-people-higher-number-carb-breakdown-genes-less-likely-be-overweight-273496>.

60. Qibin Qi et al., *Fried Food Consumption, Genetic Risk, and Body Mass Index: Gene-Diet Interaction Analysis in Three US Cohort Studies*, BMJ (Mar. 19, 2014), <http://www.bmj.com/content/bmj/348/bmj.g1610.full.pdf>.

61. For example, it seems ridiculous to tell people with the FTO mutation to exercise. We should all exercise.

62. I have commented on this reality in other publications. See Timothy Caulfield, *Direct-to-Consumer Testing: If Consumers Aren't Anxious, Why Are Policymakers?*, 130 HUM. GENETICS 23 (2011); Timothy Caulfield & Amy L. McGuire, *Direct-to-Consumer Genetic Testing: Perceptions, Problems and Policy Responses*, 63 ANN. REV. MED. 23 (2012); James P. Evans et al., *Deflating the Genomic Bubble*, 331 SCI. 861 (2011). To be fair, there is some evidence that customers of DTC companies report increased motivation. Such self-reported data, however, is far from reliable and the sample bias—people who buy DTC services—has implications for the generalizability of the results. See, e.g., Corin Egglestone

available evidence, published in 2010, concluded thus: “communicating genetic test based disease risk estimates may have little or no effect on behaviour.”⁶³ Other studies have found that personalized genetic risk counseling—which is at the core of the rationale for the broader personalized medicine movement—does not increase people’s motivation to lose weight or adhere to preventative health strategies.⁶⁴ Moreover, the studies that have looked at this in the context of obesity have come to the same conclusion.⁶⁵ A 2012 analysis that examined the value of obesity-related genetic tests concluded “their accuracy to predict obesity is poor and not competitive with the predictive ability of traditional risk factors” and, more importantly, that there is little evidence to suggest “they could have a beneficial effect on behavior.”⁶⁶

This is not to say that discussing the genetics of obesity with individuals seeking to lose weight has absolutely no effect. There is some data that suggest discussing the role of heredity may reduce feelings of self-blame and allow individuals to set more realistic weight loss goals.⁶⁷ Conversely, this does not imply that genetic

et al., *Effect of Direct-to-Consumer Genetic Tests on Health Behaviour and Anxiety: A Survey of Consumers and Potential Consumers*, 22 J. GENETIC COUNSELING. 565, 565 (2013).

63. Marteau TM et al., *Effects of Communicating DNA-Based Disease Risk Estimates on Risk-Reducing Behaviours*, 10 COCHRANE DATABASE SYS. REV. CD007275 3 (2010) (U.K.); see also Cinnamon S. Bloss, Nicholas J. Schork & Eric J. Topol, *Effect of Direct-to-Consumer Genome Wide Profiling to Assess Disease Risk*, 364 NEW ENG. J. MED. 524 (2011).

64. Richard W. Grant et al., *Personalized Genetic Risk Counseling to Motivate Diabetes Prevention: A Randomized Trial*, 36 DIABETES CARE 13, 13 (2013) (“Diabetes genetic risk counseling with currently available variants does not significantly alter self-reported motivation or prevention program adherence for overweight individuals at risk for diabetes.”); see also Christopher A. Harle et al., *Effectiveness of Personalized and Interactive Health Risk Calculators: A Randomized Trial*, 32 MED. DECISION MAKING 594, 603 (2012) (finding that personalized risk estimates did not improve risk perceptions or motivate healthy behaviors).

65. There is, in fact, a large literature on how difficult it is to change behavior with information alone. See, e.g., Jeanne P. Goldberg & Sarah A. Sliwa, *Communicating Actionable Nutrition Messages: Challenges and Opportunities*, 70 PROC. NUTRITION SOC’Y 26, 26 (2011) (“Communication alone has not been, and will not be, sufficient for consumers to adopt the behavioural changes endorsed by experts. Broad environmental interventions coupled with individual skills development will need to be part of the process.”).

66. Loos, *supra* note 21, at 211.

67. Matthias Conradt et al., *A Consultation with Genetic Information About Obesity Decreases Self-Blame about Eating and Leads to Realistic Weight Loss Goals in Obese Individuals*, 66 J. PSYCHOSOMATIC RES. 287, 287 (2009); S.F. Meisel & J. Wardle, ‘*Battling My Biology*’: *Psychological Effects of Genetic Testing for Risk of Weight Gain*, 23 J. GENETIC COUNSELING. 179 (2014). It should be noted, however, that given the evidence on how people respond to genetic risk information, I suspect that even this effect would dissipate relatively quickly.

testing is warranted—a healthcare provider could simply discuss the role of genetics—or that the provision of genetic information will improve long-term weight loss outcomes.

Of course, the lack of behavior change should be no surprise. Few people change their behavior based on the risk information they receive from the number on a weigh scale,⁶⁸ which is far more predictive of future health than almost any bit of genetic risk information.

Even if one does believe the idea that genetic testing can motivate significant behavior change (and, it can not be emphasized enough, there is little evidence to support this ubiquitous idea), we need to accept the possibility that it may also result in unhealthy behavior change. For every individual that is at increased risk for obesity, there will be someone who is at decreased risk.⁶⁹ Will the latter individuals be motivated to eat more and exercise less? And perhaps those with an increased risk for obesity will behave fatalistically, believing that since obesity is in their genes, there is nothing they can do, so they might as well live it up.

In fact, once again, there is little evidence to support the idea that individuals respond this way to genetic predisposition information.⁷⁰ Nevertheless, it is worth considering this scenario as a counterbalance to the pitch that genetics can motivate healthy behavior change—a central plank in the push for personalized medicine. If genetics can motivate healthy behaviors, it must also be able to motivate unhealthy change. You cannot have one

68. A significant portion of patients do not change their behavior after heart attacks and strokes. See Will Campbell, *Many Cardiac Survivors Don't Change Bad Habits, Poll Finds*, GLOBE & MAIL (Feb. 3, 2014, 12:01 AM), <http://www.theglobeandmail.com/news/national/many-cardiac-survivors-dont-change-bad-habits-poll-finds/article16658811>.

69. In fact, as highlighted by the test results I received from 23andMe, *supra* note 49, most individuals are likely to have a complex mix of predispositions which will make a definitive genetic risk estimate far from clear. For the sake of argument, however, let us assume that a definitive genetically informed obesity risk could be provided.

70. Indeed, there is no evidence that people respond fatalistically or become discouraged. See, e.g., Jean Harvey-Berino et al., *Does Genetic Testing for Obesity Influence Confidence in the Ability to Lose Weight? A Pilot Investigation*, 101 J. AM. DIETETIC ASS'N 1351, 1353 (2001) (“In this study, we found that a positive obesity gene status does not adversely affect people’s confidence in their ability to lose weight, or control their eating behaviour in difficult situations.”); see also Liesbeth Claassen et al., *Fatalistic Responses to Different Types of Genetic Risk Information: Exploring the Role of Self-Malleability*, 25 PSYCHOL. & HEALTH 183 (2010) (finding that people do not respond fatalistically to genetic information).

without the other. If genetic testing does alter behavior, the aggregate level result would be, at best, a wash—with some people behaving healthier and some less healthy. This is far from the revolutionizing result the advocates of personalized medicine are hoping for.

IV. POLICY PROBLEMS?

A focus on the genetic causes of obesity may also have a range of adverse effects on the development of obesity related policies.⁷¹ It may, for example, cause a shift in attention away from broad social change—which, as suggested in the recent *Lancet* study, is urgently needed⁷²—toward policies that focus on biomedical responses and individual action.⁷³ At the current time, the public is somewhat supportive of government interventions to address the obesity issue.⁷⁴ However, this support appears to be

71. I recognize that the evidence around the success of government obesity policy is, at best, mixed. See, e.g., Nola Ries, *Legal and Policy Measures to Promote Healthy Behaviour: Using Incentives and Disincentives to Control Obesity*, 6 MCGILL J. L. & HEALTH 3 (2012). An analysis of the (lack of) effectiveness of various obesity policies—such as menu labeling, food bans, etc.—is beyond the scope of this paper. Still, most in the public health community recognize the need for a multipronged approach that embraces a range of policy strategies. See, e.g., GOV'T OFFICE FOR SCI., TACKLING OBESITIES: FUTURE CHOICES—SUMMARY OF KEY MESSAGES, GOV.UK (2d ed. 2007) at 3, available at http://www3.open.ac.uk/events/8/20071114_33718_o1.pdf; see also NATIONAL RESEARCH COUNCIL, PREVENTING CHILDHOOD OBESITY: HEALTH IN THE BALANCE 5–6 (2005); see also PUBLIC HEALTH AGENCY OF CANADA, *supra* note 5, at 30.

72. Bauer et al., *supra* note 54.

73. See Walter S. Carlos Poston II & John P. Foreyt, *Obesity is an Environmental Issue*, 146 ATHEROSCLEROSIS 201, 207 (1999) (“We believe that acknowledging the significant role of the environment in the etiology of obesity will help us to stop focusing on the individual, which is encouraged by genetic and biological explanations, and to begin focusing on changing the toxic environment. Until we do this, we will not make substantial progress in addressing the public health epidemic of obesity.”). This is not to say, of course, that individual responsibility is irrelevant. But how we engage individual responsibility—via legislative and social policy that enhance healthy choices—may be altered by how we conceptualize “blame.” See, e.g., Kelly D. Brownell et al., *Personal Responsibility and Obesity: A Constructive Approach to a Controversial Issue*, 29 HEALTH AFF. 379, 379 (2010).

74. See generally Stephanie Morain & Michelle Mello, *Survey Finds Public Support For Legal Interventions Directed At Health Behavior To Fight Noncommunicable Disease*, 32 HEALTH AFF. 486 (2013). But see *Public Agrees on Obesity's Impact, Not Government's Role*, PEW RESEARCH CENTER (Nov. 12, 2013), <http://www.people-press.org/2013/11/12/public-agrees-on-obesitys-impact-not-governments-role> (stating that while the majority see obesity as a major health issue, only forty-two percent believe the government should play a significant role in fighting the problem).

relatively fragile. Survey research has found that, despite the truism that the increase in the rates of obesity is the result of the interaction of a complex array of social and biological factors,⁷⁵ most people blame individuals.⁷⁶ It is the fault of individuals—or so most people think, and not socioeconomic factors—aggressive marketing of junk food to children, increases in portion sizes, etc., that have caused increases in obesity. Given this fact, an emphasis on genetics might make the introduction of social programs even more challenging. A 2009 study from Yale University, for example, found that framing obesity as linked to inherited traits makes government action seem impractical and, as a result, may have the “unintended consequence of stifling public policy action.”⁷⁷

To be clear, I am not suggesting that pharmaceutical or biomedical responses, such as bariatric surgery, have no place. On the contrary, research shows that for many individuals, particularly the clinically obese, these may be the most effective approaches, from the perspective of both outcomes⁷⁸ and patient satisfaction.⁷⁹ However, there appears to be a consensus that reversing the obesity trend will require the use of a range of policy tools, including government action aimed at the modification of environmental contributors.⁸⁰

75. See GOVERNMENT OFFICE FOR SCIENCE, *supra* note 71, which identified hundreds of different variables relevant to the social problem of obesity.

76. See Jayson L. Lusk & Brenna Ellison, *Who is To Blame for the Rise in Obesity?*, 68 *APPETITE* 14, 14 (2013) (“Eighty percent said individuals were primarily to blame for the rise in obesity. Parents were the next-most blameworthy group, with fifty-nine percent ascribing primary blame.”).

77. See Colleen L. Barry et al., *Obesity Metaphors: How Beliefs about the Causes of Obesity Affect Support for Public Policy*, 87 *MILBANK Q.* 7, 41 (2009); see also Regina G. Lawrence, *Framing Obesity: The Evolution of News Discourse on a Public Health Issue*, 9 *INT’L. J. PRESS/POL.* 56, 57 (2004) (“Defining a problem in individualized terms limits governmental responsibility for addressing it, while systematic frames invite governmental action.”).

78. See Lars Sjöström et al., *Bariatric Surgery and Long-term Cardiovascular Events*, 307 *JAMA* 56, 56 (2012); see also Paul O’Brien et al., *Long-Term Outcomes After Bariatric Surgery: Fifteen-Year Follow-Up of Adjustable Gastric Banding and a Systematic Review of the Bariatric Surgical Literature*, 257 *ANNALS SURGERY* 87, 87 (2013).

79. Endocrine Society, *Among Weight Loss Methods, Surgery and Drugs Achieve Highest Patient Satisfaction*, *SCIENCEDAILY* (June 23, 2014), <http://www.sciencedaily.com/releases/2014/06/140623141903.htm>.

80. See, e.g., L.K. Khan et al., *Recommended Community Strategies and Measurements to Prevent Obesity in the United States: Implementation and Measurement Guide*, CDC (July 2009), http://www.cdc.gov/obesity/downloads/community_strategies_guide.pdf; see also

Another public health issue associated with an emphasis on genetic predispositions and personalized approaches to diets is that they have the potential to further confuse an already confused public about the nature and significance of a healthy diet. Survey research has consistently found that the general public is profoundly uncertain about how they should eat or even the relationship between diet and disease.⁸¹ For example, a recent survey found that nearly half of the respondents did not know there was a relationship between diet and cancer.⁸² Pushing the suggestion that everyone should eat differently based on their genes or that certain people might be more or less susceptible to diet related health issues, including obesity, seems likely to only confuse the message even further.⁸³ It will add another layer of unnecessary complexity—thus obscuring the simple, evidence-based reality that for the vast majority of individuals, the parameters of a healthy diet are remarkably straightforward.⁸⁴

PUBLIC HEALTH AGENCY OF CANADA, *supra* note 5, at 30; *see also* WORLD HEALTH ORGANIZATION, *supra* note 26, at 167.

81. *See* INT'L FOOD INFO. COUNCIL FOUND., *2012 Food & Health Survey: Consumer Attitudes Toward Food Safety, Nutrition & Health* (Exec. Summary), FOODINSIGHT.ORG (May, 2012), <http://www.foodinsight.org/Content/3840/FINAL%202012%20Food%20and%20Health%20Exec%20Summary.pdf> (finding, *inter alia*, that the majority of Americans find doing their own taxes simpler than figuring out how to improve their diet. In addition, only thirty percent “correctly believe that all sources of calories play an equal role in weight gain.”).

82. *See* ‘Alarming’ Level of Naivete About Cancer’s Link to Poor Diet, Report Warns, CTIV NEWS (Feb. 4, 2014), <http://www.ctvnews.ca/world/alarming-level-of-naivete-about-cancer-s-link-to-poor-diet-report-warns-1.1670284>; *World Cancer Day: Half Don’t Know About Link Between Diet and Cancer*, MED. NEWS TODAY (Feb. 4, 2014), <http://www.medicalnewstoday.com/releases/272095.php>.

83. *See, e.g.*, Rebekah H. Nagler, *Adverse Outcomes Associated With Media Exposure to Contradictory Nutrition Messages*, 19 J. HEALTH COMM.: INT’L PERSP. 24, 35 (2014) (“We found evidence that confusion and backlash beliefs, in turn, may lead people to doubt nutrition and health recommendations more generally—including those that are not surrounded by conflict and controversy (e.g., fruit/vegetable consumption, exercise.)”); *see also* Ruth Patterson et al., *Is There a Consumer Backlash Against the Diet and Health Message?*, 101 J. AM. DIETETIC ASS’N 37, 40 (2001).

84. As nicely summarized by the well-known nutritionist, Marion Nestle, in a recent article critiquing the gluten-free craze, “[t]here really isn’t much better dietary advice than eating your veggies, exercising and limiting calories People just seem to like making eating difficult for themselves.” Kim Severson, *Gluten-Free Eating Appears to be Here to Stay*, N.Y. TIMES, June 16, 2014, http://www.nytimes.com/2014/06/18/dining/gluten-free-eating-appears-to-be-here-to-stay.html?_r=0; TIMOTHY A. CAULFIELD, *THE CURE FOR EVERYTHING: UNTANGLING TWISTED MESSAGES ABOUT HEALTH, FITNESS, AND HAPPINESS* (1st ed. 2012).

Indeed, there has been a move within the public health community to simplify and unify the messaging around obesity, diet, and exercise in order to make it more consistent, actionable, and easier to communicate.⁸⁵ There are many good reasons for an emphasis on simplicity and clarity, including increasing the likelihood of long-term adherence. Complicated diets are, for example, much harder to maintain. The more special requirements and tweaks that are required—which, of course, is exactly what a personalized approach promises to provide—the sooner an individual will give up on the diet.⁸⁶ The messaging associated with a genetically informed personalized approach—the idea that we all must emphasize different food groups, worry about different kinds of physical activity, etc.⁸⁷—pushes us in the wrong direction.⁸⁸

Finally, there are policy concerns associated with stigmatization and weight bias that are worth considering.⁸⁹ Some have speculated that articulating a genetic cause for obesity may attenuate obesity bias by providing a biological cause beyond the control of the individual,⁹⁰ though the evidence to support this

85. See Judith L. Buttriss, *Complex Science into Life-Course Health Promoting Strategies*, 70 PROC. NUTRITION SOC'Y 38 (2011); David R. Jacobs, Jr. & Michael J. Orlich, *Commentary, Diet Pattern and Longevity: Do Simple Rules Suffice? A Commentary*, 100 AM. J. CLINICAL NUTRITION 313S, 315S (2014); Phillip Sparling et al., *Commentary, Energy Balance: The Key to a Unified Message on Diet and Physical Activity*, 33 J. CARDIOPULMONARY REHABILITATION PREVENTION 12 (2013).

86. See Jutta Mata et al., *When Weight Management Lasts. Lower Perceived Rule Complexity Increases Adherence*, 54 APPETITE 37, 41 (2010).

87. For example, the website for CrossFit has a commentary used to market a genetic test. See Jen Doehring, *Test-Don't Guess: Genetic Testing Gains Knowledge on Metabolism Insight* CROSSFIT COLLINSVILLE (June 13, 2014), <http://crossfitcollinsville.com/2014/06/test-dont-guess-genetic-testing-gains-knowledge-on-metabolism-insight>.

88. See Jeanne H. Freeland-Graves & Susan Nitzke, *Position of the Academy of Nutrition and Dietetics: Total Diet Approach to Healthy Eating*, 113 J. ACAD. NUTRITION & DIETETICS 307, 307 (2013) (“Focusing on variety, moderation, and proportionality in the context of a healthy lifestyle, rather than targeting specific nutrients or foods, can help reduce consumer confusion and prevent unnecessary reliance on supplements.”).

89. Friedman and Puhl made an excellent observation that, “[i]n a country where two out of three adults and one out of three children are overweight or obese, weight bias affects millions, at a steadily increasing rate.” ROBERTA R. FRIEDMAN & REBECCA M. PUHL, YALE RUDD CENTER FOR FOOD POL'Y & OBESITY, WEIGHT BIAS: SOCIAL JUSTICE ISSUE, 2 (2012), available at http://www.yaleruddcenter.org/resources/upload/docs/what/report_s/Rudd_Policy_Brief_Weight_Bias.pdf.

90. See, e.g., Meisel & Wardle, *supra* note 67; see also Mary E. Segal, Pamela Sankar & Danielle R. Reed, *Research Issues in Genetic Testing of Adolescents for Obesity*, 62 NUTRITION

idea remains equivocal.⁹¹ While a theoretically reasonable idea, it must not be forgotten that a genetic explanation of obesity still emphasizes the individual, which has the potential impact of removing responsibility from other social actors, such as government and the food industry. Indeed, even if obesity is viewed largely as a genetic phenomenon—and therefore beyond the control of the individual—this will not necessarily remove the perceived responsibility or blame from the individual. Indeed, one could argue that a personalized approach *increases* the burden on the individual patient to act on the provided genetic risk information and, as such, may stigmatize those individuals who have unsuccessfully responded to their genetic risk information.⁹² The personalized approach demands personal action.

V. CONCLUSION

There are many public health issues associated with the use of genetics and personalized approaches to combat the social problem of obesity. This paper has sought to highlight several of the most fundamental issues, including the reality that the predictive power of genetics remains relatively weak, that there is no evidence that personalized advice is clinically beneficial or useful for motivating healthy behavior change, and that there are

REV. 307, 312 (2004) (speculating that “information that shifts the responsibility from the obese and describes them as not responsible for their condition may mitigate this stigma [of obesity]”).

91. See, e.g., B.A. Teachman et al., *Demonstrations of Implicit Anti-Fat Bias: The Impact of Providing Causal Information and Evoking Empathy*, 22 HEALTH PSYCHOL. 68, 76 (2003) (finding that “giving participants information about genetic contributions [to obesity] did not lead to lower bias.”). In another study it was found that obese individuals feel more stigmatized by simplistic messages about causation. See Lewis et al., *I Don't Eat a Hamburger and Large Chips Every Day! A Qualitative Study of the Impact of Public Health Messages About Obesity on Obese Adults*, 10 BMC PUB. HEALTH 309, 309 (2010) (noting that messaging should recognize “the complexity of obesity and focus on encouraging healthy behaviours for individuals of all sizes”).

92. See, e.g., Eric Juengst et al., *Personalized Genomic Medicine and the Rhetoric of Empowerment*, 42 HASTINGS CENTER REP. 34, 39 (2012) (“[E]mphasizing patient empowerment [through personalized medicine] might unfairly inflate patient’s responsibilities for their health, either by tying their empowered role to obligations to make decisions that conform to social expectations and interests, by enlisting patients in quasi-contractual ways to help secure the success of the personalized medicine paradigm through their decision-making, or by abandoning other environmental, structural, and regulatory approaches to health promotion and risk reduction in order to force the ‘choices’ that individuals must make for themselves.”).

reasons to believe it may have an adverse impact on the development of both nutrition and obesity-related policies.⁹³

Moreover, there is no evidence that, to date, genetic predisposition information provides useful information for the purposes of targeting population level prevention strategies.⁹⁴ When over sixty percent of the population is either overweight or obese—which is the case in many developed countries⁹⁵—it seems near absurd to parse the biological predisposition. We are all predisposed.

To be clear, I am not arguing against the need for research in this area. Research on the genetics of obesity seems likely to yield useful insights into the complex biological process that contribute to weight gain, to the identification of relevant rare genetic conditions that lead to obesity, and to the development of new pharmaceuticals that may help individuals control or lose weight.⁹⁶ It may even help to illuminate which patients will respond best to bariatric surgery.⁹⁷ However, for the reasons outlined in this paper, it seems a grave mistake to place too much emphasis on genetic testing and personalized approaches as the

93. I am, of course, not alone in this rather damning view of a genomics-informed approach to obesity prevention. *See, e.g.*, Aston & Kroese, *supra* note 18, at 44 (“[I]n common or polygenic obesity, there is negligible utility of knowledge of the genotype. This knowledge will not change the approach to management and it is unclear whether awareness of increased risk will increase motivation for behaviour change, and may even decrease this.”).

94. Interestingly, some entities, such as the CDC, note the limited value of genetic testing at the current time, but remain hopeful for the future. For example, on its public education website, the CDC explicitly notes that “genetic tests are not useful for guiding personal diet or physical activity plans” and “[r]esearch on genetic variation that affects response to changes in diet and physical activity is still at an early stage.” *See Genomics and Health*, CENTERS FOR DISEASE CONTROL & PREVENTION, <http://www.cdc.gov/genomics/resources/diseases/obesity> (last visited Sept. 2, 2014). But, despite evidence to the contrary, the CDC also suggests “explaining obesity in terms of genes and environment factors could help encourage people who are trying to reach and maintain a healthy weight.” *Id.*

95. *E.g.*, *Obesity and Overweight*, CENTERS FOR DISEASE CONTROL & PREVENTION, <http://www.cdc.gov/nchs/fastats/obesity-overweight.htm> (last visited Sept. 7, 2014); *see Obesity and the Economics of Prevention: Fit Not Fat*, ORG. FOR ECON. CO-OPERATION & DEV., <http://www.oecd.org/els/health-systems/46044572.pdf> (last visited Sept. 7, 2014).

96. For a useful review of the potential benefits of genetic research in this context, *see* Aston & Kroese, *supra* note 18, at 18. *See also* I.S. Farooqi & S. O’Rahilly, *Genetic Factors in Human Obesity*, 8 *OBESITY REV.* 37, 37 (2007).

97. *See* Andrew A. Butler & Robert W. O’Rourke, *Bariatric Surgery in the Era of Personalized Medicine*, 144 *GASTROENTEROLOGY* 497, 499 (2013).

primary strategy in our fight against the rise in obesity rates. Indeed, despite the claims made by the DTC genetic testing industry, a genetics-informed approach seems unlikely to have much use even at the level of the individual. There is, to date, no good evidence that it facilitates long-term and sustained weight loss.

One can only speculate as to why this genetics-informed approach has gained so much traction, but it seems likely to be associated with the framing of personalized medicine—by scientists and research institutions, among others—as a health care revolution.⁹⁸ This rhetoric, which is necessary to attract research funding and support from both the private and public sectors,⁹⁹ has created a great deal of hype about the potential value of genetic testing and personalized approaches. Direct-to-consumer companies have leveraged this hype in order to advertise personalized (and largely unproven) weight loss and prevention services.¹⁰⁰ This adds an additional layer of market-driven public representations about the promise of genetics and personalized medicine.

However, both individuals and public health policymakers should largely shut out the noise about personalized approaches to obesity. Evidence tells us that the public remains confused about nutrition, calories, and how best to address obesity. It seems the wrong time to be pushing a personalized, genetically informed approach. We need to stick to the evidence-based basics.

98. I have addressed the issues associated with this trend elsewhere. Timothy Caulfield, *The Paradoxes of Pop Science*, POLY OPTIONS (Sept. 2013), <http://policyoptions.irpp.org/issues/the-age-of-man/caulfield>; Timothy Caulfield, *Commercialization Creep*, POLY OPTIONS (Dec. 2012), <http://policyoptions.irpp.org/issues/talking-science/caulfield>; Timothy Caulfield, *Why Science Has To Promise Profits*, GLOBE & MAIL (Apr. 23, 2012, 2:00 AM), <http://www.theglobeandmail.com/globe-debate/why-science-has-to-promise-profits/article4210388>.

99. See James P. Evans et al., *Deflating the Genomic Bubble*, 331 SCIENCE 861, 861–862 (2011).

100. This phenomenon of leveraging an exciting area of science to sell products is called “scienceploitation.” See Timothy Caulfield, *Blinded by Science*, THE WALRUS, Sept. 2011, available at <http://thewalrus.ca/blinded-by-science>.