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Opinion

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Precision nutrition: hype or hope for public health interventions to reduce obesity?

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Abstract

High-income countries are experiencing an obesity epidemic that follows a socioeconomic gradient, affecting groups of lower socioeconomic status disproportionately. Recent clinical findings have suggested new perspectives for the prevention and treatment of obesity, using personalized dietary approaches. Precision nutrition (PN), also called personalized nutrition, has been developed to deliver more preventive and practical dietary advice than 'one-size-fits-all' guidelines. With interventions becoming increasingly plausible at a large scale thanks to artificial intelligence and smartphone applications, some have begun to view PN as a novel way to deliver the right dietary intervention to the right population. We argue that large-scale PN, if taken alone, might be of limited interest from a public health perspective. Building on Geoffrey Rose's theory regarding the differences in individual and population causes of disease, we show that large-scale PN can only address some individual causes of obesity (causes of cases). This individual-centred approach is likely to have a small impact on the distribution of obesity at a population level because it ignores the population causes of obesity (causes of incidence). The latter are embedded in the populations' social, cultural, economic and political contexts that make environments obesogenic. Additionally, the most socially privileged groups in the population are the most likely to respond to large-scale PN interventions. This could have the undesirable effect of widening social inequalities in obesity. We caution public health actors that interventions based only on large-scale PN are unlikely, despite current expectations, to improve dietary intake or reduce obesity at a population level.

Key words: Precision nutrition, personalized nutrition, obesity, population interventions, social inequalities in health, obesogenic environments

Key Messages

- Some public health actors have begun to view large-scale precision nutrition as a novel opportunity to provide the right dietary intervention to the right population at the right time.
- Large-scale precision nutrition is an individual-centred approach focusing on behavioural modification in large numbers, and not a true population approach as defined by Geoffrey Rose.
- Large-scale precision nutrition is likely to have a limited impact on obesity at a population level as it neglects population causes of obesity that are rooted in obesogenic environments.
- Early adoption and achievement of improved dietary habits based on precision nutrition are more likely among more socially privileged members of the population, which would exacerbate socioeconomic inequalities in diet and obesity.
- If taken alone, interventions based on large-scale precision nutrition are unlikely to improve dietary intake or reduce obesity at a population level.

Introduction

Most high-income countries are experiencing an obesity epidemic, since 1975.[1] For example, in the USA, more than one in three adults and one in six children were estimated to be obese in 2015.[2] Obesity has been linked to numerous non-communicable diseases such as diabetes, cardiovascular disease, osteoarthritis and certain forms of cancer.[3–5] According to the 2016 Global Burden of Disease,[6] an unbalanced diet, obesity, and high fasting plasma glucose were among the top six leading risk factors for disability-adjusted life-years in high-income countries. In these countries, the incidence and prevalence of obesity follows a socioeconomic gradient, whereby individuals with lower education, occupation and income are disproportionally affected.[7–9] In Spain, Italy and France for instance, the least educated women are over four times as likely to be obese as the most educated ones.[10]

Diet is a major modifiable determinant of obesity. Multiple public health interventions to improve population dietary intake have been implemented to date. Some individual-centred interventions have aimed at providing information about healthy eating. They used, for example, mass campaigns to disseminate dietary guidelines (e.g. '5 a day') and food guides (e.g. MyPlate in the USA).[11–13] More recent interventions have focused on shaping the food environment through structural measures. Classical examples are compulsory nutritional standards for school meals[14,15] or taxes on sugar-sweetened beverages.[16–18] So far outcomes have been disappointing. People largely fail to follow the dietary guidelines.[19–22] As for obesity, the prevalence has not declined,[2,23] and social inequalities in diet[24] and obesity[10,25,26] have persisted or even increased.

Recent research findings,[27–35] particularly by Zeevi *et al.*[36] have suggested new perspectives for the prevention and treatment of obesity-related diseases, using personalized dietary approaches. Precision nutrition (PN), also called personalized nutrition, is based on the postulate that the optimal diet is not the same for everyone. In brief, PN aims at delivering tailored nutritional recommendations based on combined information from individuals' gut microbiota, genetic, physiological, and behavioural backgrounds.[37–42]

Following these promising results in clinical research,[27–36] some large public research funders, such as the EU Horizon 2020 programme,[43] have encouraged researchers to test solutions providing tailored nutritional advice to large numbers of people, including healthy individuals. An international trial, Food4Me, was recently launched with 1600 volunteers to test the opportunities and challenges of PN in the general population.[44] Within this context, some[39,42,45,46] have begun to consider PN as an emerging tool for public health to reduce obesity and obesity-related diseases, notably because precision approaches have a marked preventive component.

In parallel, advances in 'omics' technologies and wearable devices facilitate less costly collection and analysis of massive data. This makes scaleable delivery of tailored nutritional advice increasingly plausible.[38,39,42] Thanks to these technical developments and the clinical context explained above, PN could be viewed as a novel opportunity to provide the right dietary intervention to the right population at the right time, and on a large scale.[47–49] In this paper, we explore the promises and potential limitations of interventions based on large-scale PN. We question their relevance in balancing individuals' diet and addressing obesity at a population level. We build our argument on Geoffrey Rose's theory[50,51] regarding the differences in individual and population causes of disease. We finally argue that large-scale PN could possibly have the unintended effect of exacerbating social inequalities in obesity.

What is large-scale precision nutrition?

Modelled after PN in clinical settings,[36] large-scale PN relies on the collection and analysis of several types of data

from eating behaviour, physical activity, deep phenotyping, nutrigenomics, microbiomics/metagenomics and metabolomics[37–42] (Table 1). These data serve to define the appropriate diet for each individual, or more realistically, each population sub-stratum.[49,52] Different amounts of data can be collected and analysed depending on the infrastructure availability and financial resources. For example, in the Food4Me trial, the intervention involved the delivery of personalized nutrition advice based on data from: (i) current diet; or (ii) diet plus phenotypic traits such as waist circumference, serum glucose, total cholesterol, carotenes and omega 3 index; (iii) diet and phenotype plus genotype (i.e. specific variants on five diet-responsive genes).[44]

Once the desired level of precision/information is defined, data can be collected on a large scale using personal smartphones and other relatively inexpensive and reliable wearable devices, such as an electronic food diary and wristband for accelerometry.[38,39] In parallel, new tools (Table 1), such as dried blood spot testing[42] already routinely used for the Guthrie test in newborns,[53] and simple stool kits,[36,54] enable biosample collection from home or a

Table 1. Potential sources of data for tailored nutritional advice in large-scale precision nutrition interventions

Data	Aims of data collection	Methods to produce data	Infrastructures and tools to collect, analyse and store data
Eating behaviour	To evaluate: • Dietary intake (e.g. food consumption, use of nutrient supplements) • Eating behaviour	Dietary assessment on several days using: • Online food diary • Smartphone applications (self-description and quantification of consumed foods) • Digital photography (semi-automatic identification and quantification of consumed foods)	• Dried blood spot testing • Saliva swabs • Stool kits • Shipment material • Local pharmacy networks • Accelerometers • Smartphone and other digital technologies • Biobanks • Linkage with electronic health records • Biomedical laboratories • Artificial intelligence etc.
Physical activity	To measure physical activity level To estimate energy expenditure	Accelerometry techniques using: • Wearable/portable devices (e.g. wristband) • Online questionnaire	
Deep phenotyping	To assess: • Body composition • Nutritional status • Other risk factors for diet-related diseases	Anthropometric measurements (e.g. weight, waist circumference, bone densitometry) Clinical chemistry from various bio-samples (e.g. plasma, urine, saliva) to assess visceral fat distribution, insulin resistance, low-density lipoprotein cholesterol, nutrient deficiencies, etc.	
Nutrigenomics	To look for genetic variants associated with diet-related diseases and/or responsive to dietary changes	DNA extraction and genotyping of selected loci from whole-blood samples	
Microbiomics/ metagenomics	To understand the interplay between diet and gut microbiota	Faeces collection to sequence the microorganisms present in the gut for microbial profiling and detection of dysbiosis	
Metabolomics	To understand how the body metabolizes/uses nutrients	Complex chemical analyses from biosamples (e.g. serum, plasma, urine) using: • Nuclear magnetic resonance spectroscopy • Mass spectrometry-based techniques	

local pharmacy. The Food4Me intervention was entirely internet-delivered, for instance. Participants themselves collected both biosamples, using the saliva swabs for genotyping and dried blood spots for phenotyping. They followed online demonstrations, and sent their biological material by conventional mail.[44] The advances of laboratory analytical techniques (e.g. DNA sequencing, mass spectrometry),[39,42] bioinformatics, and artificial intelligence (e.g. machine-learning algorithms, deep learning)[36,38,55,56] render the analysis and interpretation of large datasets less and less expensive and time-consuming.

Lastly, smartphone applications allow large-scale dissemination of personalized advice directly to individuals. For instance, the applications delivered by the companies DayTwo[57] and Viome[58] can provide a personal score for foods or recipes regarding their potential positive or negative impact on blood glucose level. The enterprise habit[59] even offers detailed menu plans to comply with personalized recommended intake in terms of protein, carbohydrate and fat.

Large-scale precision nutrition: promises and challenges

The central promise of large-scale PN is personalized interventions based on more: (i) preventive (predictive and accurate); (ii) practical (understandable and implementable); and (iii) dynamic nutritional advice than 'one-size-fits-all' guidelines.[39] First, PN advocates presume that nutritional advice is likely to be more predictive because the personal risk of developing specific diseases (e.g. based on polygenic risk scores) and biomedical context can be considered.[40,49,60] Advice could also be more accurate due to more precise dietary intake and nutritional status assessment[61–65] and better anticipation of interpersonal variability in food metabolic response.[36,66,67] Second, personalized nutritional advice may be easily understood, as messages could be delivered in a simpler way using modern communication techniques.[68,69] Advice may also be more implementable as adapted to actual food consumption, personal food preferences and lifestyle.[68–70] Third, nutritional advice would evolve following the personal dietary and biomedical evolutions of each individual as automatically processed and refined over time through new data.[39] In sum, large-scale PN promises better individual risk identification through comprehensive screening and behavioural modification in line with these identified risks.

At present, large-scale PN faces two main challenges, however. On the one hand, its application on a large scale raises organizational, legal and ethical questions, notably regarding biobank management, data protection and informed consent.[42,52,71] However, these technical challenges are currently being addressed by some countries that have launched large-scale precision medicine projects, such as the Precision Medicine Initiative in one million US residents,[72] and the human biomonitoring project (HBM4EU) in 28 European countries.[73] On the other hand, the effectiveness associated with both identifying the individual risk and delivering personal messages for prevention and treatment of obesity-related disease is disputed.[4,38,40,74–77] The 2018 *Lancet* review by Wang and Hu[38] concluded that evidence is currently lacking to support the additional benefits of PN over 'one-size-fits-all' nutrition intervention in the prevention and treatment of type 2 diabetes. Evidence regarding effectiveness and cost-effectiveness of large-scale PN in the general population is even scarcer. To date, the Food4Me trial has determined that participants receiving personalized advice had a healthier diet compared with controls receiving standard guidelines after the 6-month intervention (completion rate: 79%).[78] However, no significant changes in weight or waist circumference were observed, even when phenotypic or genotypic data were considered to personalized diet. The question of effectiveness on population health will probably remain open for some years.

Obese individuals and obese populations

In public health, two main traditional strategies have existed for preventive interventions: high-risk and population approaches.[50,51] The traditional population approach seeks an improvement of overall population health by shifting the distribution of exposure risk in a favourable direction in the entire population (**Figure 1A**). With the assumption that 'a large number of people at a small risk may give rise to more cases of disease than the small number who are at a high risk', the population approach contrasts with the high-risk approach.[50] The high-risk approach proposes targeted interventions addressed only to individuals screened for their higher probability of developing the disease.[50]

Large-scale PN targets the whole population in the spirit of a traditional population approach. Both preventive strategies can be used for primary and secondary prevention. However, large-scale PN interventions substantially differ from the traditional population interventions, in the way of achieving the distribution shift. The former targets individual risk with precision behavioural measures in large numbers, whereas the latter targets overall population risk with structural/environmental measures, as shown below.

In the 1985 seminal article 'Sick individuals and sick populations',[50] still considered relevant for modern public health,[79] Geoffrey Rose suggested a distinction be made

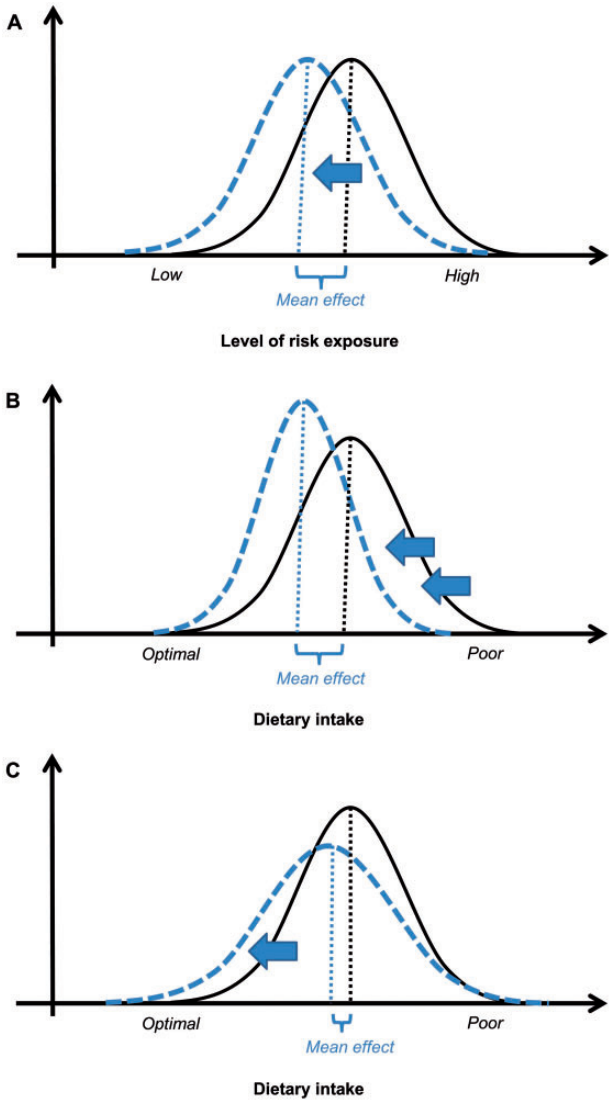

Figure 1. Impact of public health interventions on health. A. Intended effect of Rose's population strategy on risk of exposure (i.e. large mean effect and unchanged standard deviation after the intervention). B. Desirable impact of public health interventions on dietary intake (i.e. large mean effect and decreased standard deviation after the intervention). C. Probable impact of large-scale precision nutrition on dietary intake (i.e. small mean effect and increased standard deviation after intervention). Solid line: distribution of risk/dietary intake before the intervention. Dashed line: distribution of risk/dietary intake after the intervention.

between two kinds of disease determinants. First, the causes of cases explain why individuals become sick (i.e. individual risk). Second, the causes of incidence explain why certain populations become sick, whereas others do not (i.e. population risk). Rose,[50,51] and later Schwartz and Diez-Roux,[80] demonstrated that the causes of cases and of incidence are not necessarily the same, even if they are often related. Using empirical examples for hypertension and hypercholesterolaemia, Rose showed that causes of cases originate generally from the individual variation in genetic, social and behavioural factors, or a mixture of them (i.e. what we call today gene-environment interactions).[51] As for the causes of incidence, they originate instead from the population variation in collective and societal characteristics.[51]

Returning to the issue of obesity, Rose would argue that the causes of some individuals becoming obese differ from the causes of some populations becoming obese. Table 2 provides examples of the distinction between causes of obesity in individuals and those in populations, knowing that inadequate diet and lack of physical activity are common causes at both individual and population levels. Based on the determinants listed in Table 2, we observe that the causes of incidence are largely related to the living conditions encouraging excessive food intake and discouraging physical activity. Others have grouped these determinants under the umbrella term of obesogenic environments.[81–88] As for large-scale PN, it accounts only for some of the causes of obesity in individuals: e.g. genetic predisposition, gut microbial dysbiosis and lack of food and nutrition literacy regarding the meaning of healthy eating. By definition, as PN is an individualized approach, it does not address any causes of incidence. That is why we define large-scale PN as an individual-centred approach in large numbers, rather than a true population approach.

Individual-centred interventions targeting behaviour change in large numbers can bring benefits to some individuals or sub-strata in the population. For example, it could allow early detection of rare forms of monogenic obesity, such as leptin deficiency due to LEP gene mutations.[89,90] However, such interventions are less valuable for overall population health, especially in the case of common diseases with reduced penetrance, such as obesity.[51,91] We will now discuss these limitations.

Limitations of individual-centred strategies for population health

Individual-centred strategies often offer temporary and palliative, rather than radical, success at a population level because they do not alter the conditions that affect the overall distribution.[50,79] In other words, helping individuals to reduce their individual level of risk exposure does not address the root of the problem determining population risk exposure.[51] For obesity, the root of the problem of inadequate dietary intake in most high-income populations is mainly the obesogenic food environment, as mentioned previously.

High-energy and ultra-processed foods rich in sodium, added sugars and saturated fats are widely available in shops and restaurants, and hence in households.[92,93] This is particularly applicable for people of lower

Table 2. Non-exhaustive list of determinants of obesity in individuals vs those of obese populations in most high-income countries

	Causes of cases: individual risk Why do some individuals in a population become obese?	Causes of incidence: population risk Why do some populations become obese whereas others do not?
Common causes	Quantitative and qualitative imbalance in diet Lack of physical activity	
Distinctive causes	Genetic predisposition Diseases, metabolic and endocrine disorders Medications associated with weight gain Lack of richness and diversity in gut microbiota Age Lack of food and nutrition literacy Psychological factors	Food markets making high-energy and ultra-processed foods widely available, low-priced, delivered in large portion sizes, and/or prominently marketed Agricultural policies and subsidies promoting the production of less healthy foods Built environment and transportation policies promoting physical inactivity School and workplace environment not encouraging healthy eating and physical activity Loss of traditional culture around food, cooking and meals Values associated with slimness and fatness

socioeconomic status (SES), who tend to experience a more prominent obesogenic food environment in their neighbourhoods.[85,94,95] For example, lack of access to shops/supermarkets to buy fresh healthy products and overexposure to fast-food restaurants have been documented in the US poorer neighbourhoods.[85,94,95] Moreover, high-energy and ultra-processed foods tend to cost less than healthier alternatives.[96–99] High-energy and ultra-processed foods are also heavily advertised,[100,101] promoting their over-consumption especially in children.[102,103] In addition, food is sold in large portion sizes encouraging overeating.[87] Of note, social and cultural norms (e.g. reduction of time and/or skills to shop, prepare and eat food, and frequent snacking) tend to favour imbalanced diets and excessive food intake.[104,105] These social, cultural, economic and political barriers hinder healthy eating on a daily basis. If these barriers persist at a population level, the weight loss success of some individuals, thanks to large-scale PN, might be attenuated by the future weight gain of their neighbours or children who are exposed to the same unchanged obesogenic environments. This puts them continually at risk of obesity.[106]

Similarly, if the root causes of disease in the population are not addressed, individual-centred strategies tend to be behaviourally and culturally difficult to maintain over time.[50,79] Namely, implementing behaviour change at an individual level becomes challenging when 'social norms' (i.e. peers and environment) are not altered. Deviation from norms necessitates constant effort to sustain alternative behaviours.[107–110] This might enlighten us as to why individual-centred programmes, aimed at changing eating behaviour and/or maintaining weight loss in a priori motivated people, have regularly yielded disappointing results in the long term.[111–113]

PN advocates could argue that knowing the higher personal risk of obesity might further motivate people to change their diet. A systematic review of seven randomized and quasi-randomized controlled trials[114] and a more recent trial[115] however did not support this hypothesis. They found that communicating DNA-based risk estimates for common complex diseases did not enhance eating behaviour compared with non-DNA-based risk estimates or no risk estimates at all. It seems, indeed, that targeting individual eating behaviour with rational advice on food choices without simultaneously tackling the social, cultural, economic and political conditions in which behaviours occur is unlikely to generate large, long-term dietary changes at a population level.

Together with efficacy, public health interventions aim at maximizing equity or at least mitigating inequity.[91,116] In other words, desirable population interventions should have a large mean effect size together with a decreased standard deviation (Figure 1B). Applied to large-scale PN, desirable interventions should reduce the gap between those with the best and worse dietary intake. This means that they should have the most impact on groups with poorer dietary intake, often those of lower SES.[24,117,118] However, several reviews have shown that individual-centred public health interventions targeting behavioural changes to improve nutrition[119,120] or health[121,122] provide less benefit to lower SES groups. For example, Sumar and McLaren[123] demonstrated that public information campaigns about the importance of folic acid intake among childbearing-aged women (i.e. an intervention requiring individual decisions to change behaviour) were more likely to increase socioeconomic inequalities in folate status than staple food fortification with folic acid (i.e. an

intervention at a policy level requiring no individual decision making).

Inequalities resulting from individual-centred interventions targeting the entire population can be understood through the 'capability approach', developed by Amartya Sen.[124,125] He stated that people with the same amount of resources at hand are not equal in capacity, that is in what they are able to actually achieve with these resources. Specifically to health, Link and Phelan's fundamental cause theory[126,127] states that individuals of higher SES have a wider range of 'flexible resources' with regard to knowledge, wealth, power and social networks than individuals of lower SES. Thanks to these resources, they can better understand information, afford and become motivated to engage in a larger range of activities focusing on their health improvement. In essence, control over the determinants of diet and the motivation to act on it is unequally distributed within a population.

These theories, plus the role of obesogenic neighbourhoods, may partly explain why individuals of higher SES have already taken the most advantage of previous public health individual-centred interventions and thus have lower obesity prevalence than less privileged individuals.[10,25,26] From this observation, and building on the fundamental cause theory,[126,127] we believe that smartphone applications delivering tailored nutritional advice, albeit free, may be more or less attractive and differentially used according to SES. Early adoption and achievement of improved dietary habits is hence more likely among more socially privileged members of the population. This could exacerbate socioeconomic inequalities in diet and in obesity. This is not only an equity concern, but also one of efficacy. Indeed, if mostly privileged groups in the population improve their eating habits, this would have a limited impact on overall population health, since they already demonstrate a lower risk of obesity and obesity-related disease.

Conclusion

Some public health actors have become enthused by the central promise of PN to better identify the individual risks and suggest targeted dietary modification. They expect PN applied at a large scale to improve populations' diet and health. We showed, however, that individual-centred interventions directed to behaviour change, such as large-scale PN, are likely to have a limited and unequal impact on diet and obesity incidence at a population level (Figure 1C), particularly if the obesogenic environments are not addressed in the first place.

We nevertheless believe that knowledge and technologies from large-scale PN (Table 1) may provide improved solutions to two recurring concerns in nutritional epidemiology: (i) the accurate assessment of food and nutrients intakes, together with physical activity, in relation to energy intake and expenditure; and (ii) the long-term monitoring of nutritional status at individual and population levels. This may improve or validate our understanding of the impact of dietary intakes and changes on the personal risk of diseases and related biological pathways.[36,38,41,65,128] Similarly, methods used in large-scale PN could complement traditional subjective and/or memory-based dietary assessment methods, such as food frequency questionnaires and 24-h dietary recalls.[38,39,41,61,63,64] Overall, this may help confirm or refine dietary guidelines for specific population sub-strata. Despite the potential for causal inference and population surveillance, we conclude that PN on a large scale would be of limited interest for public health interventions in the prevention of common polygenic diseases, such as obesity. The impact of large-scale PN on populations' health is likely to be minor and unevenly distributed in the populations in the absence of complementary social and structural/environmental measures.

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Author Contributions

AC conceptualized the manuscript under the supervision of KLF. MB provided inputs to the manuscript. All authors read and approved the final manuscript.

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References

1. World Health Organization. *Obesity and Overweight. Key Facts*. http://www.who.int/mediacentre/factsheets/fs311/en/ (13 June 2018, date last accessed).
2. Hales CM, Fryar CD, Carroll MD, Freedman DS, Ogden CL. Trends in obesity and severe obesity prevalence in US youth and adults by sex and age, 2007-2008 to 2015-2016. *JAMA* 2018;**319**:1723–25.
3. Pi-Sunyer X. The medical risks of obesity. *Postgrad Med* 2009; **121**:21–33.

4. Goodarzi MO. Genetics of obesity: what genetic association studies have taught us about the biology of obesity and its complications. *Lancet Diabetes Endocrinol* 2018;**6**:223–36.
5. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA* 1999;**282**:1523–29.
6. Global Burden of Disease Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 2017;**390**: 1345–422.
7. Drewnowski A. Obesity, diets, and social inequalities. *Nutr Rev* 2009;**67**(**Suppl 1**):S36–39.
8. Robertson A, Lobstein T, Knai C. *Obesity and ocio-economic Groups in Europe: Evidence Review and Implications for Action. Report SANCO/2005/C4-NUTRITION-03*. Brussels: European Commission, 2007.
9. Siddiqi A, Brown R, Nguyen QC, Loopstra R, Kawachi I. Cross-national comparison of socioeconomic inequalities in obesity in the United States and Canada. *Int J Equity Health* 2015;**14**:116.
10. Devaux M, Sassi F. Social inequalities in obesity and overweight in 11 OECD countries. *Eur J Public Health* 2013;**23**: 464–69.
11. Food and Agriculture Organization. *Food-based Dietary Guidelines*. http://www.fao.org/nutrition/nutrition-education/food-dietary-guidelines/en/ (13 June 2018, date last accessed).
12. United States Department of Agriculture. *2015–2020 Dietary Guidelines for Americans*. https://www.choosemyplate.gov/dietary-guidelines (13 June 2018, date last accessed).
13. Public Health England. *The Eatwell Guide*. https://www.gov.uk/government/publications/the-eatwell-guide (13 June 2018, date last accessed).
14. United States Department of Agriculture. Nutrition standards in the national school lunch and school breakfast programs. *Federal Register* 2012;**77**:4088–167.
15. United Kingdom Department of Education. *The Requirements for School Food Regulations*, 2014, No. 1603.
16. Backholer K, Blake M, Vandevijvere S. Sugar-sweetened beverage taxation: an update on the year that was 2017. *Public Health Nutr* 2017;**20**:3219–24.
17. Silver LD, Ng SW, Ryan-Ibarra S, Taillie LS, Induni M, Miles DR. Changes in prices, sales, consumer spending, and beverage consumption one year after a tax on sugar-sweetened beverages in Berkeley, California, US: a before-and-after study. *PLoS Med* 2017;**14**:e1002283.
18. Colchero MA, Molina M, Guerrero-Lopez CM. After Mexico implemented a tax, purchases of sugar-sweetened beverages decreased and water increased: difference by place of residence, household composition, and income level. *J Nutr* 2017;**147**: 1552–57.
19. Rehm CD, Penalvo JL, Afshin A, Mozaffarian D. Dietary intake among US adults, 1999-2012. *JAMA* 2016;**315**:2542–53.
20. Bates B, Lennox A, Prentice A *et al*. *National Diet and Nutrition Survey: Results from Years 1, 2, 3 and 4 (Combined) of the Rolling Programme (2008/2009–2011/2012)*. London: Public Health England & Food Standard Agency, 2014.
21. Diethelm K, Jankovic N, Moreno LA *et al*. Food intake of European adolescents in the light of different food-based dietary guidelines: results of the HELENA (Healthy Lifestyle in Europe by Nutrition in Adolescence) Study. *Public Health Nutr* 2012;**15**:386–98.
22. Chatelan A, Beer-Borst S, Randriamiharisoa A *et al*. Major differences in diet across three linguistic regions of Switzerland: results from the First National Nutrition Survey menuCH. *Nutrients* 2017;**9**. doi: 10.3390/nu9111163.
23. Non-communicable Disease Risk Factor Collaboration. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. *Lancet* 2017;**390**:2627–42.
24. Wang DD, Li Y, Chiuve SE, Hu FB, Willett WC. Improvements in US diet helped reduce disease burden and lower premature deaths, 1999-2012; overall diet remains poor. *Health Aff (Millwood)* 2015;**34**:1916–22.
25. Stamatakis E, Primatesta P, Chinn S, Rona R, Falascheti E. Overweight and obesity trends from 1974 to 2003 in English children: what is the role of socioeconomic factors? *Arch Dis Child* 2005;**90**:999–1004.
26. Singh GK, Siahpush M, Hiatt RA, Timsina LR. Dramatic increases in obesity and overweight prevalence and body mass index among ethnic-immigrant and social class groups in the United States, 1976-2008. *J Community Health* 2011;**36**: 94–110.
27. Qi Q, Chu AY, Kang JH *et al*. Sugar-sweetened beverages and genetic risk of obesity. *N Engl J Med* 2012;**367**:1387–96.
28. Price ND, Magis AT, Earls JC *et al*. A wellness study of 108 individuals using personal, dense, dynamic data clouds. *Nat Biotechnol* 2017;**35**:747–56.
29. Wang T, Huang T, Zheng Y *et al*. Genetic variation of fasting glucose and changes in glycemia in response to 2-year weight-loss diet intervention: the POUNDS LOST trial. *Int J Obes* 2016;**40**:1164–69.
30. Qi Q, Bray GA, Smith SR, Hu FB, Sacks FM, Qi L. Insulin receptor substrate 1 gene variation modifies insulin resistance response to weight-loss diets in a 2-year randomized trial: the Preventing Overweight Using Novel Dietary Strategies (POUNDS LOST) trial. *Circulation* 2011;**124**:563–71.
31. Huang T, Ley SH, Zheng Y *et al*. Genetic susceptibility to diabetes and long-term improvement of insulin resistance and beta cell function during weight loss: the Preventing Overweight Using Novel Dietary Strategies (POUNDS LOST) trial. *Am J Clin Nutr* 2016;**104**:198–204.
32. Corella D, Arregui M, Coltell O *et al*. Association of the LCT-13910C>T polymorphism with obesity and its modulation by dairy products in a Mediterranean population. *Obesity (Silver Spring)* 2011;**19**:1707–14.
33. Goni L, Cuervo M, Milagro FI, Martinez JA. A genetic risk tool for obesity predisposition assessment and personalized nutrition implementation based on macronutrient intake. *Genes Nutr* 2015;**10**:445.
34. Wu GD, Compher C, Chen EZ *et al*. Comparative metabolomics in vegans and omnivores reveal constraints on diet-dependent gut microbiota metabolite production. *Gut* 2016; **65**:63–72.

35. Ortega-Azorin C, Sorli JV, Estruch R *et al*. Amino acid change in the carbohydrate response element binding protein is associated with lower triglycerides and myocardial infarction incidence depending on level of adherence to the Mediterranean diet in the PREDIMED trial. *Circ Cardiovasc Genet* 2014;**7**: 49–58.
36. Zeevi D, Korem T, Zmora N *et al*. Personalized nutrition by prediction of glycemic responses. *Cell* 2015;**163**:1079–94.
37. Ferguson LR, De Caterina R, Gorman U *et al*. Guide and position of the International Society of Nutrigenetics/Nutrigenomics on Personalised Nutrition: Part 1—Fields of Precision Nutrition. *J Nutrigenet Nutrigenomics* 2016;**9**:12–27.
38. Wang DD, Hu FB. Precision nutrition for prevention and management of type 2 diabetes. *Lancet Diabetes Endocrinol* 2018; **6**:416-26.
39. de Toro-Martin J, Arsenault BJ, Despres JP, Vohl MC. Precision nutrition: a review of personalized nutritional approaches for the prevention and management of metabolic syndrome. *Nutrients* 2017;**9**. doi: 10.3390/nu9080913.
40. Heianza Y, Qi L. Gene-diet interaction and precision nutrition in obesity. *Int J Mol Sci* 2017;**18**. doi: 10.3390/ijms18040787.
41. Ferguson JF, Allayee H, Gerszten RE *et al*. Nutrigenomics, the microbiome, and gene-environment interactions: new directions in cardiovascular disease research, prevention, and treatment: a scientific statement from the American Heart Association. *Circ Cardiovasc Genet* 2016;**9**:291–313.
42. Kohlmeier M, De Caterina R, Ferguson LR *et al*. Guide and position of the International Society of Nutrigenetics/ Nutrigenomics on Personalized Nutrition: Part 2—ethics, challenges and endeavors of precision nutrition. *J Nutrigenet Nutrigenomics* 2016;**9**:28–46.
43. European Commission. *EU Programmes 2014–2020. Horizon2020. TOPIC: Personalized Nutrition.* http://ec.europa.eu/research/participants/portal/desktop/en/opportunities/h2020/topics/dt-sfs-14–2018.html (13 June 2018, date last accessed).
44. Celis-Morales C, Livingstone KM, Marsaux CF *et al*. Design and baseline characteristics of the Food4Me study: a web-based randomised controlled trial of personalised nutrition in seven European countries. *Genes Nutr* 2015;**10**:450.
45. Newell A, Zlot A, Silvey K, Arail K. Addressing the obesity epidemic: a genomics perspective. *Prev Chronic Dis* 2007;**4**:A31.
46. Claus SP. Development of personalized functional foods needs metabolic profiling. *Curr Opin Clin Nutr Metab Care* 2014;**17**: 567–73.
47. Khoury MJ, Iademarco MF, Riley WT. Precision public health for the era of precision medicine. *Am J Prev Med* 2016;**50**: 398–401.
48. Vaithinathan AG, Asokan V. Public health and precision medicine share a goal. *J Evid Based Med* 2017;**10**:76–80.
49. Belsky DW, Moffitt TE, Caspi A. Genetics in population health science: strategies and opportunities. *Am J Public Health* 2013; **103**(**Suppl 1**):S73–83.
50. Rose G. Sick individuals and sick populations. *Int J Epidemiol* 1985;**14**:32–38.
51. Rose G. *The Strategy of Preventive Medicine*. Oxford, UK: Oxford University Press, 1992.
52. Burton H, Sagoo GS, Pharoah P, Zimmern R-L. Time to revisit Geoffrey Rose: strategies for prevention in the genomic era? *Ital J Public Health* 2012;**9**. doi: 10.2427/8665.
53. Guthrie R, Susi A. A simple phenylalanine method for detecting phenylketonuria in large populations of newborn infants. *Pediatrics* 1963;**32**:338–43.
54. Human Microbiome Project Consortium. Structure, function and diversity of the healthy human microbiome. *Nature* 2012; **486**:207–14.
55. Rimbach G, Minihane AM. Nutrigenetics and personalised nutrition: how far have we progressed and are we likely to get there? *Proc Nutr Soc* 2009;**68**:162–72.
56. Beam AL, Kohane IS. Big data and machine learning in health care. *JAMA* 2018;**319**:1317–18.
57. DayTwo. *DayTwo: Personalized Nutrition Based On Your Gut Microbiome*. https://www.daytwo.com (13 June 2018, date last accessed).
58. Viome. *Viome: Gut Microbiome and Wellness*. https://www.viome.com (13 June 2018, date last accessed).
59. Habit. *Habit: Personalized Nutrition Designed for Better Health & Weight Loss*. https://habit.com (13 June 2018, date last accessed).
60. Weber GM, Mandl KD, Kohane IS. Finding the missing link for big biomedical data. *JAMA* 2014;**311**:2479–80.
61. Garcia-Perez I, Posma JM, Gibson R *et al*. Objective assessment of dietary patterns by use of metabolic phenotyping: a randomised, controlled, crossover trial. *Lancet Diabetes Endocrinol* 2017;**5**:184–95.
62. Vernocchi P, Vannini L, Gottardi D *et al*. Integration of datasets from different analytical techniques to assess the impact of nutrition on human metabolome. *Front Cell Infect Microbiol* 2012;**2**:156.
63. Lloyd AJ, Beckmann M, Haldar S, Seal C, Brandt K, Draper J. Data-driven strategy for the discovery of potential urinary biomarkers of habitual dietary exposure. *Am J Clin Nutr* 2013;**97**: 377–89.
64. Martin CK, Correa JB, Han H *et al*. Validity of the Remote Food Photography Method (RFPM) for estimating energy and nutrient intake in near real-time. *Obesity (Silver Spring)* 2012; **20**:891–99.
65. Vazquez-Fresno R, Llorach R, Urpi-Sarda M *et al*. Metabolomic pattern analysis after mediterranean diet intervention in a nondiabetic population: a 1- and 3-year follow-up in the PREDIMED study. *J Proteome Res* 2015;**14**:531–40.
66. Connaugton RM, McMorrow AM, Healy ML, McGillicuddy FC, Lithander FE, Roche HM (eds). An anti-inflammatory nutritional intervention selectively improves insulin sensitivity in overweight and obese adolescents wherein baseline metabotype predicts response. *Proceedings of the Irish Section Meeting, Changing Dietary Behaviour: Physiology through to Practice, 18-20 June 2014*. University of Ulster, UK, 2014.
67. Sevilla-Villanueva B, Gibert K, Sanchez-Marre M, Fito M, Covas MI. Evaluation of adherence to nutritional intervention through trajectory analysis. *IEEE J Biomed Health Inform* 2017;**21**:628–34.
68. Nielsen DE, El-Sohemy A. A randomized trial of genetic information for personalized nutrition. *Genes Nutr* 2012;**7**:559–66.

69. Brug J, Campbell M, van Assema P. The application and impact of computer-generated personalized nutrition education: a review of the literature. *Patient Educ Couns* 1999;**36**:145–56.
70. Afshin A, Babalola D, McLean M *et al*. Information technology and lifestyle: a systematic evaluation of internet and mobile interventions for improving diet, physical activity, obesity, tobacco, and alcohol use. *J Am Heart Assoc* 2016;**5**. doi: 10.1161/JAHA.115.003058.
71. Stewart-Knox B, Kuznesof S, Robinson J *et al*. Factors influencing European consumer uptake of personalised nutrition. Results of a qualitative analysis. *Appetite* 2013;**66**:67–74.
72. Collins FS, Varmus H. A new initiative on precision medicine. *N Engl J Med* 2015;**372**:793–95.
73. German Environment Agency. *Human Biomonitoring for Europe. HBM4EU*. https://www.hbm4eu.eu (13 June 2018, date last accessed).
74. Gardner CD, Trepanowski JF, Del Gobbo LC *et al*. Effect of low-fat vs low-carbohydrate diet on 12-month weight loss in overweight adults and the association with genotype pattern or insulin secretion: the DIETFITS randomized clinical trial. *JAMA* 2018;**319**:667–79.
75. Livingstone KM, Celis-Morales C, Papandonatos GD *et al*. FTO genotype and weight loss: systematic review and meta-analysis of 9563 individual participant data from eight randomised controlled trials. *BMJ* 2016;**354**:i4707.
76. Look AHEAD Research Group. Prospective association of GLUL rs10911021 with cardiovascular morbidity and mortality among individuals with type 2 diabetes: the Look AHEAD study. *Diabetes* 2016;**65**:297–302.
77. Tyrrell J, Wood AR, Ames RM *et al*. Gene-obesogenic environment interactions in the UK Biobank study. *Int J Epidemiol* 2017;**46**:559–75.
78. Celis-Morales C, Livingstone KM, Marsaux CF *et al*. Effect of personalized nutrition on health-related behaviour change: evidence from the Food4Me European randomized controlled trial. *Int J Epidemiol* 2017;**46**:578–88.
79. Doyle YG, Furey A, Flowers J. Sick individuals and sick populations: 20 years later. *J Epidemiol Community Health* 2006; **60**:396–98.
80. Schwartz S, Diez-Roux AV. Commentary: causes of incidence and causes of cases—a Durkheimian perspective on Rose. *Int J Epidemiol* 2001;**30**:435–39.
81. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. *Science* 1998;**280**:1371–74.
82. Egger G, Swinburn B. An "ecological" approach to the obesity pandemic. *BMJ* 1997;**315**:477–80.
83. Swinburn B, Egger G, Raza F. Dissecting obesogenic environments: the development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Prev Med* 1999;**29**(6 Pt 1):563–70.
84. Drewnowski A. Obesity and the food environment: dietary energy density and diet costs. *Am J Prev Med* 2004;**27**(**Suppl 3**): 154–62.
85. Papas MA, Alberg AJ, Ewing R, Helzlsouer KJ, Gary TL, Klassen AC. The built environment and obesity. *Epidemiol Rev* 2007;**29**:129–43.
86. Booth KM, Pinkston MM, Poston WS. Obesity and the built environment. *J Am Diet Assoc* 2005;**105**(**5 Suppl 1**):S110–17.
87. Osei-Assibey G, Dick S, Macdiarmid J *et al*. The influence of the food environment on overweight and obesity in young children: a systematic review. *BMJ Open* 2012;**2**. doi: 10.1136/bmjopen-2012-001538.
88. Glanz K, Sallis JF, Saelens BE, Frank LD. Healthy nutrition environments: concepts and measures. *Am J Health Promot* 2005;**19**:330–33, ii.
89. Fruhbeck G, Kiortsis DN, Catalan V. Precision medicine: diagnosis and management of obesity. *Lancet Diabetes Endocrinol* 2018;**6**:164–66.
90. ShabanaHasnain S. The p. N103K mutation of leptin (LEP) gene and severe early onset obesity in Pakistan. *Biol Res* 2016; **49**:23.
91. Keyes KM, Galea S. *Population Health Science*. New York, NY: Oxford University Press, 2016.
92. Pan American Health Organization (PAHO). *Ultra-processed Food and Drink Products in Latin America: Trends, Impact on Obesity, Policy Implications. Chapter 3. Ultra-processed Product Sales*. Washington, DC: PAHO, 2015.
93. Monteiro CA, Moubarac JC, Levy RB, Canella DS, Louzada M, Cannon G. Household availability of ultra-processed foods and obesity in nineteen European countries. *Public Health Nutr* 2018;**21**:18–26.
94. Lovasi GS, Hutson MA, Guerra M, Neckerman KM. Built environments and obesity in disadvantaged populations. *Epidemiol Rev* 2009;**31**:7–20.
95. Cummins S, Macintyre S. Food environments and obesity—neighbourhood or nation? *Int J Epidemiol* 2006;**35**:100–04.
96. Rao M, Afshin A, Singh G, Mozaffarian D. Do healthier foods and diet patterns cost more than less healthy options? A systematic review and meta-analysis. *BMJ Open* 2013;**3**. doi: 10.1136/bmjopen-2013-004277.
97. Jones NR, Conklin AI, Suhrcke M, Monsivais P. The growing price gap between more and less healthy foods: analysis of a novel longitudinal UK dataset. *PLoS One* 2014;**9**:e109343.
98. Drewnowski A, Darmon N. The economics of obesity: dietary energy density and energy cost. *Am J Clin Nutr* 2005;**82**(**Suppl 1**):265S–73S.
99. Andrieu E, Darmon N, Drewnowski A. Low-cost diets: more energy, fewer nutrients. *Eur J Clin Nutr* 2006;**60**:434–36.
100. Batada A, Seitz MD, Wootan MG, Story M. Nine out of 10 food advertisements shown during Saturday morning children's television programming are for foods high in fat, sodium, or added sugars, or low in nutrients. *J Am Diet Assoc* 2008;**108**: 673–78.
101. Sixsmith R, Furnham A. A content analysis of British food advertisements aimed at children and adults. *Health Promot Int* 2010;**25**:24–32.
102. Dovey TM, Taylor L, Stow R, Boyland EJ, Halford JC. Responsiveness to healthy television (TV) food advertisements/commercials is only evident in children under the age of seven with low food neophobia. *Appetite* 2011;**56**:440–46.
103. Halford JC, Boyland EJ, Hughes G, Oliveira LP, Dovey TM. Beyond-brand effect of television (TV) food advertisements/commercials on caloric intake and food choice of 5-7-year-old children. *Appetite* 2007;**49**:263–67.
104. Smith LP, Ng SW, Popkin BM. Trends in US home food preparation and consumption: analysis of national nutrition surveys

and time use studies from 1965-1966 to 2007-2008. *Nutr J* 2013;**12**:45.
105. Higgs S, Thomas J. Social influences on eating. *Curr Opin Behav Sci* 2016;**9**:1–6.
106. Syme SL. Strategies for health promotion. *Prev Med* 1986;**15**: 492–507.
107. Frohlich KL, Poland B, Sharek M, Contrasting entry points for intervention in health promotion practice: situating and working with context. In: Rootman I, Pederson A, Frohlich KL, Dupéré S (eds). *Health Promotion in Canada New Perspectives on Theory, Practice, Policy, and Research*. 4th edn. Toronto, ON: Canadian Scholars, 2017.
108. Higgs S. Social norms and their influence on eating behaviours. *Appetite* 2015;**86**:38–44.
109. Delormier T, Frohlich KL, Potvin L. Food and eating as social practice—understanding eating patterns as social phenomena and implications for public health. *Sociol Health Illn* 2009;**31**: 215–28.
110. Hargreaves K, Amos A, Highet G *et al*. The social context of change in tobacco consumption following the introduction of 'smokefree' England legislation: a qualitative, longitudinal study. *Soc Sci Med* 2010;**71**:459–66.
111. Dombrowski SU, Knittle K, Avenell A, Araujo-Soares V, Sniehotta FF. Long term maintenance of weight loss with non-surgical interventions in obese adults: systematic review and meta-analyses of randomised controlled trials. *BMJ* 2014;**348**:g2646.
112. Mead E, Brown T, Rees K *et al*. Diet, physical activity and behavioural interventions for the treatment of overweight or obese children from the age of 6 to 11 years. *Cochrane Database Syst Rev* 2017;**6**:CD012651.
113. Douketis JD, Macie C, Thabane L, Williamson DF. Systematic review of long-term weight loss studies in obese adults: clinical significance and applicability to clinical practice. *Int J Obes* 2005;**29**:1153.
114. Hollands GJ, French DP, Griffin SJ *et al*. The impact of communicating genetic risks of disease on risk-reducing health behaviour: systematic review with meta-analysis. *BMJ* 2016;**352**: i1102.
115. Godino JG, van Sluijs EM, Marteau TM, Sutton S, Sharp SJ, Griffin SJ. Lifestyle advice combined with personalized estimates of genetic or phenotypic risk of type 2 diabetes, and objectively measured physical activity: a randomized controlled trial. *PLoS Med* 2016;**13**:e1002185.
116. Benach J, Malmusi D, Yasui Y, Martinez JM. A new typology of policies to tackle health inequalities and scenarios of impact based on Rose's population approach. *J Epidemiol Community Health* 2013;**67**:286–91.
117. Darmon N, Drewnowski A. Does social class predict diet quality? *Am J Clin Nutr* 2008;**87**:1107–17.
118. Giskes K, Avendaño M, Brug J, Kunst AE. A systematic review of studies on socioeconomic inequalities in dietary intakes associated with weight gain and overweight/obesity conducted among European adults. *Obes Rev* 2009;**11**:413–29.
119. Adams J, Mytton O, White M, Monsivais P. Why are some population interventions for diet and obesity more equitable and effective than others? The role of individual agency. *PLoS Med* 2016;**13**:e1001990.
120. Lorenc T, Petticrew M, Welch V, Tugwell P. What types of interventions generate inequalities? Evidence from systematic reviews. *J Epidemiol Community Health* 2013;**67**:190–93.
121. Beauchamp A, Backholer K, Magliano D, Peeters A. The effect of obesity prevention interventions according to socioeconomic position: a systematic review. *Obes Rev* 2014;**15**:541–54.
122. Frohlich KL, Potvin L. Transcending the known in public health practice: the inequality paradox: the population approach and vulnerable populations. *Am J Public Health* 2008; **98**:216–21.
123. Sumar N, McLaren L. Impact on social inequalities of population strategies of prevention for folate intake in women of childbearing age. *Am J Public Health* 2011;**101**:1218–24.
124. Sen A. *Inequality Re-examined*. Cambridge, MA: Harvard University Press, 1992.
125. Frohlich KL. The social determinants of what? *Int J Public Health* 2010;**55**:235–36.
126. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav* 1995;Spec No:80–94.
127. Phelan JC, Link BG, Tehranifar P. Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. *J Health Soc Behav* 2010;**51**(**Suppl**):S28–40.
128. Esko T, Hirschhorn JN, Feldman HA *et al*. Metabolomic profiles as reliable biomarkers of dietary composition. *Am J Clin Nutr* 2017;**105**:547–54.