



Perspective

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Nutrigenetics and personalized nutrition: are we ready for DNA-based dietary advice?

Common genetic variation affects individual nutrient requirements and the use of DNA-based dietary advice, derived from nutrigenetics, has been growing. The growth is about to accelerate as the cost of genotyping continues to fall and research results from major nutrigenetics projects are published. There is still some skepticism; some barriers remain including some commercial tests, which make exaggerated, incorrect claims. There is a need for more public resources dedicated to unbiased, objective review and dissemination of nutrigenetics information; however, nutrigenetics evidence should be assessed in the context of standard nutritional evidence and should not require higher standards. This article argues that we are ready for some DNA-based dietary advice in general nutrition and it can be beneficial. Examples of the scientific validity and health utility of gene–diet interactions will be given and the development of guidelines for assessment and validation of benefits will be discussed.

Keywords: food4me • nutrigenetics • nutrigenomics • nutrition genetics • personal genetics • personalized nutrition

Individual genetic variation affects nutrient requirements and there has been good evidence available for at least 10 years that: “With the identification of polymorphisms, or common mutations, in vitamin metabolism, large percentages of the population may have higher requirements for specific vitamins” [1]. At about the same time as this statement was published, the first genetic-based personalized nutrition tests appeared on the market in the UK [2], yet there is a belief held by some that, more than a decade later, it is still too early to incorporate genetic information into nutritional advice [3].

DNA-based dietary advice is derived from nutrigenetics studies and is a component of what is known as ‘personalized nutrition’, which itself is not new – it is already part of daily life for people with allergies, diabetes (Type 1 and 2), celiac disease sufferers and, of course, people trying to lose weight. There are also well-established gene–diet interactions for which specific nutrition is necessary such as phenylketonuria where the treatment

for this genetic disorder is a lifetime phenylalanine-restricted diet [4]. While the phenylketonuria genetic mutation is rare, the scientific literature of the last 15–20 years contains reproducible evidence of several more common gene–diet interactions; indeed Lee *et al.* describe the creation of a database of over 550 gene–environment interactions related to lipids, cardiovascular disease and Type 2 diabetes [5].

This article will examine the current status of nutrigenetics, the evidence requirements and the context in which that evidence should be assessed. Based on assessments of scientific validity and health utility, it will argue that for some gene–diet interactions the evidence is at least as strong as that accepted for standard nutritional guidelines. It will also acknowledge that there are some dubious nutrigenetic tests on sale and will discuss measures to identify those with value and those without. The overall aim is to provide a strong case for the routine inclusion of personal genetic information in the process of formulating dietary advice.

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Nutrigenetics & nutrigenomics

Nutrigenetics involves the study of how individual genetic variation affects interaction with components of the diets, including micro- and macro-nutrients and toxins. Genetic variation has been demonstrated to affect uptake, transport, metabolism and elimination of food components and also affects individual daily requirements for some essential nutrients. It is sometimes also referred to as nutrigenomics, which actually is an umbrella term for all aspects of gene and diet interactions, including the effects of dietary molecules on gene expression and metabolic profiles [6]. This article is focused specifically on potential uses of nutrigenetics and the effect of genetic variation on nutrient requirements. Nutrigenetic studies assess how genes and diet (and sometimes lifestyle) interact, where the effect of one component is dependent on the status of the other. A classic example is that involving lactose intolerance. In the majority of humans, the enzyme lactase is only produced for the first few years of life when the infant is dependent on milk for nutrition, after which production of the enzyme reduces almost completely. Milk is the only naturally occurring source of lactose and since milk and dairy products have only recently, in evolutionary terms, been common components of the human diet, there was no requirement for the enzyme throughout adult life. A few thousand years ago in Central Europe, a mutation appeared upstream of the lactase gene and the effect was continued production of the enzyme lactase throughout adult life; the mutation proliferated through the generations, presumably because of a survival advantage and is now a very common polymorphism in Europe (range: 30–95%) [7,8]. Another example of genetics affecting nutrition is seen in taste reception. The perception of bitter taste varies among individuals and the population is divided into ‘super’-tasters, tasters and nontasters. The phenotypes are almost completely accounted for by three common SNPs in the gene of the bitter taste receptor *TAS2R38* [9].

The context of nutrigenetics

It is important to carefully define the context in which nutrigenetics may be beneficial and to show clearly how it can be used, because despite the claims of some commercial tests, it is not possible to devise a completely personalized diet from genetic data alone. Nutrigenetic information can be used to modify existing standard guidelines and provide an element of personalization to the otherwise ‘one-size-fits-all’ advice. Genetic information can be beneficial when it is added to other information such as gender, height, weight, age, state of health and so on; it is not used in isolation nor does it override other parameters. Nutrigenetics is part of

everyday nutrition – it is not specifically therapeutic and does not depend on the use of nutraceuticals or supplements. In general use, it is not intended for specific disease prevention, but as an aid in optimizing diet and lifestyle for promoting long-term health based on the best evidence that is available. Nutrigenetic and, indeed, nutritional advice in general, is useful for maintaining health and its primary purpose is not for treating disease. These points are all important for determining the threshold of evidence required to support nutrigenetic advice and, in this context, the appropriate level of evidence should be the same as that applied to existing nutritional guidelines in the first place.

From the point of view of evidence assessment, nutrigenetic information should not be considered to be separate or treated differently in any way from the standard nutritional guidelines. There is no reason or justification to require a higher evidence threshold simply because genetic information is included. It is worth pointing out here that no component standard nutritional guidelines (recommended vitamin intakes, salt and saturated fat reductions, fruit and vegetables, and so on) has been assessed and ‘proven’ to prevent disease in randomized clinical trials (RCTs) in healthy individuals. It is indeed unlikely that such a level of evidence will ever be reached; RCTs may have demonstrated that reducing salt in hypertensive individuals can reduce blood pressure, but there are no RCTs on the effects of lifelong low-salt diets and the reduction of heart disease [10]. While the therapeutic applications of nutrition might be amenable to RCT, the proposed benefits of general nutritional guidelines cannot be tested in this way (see [11] for extended discussion). Thus nutrigenetics should not be held to a higher (unreachable) standard as has sometimes been suggested [12,13]. It should be supported by the type of evidence used, for example, to set vitamin, salt and saturated fat recommendations in healthy people, which is mainly epidemiological and interventional in nature [3].

Current nutrition guidelines are formulated by a variety of organizations at various levels (Box 1). This information is used by scientists, dieticians and other health professionals to communicate nutritional advice to the general public through scientific publications, the mass media and personal consultation. In formulating the guidelines, the expert committees have to make decisions based on the best evidence available; it is clear that there are gaps in the evidence, but it is acknowledged that a scientific judgment of some sort has to be made, that “no decision is not an option” as we all have to consume nutrients daily [14]. The correct application of nutrigenetics is to use these guidelines

Box 1. Tiers of general nutritional advice for public health purposes.

- Official public health guidelines recommend specific intakes of various essential vitamins and minerals, with the objective of achieving health benefits at the population level. They are formulated by expert committees, which assess evidence of public health utility on behalf of such bodies as the UK Food Standards Agency and the Institute of Medicine in the USA [15,16]
- More general advice about all food components such as fats, carbohydrates, caffeine, fiber, fruit and vegetables, red meat, and so on, is issued by various institutions both governmental and nongovernmental (e.g., Departments of Health Nutrition, WHO and British Nutrition Foundation) [17–19]
- Targeted nutritional advice aimed at specific subgroups, for example, breast feeding and timing of gluten introduction for infants with genetic risk of celiac disease communicated by the European Society for Paediatric Gastroenterology Hepatology and Nutrition [20]

as the starting point and to examine where there is evidence that incorporating genetic information to modify intakes of specific nutrients may be beneficial. In order to validate nutrigenetic advice for a particular nutrient the question should be: is the evidence of the same or higher standard compared with that used to justify the standard recommendations? For example, if there is evidence that a genetic variation increases daily requirements for a specific vitamin, the quality of the evidence should be compared with that used to formulate the standard recommended daily allowance for that vitamin for the general population. Nutrigenetic evidence can be formally assessed to determine if the required criteria, scientific validity and health utility, are met.

How to assess the evidence: scientific validity & health utility

There is currently a small but growing commercial market for nutrigenetic tests, which claim to interpret and translate gene–diet information into benefits for the consumer. These tests, just like other routine laboratory tests, could be potentially useful for the individual or health professional but, as with any test, before they should be used, it is necessary to know how reliable the evidence is and how useful is the result for the individual? These questions are encapsulated in the Analytical and Clinical Validity, Clinical Utility and Ethics definitions of ‘scientific validity’ and ‘clinical utility’ (Box 2) [21], which were developed for evaluation of medical tests. These guidelines are helpful for the assessment of nutrigenetic information, with the caveat that the context is not that for a medical test and we should refer to ‘health’ rather than ‘clinical’

utility. For any given gene–diet interaction that satisfies the criteria of scientific validity and health utility, it is a logical conclusion that this specific nutrigenetic information is ready to be, and should be, incorporated into individual dietary advice.

The two gene–diet interactions mentioned in the introduction can be assessed according to these criteria. Both lactose intolerance and bitter taste reception are well established as scientifically valid, but the question about utility is less clear cut. In the case of lactose/lactase it is relatively straightforward – the dietary advice to reduce exposure to lactose will almost certainly lead to the disappearance of the related unpleasant effects caused by lactose fermentation – a definite health benefit.

Regarding taste receptors, it is an attractive hypothesis; individuals who are highly sensitive to bitterness may eat fewer bitter foods, and since these include many vegetables, this genetic variation could have an important impact on diets. It might be useful, for example, to identify bitter tasters at an early age to ensure that an adequate intake of vegetables is not neglected. However bitter taste does not necessarily mean unpleasant taste. Personal taste preference will be influenced by genetic variation in taste receptors, but it is likely to be a complex phenotype under several psychological, behavioral, social and environmental influences. So far the studies of vegetable intake according to taste perception have been inconsistent and the hypothesis that bitter tasters will consume fewer vegetables remains inadequately supported [22–24].

It can be concluded that *LCT* testing does demonstrate health utility, but *TAS2R38* testing does not; however, the latter does provide interesting, potentially

Box 2. Evidence requirements for acceptance of health-related advice.

- Scientific validity: concerns the accuracy of the interpretation (i.e., the accuracy with which a test predicts an outcome). For example, a certain genetic variant may be predicated to influence low-density lipoprotein cholesterol levels according to dietary saturated fat levels
- Clinical utility: the measure of the likelihood that the recommended intervention will lead to a beneficial outcome (e.g., does reducing saturated fats in the diet of individuals with specific genetic variant(s) reduce and/or maintain low-density lipoprotein cholesterol levels?)

useful, information for the individual, which could be described as ‘personal utility’, as long as it is clear that no health claim is made. There is frequently a subjective aspect and sometimes the information itself can be interesting and useful to an individual, even if there is no precise action to be taken regarding the results. This has come to be referred to as ‘personal utility’ rather than clinical or direct health utility; an example is Alzheimer’s disease and the *APOE* genotype, where, even though there is no known prevention or cure, some people would still like to know their own genetic risk [25].

Assessment of the validity & utility of some gene–diet interactions

The following will describe briefly some good and some bad examples of gene–diet interactions, which have been included in commercial genetic tests. Further examples of gene–diet interactions for which there is extensive good quality evidence that dietary modification is likely to have health benefits are listed in Table 1.

MTHFR–folic acid

Probably the most widely studied is that between the *MTHFR* gene C677T polymorphism, folic acid and homocysteine. It has been reliably demonstrated that the 677T version of the enzyme has only approximately 35% of the activity of the 677C version and inadequate folic acid in TT individuals leads to high levels of homocysteine [55], which is a risk factor for

several diseases, including stroke and cardiovascular disease [26,56,57]. For individuals with the *MTHFR* 677CC genotype, the recommended daily requirement for folic acid is sufficient to maintain normal homocysteine levels, while for individuals with the *MTHFR* 677TT genotype, this is not sufficient and they require higher levels. This has been repeatedly demonstrated and it can be concluded with high confidence that this nutrigenetic advice, specific for *MTHFR* 677TT individuals, will ensure normal homocysteine homeostasis. Homocysteine is a risk marker for cardiovascular disease, whether it is a cause or effect is not established, although there is reasonable evidence for it being involved causatively [56,58]. Homocysteine has also been reported to have damaging effects on genome stability [59] and high levels have been linked to a host of diseases from osteoporosis to dementia [57]. Earlier fears about possible links of folic acid intake with some cancers have not been supported by recent large-scale studies [60,61]. With this nutrigenetic information, the 677TT individual (or his/her healthcare advisor) can make an informed choice between following the standard guidelines for B vitamins and as consequence, probable lifetime high levels of homocysteine, or choosing to increase B-vitamin intake, staying well within the tolerable upper limits, and ensuring normal levels of homocysteine.

APOA2–saturated fats

APOA2 is the second most abundant protein on high-density lipoprotein (HDL) cholesterol particles and

Table 1. Examples of gene–diet interactions that can be considered to be scientifically valid and have potential benefit.

Gene	Diet component	Phenotype	Nutrigenetic vs standard guidelines	Ref.
<i>MTHFR</i>	Folic acid	Homocysteine	Reduced homocysteine	[26–28]
<i>MTHFR</i>	Riboflavin	Hypertension	Blood pressure reduction	[29–32]
<i>SOD2</i>	Antioxidants	DNA damage, cancers	Reduced DNA damage and cancer risk	[33–37]
<i>APOA2</i>	Saturated fats	BMI	Increased BMI with high saturated fats	[38–41]
<i>APOE</i>	Dietary lipids, alcohol	LDL cholesterol	Saturated fat and alcohol reduction	[42–46]
<i>CYP1A2</i>	Caffeine	Cardiovascular disease	Reduce caffeine in slow metabolizers to reduce CVD risk	[47–49]
<i>GSTM1, GSTT1</i>	Cruciferous vegetables	DNA damage, cancer	Increased cruciferous reduces DNA damage and cancer risk	[50–53]
<i>GSTM1, GSTT1</i>	Vitamin C intake	Serum ascorbic acid levels	Maintain required levels of ascorbic acid	[54]

CVD: Cardiovascular disease; LDL: Low-density lipoprotein.

has been repeatedly linked to obesity, although the precise mechanism is not well known. A promoter polymorphism *APOA2* 2265T > C has been reproducibly associated with increased BMI in several different populations (European, Asian, Puerto Rican and most recently in the USA). In all studies, the group with the CC genotype and high consumption of saturated fat had, on average, greater BMI. This can be useful information for weight control – an individual with the CC genotype is likely to be more sensitive to saturated fats and in order to either lose weight or maintain weight may benefit more than those with CT or TT genotypes, by reducing intake, replacing them with unsaturated fats [38–41].

APOA1–polyunsaturated fatty acids

The case of a common *APOA1* polymorphism is interesting as it illustrates how information of dubious utility can find its way into a commercial nutrigenetic test. ApoA1 is the major apoprotein constituent of HDL. A common promoter polymorphism (-75G/A; rs670) may affect gene expression and response to dietary lipids. In a paper published in 2002 by Ordovas *et al.*, it was reported in the abstract that when polyunsaturated fatty acid (PUFA) intake was low, the GG subjects had higher HDL cholesterol concentrations compared with carriers of the A allele. Conversely, when PUFA intake was high, HDL cholesterol concentrations in carriers of the A allele was higher than those of G/G subjects [62].

These results appear to have been misinterpreted in some commercial tests that have taken this to mean that higher PUFA caused a reduction in HDL levels in GG subjects; however, data in the paper itself show that the reported effect was wholly or mainly due to an increase in HDL levels in AA subjects when dietary PUFA was high. There was actually no significant reduction in HDL levels in GG subjects. However, *APOA1* has appeared in at least two commercial tests, which advised GG individuals (the majority) to reduce dietary PUFA [63] or that Omega3 will not be effective in raising HDL [64]. It should also be noted that the G allele is the most common and that GG carriers include 50–70% of the population. This is clearly a mistaken recommendation; it is even potentially harmful given that the standard guidelines recommend high PUFA consumption. A possible interpretation of this data for a genetic test could be that AA carriers may benefit by increased HDL levels with a PUFA-rich diet; however, in this study the effect was only seen in females and also has never been repeated and this gene–diet interaction does not even meet the requirements for scientific validity, let alone utility.

Another example of a potentially negative recommendation was due, not to misinterpretation, but

rather to eagerness to sell a ‘compelling’ genetic test before confirmation of the first report. In 2007, a study was published by Caspi *et al.* reporting that breast feeding resulted in an eventual increase in IQ, of around 7 points, but only in children who were not GG for the *FADS2* rs174575 SNP. The study was large and involved two geographically distinct cohorts from Britain and New Zealand; with a total of nearly 3000 children tested, the prospects for replication looked good [65]. Based on this study, a genetic test was put on the market advising that infants with a certain version of the *FADS2* gene will have an increase of approximately 7 IQ points, whereas infants without the variant will not experience any IQ boost from breastfeeding [66].

However, in a subsequent larger study, Steer *et al.* attempted replication in 5394 children [67]. They were not able to reproduce the results; in fact they reported the opposite, that breastfed GG children performed better by 5.8 IQ points – GG children actually showed the greatest difference. In another study, there was a possible positive effect of breast feeding, but it applied to all children and there was definitely no *FADS2* involvement [68]. The company that first offered the test is no longer in business, but unfortunately the *FADS2*/breastfeeding/IQ relationship is still reported by 23andme, citing just the 2007 paper as a ‘preliminary research report’ [69].

Developing evidence guidelines

A nutrigenetic test, like any health-related test, needs to fulfill the criteria for scientific validity and health utility regarding a nutritional recommendation. As yet, there are no formal regulations controlling the sale of nutrigenetic tests and, unfortunately, the quality in the marketplace is highly variable. As with most areas of nutrition, it is yet another where the vulnerable are being exploited by the unscrupulous. For the full benefits of nutrigenetics to be realized, it will be important that reliable, easily accessible and up-to-date information is available in order for health professionals and customers to be able to assess the quality of the various offerings. This is actually far more important than regulations, which would be hard to develop and enforce, and anyway offer no guarantee of quality.

A major EU research project is underway called Food4Me, which is exploring several aspects of personalized nutrition and nutrigenetics, including ethics and evidence requirements [70]. One part of the project is the engagement of an expert group to develop a set of guidelines for the assessment of nutrigenetic evidence and to establish a framework, which can be used to decide whether the evidence for a particular gene–diet interaction has reached the required level at which DNA-based dietary advice would be appropriate. The

first step will be to establish the scientific validity of the gene–diet interaction and the second step will be an assessment of the utility, the potential benefit, of the related dietary advice. Evidence is being assessed according to multiple criteria including:

- Quality and types of study (intervention, observation, case–control, cohort and meta-analysis, among others, and numbers of subjects studied);
- Type of interaction (e.g., simple or complex phenotype);
- Magnitude of effect;
- Biological plausibility;
- Type of intervention (e.g., does it require simple dietary change or would supplements be required?);
- Probability of benefit compared with standard guidelines – the ‘utility’.

It is often hard to quantitatively demonstrate health utility and this is especially so for nutrition where the effects of diet and lifestyle on long-term health begin even before birth [71]. The framework we propose in Food4Me has the objective of providing at least a semi-quantitative scientific judgement of clinical validity with an evidence-based opinion on clinical utility. The ultimate goal will be to provide the resources and the information required for the end user (health professional or consumer) to make an informed decision.

Conclusion

Nutrigenetics has value and can make a definite contribution to individual health and wellbeing provided it is used in the right way. Gene–diet evidence should be assessed at the same level as other nutritional evidence that is used to formulate advice and guidelines and, for continued progress, it will be important to develop reliable information resources giving access to such assessments and to help professional to provide good quality nutrigenetic services.

Nutrigenetics represents right now a valuable tool in the hands of the health professional, especially for dieticians and nutritionists who should incorporate evidence-based gene/diet information when devising nutrition programs for their clients. Health professionals routinely evaluate a range of biological data (biomarkers, height, weight, gender, ethnicity, health issues, and so on) when formulating personalized diets and it is entirely logical that genotype should also be included where the evidence is sufficient. This is the case for several gene–diet interactions and there is evidence that nutrigenetic advice is better understood and more likely to be followed compared with

general dietary advice [72], plus it can be beneficial, for example, in long-term weight control [73].

The evidence for some DNA-based dietary advice is very strong and this will increase significantly as research progresses. Lee *et al.* recently published details of a research studies database containing in excess of 550 examples of gene–diet interactions; it is likely that a significant proportion will reach scientific validity and health utility [5].

Strong evidence is crucial of course, but translating the information from gene–diet studies into personalized advice is not as straight forward as it may seem at first sight and misinterpretations and incorrect advice have appeared in commercial genetic tests. There is currently little or no regulation of personal genetic testing [74,75] and even if current proposals are enacted, just restricting testing to medical practitioners will not guarantee the quality and accuracy of the products. The non-valid *FADS2/IQ* test described above, for example, was sold only through medically qualified doctors. It is also highly questionable whether the medical environment is appropriate for delivering nutritional advice and information that is intended for maintaining health and wellbeing rather than having a specific therapeutic use. Irrespective of the regulatory framework, it will be important to promote serious and beneficial nutrigenetics via provision of information and resources. This is an objective of the Food4Me project, to assess the existing evidence using similar criteria for conventional nutritional recommendations and to make the information available in a transparent way to healthcare practitioners and the general public to enable informed decisions and wise choices to be made. Information dissemination will be the most effective way of promoting the benefits of DNA-based dietary advice and ensuring that good quality services are available to the public.

Future perspective

Since nutrigenetics was introduced as a service over 10 years ago, the progress with its uptake has been slower than many expected. Maybe expectations were overenthusiastic, as were many expectations at the time regarding the ‘genetic revolution’. With hindsight, it is clear that, while technology has made such enormous advances in costs and speed of genotyping, it cannot change the fact that complex research on long-term human health and nutrition cannot go any faster than laws of nature. Despite this, we can expect solid progress in the application of nutrigenetics. This will be helped by the development and convergence of new technologies, including self-monitoring devices – wearable sensors that record useful health-

related information such as physical activity, sleep quality, blood pressure, heart rate and nutrition metabolites, among others [76,77].

It is not expected that the majority of the population will imminently join the so-called ‘quantified-self’ movement [78], but there is likely to be a highly significant impact on research and discovery, yielding results that will be applicable to all. Self-monitoring technology and the establishment of ethical and scientific procedures for voluntary cohort research should enable much more rapid discovery of how specific aspects of diet and lifestyle interact with genetics and genomics to influence the maintenance of health. In one sense, this has been described as a “new form of contract research organization” [79], and

is the central feature of the European Nutrigenomics Organisation Nutrition Research Cohort [80,81]. ‘Crowdsourced’ research is already established in the scientific literature, with recent publications on the discovery of new genetic associations in hypothyroidism and Parkinson’s disease using customers of the commercial genetic profiling service, 23andMe, as the research subjects [82,83].

The EU-funded Food4Me project will be reporting results in 2014 of a seven-country ‘proof-of-principle’ study, looking at the effect of varying levels of personalized dietary advice. Other areas of research in this 4-year project include ethical and social aspects, assessment of scientific evidence, gene–diet database creation, business development, and development

Executive summary

Background

- Common genetic variants affect individual nutritional requirements.
- Commercial nutrigenetic tests have been available for >10 years.
- How confident can we be that nutrigenetic advice is valid and useful?

The context of nutrigenetics

- Nutrigenetics is different from clinical genetics, it operates in the realm of health maintenance rather than diagnosis and treatment.
- It is used to establish personal nutritional guidelines, it is not a therapy nor is it medical practice.
- DNA-based advice builds upon standard nutrition guidelines modifying them where the evidence is strong that it would be beneficial to do so.

Scientific validity & health utility

- Evidence needs to demonstrate both scientific validity and health utility.
- The quality of evidence required should be the same level as the evidence for standard nutritional guidelines. Nutrigenetic evidence should not be held to a higher standard.

Assessment of the validity & utility of some gene–diet interactions

- The quality and accuracy of commercial nutrigenetic tests is highly variable and many are associated with exaggerated, unsupported claims and misinterpretations of research results.
- Some commercial tests are valid, others are not.

Developing evidence guidelines

- There is little regulation of the personal genetics market and it is important to set some standards.
- One aspect of the Food4Me project is to establish a framework for determining whether a specific gene–diet interaction is scientifically valid and has potential health benefit.
- The objective is to establish transparent, public resources to assess whether DNA-based advice is likely to be more beneficial compared with standard nutrition advice.

Conclusion

- Nutrigenetics has value and can contribute to individual health and wellbeing provided it is used correctly, as a part of the overall nutrition guidelines.
- There is evidence of health benefits and that DNA-based advice is better understood and more likely to be followed compared with general dietary advice.
- Reliable information and education resources need to be developed to protect the growth in adoption of serious and good quality nutrigenetic services.

Future perspective

- The ‘genetic revolution’ has been less dramatic than predicted, due to overenthusiastic expectations.
- Personal self-monitoring technology is growing rapidly and will, together with genetics, accelerate research and development in nutrigenetics.
- Recent initiatives such as the European Nutrigenomics Organisation Nutrition Research Cohort and others will harness genetics, self-quantification and crowdsourcing, introducing new types of research organization.
- Further reductions in costs will make genotyping routine and DNA-based dietary advice will become part of standard recommendations – where the evidence is supportive.

and application of advanced metabolic profiling techniques [3,84,85].

It is inevitable that genotyping will become ever more widespread as costs reduce even further and ethical issues are addressed – this will overcome one of the significant barriers to widespread use of DNA-based dietary advice by health professionals: the cost and time required for genotyping. In the future, it will be possible to generate personalized advice in real-time based on existing genetic information. It is also predicted that some genotype-specific information will begin to be included in public health guidelines and in the formulation of the recommended daily intakes of vitamins and minerals.

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K Grimaldi was Science Director of Sciona Inc. (a UK/USA company and provider of genetic testing services) from 2002–2008. He is currently Chief Science Officer of DNAFit Ltd and founding director of the personal genetics services company Eurogenetica, which is a member of NuGO (www.nugo.org) and receives funding as a participant in the Food-4Me project. The author has no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript apart from those disclosed.

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