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Integrating nutrition science and consumer behaviour into future food policy

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Abstract

The session 'Advancing risk assessment science – Nutrition' at EFSA's third Scientific Conference 'Science, Food and Society' aimed to foster the ongoing debate on the extent to which single nutrients, whole foods and overall diets may impact human health in wealthy populations, and to explore how societal and technological developments could affect food choices and diets in the future. The overarching goal of the session was to discuss how dietary guidelines could evolve to account for the switch from single nutrient deficiencies to diseases of malnutrition in all its forms as the predominant public health concern in developed countries. Speakers addressed the contribution of single nutrients to the prevalence of chronic metabolic diseases, discussed the need to move towards diets focusing on whole foods and overall eating patterns, provided insides on food innovation and consumer behaviour and stressed the need for multidisciplinary approaches to face these challenges.

Keywords: chronic metabolic diseases, consumer behaviour, controlled trials, food policy, nutrition, sugars, saturated fats

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Prof Graham Mc Gregor (Queen Mary University of London) contributed as a speaker to the session but declined the invitation to co-author the present paper.

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1. Introduction

The discovery in the first half of last century that insufficient intakes of specific vitamins could be the single cause of various diseases of public health concern underpinned the research and policy agenda in the nutrition area. Research focused on the estimation of nutrient requirements compatible with prevention of clinical nutrient deficiency diseases, on the quantification of nutrients in foods, and on the prevention of hunger. Therefore, policy aimed to ensure adequate provision of specific isolated nutrients as well as total calories through agricultural production of staple crops, food processing, fortification and distribution, dietary advice and labelling. Towards the 1980s, overt vitamin and mineral deficiencies became rare in wealthy countries, whereas the prevalence of chronic metabolic diseases such as obesity, cardiovascular diseases (CVDs) and type 2 diabetes (T2DM) increased steadily. The leading causes of morbidity and mortality of western populations shifted as a result, and so did nutrition research.

Consistent with paradigms for treating nutrient deficiencies, studies on the prevention of metabolic cardiometabolic diseases initially focused mainly on single dietary components (e.g. on reducing intakes of energy, total fat, saturated fats, *trans*-fats, sugars, sodium; and on increasing intakes of dietary fibre, potassium). More recently, however, the role of specific foods and overall dietary patterns on disease prevention has gained attention, also in the context of other lifestyle modifications.

This publication builds upon presentations made and discussions held during the breakout session 'Advancing risk assessment science – Nutrition' at EFSA's third Scientific Conference 'Science, Food and Society' (Parma, Italy, 18–21 September 2018).[1]

The session aimed to foster the ongoing debate on the extent to which single nutrients, whole foods and overall diets may impact human health in wealthy populations, and to explore how societal and technological developments could impact food choices and diets in the future. The overarching goal of the session was to discuss how dietary guidelines could evolve to address the switch from nutrient deficiencies to diseases of excess as the predominant public health concern in developed countries.

The specific objectives of the session were to:

- foster the scientific debate about whether and how the consumption of two classes of nutrients, sugars and saturated fats, is causally related to the high prevalence of chronic metabolic diseases;
- explore the health impact of diets selected by their energy content, the content of a single nutrient, or personal priorities and beliefs versus the health impact of diets selected on the basis of whole foods and overall eating patterns;
- understand the role of food innovation on modern diets in response to consumer trends, and as driver of consumer choices;
- investigate how societal and technological changes could impact food choices of users in post-industrial societies in the coming years.

2. From single nutrients to whole foods and diets in post-industrial societies

In the context of Socratic debates, Graham McGregor and John Sievenpiper were invited to argue in favour and against sugars being one of the nutrients to tackle in the prevention of chronic metabolic diseases. Ronald Mensink and Philippe Legrand discussed the causal role of saturated fats in chronic disease development, primarily coronary heart disease (CHD). The aim was to identify reasons for discrepant views and points of agreement to draw a balanced picture on these matters. Dariush Mozaffarian provided an overview on how nutrition science has evolved in the last century to understand why different dietary approaches aiming to prevent chronic metabolic diseases have been relatively unsuccessful so far and the lessons to be learnt from a scientific and policymaking perspective. Finally, Petra Klassen Wigger discussed how socio-demographic changes and consumer trends in information societies could impact food availability, food choices and dietary patterns, as well as on the challenges and opportunities that this could create in the journey towards healthier diets.

Below follows a summary of the ideas, statements, opinions and challenges raised by the panellists during the session.

[1] All conference materials are available at https://www.efsa.europa.eu/en/events/event/180918

2.1. Impact of single nutrients on the prevalence of chronic metabolic diseases

Sugars and saturated fats have been singled out at different points in time as being responsible for the increasing epidemic of obesity, CVD and diabetes in affluent societies. Their causal role and contribution to the development of multifactorial diseases, however, has been long debated in the scientific literature. Mass media and social media have highlighted that scientific debate shapes different views.

Speakers agreed that unhealthy diets are a major cause of death and preventable disability worldwide. In wealthy countries, there was consensus that excessive consumption of added sugar and salt is to be avoided. The questions are how much is too much, whether the food source matters, and which could be the most effective ways to change consumers' behaviour. More complexity was evident on saturated fat, with differences in views depending on the replacement nutrient and type of saturated fatty acids (SFA).

Arguments in favour of added sugars causing chronic disease focused on sugar-sweetened beverages (SSBs). Sugar has an unquestionable causal role in caries development. With respect to obesity, diabetes and fatty liver disease, the effect appears to be at least in part mediated by overconsumption of SSBs, and so energy, which is not compensated by an increase in physical activity. Some data from short-term controlled trials keeping energy intake constant do not support an independent role of sugars in chronic disease development, but this was deemed to be less relevant because SSBs are rather consumed in free-living conditions and not within energy limits. Other studies support the independent adverse effects of added sugars, at high dose and flux, on hepatic steatosis, insulin resistance, associated metabolic abnormalities and metabolic expenditure. Among the possible ways to address the public health problem of excessive consumption of 'empty' calories from SSBs, particularly by children, taxation, advertisement bans, colour labelling, reduction of portion sizes, step-wise product reformulation and strict dietary guidelines were mentioned.

Opponents to a causal role of sugars in chronic disease development called for a reformulation of the question: are sugars, independent of food form and calories, associated with chronic diseases? In prospective cohort studies, no association has been observed between total sugars, sucrose, or fructose and the incidence of T2DM, whereas a threshold for an adverse association of total sugars, added sugars and fructose with CVD outcomes has been reported. There appears to be an independent positive association between fructose and risk of gout and CVD at high intakes. Positive dose–response associations have been also described for SSBs and body weight gain, risk of hypertension, T2DM, cardiovascular death and gout. In contrast, no association or even inverse associations are found for other sugar-containing foods, such as 100% fruit juice (with no added sugars), fruits, yogurt, chocolate, ice cream or breakfast cereals. Evidence from randomised controlled trials (RCTs), which are in principle more protected from bias than observational studies, shows no adverse effects of fructose on body weight and even a beneficial effect on blood glucose control and blood pressure when compared to refined starches in eucaloric conditions (substitution trials), suggesting that a reformulation strategy that replaces fructose-containing sugars with refined starches is unlikely to be of any benefit. In trials in which sugars are added to the usual diet as a source of excess calories, however, high intakes of fructose induced body weight gain, insulin resistance, a more atherogenic blood lipid profile and increases in serum uric acid, plasma glucose and liver fat. Similar effects have been described for SSBs. Overall, the available evidence suggests that the adverse health effects of fructose and SSBs are mediated at least partly by their contribution to excess calorie consumption.

In this context, a risk was raised of blaming sugars as a whole for the chronic disease epidemic, which may induce consumers to select foods and diets on the basis of the sugar content only, ignoring other nutritional characteristics. An example of how a narrow dietary strategy for disease prevention can fail is given by the low-fat paradigm, by which reducing fat intake by increasing availability of low-fat products in the USA resulted in a parallel increase in the incidence of obesity and related diseases. Therefore, attention should be paid to the nutritional properties of foods as a whole, rather than their sugar content alone, shifting from 'nutricentric' recommendations to food and dietary pattern-based guidelines. Panellists agreed that decreasing consumption of SSBs and other liquid sources of 'empty' calories was a sensible recommendation to make. It was also discussed that decreasing the sugar content of solid foods through food reformulation can be challenging in some cases, also because the nutrient or substance to be used in replacement should be considered carefully: replacing sugars with refined complex carbohydrates may not have a positive impact on chronic disease prevention; fibre would be the preferred alternative, but achieving acceptable palatability could be an issue.

The discussion on saturated fats started with an overview of the specific physiological functions of different SFA depending on their chain length. Besides being a source of energy, SFA participate in the metabolism of n-3 and n-6 polyunsaturated fatty acids (PUFA), in protein acetylation and activation pathways, and are structural components of sphingolipids and phospholipids, among others. Metabolic specificities of some SFAs were described. Whereas endogenously synthesised and perhaps dietary palmitic acid (C16:0) in cells may induce adverse effects, short- and medium-chain SFA ($\leq$ C10:0; e.g. butyric acid) and some long-chain SFA (e.g. myristic and lauric acid) do not. Also, dietary stearic acid (C18:0) desaturates to *cis*-oleic acid (C18:1) in phospholipids and may partly mimic the metabolic effects of its monounsaturated derivative.

It was argued that most RCTs available showed adverse effects of SFA on intermediate markers of disease [e.g. low-density lipoprotein (LDL) cholesterol] rather than on hard CVD outcomes (e.g. myocardial infarction); that most studies have compared SFA with PUFA without specifying the type of SFA being investigated; and that prospective cohort studies or meta-analysis on the association between SFA intake and CHD risk were inconsistent, so that there was no evidence to consider SFA as a single group of nutrients of public health concern. Adverse metabolic effects have been documented for palmitic acid if consumed in excess, but excessive consumption of added sugars, refined complex carbohydrates and alcohol could lead to similar metabolic effects, as these molecules are converted to palmitic acid by the liver and contribute to the LDL cholesterol load, liver fat accumulation and insulin resistance. In this context, replacement of palmitic acid with n-3 PUFA was indicated as the option that could bring the highest health benefits.

Conversely, it was argued that the causal relationship between LDL cholesterol and CHD risk has convincingly been established to rely on this biomarker of effect, as supported by clinical guidelines on disease prevention, even if the number of RCTs on hard CVD outcomes is limited. It was emphasised that the effects of SFA on LDL cholesterol depend on the nutrient that is taken as reference. Stearic acid decreases LDL cholesterol concentrations as compared with palmitic acid to the same extent as carbohydrates, but increases LDL cholesterol concentrations as compared with PUFA. In addition, it was highlighted that fatty acids are found as mixtures in edible oils and fats, and that there are very few intake data on individual SFA. Such data, however, show that palmitic acid is quantitatively the main SFA consumed, about two times more than stearic acid, and that the intake of myristic and lauric acid is lower.

With respect to the apparently conflicting observational data on the relationship between SFA intake and CHD risk, some speakers noted that: (a) estimations of individual habitual intakes of SFA in prospective cohort studies are likely to be imprecise due to the limitations of the assessment methods used; and (b) the influence of dietary factors on LDL cholesterol is relatively small, but not negligible, compared with genetic and other environmental factors. Therefore, these speakers found it not surprising that some cohort studies do not show an association between SFA and CHD risk, and concluded that the effect of a mixture of SFA on LDL cholesterol concentrations from RCTs was the best evidence available to conclude that an upper limit for the consumption of SFA contributes to decrease the burden of chronic metabolic diseases, despite the different metabolic effects of SFA.

So, the same scientific evidence led to different conclusions on the recommendations to be given for dietary SFA. One view was that dietary guidelines should be based on the metabolic effects of nutrients, giving more weight to the physiological functions of individual SFA and less weight to observed associations with clinical disease endpoints or biomarkers of disease. In that case, butyric acid should be grouped with 'probiotics' and dietary fibres, myristic acid with n-3 long-chain PUFA, stearic acid with oleic acid, and palmitic acid with linoleic acid, sugars, total energy and alcohol when providing nutritional recommendations for populations. The second view advocated to recommend replacement of food sources high in SFA by food sources rich in *cis*-unsaturated fatty acids for CHD prevention, rather than providing guidelines for total SFA form all food sources or for individual SFA, which rarely occur alone in foods.

2.2. Towards diets focusing on whole foods and overall eating patterns

An historical perspective of nutrition concerns in high-income countries was given. Nutrition became a matter of national security in the first half of the 1900s. That was the time when all known vitamins were isolated, synthesised and proved to be at the basis of severe deficiency diseases that could be cured with food or synthetic sources. Establishing Recommended Dietary Allowances (RDA) for vitamins, some minerals, protein and energy became a priority during the Great Depression and World War II. In the 1960s and 1970s, efforts were made to ensure an adequate supply of energy and selected micronutrients to the global population, such as through increased agricultural production of starchy staples and also through food fortification and supplementation. World-wide, the debate around fat and sugars as responsible nutrients for CHD high-income countries, and of energy and protein shortage as the cause of infant and child malnutrition in low-income countries, took centre stage. In the 1980s and 1990s, the interest shifted from nutrient deficiency diseases to chronic disease prevention in high-income countries.

This new challenge was approached with the single nutrient, reductionist approach that had been so successful for nutrient deficiency diseases. Therefore, reductions of either energy, fat, SFA, *trans*-fats, sodium or sugar intakes were proposed at different points in time to face diseases that are multifactorial and are influenced by complex characteristics of foods and diet patterns through different metabolic pathways. Single nutrient emphasis may be appropriate for additives such as salt, industrial *trans*-fats and added sugars in beverages, which have been successfully reduced in foods through reformulation and these measures could contribute to CVD prevention. A reduction in the sugar content of beverages has also been effectively pushed forward in some countries through taxation, which could limit their intake of empty calories particularly by children and adolescents and eventually contribute to lower weight gain and improved metabolic health. However, there was agreement among the experts that, beyond additives, the relationship between diet and health is complex, and so the proposed solutions should probably reflect that complexity. For example, instead of tackling one metabolic pathway at a time, focusing on single nutrients with potential adverse health effects (e.g. total SFA reduction to lower LDL cholesterol, total calorie restriction for weight loss), more attention should be paid to whole foods and their complex health effects. Foods represent mixtures of nutrients that can affect metabolic pathways in very different ways. In some cases, the presence of certain nutrients in high quantities (e.g. n-3 PUFA, dietary fibre) may be as important as the low content of other nutrients (e.g. sodium, SFA) to achieve beneficial health effects. For example, promoting consumption of fruits and vegetables, whole grains and fish could be more effective in tackling obesity and disease prevention than restricting fat intake through consumption of low-fat products, which can be high in added sugars, starch and salt.

In the same line, modification of consumer behaviour for chronic disease prevention through labelling single nutrients could be appropriate in some instances mostly for additives (e.g. for salt reduction, to limit intake of SFA or *trans*-fats, to decrease added sugar intake from beverages), but otherwise food choices based on the content of single macronutrients could be unrelated to or even adverse for health. Similarly, the conventional view that energy balance (understood as energy 'in' minus energy 'out') is the main aspect to consider for obesity and chronic disease prevention is increasingly controversial: there is increasing evidence that certain foods and diets can significantly modify the metabolic risk profile without major changes in body weight, and that long-term weight loss is not necessarily achieved by selecting foods on the basis of their energy content only.

2.3. Food innovation and consumer behaviour

Consumers in post-industrial societies have evolved along with urbanisation, changes in the food supply chain, technological developments and the emergence of the digital world.

In times of shortage, the nutritional information provided in food labels was a tool to meet minimum nutrient requirements and be in good health. Today, the interest of consumers in food goes beyond clinical nutrient deficiencies and food safety, extending to chronic diseases, personal values such as social justice and the future of the planet. Sustainability, social welfare and animal welfare drive food choices more than ever before (e.g. fair trade, vegetarian, local, organic). Health concerns have also changed, together with the concept of health benefit, in relation to food. For example, gluten-free or lactose-free products are food choices that go beyond the treatment of coeliac disease or lactose intolerance; gut health, sports performance and alertness are most looked for in food by the young.

Consumers have also shifted their interest from nutrients to popular diets (e.g. vegan, palaeo, ketogenic) and personalised nutrition, and from 'what I think is good for me' to 'what I know is good for me'. The new generations are more interested than ever before in nutrition, but are also highly exposed to misinformation and confusion. As an example, the proactive approach of people to their health has fostered the proliferation of home test kits, motivating individual decisions on food choices and avoidances, the scientific basis of which is disputable.

Traditional (price, taste and convenience) and new (health, safety, societal impact, experience, trust and sustainability) purchase drivers offer both challenges and opportunities to reinforce current food dietary guidelines. Still, it is important to understand the paradoxical impact that food labelling can have on consumer choices. Labelling of low-fat and low-energy foods could lead to over-consumption if these foods are perceived as healthier, while healthier foods can be rejected as found to be less tasty and more expensive. In this context, identifying the behavioural barriers to move from 'knowing' to 'doing' is of paramount importance. For example, fruits and vegetables could be promoted through values (natural, simple, flexible, healthy), but they also need to be cheap and convenient to increase consumption.

The food industry is trying to meet consumer expectations (e.g. for a reduced use of food additives and food processing) and manufacture healthier foods (e.g. with less sugar, salt and SFA; with more fibre) through reformulation. However, achieving these targets while maintaining an acceptable price, taste and convenience of the final product was presented as a major challenge.

2.4. Towards a multidisciplinary solution

The panel discussion included the need to adapt nutrition recommendations, nutrition education, food information to consumers and food policies to the era of digital nutrition and chronic disease prevention.

Healthy diets are made of foods with very different nutrient profiles that, in combination, can affect metabolic pathways in different ways. Promoting the consumption of foods with beneficial effects on health, as well as discouraging consumption of other foods that may be unhealthy if consumed in excess, requires the simultaneous implementation of multiple strategies that are likely to fail in isolation. Healthy diets need to be not only nutritious, but also tasty, sustainable, socially acceptable, affordable and adaptable to individual values and preferences.

Taxation of some foods to discourage consumption should be accompanied by subsidies on other foods to achieve the opposite. This should probably be coupled with sister measures on food availability and achievable reformulation targets for the food industry. Nutrition labelling should be meaningful health wise and easy to interpret to support individual food choices. It was suggested that the ratio of unsaturated to saturated fats, the ratio of sugars and refined starches to fibre, and possibly the ratio of sodium to potassium could be informative for chronic disease prevention. Finally, a better understanding of the drivers for food choices and of the barriers for long-term changes in dietary habits could offer great opportunities to update nutrition messages and build effective communication with consumers of the digital era.

3. Conclusions and recommendations

The discussion held at this session of the conference provided perspectives on the several factors that are impacting and will influence the future of nutrition sciences. As outcome of the session, the following recommendations should be considered:

- Policies for chronic disease prevention should consider specific foods and diets in addition to single nutrients.
- Conduct large controlled trials of complex interventions such as specific foods and whole dietary patterns and composite chronic disease outcomes to better inform such policies.
- Anticipate the impact of socio-demographic and consumer trends on food availability, product reformulation and dietary patterns;
- Gather understanding on the barriers that prevent consumers from complying with science-based dietary guidelines and recommendations and foster research on new approaches for trusted public communication on nutrition;
- Gather evidence on effective systems level change (multifaceted strategies) to drive behavioural changes in dietary habits.
- Promote continuous engagement of policy makers, risk assessors, food industry, NGOs, academia and the society as a whole to tackle the epidemic of obesity, diabetes and heart disease in modern societies.

Abbreviations

CHD coronary heart disease
CVD cardiovascular disease

LDL	low-density lipoprotein
PUFA	polyunsaturated fatty acid
RCT	randomised controlled trial
RDA	Recommended Dietary Allowances
SFA	Saturated fatty acids
SSB	sugar-sweetened beverage
T2D	type 2 diabetes mellitus