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Nutrition in the prevention of chronic disease

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Nutrition in the prevention of chronic disease

Abstract

Increasing prevalence rates of chronic disease requires a more sophisticated view of the effects of food on health. This review examines the evidence base for the effects of food on health and discusses food based health strategies.

Keywords

nutrition, chronic disease, prevention

Disciplines

Arts and Humanities | Life Sciences | Medicine and Health Sciences | Social and Behavioral Sciences

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Nutrition in chronic disease

Nutrition in the Prevention of Chronic Disease

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ABSTRACT

The importance of nutrition in the prevention of chronic disease has been recognised for some time in the global community. In developing countries, the problem is sometimes referred to as double burden of disease, where malnutrition exists in the company of growing rates of lifestyle related diseases such as obesity, diabetes and cardiovascular disease. The frontiers of science have brought forth new understanding of the links between early under-nutrition and the later development of chronic lifestyle related disease, challenging the nutrition scientist and practitioner to evaluate practice to better support health throughout the life course. The observation of the impact of birth size on later nutritional challenges underpins the importance of maternal nutrition, not only in pregnancy but perhaps also in the pre- pregnancy period. Further research continues on the significance of subsequent nutritional practices at critical times in growth and development. Underpinning this understanding is the role of genetic background on nutritional requirements (nutrigenetics) and the effect of nutrients and food bioactives on genetic expression (nutrigenomics). This whole new enterprise has significant implications for the development of the food supply and of dietary advice to support health. An appreciation of the biological significance of whole foods also becomes a necessary parallel activity to that of consuming food in a way that matches and supports human health.

INTRODUCTION

The importance of nutrition in the prevention of chronic disease such as obesity, cardiovascular disease and diabetes has been recognized for some time [1]. In 2001 chronic disease contributed to 60% of reported deaths and 46% of the global disease burden, and this latter figure is expected to increase to 57% by the year 2020 [1]. Obesity is of prime concern, not least because of the rate at which the prevalence is increasing, but also because its reach extends across the globe. Since 1980 rates have tripled in areas like North America, Eastern Europe, the Middle East, Australia and China [2]. Likewise, the prevalence of diabetes is increasing, estimated at 2.8% in 2000 (171 million people) to 4.4% in 2030 (366million people) [3]. Linked to these conditions, cardiovascular disease is set to become the leading cause of death and disability, with 24 million cases predicted for 2030 [4].

In developing countries, nutrition related problems are sometimes referred to as a double burden, where obesity, diabetes, and heart disease rest in the company of malnutrition, usually in different locations. For example, a prospective study of schoolchildren in the Gauteng province of South Africa between 1962 and 1999 saw the percent energy from carbohydrate decrease from 72-60% and the fat consumption increase from 17 to 25.8%. The urban children were more likely to change their dietary habits and be overweight, but micronutrient deficiency and stunting were still prevalent [5]. In addition to the nutrition transition phenomenon, variation is also seen in prevalence rates amongst different sub-groups within a population. For example, in the USA, ethnicity appears to play a major role in the prevalence of metabolic syndrome [6], a term used to link risk factors associated with overweight, diabetes and heart disease [www.diabetes-symposium.org]. With these issues in mind, this review considers the role of nutrition in the prevention chronic disease, and the implications for the development of the food supply and associated dietary advice.

ORIGINS OF CHRONIC DISEASE

By nature, the development of chronic disease rests in the course of events over time. Food directly contributes to body composition and the processes of the human biological system. With the discovery of the human genome, the complexity of this interrelationship has become more exposed, particularly in view of the interactions between food components and genetic expression. The implications are that diet is important at all points in time, that poor diet is likely to have a deleterious effect if prolonged or occurring at critical points of the development phase, and that modifications in diet may play a pivotal role in managing the disease process.

The mechanisms by which diet influences health are complex. They relate to physiological mechanisms in multiple organs and are linked to regulation at the level of genes, gene expression, proteins and metabolites [7]. Fatty acids, for example, are known to influence gene expression in relation to the development of obesity [8], and this has lead to considerations of lifestyle-drug therapies in the management of metabolic syndrome [9]. Overall, the interaction between nutrients and genes is considered to have a central impact on lifespan and disease development [10].

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The relationship, however, is not linear, with incremental environmental exposure a critical factor in genetic expression. Perhaps one of the most significant observations here is that early exposure to food components influences long term health. In a longitudinal study of 4630 males followed for 12 years in Helsinki, Finland, Barker and colleagues noted that low birth weight and subsequent poor growth during infancy was related to increased risk of developing lifestyle related disease reflective of weight gain [11]. A subsequent analysis of 8760 boys born between 1934 and 1944 found that later development of type 2 diabetes was not related to rate of infant growth following low birthweight ($\leq 3.5\text{kg}$), but was related following birthweight of $>3.5\text{kg}$ if there was slow growth in length between birth and 3 months. In both groups, rapid gain in BMI after 2yrs age was associated with increased risk, and this gain was linked to socioeconomic factors. Thus faltering growth may result in impaired insulin metabolism that cannot meet demands of subsequent increases in BMI [12]. Differences between boys and girls in the pathways for development of coronary heart disease (CHD) were found in a study of 4130 girls from the same cohort. Girls who developed CHD, rather than thin, were short at birth, compensated for this during infancy then became thin and later developed a rapid increase in BMI. Girls were seen as less vulnerable to under nutrition in utero and better able to compensate in an adverse post natal environment [13]. In contrast for boys, poor growth during infancy and small body size at 12 months was a stronger predictor of CHD than low birth weight [14].

The links between early growth and development of later disease have been generally confirmed through studies of cohorts from other parts of the globe. In a study of 356 Guatemalan children, positive associations were found between length at age 2 and fat mass, height and weight, with birth weight directly related to waist hip ratio in females [15]. Research on persons conceived during the 1944-45 Dutch famine found an earlier onset of coronary artery disease compared to unexposed persons [hazard ratio adjusted for sex 1.9; 95%CI 1.0-3.8] [16], but a subsequent study could not confirm that this led to increased adult mortality, although the cohort was only studied to 57yrs [17]. Thus, while early nutrition may play a role in programming disease, there appears to be opportunity to manage the risk later in life. In the Herfordshire Cohort Study [37,615 men and women born 1911-1939] lower birth weight was associated with increased risk of mortality from cardiovascular disease in men and women [18]. Interestingly a subsequent study of a 59-71yr old subset found that high intakes of total and saturated fat were associated with adverse HDL:total cholesterol ratios in men with low birth weights ($\leq 3.2\text{kg}$) (interaction effects $P=0.02$ for total fat and 0.01 for saturated fat, increasing to 0.008 and 0.006 respectively with those taking cholesterol lowering medications excluded) [19]. This finding could be interpreted as evidence of gene nutrient interactions, whereby the effects of nutrients (in this case fat) are mediated by the genetic profile of the organism.

The initial observations of Barker that low birthweight infants were at greater risk of chronic lifestyle related diseases, has helped to develop to the concept of 'developmental plasticity' in humans. It explains how different environmental conditions during development can result in different physiological or morphological states from one

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genotype [20]. More specifically, it explains how insulin resistance [21] and hypertension [22] may emerge in persons with low birthweight and thinness at 2yrs followed by an increase in BMI after 2 yrs. The implications for growth protection during the first 2 yrs of life and the prevention of rapid increases in BMI after that age then represent major challenges for nutritional strategies for the health of populations.

If we take the position that the chronic diseases such as obesity, cardiovascular disease and diabetes have their origins in early development, then it follows that nutrition intervention is important throughout the lifespan, not just at the time where risk factors reach a certain 'treatable' point. In any case, with increasing obesity rates, these cut off points are working their way down from the middle aged to the young. The term 'adult onset diabetes' is now being challenged as obese children emerge with type 2 diabetes mellitus, and the sequence of events leading to this situation are beginning to unfold. Nutrition practice will need to draw on scientific advances such as that described above where knowledge of a person's nutritional history will form a part of clinical decision making and personalized nutrition advice will become an imperative.

DEMOGRAPHIC TRENDS

The changes in demographic trends across the globe fuel the need for prevention of morbidities associated with chronic lifestyle related disease. Life expectancy will increase in all sectors and the percentage of the population aged over 65 yrs is expected to grow substantially by 2025 [23, 24]. While the so called 'baby boomer' generation is likely to have a significant effect on health care services in Western societies, the number of people in the age range 20-45yrs - those likely to show early signs of lifestyle related disease - is significant across the globe. In addition, while the percentage of the population below the age of 14yrs may be reducing, this will be reflected in the number of children per household who will then gain attention for health and nutrition [23, 24]. The risk is that these children may become overfed and overweight [25], but there is also likely to be a concomitant desire to ensure these children receive optimum nutrition, not just for growth but also for cognition and performance.

These trends are likely to produce demands for better quality food. With increasing knowledge of the impact of nutrition on health and longevity, developments in nutrition research stand to influence the development of the food supply. This represents a challenge for science and food marketing alike, as food standards legislation references the quality of the evidence that substantiates these benefits.

EVIDENCE BASE

Establishing evidence for practice has become a significant activity in nutrition, whether this practice relates to dietary guidelines for the general population, clinical practice guidelines for disease management or food standards legislation governing health claims on foods [26-30]. In common is the need for research in humans (preferably experimental but including good quality observational studies), plausible dose responses, explanatory mechanisms and consistency in the evidence base. Levels of evidence tend to be reported as convincing, probable and possible.

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In the review of the WHO Technical Report series 916, Diet, Nutrition and the Prevention of Chronic Diseases [1], convincing evidence was found for increasing risk of obesity with high intake of energy dense foods, of type 2 diabetes mellitus with the presence of abdominal obesity, overweight and obesity, physical inactivity and maternal diabetes, and of cardiovascular disease with dietary saturated (myristic and palmitic) and trans fatty acid, dietary sodium, high alcohol intakes and the presence of overweight and obesity (Table 1). A decreased risk of developing obesity was found with high intakes of non-starch polysaccharides, and of fruits and vegetables and with the practice of regular physical activity. Likewise, decreasing risk of type 2 diabetes mellitus was found with voluntary weight loss and regular physical activity, whereas decreasing risk was found for cardiovascular disease, with intakes of linoleic acid and fish oils (and of fish itself), and of potassium, fruits and vegetables, with low to moderate alcohol intakes and the practice of regular physical activity.

[INSERT TABLE 1]

Put together this evidence base can be seen as a mixture of behaviors relating to physical activity and the consumption of some whole foods and of specific individual nutrients, and the presence of pre-existing conditions such as overweight/obesity, which is modifiable, and maternal diabetes, which is not modifiable (from the offspring's perspective, although presumably it can be prevented in the mother prior to the conception of the child). The evidence itself reflects the types of studies that are available for review, and these were not necessarily conducted for the purposes of establishing the evidence base. Nevertheless, from a food/nutrient perspective, there appears to be enough evidence linking patterns of consumption of fatty acids, sodium and potassium, and fruits, vegetables and fish with the development (or not) of chronic lifestyle related disease. These dietary components could be seen as the first to emerge consistently in a complex picture of diet and health.

Food standards and the drive for health claims related to food may forge an acceleration of this process. Pre-approved health claims in Western societies have also linked reduced risk of cardiovascular disease with foods such as nuts and those containing wholegrains, fibre or soy [29]. There remain challenges, however, in communicating the degree of confidence the evidence base carries with respect to outcomes an individual might expect from including these foods in the diet. On reviewing the PASSCLAIM project outcomes [26] it would seem that extent of scientific support in terms of developed methodologies at least, was promising for foods that targeted appetite control and risk factors for lifestyle related disease, but there was a need for better quality interventions (particularly with respect to background diet and dietary intervention modeling) and for a consideration of genetic factors.

DIETARY FAT, FOOD AND LIFESTYLE RELATED DISEASE

Perhaps one of the most extensively studied areas of nutrition in relation to the development of diet-related disease has been that of dietary fat. Extensive mechanistic studies have exposed the relatively positive effects of unsaturated versus saturated fatty acids on insulin action [31]. The related benefits of manipulating dietary fatty acids have

been supported with observational and experimental studies in humans [32-35]. To be easily followed though, nutritional advice needs to be given in terms of foods not nutrients. Our team developed research methodologies that targeted whole food sources of unsaturated fatty acids (in this case, walnuts) to produce desired dietary fatty acid profiles [36], which in turn resulted in improved lipid profiles in subjects with type 2 diabetes mellitus [37]. Total anti-oxidant status improved significantly during the intervention and this was attributed to the anti-oxidants delivered concomitantly with the walnuts and the recommended 5 serves/day of vegetables and fruits. In another study, the inclusion of walnuts and olive oil in test meals respectively was found to preserve the protective phenotype of endothelial cells through examinations of flow mediated dilatation, a quasi measure of inflammatory activity following feeding with a high fat meal [38]. The two studies of whole foods are significant for two reasons. First, inflammation may well become the new target for nutrition strategies in the prevention of chronic disease [39], and positive related effects were observed with the dietary strategies. Secondly, while the delivery of unsaturated fats was a primary focal point of each study, there was recognition of the nutrient or bioactive package that the whole food delivered. Jacobs and Steffen argue that this concept of food synergy is an important consideration in understanding dietary exposure and the development of chronic disease [40]. To convert this knowledge to practice, methodologies are required to differentiate between foods in terms of attributes such as nutrient density [41]. Thus just as the evidence base for risk linking diet with disease has identified certain elements that need to be considered together, the experimental assessment of these elements drives us back to whole foods, and then to whole diets to adequately explain effects and cover all the known bases in disease prevention.

FOOD BASED HEALTH STRATEGIES

Translating the scientific evidence to policies and strategies for health runs in parallel to advances in nutritional science. There are a number of policies in place that clearly outline the means by which governments and health agencies need to take action. The WHO Global Strategy on diet, physical activity and health outlines the need for leadership, effective communication, functioning alliances and partnerships and enabling environments. Strategies relate to surveillance, informed choices, standards and legislation, availability of healthy diets, intersectoral activity and health service provision [42]. In Australia, the national action agenda for children and young people and their families, Heathy Weight 2008 [43], outlines a matrix model of four national strategies (co-ordination / capacity building, evidence / performance monitoring, whole of community demonstration areas, and support for families / community-wide education) across nine settings strategies (childcare, schools, primary care services, family / community care services, maternal and infant health, neighborhood / community organizations, workplaces, food supply, and media / marketing) (Fig 1)

INSERT FIG 1 HERE

The components of these policies are reflected in the new paradigm for nutrition was proclaimed at the 2005 meeting of the International Union of Nutrition Scientists. In view of the context in which nutrition is likely to have an impact, this new paradigm

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called for partnerships to solve nutrition problems. These partnership would see the traditional biological bases for nutrition better integrated with knowledge from the environmental and social sciences [44]. This broadens the potential for problem solving of nutrition related problems, but it will be necessary for the nutrition scientist to retain a discipline identity and for advances in the understanding of biological processes to be applied.

An important consideration is the continuing acknowledgement that food lies at the heart of nutrition. The European PASSCLAIM project [26] provides a good example of productive inter sectoral engagement targeting an improved food supply. This project integrated knowledge on marketing claims for foods with scientific methodologies and evidence frameworks. The resultant framework provides those working in the field of food and nutrition with direction on the types of measurable effects that can be addressed and thereby identifies targets for the development of healthier food products. The ability to lower cholesterol levels, reduce body weight, improve insulin sensitivity, reduce risk of type 2 diabetes are all tangible targets for food product development in the context of a healthy diet, based on scientific methodologies. ILSI Europe has also produced a review of interventions and programmes targeting the prevention of overweight and obesity in children and adolescents [45], with recommendations relating to outcome measures, participation rates, heterogeneity in intervention groups, tailoring, changes to the physical and/or social environment, sustainability, impact, adverse effects and publications of results. These are all positive steps to improve the scientific quality of interventions to greater public good.

At a policy level, the emergence of evidence frameworks for clinical practice guidelines, and for guidelines for disease prevention and health promotion is another significant development [27,30]. These developments also demonstrate the need for building our understanding of the way in which food and nutrition translates to health and the means by which we may expose it through scientific practices.

CONCLUSION

With the increasing prevalence rates of chronic disease related to lifestyle comes a more sophisticated view of food and nutrition. This sees food as a biological system in itself comprising sets of bioactive components that interact with human biology in ways that can only at times be imagined. There is much to be done, however, with the knowledge that is currently at hand. The challenge lies in integrating different forms of knowledge and rigorously testing the evidence that assumptions made actually hold true following changes in diet and physical activity. On a broader scale, policies that govern food and nutrition need to be accommodating of the various sectors' interests and contributions to problem solving, for example the development of dietary advice and the concomitant development of the food supply. There needs to be synergy in the efforts of academia, government, industry and healthcare providers [46]. The advent of nutrigenomics and nutrigenetics heralds a new era of personalized nutrition that considers the impact of the nutritional environment on programming nutritional requirements and disease risk later in life. One of the primary platforms for nutrition today and tomorrow will be the

prevention of chronic disease through an understanding of the interactions between genes and the nutritional environment.

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Table 1: Levels of evidence of lifestyle factors for and against conditions of the metabolic syndrome. Adapted from [1]

Evidence	Decreases risk	Increases risk
Convincing	<ul style="list-style-type: none"> * Regular physical activity^{a,b,c} * High intake of non-starch polysaccharides (dietary fibre)^a * Voluntary weight loss^b * Fish and fish oils * Vegetables and fruits * Potassium * Low to moderate alcohol 	<ul style="list-style-type: none"> * Sedentary lifestyle^{a,b} * High intake of micronutrient-poor, energy-dense foods^a * Abdominal obesity^b * Overweight/obesity^{b,c} * Maternal diabetes^b * Palmitic and myristic acids^c * Trans fat intake^c * High intake of sodium^c * Excess alcohol^c
Probable	<ul style="list-style-type: none"> * Environments that support health food choices for children^a * Breastfeeding^{a,b} * Non-starch polysaccharides^{b,c} * alpha linolenic acid^c * oleic acid^c * wholegrain cereals^c * unsalted nuts^c * plant stanols and sterols^c * folate^c 	<ul style="list-style-type: none"> * Heavy marketing of fast-food outlets and energy-dense foods^a * Fruit juices and sugar-sweetened drinks^a * Difficult social and economic conditions^a * Saturated fats^b * Intrauterine growth retardation^b * Dairy cholesterol^c * Unfiltered coffee (boiled)^c
Possible	<ul style="list-style-type: none"> * Low Glycaemic index foods^{a,b} * Omega-3 fats^b * Flavonoids^c * Soy products^c 	<ul style="list-style-type: none"> * Large portion sizing^a * A large proportion of foods prepared outside the home^a * Eating patterns such as rigid restraint or periodic disinhibition^a * Fat intake (total)^b * Trans fat intake^b * Lauric acid-rich fats^c * Impaired foetal nutrition^c * Beta-carotene supplementation^c
Insufficient	<ul style="list-style-type: none"> * Increased frequency of eating^a * Vitamin E^b * Chromium^b * Magnesium^{b,c} * Moderate alcohol^b * Calcium^c * Vitamin C^c 	<ul style="list-style-type: none"> * Excess alcohol^{a,b} * Carbohydrates^c * Iron^c

Related to ^a Overweight/Obesity, ^b Type 2 Diabetes Mellitus, ^c Cardiovascular disease

Figure 1: National and setting-based action strategies for children and young people
Adapted from [43].

