Nutrition and Health: Focus on fats from the personalised perspective

Expert Statement: Royal Irish Academy Life and Medical Sciences Committee

Professor Helen Roche

October 2015
There is no doubt that good food and nutrition promote health and quality of life. It is also becoming increasingly apparent that a more personalised approach to people’s nutritional requirements to maintain health is necessary—one size does not fit all. Dietary fats are an essential nutrient, yet in certain circumstances they can also have adverse effects. Depending on their nature or type, fatty acids can have very different effects on individuals, and even on various systems within a single individual, highlighting the need for a more personalised approach to nutrition and health.

**Dietary fats—good and bad**

Fat is an excellent source of energy, but, in terms of health, not all fats are the same. Plant and marine sources provide essential polyunsaturated fatty acids (PUFAs). PUFAs are essential because they are needed for normal body functioning, but they are also essential in the diet because we cannot synthesise these key fatty acids ourselves. There are two main categories of PUFAs: n-3 and n-6 types. Dietary sources of n-3 PUFAs include linseed oil. The long chain n-3 PUFAs (LC n-3 PUFAs), which are particularly healthful, include eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA); these are exclusively found in fish, particularly the oily variety, including salmon, trout and fresh tuna. Fish are a good source of LC n-3 PUFAs because fish consume plankton, which is the only organism that can synthesise LC n-3 PUFAs, and so when we eat fish these essential fats moves up the food chain to people.

In terms of promoting health, we tend to fall below the recommended intake of LC n-3 PUFAs. These particular polyunsaturates are a unique class of fatty acids which have benefits of improving lipid metabolism, modulating the inflammatory response and aiding anti-thrombotic (clotting) potential. In terms of anticipating the nutritional needs of an aging population, it is important to note that low LC n-3 PUFA-status tends to be associated with cognitive decline and Alzheimer’s disease. DHA is the most abundant fatty acid in the brain; levels do decline, and decreased levels are associated with age-related cognitive decline.

Other healthy fats include the monounsaturated fatty acids (MUFA). These are not essential in the diet, as we can synthesise them ourselves from saturated fat sources, but dietary sources include dairy products, meat and olive oil. Saturated fats tend to have a bad reputation, but saturated fatty acids (SFAs) only have adverse health effects when they are consumed in excess. In moderation SFAs, like other dietary fats, are key nutrients, providing energy and acting as a vehicle to promote absorption of the essential fat soluble vitamins A, D and E.

Health issues arise when we have an excess of fat, and in particular of SFAs, in association with surplus calories from carbohydrates and protein. These excess calories will in turn be converted into SFAs for storage in adipose tissue (fat tissue).
Long-term over-abundance of fats leads to overweight and obesity, which in turn can lead to cardiovascular disease and Type 2 diabetes. The particularly adverse effects of SFAs relate to cholesterol metabolism: most SFAs increase cholesterol concentrations, and this is a key risk factor for cardiovascular disease. Reducing saturated fat intake or replacing saturates with MUFAs or PUFAs is an effective strategy for reducing cholesterol concentrations to promote heart health. There is also good evidence that high-fat SFA-enriched diets promote insulin resistance, which is a key factor that subsequently leads to the development of Type 2 diabetes. Again, reduction or replacement of SFAs can attenuate Type 2 diabetes risk. The bottom line with dietary fats is that the quality as well as the quantity consumed is highly important—as is the proportion of the key unsaturated fatty acids (LC n-3 PUFA, n-6 PUFA and MUFA) in an individual’s diet in comparison to the level of SFAs.

But one diet does not fit all

Personalised Nutrition is a relatively new concept; it seeks to promote health through optimal food and nutrient intake, based on individual needs and considering family history / genetic background and environmental / lifestyle factors. Diet-related diseases such as obesity, diabetes and cardiovascular disease are dependent on both genetic and environmental factors, of which diet is one important component. Diet can be viewed either as a risk factor or a potential preventative strategy / therapy against disease. For example, in those who are more susceptible to diabetes or heart disease due to their family history or genetic background, a high-fat, SFA-rich diet can greatly increase risk. Conversely, an individual can choose to reduce the risk of developing such diseases and promote health through modifying dietary fat intake.

Interestingly, some individuals respond more effectively to dietary interventions than others. In any dietary intervention a significant proportion of people will not respond to a nutritional intervention, despite being compliant. They represent a group of ‘non-responders’ who, based on their genetic make-up, cannot improve their health profile through a given dietary intervention. Therefore we need to treat only those who respond and seek a more effective alternative therapeutic strategy for those who are non-responsive to dietary intervention. The ultimate challenge is to understand why some people have great sensitivity to or resistance against a given dietary / nutritional environment. A ‘gene-nutrient interaction’ is the process whereby individuals show differential risk or differential response to a given dietary environment or nutritional intervention, based on their genetic background. To date, most of the work on such interactions has been based on relatively few genes that interact with the dietary environment. While this research demonstrates proof of concept, an individual’s genetic background reflects several thousand genes, some of which will confer risk while others may interact to give a protective effect.

The challenge for the future is to enhance our understanding of the personalised component of an individual’s health status or risk of diet-related diseases, in order to build a more comprehensive evidence base that underpins more effective nutrition to promote health. Foods and their fat content uniquely affect you. Think, learn and eat to be healthy.

Helen Roche is Associate Professor of Nutrigenomics at UCD Conway Institute, affiliated with the UCD Institute of Food and Health and the UCD School of Public Health.
October 2015