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Dietary fibre as an essential nutrient

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Despite strong evidence of dietary fibre's health benefits and its role in reducing chronic disease risk, it is not considered to be an essential nutrient. Recognizing its essentiality and confirming reference values is a critical step to drive clinical and public health recommendations, policies and interventions.

Establishing a nutrient as essential has important clinical and public health implications regarding how it is prioritized and communicated. Based on extensive evidence from epidemiology, gastroenterology, endocrinology and microbiology, we propose dietary fibre as an essential nutrient. We also offer an approach for providing further evidence to support this proposal and discuss the benefits and consequences of acknowledging fibre's essentiality.

There is no universally accepted definition of an essential nutrient. However, common features are that they are required for a critical function, cannot be synthesized in vivo or are produced in insufficient quantities to meet requirements, and deficiency is associated with characteristic clinical features that are reversed when requirements are met. While not specified, these attributes of essentiality clearly indicate that lack of an essential nutrient is causally implicated in adverse health outcomes. Nutrients considered to be essential for humans include a range of amino acids, vitamins, minerals and fatty acids. Dietary fibre and other carbohydrates are not currently considered essential.

With the transition from agrarian to industrialized lifestyles, intakes of dietary fibre have fallen from ancestral amounts of around 100 g or more per day to historically low levels of 12–22 g per day[1]. In parallel with this has been an increase in several non-communicable diseases, notably coronary heart disease, type 2 diabetes and colorectal cancer, which are major contributors to the global burden of disease. While such associations may be a consequence of confounding factors, the benefits of high dietary fibre intakes are supported by more than 100 years of research into its chemistry, physical properties, physiology and metabolic effects, and epidemiological and clinical observations. A hypothesis, initially based on clinical observations and first proposed by Trowell and Burkitt in the early 1970s, suggested that inadequate intakes of dietary fibre could explain high rates of a wide range of diseases typically seen in people consuming 'western diets'[2,3]. More recently, meta-analyses of cohort studies have shown reductions in the risk of cardiovascular diseases, type 2 diabetes, colorectal cancer and premature mortality with higher dietary fibre intakes[4]. Striking dose–response relationships between increasing fibre intakes and risk reduction support a causal association[4]. These findings, based on generally healthy populations, are reinforced by separate meta-analyses of prospective observational studies in those with diabetes[5], hypertension[6] or heart disease[6].

Further evidence of causality, function and essentiality is provided by meta-analyses of randomized controlled trials that have demonstrated improvements in established cardiometabolic risk factors with higher dietary fibre intakes[4–6]. The cardio-metabolic benefits have been largely attributed to the effects on blood lipids and glycaemia. Increasing dietary fibre intake results in reductions in total and low-density lipoprotein (LDL) cholesterol[4] through pathways such as promoting bile acid excretion and increasing LDL clearance. Dietary fibre lowers postprandial glycaemia, measures of glycaemic variability and, when consumed regularly, can improve long-term glycaemic control as measured by glycated haemoglobin[4–6]. Further established pathways moderated by higher fibre intakes include incretin secretion[7], satiety and appetite[7], body weight regulation[4–7], and increased stool weight and transit time[7] leading to reduced colorectal cancer incidence[4]. Such physiological mechanisms provide the rationale for the observed dose–response associations between increasing dietary fibre intakes and reductions in the risk of cardiovascular disease and type 2 diabetes[4]. It is largely owing to these confirmed associations between fibre and both cardiometabolic risk factors and non-communicable disease outcomes that the World Health Organization released its most decisive global dietary guidelines in 2023, promoting foods rich in dietary fibre to achieve an individual intake of at least 25 g dietary fibre per day[8]. These global recommendations take into account that dietary fibre is not a single chemical entity and that no discernible differences in health outcomes were observed when considering fibre type or source.

An emerging pathway between dietary fibre and human health is via the gut microbiome, which we define here as the total mass of bacteria in the colon. The diversity and functionality of the gut microbiome is dependent on dietary fibre as its key substrate for growth and maintenance[9], with gut microbiome composition responding rapidly to changes in fibre intake[10]. Dietary fibre is degraded by the gut microbiome through fermentation, the only anaerobic process within human digestion and essential to the survival of many animal species. In addition to its effects on the gut microbiome diversity and density, the main products of dietary fibre fermentation are the short-chain fatty acids – acetate, propionate and butyrate – as well as gases such as hydrogen, carbon dioxide and methane[11]. Acetate is absorbed and largely passes through the liver to the peripheral circulation, where it is a readily available substrate for intracellular energy metabolism via a process that does not influence blood sugar levels nor elicit an insulin response. Dietary fibre, a carbohydrate, therefore, provides energy to the host by a non-glycaemic non-insulinaemic pathway. Butyrate provides the principal energy source for the colonic epithelium and is also thought to act intracellularly by inhibiting histone deacetylase. Both propionate and butyrate may be involved in the systemic responses to dietary fibre that are known to lead to weight loss, improved glycaemic control and lipid metabolism[4–6]. Short-chain fatty acids have been shown to interact with the gut immune system where they act as signalling molecules that counteract inflammation and, in the case of butyrate, reduce the risk of cancer[11]. When dietary fibre intakes are limited, or animal protein intakes are high, the gut microbiome obtains maintenance energy from the breakdown of protein and fermentation of the resultant amino acids. The end products of this limited fibre

intake include branched-chain fatty acids and potentially toxic substances such as ammonia and sulphide[12].

What constitutes a healthy gut microbiome and its interaction with the host is still under debate. To date, the health of the gut microbiome has been linked to an expanding list of benefits, including its role in the pathogenesis and progression of Parkinson's disease through the gut–brain axis, the development of demyelinating disease, the moderation of colonic mucosal inflammation and immunity, and the bioavailability and metabolism of orally administered drugs in limiting atherosclerosis and promotion of weight loss[13]. These known and emerging interactions between the gut microbiome and host provide additional biological plausibility for many of the clinical benefits associated with dietary fibre[4–6].

Despite the impressive body of known and emerging evidence, dietary fibre has not yet been designated an essential nutrient principally because a reversible deficiency state has not been established. Given the current evidence available on dietary fibre as an essential substrate for a healthy gut microbiome and the consequent benefits for the host, we propose that a dysfunctional gut microbiome is a deficiency state due to inadequate dietary fibre intake. Confirming this hypothesis will require further research regarding the gut microbiome as an ecosystem, its symbiosis with the human host, and interactions between dietary fibre and functional differences in the gut. While it is highly unethical and implausible to manufacture a complete and sustained fibre deficiency state, early animal studies indicate that adding fibre to fibre-free parental nutrition can reduce physiological gut damage, harmful bacteria growth and bacterial translocation[14]. Data from more recent human studies indicate that features of gut dysbiosis (which we define here as taxa imbalance leading to disturbance of the gut microbiome–host relationship) are reversed by increasing intakes of dietary fibre[15], offering direction for future research to confirm the essentiality of dietary fibre as we propose here. A range of established and emerging 'omics' technologies confer an ability to better observe interactions between the host and the gut microbiome following consumption of dietary fibre, and therefore identify the features of dietary fibre deficiency. From this basis and the existing experimental and observational data, appropriate dietary fibre reference values for restoring optimal function may be tested and developed.

Notwithstanding the overwhelming body of scientific evidence indicating causal associations and global recommendations to increase intakes, fibre intakes remain low (12–22 g per day)[1]. Acknowledgement of dietary fibre as an essential nutrient will probably prioritize it as a key carbohydrate metric and ensure greater emphasis on increasing intakes in dietary guidelines, increase awareness amongst health professionals of its importance, enhance nutrition education programmes and ensure ongoing monitoring. Reformulation of food products, changes in food labelling and fiscal incentives have the potential to further encourage greater intakes of fibre-containing foods such as whole grains, legumes, vegetables and whole fruit.

Appreciable increases in dietary fibre intakes could be a simple and effective approach to reducing major contributors to the global burden of disease, once the causal role of dietary fibre in human health is fully recognized. Accepting its essentiality is one upstream approach to doing so, with further research into the gut microbiome and dietary fibre deficiency needed to confirm this hypothesis and enable a much-needed prioritization of dietary fibre intakes for improved human health.

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Competing interests

The authors declare no competing interests.

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