



SPECIAL ARTICLE

Functional foods and cardiometabolic diseases[☆]

International Task Force for Prevention of Cardiometabolic Diseases



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Abstract Mounting evidence supports the hypothesis that functional foods containing physiologically-active components may be healthful. Longitudinal cohort studies have shown that some food classes and dietary patterns are beneficial in primary prevention, and this has led to the identification of putative functional foods. This field, however, is at its very beginning, and additional research is necessary to substantiate the potential health benefit of foods for which the diet–health relationships are not yet scientifically validated. It appears essential, however, that before health claims are made for particular foods, in vivo randomized, double-blind, placebo-controlled trials of clinical end-points are necessary to establish clinical efficacy. Since there is need for research work aimed at devising personalized diet based on genetic make-up, it seems more than reasonable the latter be modeled, at present, on the Mediterranean diet, given the large body of evidence of its healthful effects.

The Mediterranean diet is a nutritional model whose origins go back to the traditional diet adopted in European countries bordering the Mediterranean sea, namely central and southern Italy, Greece and Spain; these populations have a lower incidence of cardiovascular diseases than the North American ones, whose diet is characterized by high intake of animal fat. The meeting

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in Naples and this document both aim to focus on the changes in time in these two different models of dietary habits and their fall out on public health.
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An overview of nutritional genomics and of the gene–environment interaction¹

The dawning of the brain versus brawn struggle

To understand the emergence of nutritional genomics, one must inevitably start with the history of human nutrition. This is traditionally traced back to about 2.5 million years ago, when soon after the appearance of Lucy, the nutritional phenotypes of the human species are thought to have appeared, and much later, in the Neolithic, when nutritional phenotypes of human populations began to emerge. A major event that was not only to condition human diets but also to shape human destiny, and that eventually transformed hunter–gatherers into digital natives, was the emergence of a frame-shift mutation that “switched-off” the myosin heavy chain (MYH16) gene that was expressed in the masticatory muscles of monkeys. Using the coding sequence of the myosin rod domains as a molecular clock, Stedman et al. [1] dated, the loss of this protein isoform, which caused a reduction in the size of these muscles, to about 2.4 million years ago. This may be considered the first example of nutritional genomics. Indeed, it is a striking case of a mutation inducing a drastic change in nutrition and thus in lifestyle.

As shown in Fig. 1A, the loss of the MYH16 protein isoform and the consequent appearance of less powerful masticatory muscles together with other random mutations in early hominins that led to the upright position, prompted these populations to search for more tender food, which in turn resulted in migration to the savannah/woodland interface to gain access to the soft bone marrow of the carcasses of carnivore preys [1]. The new food provoked epi-mutations, which, probably with germinal mutations, promoted a hierarchy of organs (Fig. 1B). In fact, encephalization, namely, brain enlargement, resulted in improved structural, cognitive and problem-solving ability that are features typical of humans. Synergistically, evolution, based also on dietary changes, produced a reduction in gut length thereby releasing energy for brain development. Thus, a virtuous circle was set in motion between brain development and nutrition, thereby generating not only the *brain versus brawn struggle* but also the *brain versus gut struggle* (Fig. 1C) [2]. This scenario vividly evokes the challenging *nature versus nurture* dilemma raised by Sir Francis Galton in 1857 of which more below.

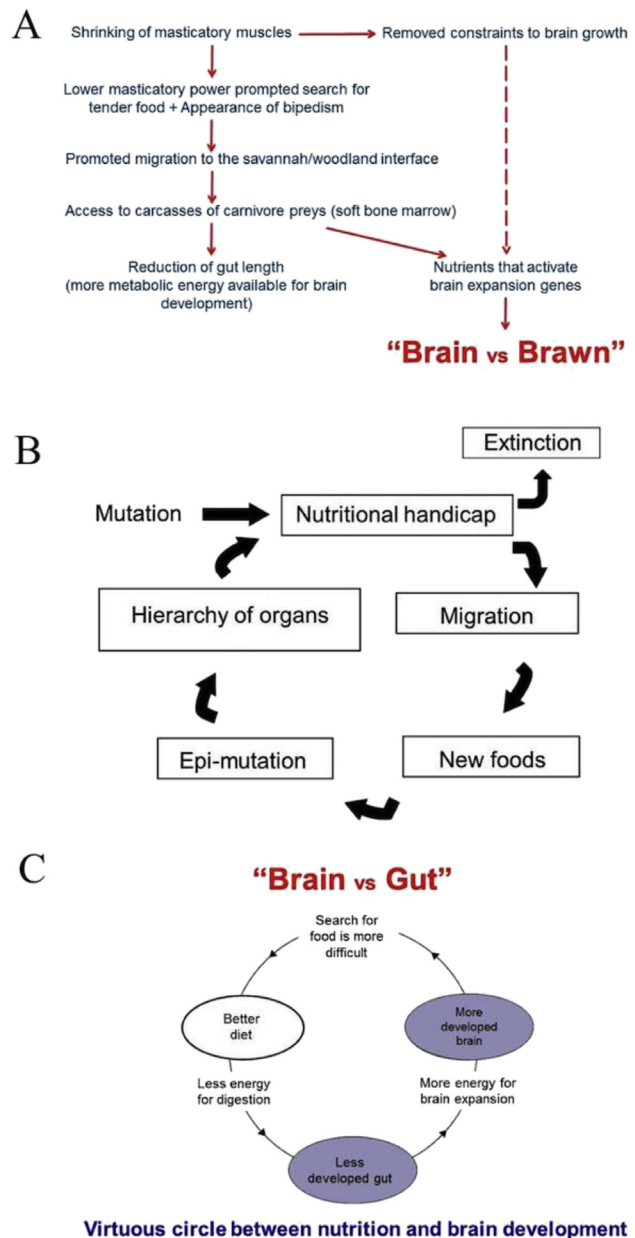


Figure 1 (A) Consequences of myosine mutations in the masticatory muscles of early hominins that ultimately provoked, on one hand, the need to look for more tender food, and on the other hand, activation of brain expansion genes (*brain versus brawn*). (B) Interaction between nutritional handicap and migration that would provoke the hierarchy of organs in the human body during evolution. (C) The virtuous circle that allowed greater brain development because of better nutrition and a less developed gut. Source: Rotilio G. Roma: Carocci, 2012.

¹ The authors of this section are Salvatore F., Daniele A., Buono P.

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