Ángeles Carbajal Azcona - carbajal@ucm.es
Dpto de Nutrición y Ciencia de los Alimentos
Facultad de Farmacia - Universidad Complutense de Madrid
https://www.ucm.es/nutricioncarbajal/
https://www.ucm.es/innovadieta/

# La evidencia científica como base de la intervención nutricional y las recomendaciones dietéticas

**Nutrición y Dietética basadas en la evidencia (NuBe)**
https://www.ucm.es/innovadieta/nube

**Documentos consenso**
https://www.ucm.es/innovadieta/documentos-consenso

**EFSA**, Nutrition and Health Claims
http://www.efsa.europa.eu/en/topics/topic/nutrition-and-health-claims

**Sánchez-Muniz FJ. Teorías, evidencias, fraude y rigor científico. Una breve reflexión**. *JONNPR. 2017;2(10):431-434*. DOI**:** 10.19230/jonnpr.1638
http://revistas.proeditio.com/jonnpr/article/download/1638/pdf1638

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**Universidad Autónoma de Nuevo León. México**
http://www.uanl.mx/
Facultad de Salud Pública y Nutrición

Programa de Habilidades en Lectura Crítica Español
http://www.redcaspe.org/herramientas

## ISOLATING EFFECTS OF SINGLE NOFS OR NOFS-CHRONIC DISEASE PATHWAYS

Isolating single NOFS effects is challenging if not theoretically impossible. Many NOFS functions are interrelated and may affect more than one biological pathway, and any one biological pathway may be affected by multiple NOFSs. In addition, there is collinearity in NOFS intake. Thus, both confounding by and interactions among NOFSs must be considered. Mapping these potential relationships in logic models or analytic frameworks helps to identify these considerations when framing questions to guide systematic reviews. Mapping the evidence identified can also be helpful for understanding relationships and patterns. For example, in Table 3-1, which is based on a World Health Organization dietary guidance report (WHO/FAO, 2003) several rows indicate associations with more than one of the six disease outcomes, and several of the columns for disease outcomes indicate associations with more than one NOFS variables. The source table for the excerpts in Table 3-1 also included food-based dietary variables. Food variables may be of interest because of their nutrient content and also content of bioactive food substances, e.g., polyphenols, with potential chronic disease risk reduction benefits as antioxidants.

**TABLE 3-1** Summary of the Strength of Evidence for Obesity, Type 2 Diabetes, Cardiovascular Disease (CVD), Cancer, Dental Disease, and Osteoporosis[a]

| | Obesity | Type 2 Diabetes | CVD | Cancer | Dental Disease | Osteoporosis |
|---|---|---|---|---|---|---|
| **Energy and fats** | | | | | | |
| High intake of energy dense foods | C↑ | | | | | |
| Saturated fatty acids | | P↑ | C↑[b] | | | |
| Trans fatty acids | | | C↑ | | | |
| Dietary cholesterol | | | P↑ | | | |
| Myristic and palmitic acid | | | C↑ | | | |
| Linoleic acid | | | C↓ | | | |
| Fish and fish oils (EPA and DHA) | | | C↓ | | | |
| Plant sterols and stanols | | | P↓ | | | |
| α-Linolenic acid | | | P↓ | | | |
| Oleic acid | | | P↓ | | | |
| Stearic acid | | | P-NR | | | |
| Nuts (unsalted) | | | P↓ | | | |
| **Carbohydrate** | | | | | | |
| High intake of NSP (dietary fiber) | C↓ | P↓ | P↓ | | | |
| Free sugars (frequency and amount) | | | | | C↑[c] | |
| Sugar-free chewing gum | | | | | P↓[c] | |
| Starch[d] | | | | | C-NR | |
| Wholegrain cereals | | | P↓ | | | |

| | | | | |
|---|---|---|---|---|
| **Vitamins** | | | | |
| Vitamin C deficiency | | | C↑[e] | |
| Vitamin D | | | C↓[f] | C↓[g] |
| Vitamin E supplements | C-NR | | | |
| Folate | P↓ | | | |
| **Minerals** | | | | |
| High sodium intake | C↑ | | | |
| Salt-preserved foods and salt | | P↑[h] | | |
| Potassium | C↓ | | | |
| Calcium | | | | C↓[g] |
| Fluoride, local | | | C↓[c] | |
| Fluoride, systemic | | | C↓[c] | P-NR[g] |
| Fluoride, excess | | | C↑[f] | |
| Hypocalcaemia | | | P↑[f] | |

NOTE: C↑ = convincing increasing risk; C↓ = convincing decreasing risk; C-NR = convincing, no relationship; DHA = docosahexaenoic acid; EPA = eicosapentaenoic acid; NSP = non-starch polysaccharides; P↑ = probable increasing risk; P↓ = probable decreasing risk; P-NR = probable, no relationship.
[a] Only convincing (C) and probable (P) evidence are included in this summary table.
[b] Evidence also summarized for selected specific fatty acids, see myristic and palmitic acid.
[c] For dental caries.
[d] Includes cooked and raw starch foods, such as rice, potatoes and bread. Excludes cakes, biscuits and snacks with added sugar.
[e] For periodontal disease.
[f] For enamel developmental defects.
[g] In populations with high fracture incidence only; applies to men and women more than 50-60 years old.
[h] For stomach cancer.
SOURCE: Reprinted from WHO/FAO (World Health Organization/Food and Agriculture Organization). 2003. *Diet, nutrition and the prevention of chronic diseases: Report of a joint WHO/FAO expert consultation.* Geneva, Switzerland: WHO.

In individuals who do not use nutrient supplements, the range of intakes may be narrow within a given population with wide day-to-day variation that makes it difficult to identify group differences. Among those who do use supplements, single nutrient supplements will be associated with a substantially higher range of doses than would be obtained from food sources, facilitating clear comparisons if supplement intake is ascertained. The same would apply to supplements of botanicals (e.g., cucurmin from turmeric). A complication that sometimes remains is that the form of the NOFS in a supplement may be qualitatively different from the form that is in food, with different pathways or potency of effect. Use of multivitamin supplements limits ability to attribute any effect of the supplement to a specific nutrient.

Intervention trials involving supplements can evaluate effects of the supplement dose as an increase over baseline intake in the study population. However, for a variety of behavioral and biological reasons, answers to DRI questions may require studies that vary NOFS intakes based on dietary advice. In this case, the intervention unavoidably involves changes in intake of other NOFSs present in the foods for which consumption is changed, and these NOFSs will vary according to participant food choices as well as the degree of compliance. Changes in targeted and non-targeted NOFSs in comparison groups can be evaluated through dietary reports or biomarkers of intake (where available) to help with the attribution of any observed changes in outcome to the intervention assignment.