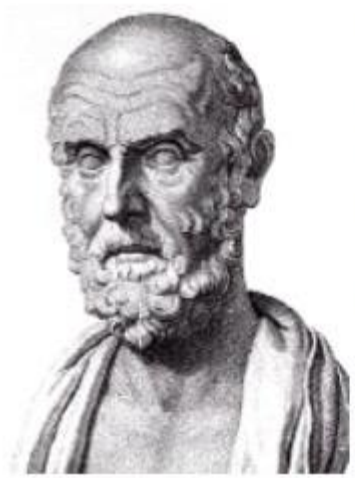
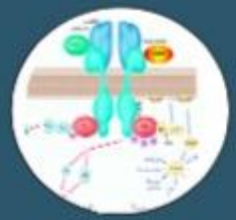
En -400 avant JC,
Hippocrate recommandait déjà de considérer
l'alimentation comme la première médecine



" l'alimentation sert à la fois à rétablir la santé et à la conserver chez les gens qui se portent bien."

" Ce qui est principalement utile et indispensable au médecin pour bien exercer son art, se réduit souvent à savoir ce qu'est l'homme par rapport à ce qu'il mange, à ce qu'il boit et les changements que chaque chose peut déterminer en lui".

Stress Oxydant

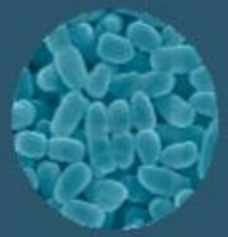


Métabolisme du glucose

Acides gras

Méthylation

Intoxication et Détoxication



Ecosystéme Intestinal

Vitamine D

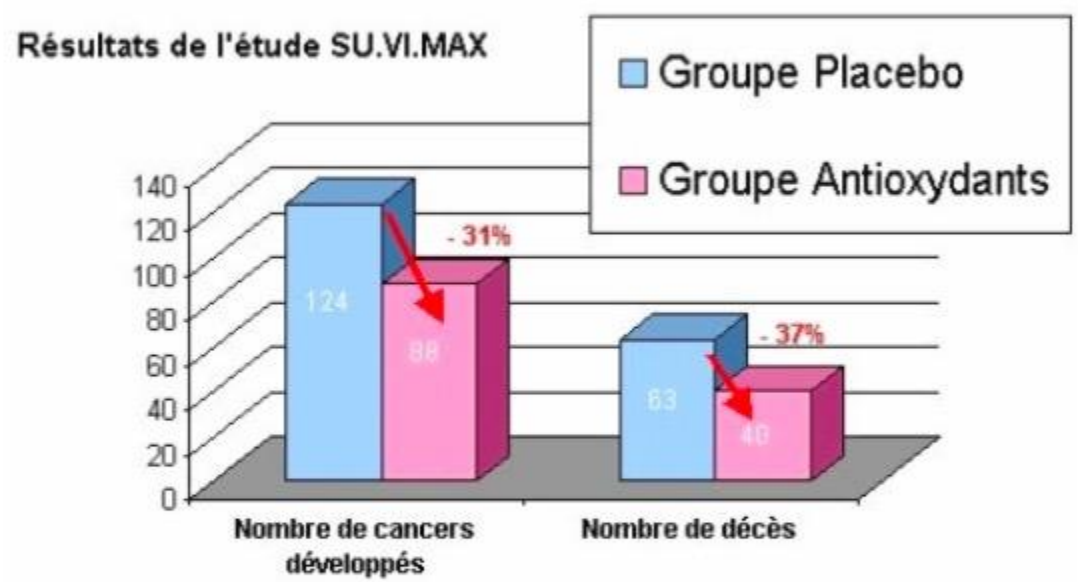
● ● ●

L'étude SU.VI.MAX est une étude épidémiologique (13 017 personnes suivies pendant 8 ans) dans le domaine de la prévention nutritionnelle des maladies chroniques.

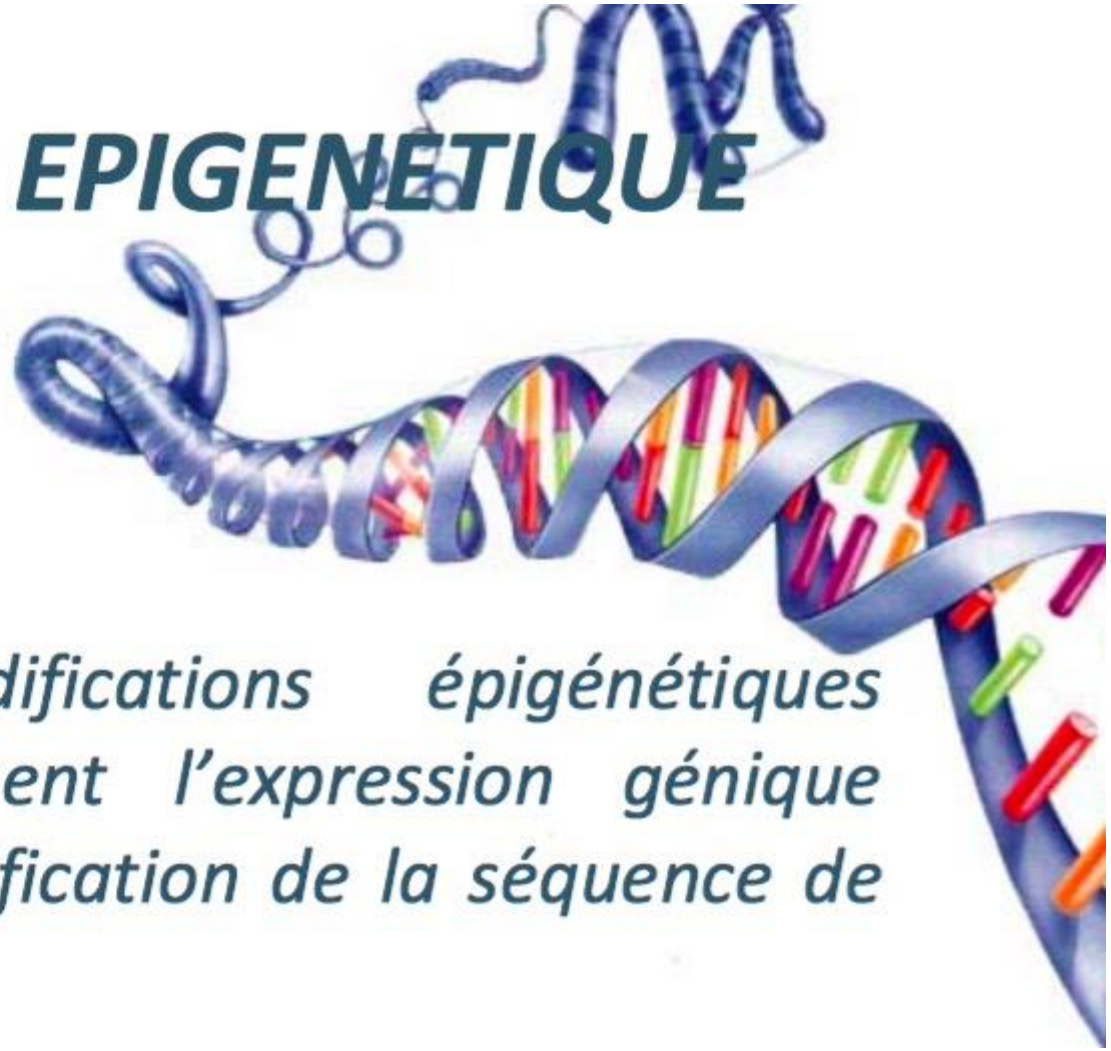
Lancée en 1994, elle vise à tester l'impact d'un apport supplémentaire en vitamines et minéraux antioxydants (bêta-carotène, vitamines E et C, zinc et sélénium), à doses nutritionnelles, dans la prévention des cancers et des maladies cardiovasculaires.

Hercberg S., Preziosi P., Briançon S., Galan P., Paul-Dauphin A., Malvy D., Roussel A-M., Favier A.
A primary prevention trial of nutritional doses of antioxidant vitamins and minerals on cardiovascular diseases and cancers in general population : The SU.VI.MAX Study. Design, methods and participant characteristics.
***Control Clin Trials**, 1998, 19: 336-351.*

SUVIMAX STUDY



EPIGENETIQUE

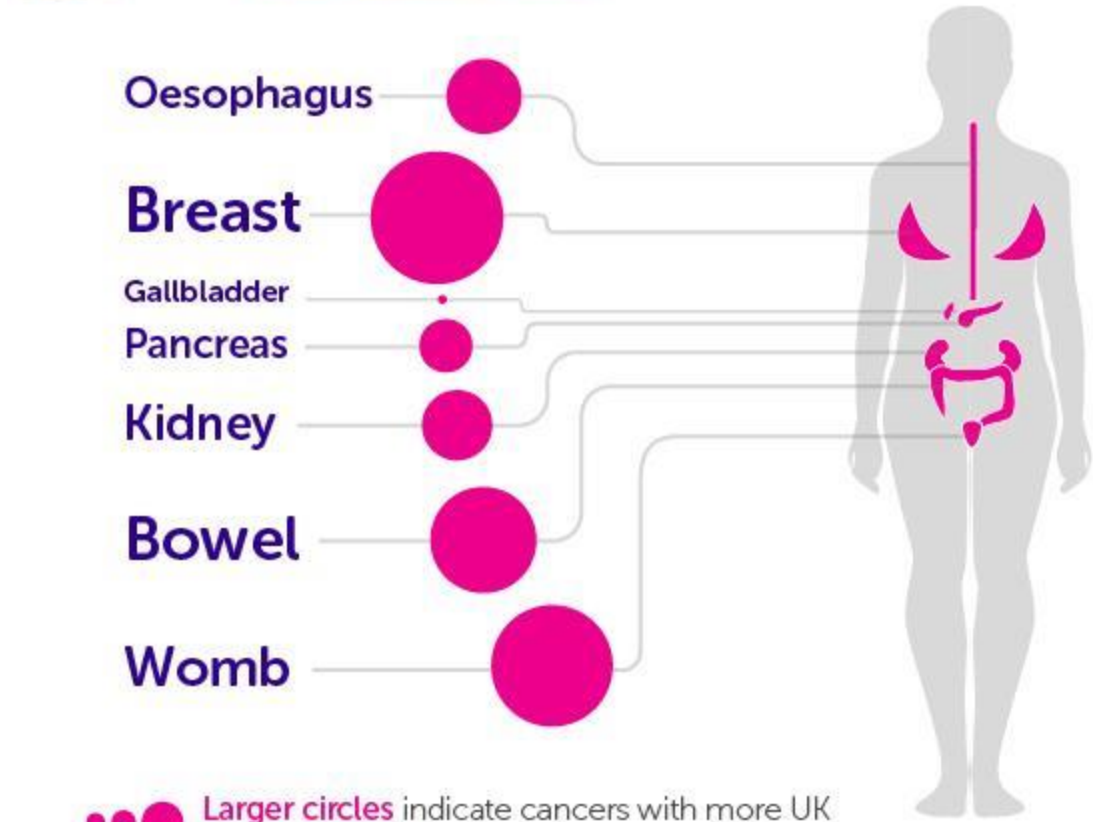


Les modifications épigénétiques conditionnent l'expression génique sans modification de la séquence de l'ADN.

Micronutrients Mitochondriaux

Vitamine A
Vitamines B1, B2, B3, B5
Vitamine E
Vitamine C
Acides Gras Oméga-3 et -6
Sélénium
Zinc
Cuivre
Acid alpha lipoïque
L-Carnitine
Coenzyme-Q10
Glutathion Réduit
Acétyl-Cystéine

BEING OVERWEIGHT CAN CAUSE AT LEAST 7 TYPES OF CANCER IN WOMEN



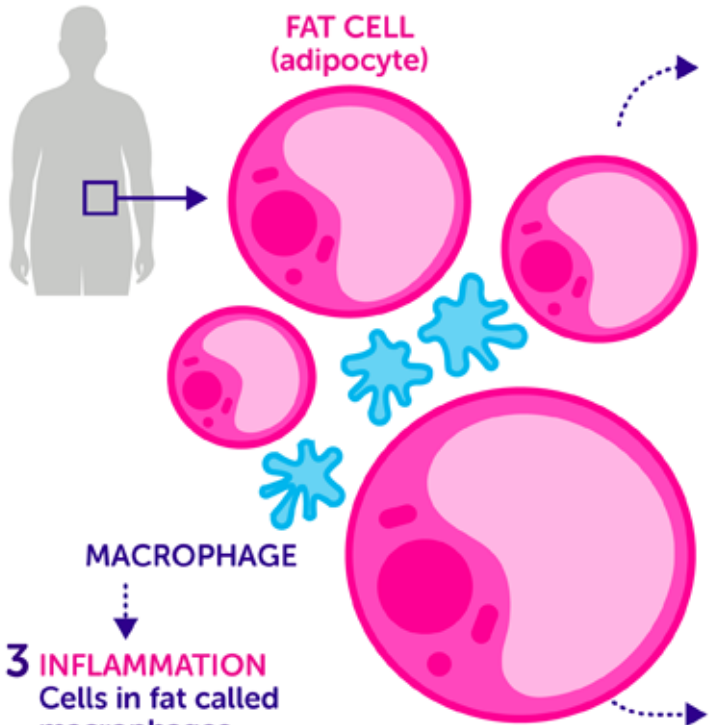
Larger circles indicate cancers with more UK cancer cases linked to being overweight or obese

WE WILL BEAT CANCER SOONER
cruk.org

CANCER RESEARCH UK

HOW COULD OBESITY LEAD TO CANCER?

Research has identified three main ways



1 OESTROGEN
After the menopause, oestrogen made by fat cells can make cells multiply faster in the breasts and womb, increasing the risk of cancer.

2 INSULIN AND GROWTH FACTORS
Excess fat can cause levels of insulin and other growth factors to rise, which can also tells cells to divide more rapidly.

3 INFLAMMATION
Cells in fat called macrophages release chemicals called cytokines, encouraging cells to divide (including cancer cells).

There are other theories too, but these are the main ideas being studied. More research is needed to understand this in more detail.

Nutrition, inflammation and cancer

Laurence Zitvogel, Federico Pietrocola & Guido Kroemer

Nature Immunology **18**, 843–850 (2017) doi:10.1038/ni.3754
Received 27 March 2017 Accepted 26 April 2017 Published online 19 July 2017

Abstract

Quantitative and qualitative aspects of nutrition have a profound effect on leukocytes and thereby affect proinflammatory carcinogenic effects or anticancer immune responses. As a result, nutrition affects the incidence, natural progression and therapeutic response of malignant diseases, both in humans and in preclinical animal models. Here we discuss the molecular mechanisms through which alimentary cues modulate metabolic, microbial and neuroendocrine circuitries and thus affect the probability of developing premalignant lesions that progress to clinically manifested disease and the response to therapeutic intervention. We examine each of the connections that compose the triangle of nutrition, immunological and inflammatory reactions and cancer while focusing on the mechanistic aspects of these relationships.

Cell Reports

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Volume 8, Issue 3, p754–766, 7 August 2014

ARTICLE

A Mitochondrial Switch Promotes Tumor Metastasis

Paolo E. Porporato, Valéry L. Payen, Jhudit Pérez-Escuredo, Christophe J. De Saedeleer, Pierre Danhier, Tamara Cop
Morgane Tardy, Thibaut Vazeille, Caroline Bouzin, Olivier Feron, Carine Michiels, Bernard Gallez, Pierre Sonveaux

Published Online: July 24, 2014

▾ Highlights

- Tumor metastasis is under metabolic control
- Both mitochondrial overload and mild respiration dysfunction promote metastasis
- Mitochondrial superoxide promotes tumor cell migration, invasion, and clonogenicity
- Scavenging mitochondrial superoxide prevents spontaneous tumor metastasis

▾ Summary

Metastatic progression of cancer is associated with poor outcome, and here we examine metabolic changes underlying this process. Although aerobic glycolysis is known to promote metastasis, we have now identified a different switch primarily affecting mitochondria. The switch involves overload of the electron transport chain (ETC) with preserved mitochondrial functions but increased mitochondrial superoxide production. It provides a metastatic advantage phenocopied by partial ETC inhibition, another situation associated with enhanced superoxide production. Both cases involved protein tyrosine kinases Src and Pyk2 as downstream effectors. Thus, two different events, ETC overload and partial ETC inhibition, promote superoxide-dependent tumor cell migration, invasion, clonogenicity, and metastasis. Consequently, specific scavenging of mitochondrial superoxide with mitoTEMPO blocked tumor cell migration and prevented spontaneous tumor metastasis in murine and human tumor models.

J. Clin. Med. **2016**, *5*(2), 15; doi:10.3390/jcm5020015

Open Access Feature Paper Review

Omega-3 Fatty Acids and Cancer Cell Cytotoxicity: Implications for Multi-Targeted Cancer Therapy

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Abstract

Cancer is a major disease worldwide. Despite progress in cancer therapy, conventional cytotoxic therapies lead to unsatisfactory long-term survival, mainly related to development of drug resistance by tumor cells and toxicity towards normal cells. *n*-3 polyunsaturated fatty acids (PUFAs), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), can exert anti-neoplastic activity by inducing apoptotic cell death in human cancer cells either alone or in combination with conventional therapies. Indeed, *n*-3 PUFAs potentially increase the sensitivity of tumor cells to conventional therapies, possibly improving their efficacy especially against cancers resistant to treatment. Moreover, in contrast to traditional therapies, *n*-3 PUFAs appear to cause selective cytotoxicity towards cancer cells with little or no toxicity on normal cells. This review focuses on studies investigating the cytotoxic activity of *n*-3 PUFAs against cancer cells via apoptosis, analyzing the molecular mechanisms underlying this effective and selective activity. Here, we highlight the multiple molecules potentially targeted by *n*-3 PUFAs to trigger cancer cell apoptosis. This analysis can allow a better comprehension of the potential cytotoxic therapeutic role of *n*-3 PUFAs against cancer, providing specific information and support to design future pre-clinical and clinical studies for a better use of *n*-3 PUFAs in cancer therapy, mainly combinational therapy. View Full-Text

Keywords: fatty acids (FAs); *n*-3 polyunsaturated fatty acids (PUFAs); docosahexaenoic acid (DHA); eicosapentaenoic acid (EPA); apoptosis; cytotoxicity; cancer therapy; combinational therapy; drug resistance; cancer stem cells