

How have you used your SOAP today?

Considering the effects of food and environment on gene expression

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Let your food be your medicine and your medicine be your food.

Hippocrates

Having practised as a family physician for more than 25 years, I have used soap regularly every day. Of course, we use the usual soap to wash away those nasty bugs and keep ourselves and others clean. But we physicians have also been well trained to use SOAP as an acronym. We all know what it stands for and use it every day on people in our practices. Yes, even after 25 years I am still *practising* medicine. We have become rather good at using this acronym as we extract histories from patients. “Can you tell me how this all began?” or “When were you last well and what symptoms did you have?” Thus we explore the Symptoms. Then we go on to the examination for discovery of the Objective findings and list them. Upon compilation of these findings, and once the differential diagnosis has been dealt with, a final Assessment is eventually made. We then devise a Plan, and this usually includes some lifestyle modification if deemed appropriate and most likely a drug or two. The emphasis is on the latter half of SOAP. This process is repeated upon seeing the patient for another complaint, and with new symptoms and signs another diagnosis might be made. Soon the patient could be taking multiple drugs. The fourth or fifth drug might be needed to counteract side effects of the first or second drug. Examples of this abound: a nonsteroidal anti-inflammatory drug followed by a proton pump inhibitor, or an antifungal after the use of an antibiotic. They are either used in concert or in tandem.

Stepping back

Is it not time to step back a little and explore how we got the dis-ease in the first place? How did we get the “SO” to begin with? Determining what environmental exposures, foods, and nutritional deficiencies or excesses could be triggering the symptoms and signs can be quite time-consuming. Despite this, would it not be better to prevent the triggering of disease in the first place than to treat already established disease with drugs?

One lifestyle modification we often recommend is that patients stop smoking. This environmental contribution can induce a number of diseases, not the least

of which is cancer¹ (although no double blind study exists to prove this). Not everyone, for example, with the genetic susceptibility will develop rheumatoid arthritis, but those who smoke² might light the fuse to a stick of dynamite that could trigger genes to be turned on or off. Once triggered, it is difficult to switch this sequence off.

You are what you eat

Recently, knowledge in the field of nutrigenomics has exploded, revealing the considerable effects that food has on gene expression.^{3,4} Vitamin D (a hormone produced by sun exposure and found in very few foods) is responsible for switching on or off more than 2000 genes. Omega-3 fatty acids (found in a limited number of foods) can switch on or off more than 500 genes—more than 50 of these genes are relevant in cardiovascular disease alone,⁴ and more than 75 of these genes are involved in colon cancer cell regulation.⁵ Inadequate intake of vitamin D and omega-3 fatty acids is common today.^{6,7} Repletion of either of these essentials improves health on its own, but if used together the results might be synergistic, just as has been suggested with the use of a combination of medications like the proposed “polypill.”⁸ The polyunsaturated fatty acids in appropriate levels might themselves act just like a polypill.⁹ Additionally, vitamin D has been shown to reduce a variety of diseases beyond rickets and osteoporosis. It decreases the risk of developing various cancers by 30% to 80%,¹⁰ decreases the risk of heart disease, prevents development of autoimmune disease, helps the innate immune system combat infection, and on and on. Taking 2000 IU or more of vitamin D in the first year of life has been shown to reduce the incidence of type 1 diabetes over the next 30 years by more than 80%.¹¹ Adequate vitamin D early in life can reduce the lifetime risk of multiple sclerosis and prevent infectious triggers from bringing on the disease.¹²

Once again it is time to broaden our view of medicine now that we know that what we eat or are exposed to can influence gene expression. This “new” medicine might not be as glamorous as the newest “biologic” used for autoimmune disease. Nevertheless, it could be the wave of the future. We need to appreciate the true influence that environmental and nutritional factors have on gene expression and thus our health. More time might need to be spent with our patients to determine the underlying causes of the SO of SOAP, and much more will need to be learned about how we can

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prevent or control detrimental gene expression, but we now know that it is not as simple as being “all in your genes.”

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Competing interests

None declared

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References

1. Khuri FR, Kim ES, Lee JJ, Winn RJ, Benner SE, Lippman SM, et al. The impact of smoking status, disease stage, and index tumor site on second primary tumor incidence and tumor recurrence in the head and neck retinoid chemoprevention trial. *Cancer Epidemiol Biomarkers Prev* 2001;10(8):823-9.
2. Klareskog L, Padyukov L, Alfredsson L. Smoking as a trigger for inflammatory rheumatic diseases. *Curr Opin Rheumatol* 2007;19(1):49-54.
3. Kaput J, Perlina A, Hatipoglu B, Bartholomew A, Nikolsky Y. Nutrigenomics: concepts and applications to pharmacogenomics and clinical medicine. *Pharmacogenomics* 2007;8(4):369-90.
4. Vanden Heuvel JP. Cardiovascular disease-related genes and regulation by diet. *Curr Atheroscler Rep* 2009;11(6):448-55.
5. Narayanan BA, Narayanan NK, Simi B, Reddy BS. Modulation of inducible nitric oxide synthase and related proinflammatory genes by the omega-3 fatty acid docosahexaenoic acid in human colon cancer cells. *Cancer Res* 2003;63(5):972-9.
6. Schwalfenberg G. Not enough vitamin D. Health consequences for Canadians. *Can Fam Physician* 2007;53:841-54.
7. Simopoulos AP. Evolutionary aspects of diet, the omega-6/omega-3 ratio and genetic variation: nutritional implications for chronic diseases. *Biomed Pharmacother* 2006;60(9):502-7.
8. Fahey T, Brindle P, Ebrahim S. The polypill and cardiovascular disease. *BMJ* 2005;330(7499):1035-6.
9. Das UN. Essential fatty acids and their metabolites could function as endogenous HMG-CoA reductase and ACE enzyme inhibitors, anti-arrhythmic, anti-hypertensive, anti-atherosclerotic, anti-inflammatory, cytoprotective, and cardioprotective molecules. *Lipids Health Dis* 2008;7:37.
10. Garland CF, Gorham ED, Mohr SB, Garland FC. Vitamin D for cancer prevention: global perspective. *Ann Epidemiol* 2009;19(7):468-83.
11. Hyppönen E, Läärä E, Reunanen A, Järvelin MR, Virtanen SM. Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study. *Lancet* 2001;358(9292):1500-3.
12. Hayes CE, Donald Acheson E. A unifying multiple sclerosis etiology linking virus infection, sunlight, and vitamin D, through viral interleukin-10. *Med Hypotheses* 2008;71(1):85-90. Epub 2008 Apr 2.

