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## Attention to Prevention

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*Bill Lands*

### Key Points

- Healthy people do not need to pay for treatment costs. Many current health care costs are for preventable chronic disorders that have excessive omega-6 hormone actions associated with a relative omega-3 deficit.
- A nutrient imbalance in foods eaten creates an imbalance in tissue hormone actions that causes much morbidity and mortality.
- An effective corporate wellness program can give food lists of Omega 3-6 Balance Scores to inform employees' personal food choices and provide each employee with fingertip blood-spot assay results of their omega-3 status.
- This simple wellness program in a self-insured corporation with 10,000 employees may allow \$20 million of annual health-related financial losses to go to more desired activities.

*What's past is prologue; what to come, in yours and my discharge.*

William Shakespeare, *The Tempest*, Act 2, Scene 1

**Key words:** Absenteeism, Atherosclerosis, Cardiovascular, Cholesterol, Food energy, Free fatty acids (FFA), Health risk assessment (HRA), Health care claim, Heart attack, Highly unsaturated fatty acids (HUFA), Hormone, Immune-inflammatory, Kilocalories (Cal), Metabolic syndrome, Omega-3, Omega-6, Omega 3-6 Balance Scores, Presenteeism, Prevention, Prediction, Psychiatric disorders, Self-insured, Symptoms, Thrombosis, Very low density lipoproteins (VLDL)

### INTRODUCTION

A major cause for excessive medical costs in the USA is excessive attention to predictive factors and neglect of causal factors. It is much better to prevent the cause than just the symptoms created by the cause. We use signs and symptoms associated with

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past harmful events as factors for predicting possible risk of future harm. Predicting future outcomes allows us to hope for ways that we might prevent future harm.

However, prediction differs from prevention. Combining all associated factors together can improve predictions, whereas focusing on only the causal factors will improve prevention. Mistaken assignments of a causal role to mere associated predictive risk factors have caused much human loss.

No matter how closely associated a predictive factor and an outcome appear to be, we must not think that thunder causes rain, that smoke causes fire, or that gray hair causes death. This logic is important, because spending time, effort, and resources to eliminate thunder, smoke, or gray hair is not an effective way to prevent loss from rain, fire, or death.

Some foods are associated with health and disease, and biochemists can define explicit molecular aspects of food that cause harmful consequences and death. Unfortunately, past failure of the biomedical community to focus on details in the chain of molecular events that causally connect food items to harmful outcomes has allowed massive waste of human resources, health, and lives (1, 2). The failure has provided our nation with 87.5% of health care claim costs due to lifestyle aspects that could be prevented (3). Claims that have been paid for treating preventable disorders have not cut the continual need to pay treatment costs.

It seems unethical to remove symptoms and create a sense of benefit while leaving the primary cause unchanged to continue harming individuals and their future generations, especially when such harm is preventable. Failure to identify and prevent causal factors allows the nation's health care costs to continue escalating, with preventable illness causing 80% of the burden of illness and 90% of all health care costs (4). It is illogical to have preventable illnesses account for eight of the nine leading categories of death in the USA (4) when we can identify and avoid the preventable cause of those costs. When disease is prevented, treatment costs are not needed.

This chapter describes two preventable imbalances in daily food habits that cause serious health disorders: an imbalance in the intake and expenditure of food energy and an imbalance in the intake of vitamin-like omega-3 and omega-6 nutrients (1, 2). What is to come from the assembled knowledge is in your and my control.

## FOOD ENERGY MANAGEMENT

General advice to “eat sensibly” and avoid “poor nutrition” lacks explicit information of how food connects to health problems. Currently popular “super-sized” meals create a body burden not suited to healthy outcomes. The meals can provide 1,000 calories (Cal) in 20 min to a basal metabolic system that uses only 0.45 Cal/h/lb or 68 Cal/h/150-lb individual. Every meal over 300 Cal brings in more food energy than will likely be expended in the next few hours, causing the extra absorbed carbohydrate, protein, and fat to be metabolized by the liver to form triglycerides and cholesterol, which are assembled into very low density lipoproteins (VLDL) and secreted into the blood stream. The larger the meal, the greater is the postprandial triglyceridemia.

As the triglyceride-rich VLDL circulates in the blood, lipoprotein lipase steadily cleaves the triglycerides into nonesterified free fatty acids (FFA) that disturb many aspects of tissue metabolism. Although the FFA can enter muscle tissue and be “burned” during muscle activity, circulating FFA can do the following:

1. Cause insulin resistance that contributes to elevated blood glucose
2. Impair endothelial integrity
3. Initiate oxidative inflammatory events in tissues
4. Enter adipose tissue where they contribute to obesity

More attention needs to be paid to the transient harmful effects of FFA released during VLDL cleavage in the bloodstream. A major cause for excessive medical costs in the USA is excessive attention to accumulated fat and neglect of the FFA that cause fat accumulation. Eating foods with low energy density and fewer calories per meal can decrease the “flood” of FFA that follows each large meal, and it can decrease the transient FFA-mediated tissue insults and the conditions that those insults cause.

“Metabolic syndrome” describes a cluster of often-discussed conditions that predict the development of cardiovascular disease (5). They include the following:

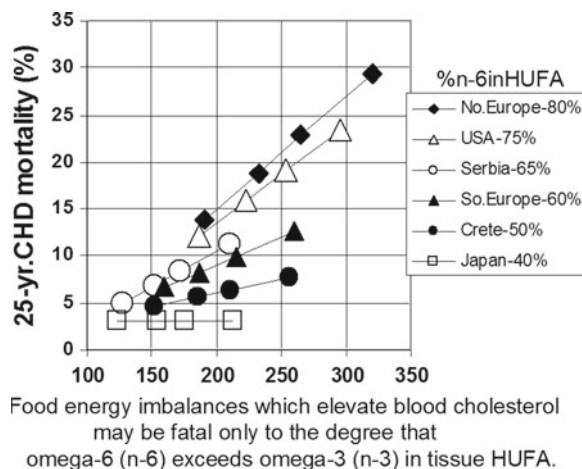
1. Serum triglycerides 150 mg/dL or above
2. Fasting blood glucose of 110 mg/dL or above
3. Abdominal obesity (waist circumference >102 cm in men or >88 cm in women)
4. HDL cholesterol 40 mg/dL or lower
5. Blood pressure of 130/85 or above

Many of these conditions can be regarded as results of food energy toxicity. While these conditions *per se* may not actually cause cardiovascular disease, factors that cause the conditions do cause the disease. For example, obesity itself is a “strong” predictive risk factor, but the body fat itself may not actually mediate cardiovascular disease or death.

Also, the beneficial enzyme activity in HDL lipoproteins that reduces oxidative stress is not found in VLDL or LDL lipoproteins, alerting us to the importance of reducing the primary causes of oxidative inflammatory events that mediate cardiovascular disease (1). Adipose tissue is increasingly recognized to acquire inflammatory cells and mediators that act while adipose tissue acquires and releases nonesterified free fatty acids (FFA) during its major interactions with the body. The cytokine mediators of inflammation, like the omega-6 hormones that enhance their release and action, are involved in many chronic diseases ranging from cardiovascular to psychiatric disorders.

Inflammatory processes, more than blood cholesterol, seem to mediate the processes causing cardiovascular disease (6–8). Cholesterol levels in blood rise with elevated food energy intakes, but they have uncertain utility worldwide for predicting cardiovascular death. Results from a 25-year follow-up of the “Seven Countries Study” (9) showed that risk of death differs widely for people with 220 mg cholesterol per deciliter (Fig. 2.1). Death correlated with blood cholesterol levels in Northern European and US people (1).

However, cholesterol was not appreciably predictive of death in Japan or Crete, where the relative abundance of omega-3 in tissue HUFA was equal to or greater than omega-6, and inflammatory factors were less prevalent (10).



**Fig. 2.1.** Food energy imbalances, cholesterol, and death.

Subsequent data from 173,539 Japanese (11) confirmed that blood cholesterol levels show no apparent risk for death of Japanese. Apparently, the food energy imbalances that elevate blood cholesterol may be fatal only to the degree that omega-6 (*n*-6) *exceeds* omega-3 (*n*-3) in the 20- and 22-carbon highly unsaturated fatty acids (HUFA) in tissues.

## BALANCING OMEGA-3 AND OMEGA-6 ACTIONS IN TISSUES

We accumulate vitamin-like omega-3 and omega-6 nutrients in tissues and convert them into hormones that have receptors on nearly every cell and tissue in the body. This fact gives a powerful role for voluntary food choices in affecting nearly every aspect of human physiology and pathology (12, 13). The diverse ethnic food habits seen around the world provide very different mixtures of omega-3 and omega-6 nutrients that maintain very different proportions of omega-3 and omega-6 in tissue hormone precursors. Figure 2.2 uses a simple measure of the relative balance in the milligrams per calorie of omega-3 and omega-6 nutrients in foods, the Omega 3-6 Balance Score (14), to show that typical average scores range worldwide from +3 to -8. As a result, blood samples from these diverse groups have proportions of omega-6 in HUFA that range from 28 to 88% (15).

The metabolic processes of digestion, elongation, desaturation, esterification, and accumulation of HUFA in tissue membrane lipids discriminate little between omega-3 and omega-6 chemical structures, making their relative abundance in the diet a major factor causing the relative proportions maintained in the tissue hormone precursors (16-17). A major cause for excessive medical costs in the USA is excessive attention to a food's calorie content diverting attention from imbalanced intake of omega-3 and omega-6 nutrients.

In contrast to the accumulation of hormone precursors in tissues, conversion of the precursors into potent hormone actions has many selective events that cause hormones

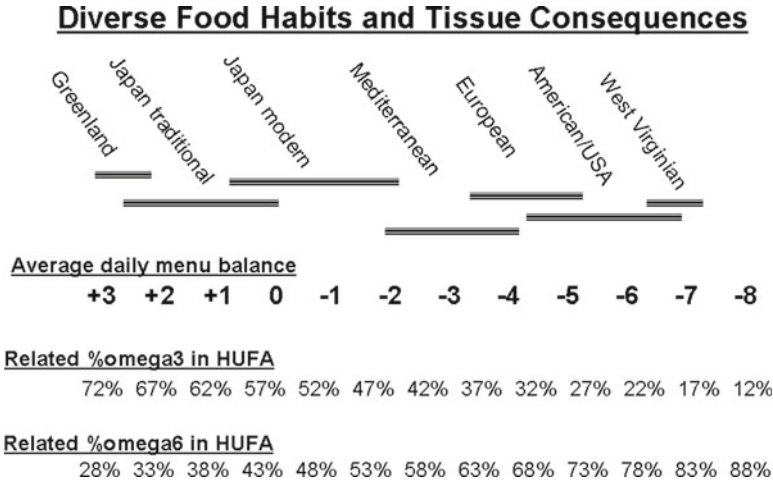


Fig. 2.2. Diverse food habits and their tissue consequences.

from omega-6 precursors to have much more vigorous actions than the omega-3 forms (18). As a consequence, the incidence and severity of cardiovascular, immune-inflammatory, and many other hormone-mediated disorders differ greatly among the different ethnic groups in Fig. 2.2. A major cause for excessive medical costs in the USA is excessive attention to signs and symptoms caused by excessive omega-6 hormone actions while neglecting the preventable tissue imbalance that causes them.

Heart attacks are infrequent for Greenland Inuits and distressingly frequent among West Virginians. The top four causes of death among elderly West Virginians—heart disease, malignant neoplasms, chronic low respiratory disease, and cerebrovascular disease—all involve excessive actions of omega-6 hormones made from the omega-6 HUFA accumulated in tissues.

The widespread actions of the many different receptors for omega-3 and omega-6 hormones (prostaglandins, thromboxanes, leukotrienes, and endocannabinoids) form a very large and expanding list of physiological and pathological processes in which essential fatty acids and the “arachidonic cascade” play an important preventable causal role (1, 2).

The list of disorders has grown to include atherosclerosis, thrombosis, arrhythmia, heart attacks, immune-inflammatory disorders, asthma, arthritis, psychiatric disorders, suicide, oppositional behavior, unproductive workplace behaviors, cancer proliferation, and length of stay in hospitals. As a result, the preventable risk for a wide range of serious health problems relates to the proportion of omega-6 in the hormone precursors accumulated in tissues (2).

The percent of omega-6 in HUFA is a health risk assessment (HRA) value easily measured in a fingertip blood-spot sample (19). It reflects the average omega 3-6 balance score of foods eaten (see Fig. 2.2), and it is useful in predicting future risk of harm from excessive omega-6 hormone actions (20). Figure 2.3 combines results from several studies (20–22) to show clearly that people with HRA values near 60% omega-6 in HUFA have much lower cardiovascular morbidity and mortality than those with 80%.

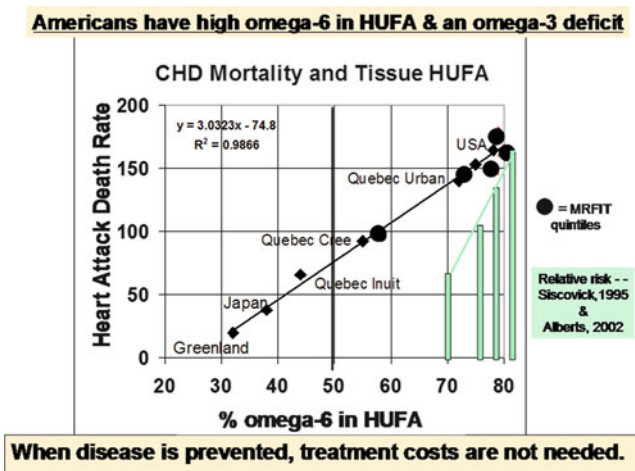


Fig. 2.3. Mortality and tissue HUFA.

In a similar way, Hibbeln et al. (23) noted how dietary availability of *n*-3 HUFA relates to attributable risk from 13 morbidity and mortality outcomes, including all-cause deaths, coronary heart disease, stroke, cardiovascular disease, homicide, bipolar disorder, and major and postpartum depressions. Commodities for 38 countries and tissue composition data were correlated to each illness in deficiency risk models. The authors noted that a healthy allowance for dietary *n*-3 HUFA in current US diets would be 3.5 g/day for a 2,000-Cal diet and likely could be reduced to one-tenth of that amount when people consume fewer competing *n*-6 fats.

To illustrate the role of imbalanced intakes of omega-3 and omega-6 nutrients in the many diverse health problems caused by excessive omega-6 hormone actions, I combined all the problems together under a single measure of health outcome, the annual overall health care claim costs (24). Fragmentary data available at this time indicate that average annual claims of \$6,408 in West Virginia relate to HRA values near 83% *n*-6 in HUFA while the USA has averages of \$5,184 with 78%.

A self-insured corporation making fish oil supplements freely available to employees averaged only \$3,930 ( $n=800$ ), and two separate samplings of interested employees had \$3,052 with 67% ( $n=61$ ) and \$2,076 with 63% ( $n=51$ ). A major cause for excessive medical costs in the USA is excessive expenditures that treat signs and symptoms and neglect to prevent imbalanced omega-3 and omega-6 mediators of those signs and symptoms (24).

A small study in a West Virginia school district this year had a 2-month intervention of “Nix the 6 and Eat the 3” by eating foods and supplements that shifted HRA values for a majority of the group from above 70% omega-6 in HUFA to values below 70% (unpublished results). The group shifted in 2 months from having many members with average daily Omega 3-6 Balance Scores near  $-7$  to values near  $-3$ . Overall, the limited data suggest that corporate wellness programs can easily collect data that confirm that the simple measure of annual claim costs for a group (a valid measure of overall health) will indeed correlate with the average percent of omega-6

in tissue HUFA (a valid HRA measure). No prescription is needed for food choices that keep people healthy and make treatment costs unneeded.

## FOLLOW THE MONEY

Several years ago, an editor invited me to evaluate the paradoxical advice to the public about dietary lipids. My review described how fragments of knowledge develop in separate societal groups with different priorities (1). Combining these fragments together can fail when “silo mentality” limits communication. As a result, incomplete information is transmitted to the public in oversimplified messages (on food energy density, saturated fat, cholesterol, omega-3, etc.) that can often be misunderstood or seen as contradictory. Paradoxes arise when two valid facts appear to contradict. They may be reconciled by an added fact that had not been presented.

The detailed review provided some “missing facts” (1), but it failed to bring any appreciable change in the continuing public misperceptions. It likely was read only by biochemists and not read by people who would apply the information for the public (24, 25). To succeed, prevention knowledge will need to be in the hands and minds of people who can use the information for their personal gain. Gain, rather than altruism, is the major motivating force for change in a market-oriented society. My attempt to “follow the money” at that time needed information that I had not yet acquired as a biochemistry professor in medical schools (1).

Knowledge is power, but I had not recognized that academic knowledge and financial acumen wield very different power in a market-oriented society. Explicit biochemical knowledge of how nutrition and metabolism cause health disorders is only one step in helping consumers prevent health-related losses.

Trillions of dollars flow every year through the current health care system. The diverse business plans operating in the “health care arena” (25) include pharmaceutical companies, doctors, nurses, hospitals, insurers, actuaries, researchers, and many health care professionals, including wellness counselors. All of them gain from their current employment.

We can examine the various plans to see who gains and who loses with successful prevention. A major cause for excessive medical costs in the USA is excessive attention to paying for treatment procedures while neglecting to account for their efficacy in preventing future cost.

I now believe that the people most likely to gain from preventing the need for treatments will be the employees and employers who pay for health care rather than the people already gainfully employed in the current health care system. A chief executive officer (CEO) has the financial acumen (and the responsibility) to make cost-saving changes for corporate gain (24). Can we transfer the needed knowledge from biochemists to corporate CEOs when they operate in different “silos” with different priorities? It is an effort worth trying.

I was surprised to learn that nearly all large US corporations are now virtually self-insured when sharing health care claim costs with employees. In contrast, small businesses outsource the risk of uncertain health claim costs to health insurers who handle them on a “cost-plus basis.” This aspect is important when seeking partners motivated

<b>Every year excess actions of omega-6 at tissue receptors cause</b>			
<b>financial loss for</b>	<b>people</b>	<b>&amp; corporations</b>	<b>&amp; the nation</b>
	<b>employee</b>	<b>10 thousand employees</b>	<b>150 million employees</b>
<b>Medical &amp; Pharmacy cost</b>	<b>\$5,184</b>	<b>\$51,840,000</b> >millions	<b>\$777,000,000,000</b> >billions
<b>Health-related Absenteeism &amp; Presenteeism loss</b>	<b>\$10,000</b>		
<b>with an Overall loss =</b>	<b>\$15,200</b>	<b>\$152,000,000</b> >millions	<b>\$2,250,000,000,000</b> >trillions

Fig. 2.4. Financial losses from preventable disorders.

to prevent health-related expenses. Self-insured corporations losing funds from preventable causes will be motivated to take direct action to stop such losses. Many have already contracted for wellness services to encourage employees to choose preventive lifestyles (25). However, explicit ethical accountability and feedback is needed continually to inform corporate executives whether the contracted services prevent actual causes and not just symptoms created by the primary cause (1, 24, 25).

The CEO managing 10,000 employees with average annual health care claim costs of \$5,184 per employee is transferring every year about \$52 million of corporate resources for medical and pharmacy claim costs that are considered mostly preventable (Fig. 2.4). More serious, and less visible, are the health-related workplace losses to absenteeism and presenteeism (at work while sick) that occupational medicine professionals estimate may be more than twice the medical and pharmacy claim costs (26). Providing employees with information on Omega-3 Balance Scores of foods (14) and results of annual fingertip blood-spot analyses (19) gives them the tools and motivation for voluntary prevention of major financial loss to themselves and their corporation. No prescriptions are needed to make informed food choices, and no treatment expenses are needed for healthy people.

Transparent accounting that compares a group's HRA measures with actual claim costs gives explicit feedback to document the efficacy of corporate expenditures for preventing health-related losses. Executives need no new biomedical research to routinely monitor simple information on HUFA proportions and annual claim expenditures. Such feedback ensures successful attention to prevention. While one employee can decide on actions that prevent a need to spend thousands of dollars, a CEO can decide on actions that prevent a need to spend many millions of dollars (see Fig. 2.4). Pfizer might inform its 100,000 employees how to prevent \$500 million in health-related losses annually. Similarly, Walmart's 1.4 million employees might annually prevent \$52 billion in health-related losses. One can envision that a trillion dollars in financial resources expended annually by employees and employers for health-related costs nationwide might move to other, more desired, activities when there is attention to prevention.



## HOW ATTENTION IS DIVERTED FROM PREVENTION

News is what is happening now, and nothing seems “older” than yesterday’s news. Researchers who make biomedical discoveries need financial support from grant proposals that emphasize newness. Funds are not awarded to study “old” topics. Essential nutrients (e.g., vitamins) were news from 1900 to 1950 before metabolic enzymes and pathways became news (1). Then attention to metabolism began to give way to molecular biology in the 1970s as new hormone-like compounds (e.g., eicosanoids and cytokines) and their receptors became news.

Now attention is diverted toward the structure and expression of genes, the other half of gene-environment interactions. Academic researchers focus on what today’s research committees approve for new funding. There are few funds to spend on “old” topics. As a result, facts known earlier about metabolism of the vitamin-like omega-3 and omega-6 nutrients in our foods are no longer news and are not a large part of ongoing scientific attention.

Fortunately, executives will need no new biomedical knowledge to help employees monitor their annual claim expenditures and simple fingertip blood-spot assays of their HUFA proportions. The needed tools already exist, and each cycle of feedback information will be relevant personal news to each employee. The CEO who arranges for such feedback will ensure successful attention to prevention of future financial losses.

The public learns health news mostly from repeated marketing messages that fit specific financial goals of marketers who have no responsibility for fully integrated valid interpretations. Such repetitive paid messages are not news. The diverse mixture of incomplete, oversimplified messages to the public about drug treatments and food benefits is often misunderstood or seen as contradictory.

A major cause for excessive medical costs in the USA is excessive public messages about profitable treatment procedures while neglecting to tell people how they can prevent the need to pay for such procedures (1, 2). The chain of molecular events by which dietary *n*-6 fats cause inflammation, thrombosis, arrhythmia, and death is known and documented in detail (e.g., see (1)) but poorly communicated to the public. Doctors fail to inform the public that aspirin acts by blocking excessive omega-6 actions. That was news in 1971. Now billions of dollars are spent producing and marketing new pharmaceuticals that moderate excessive omega-6 actions without informing the public how beneficial moderation can also come by shifting the high dietary supply of omega-6 nutrients toward more omega-3 nutrients.

Drug companies carefully design and finance clinical trials to integrate with their marketing priorities and messages. The fortunate fact that serious disease or death occurs infrequently means that trials must monitor thousands of people for years to observe enough definite clinical outcomes of morbidity and mortality. As a result, many trials depend on predictive biomarkers as surrogates for actual clinical outcomes (Fig. 2.5).

However, the biomarkers used may not mediate causal pathways and may not be valid surrogates for true clinical outcomes (1, 2, 27). Cause differs from consequence, and prevention differs from prediction. Some predictive risk factors divert attention from preventing the causal factors.

Figure 2.5 shows causal intermediates that connect food to morbidity and mortality. The %*n*-6 in HUFA and excessive *n*-6 hormones (left side of Fig. 2.5) amplify transient

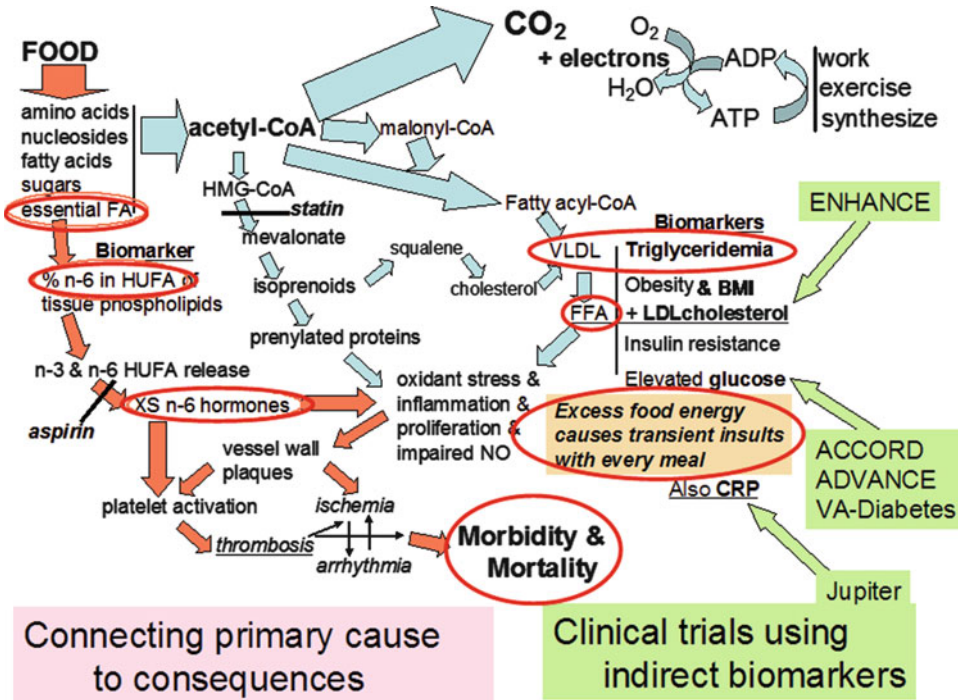


Fig. 2.5. Attending to causal mediators rather than associated surrogates.

insults from FFA into chronic inflammatory conditions that mediate morbidity from many different disorders and cause mortality.

When designing and interpreting clinical studies (e.g., far right side of Fig. 2.5), the associated biomarkers, obesity, body mass index (BMI), LDL cholesterol, elevated glucose, and C-reactive protein (CRP), that result when FFA are released from VLDL often divert attention away from causal mediators (1, 2). Very misleading oversimplifications came from giving “good” and “bad” attributes to the cholesterol carried by circulating lipoproteins (1). Also, markers like BMI, waist/height ratios, and body mass are secondary to the transient postprandial rises in blood FFA levels that cause the vascular insults that lead to death from cardiovascular disease (CVD).

The large ENHANCE trial (28) made news when it lowered blood cholesterol without a corresponding lowering of coronary heart disease (CHD) clinical events. The evidence prompted open questions in the mass media about whether blood cholesterol is truly the cause of death and whether “cholesterol drugs” actually do any good (29–31). The public learned “news” of the ongoing search for proof of whether elevated LDL-cholesterol is an actual cause of death or merely a surrogate marker caused by factors that do cause death (see Figs. 2.1 and 2.5). However, the passage of time gave later news cycles and repeated marketing messages that diverted attention away from the observed results and gave little further understanding.

In that context, my 2008 review (1) evaluated events around the 1984 NIH Conference that led the US Food and Drug Administration (FDA) to permit marketers to sell billions of dollars of statin drugs without filing evidence of their efficacy for preventing CVD

morbidity and mortality (32). The conference report stressed that maximal diet therapy (caloric restriction and weight loss) should be continued “even when use of drugs seems appropriate” (33). The committee clearly regarded CHD as a diet-induced disease caused by imbalanced food energy. However, the conference report was followed by widespread public information (especially from statin marketers) focused on blood cholesterol. More than two decades of diverting attention away from effective management of food energy has produced an obesity epidemic accompanied by widespread use of statins and little decrease in the incidence and prevalence of CVD (34).

Data accumulated by the Centers for Disease Control and Prevention confirm that its actions in serving the nation have not prevented the high burden of preventable CVD disease (2). An estimated 82,600,000 American adults (>1 in 3) have one or more types of CVD. CVD accounted for 1 of every 2.9 deaths in 2007 in the USA (35). A major cause for excessive medical costs in the USA is excessive diversion of attention toward treating signs and symptoms while neglecting to prevent the factors that cause them.

A clinical trial on macrovascular mortality, Action to Control Cardiovascular Risk in Diabetes (ACCORD), involved more than 10,000 patients with type 2 diabetes. The results made news by indicating that lowering the symptom of elevated glucose did not let diabetics live longer and may actually increase mortality risk (36, 37). The ACCORD, ADVANCE, VA-Diabetes trials and the NICE-SUGAR Study (38) all had a brief news cycle informing the public that lowering a predictive risk factor for CVD—elevated blood glucose—failed to lower the cause of CVD morbidity and mortality.

An additional news item came when the JUPITER trial showed that statin treatment lowered elevated levels of C-reactive protein (CRP), an acute stress protein released in inflammatory conditions (39). The news cycle for that result again opened public questions of whether cholesterol or inflammation is more important in mediating CVD morbidity and mortality (30).

Employees and employers paying for health care deserve more logical discussions by the biomedical community regarding how prediction differs from prevention. Employers can arrange for data feedback to employees to encourage food choices that give healthy HRA values that prevent the need for healthcare treatments.

Marketing messages from pharmaceutical companies describe drug treatments that decrease signs and symptoms caused by excessive omega-6 hormones while neglecting to comment on the dietary origin of the omega-6 mediators. Similarly, marketing messages from food companies describe beneficial dietary omega-3 contents of foods while not informing about the competing omega-6 contents. Both types of marketing message focus on treating signs and symptoms while diverting attention from preventing imbalanced intakes of omega-3 and omega-6 nutrients, which maintain high proportions of omega-6 in tissue HUFA (see Fig. 2.2) that cause the signs and symptoms (see Fig. 2.5).

Without informing the public of explicit consequences, corporations processing and marketing food have altered the types and amounts of food oils eaten in the USA (40). The estimated per capita consumption of soybean oil in the USA increased >1,000-fold from 1909 to 1999, increasing the availability of omega-6 linoleic acid from 2.79 to 7.21% of food energy. The food industry controls foods to fit its own priorities (e.g., shelf-life stability and profit margins) rather than the public’s priority for better health (41). Dr. Nestle gave authoritative insights on the sharp disparity in ethical

standards between corporate executives, members of the US Congress, and staff members in regulatory agencies (41).

A complex (and little known) set of subsidies financially favors some of the agricultural commodities that go into mass-produced prepared foods (42). The large annual subsidies paid by the public to large profit-making corporations are not described by annual news messages in the mass media. Benefit–cost estimates of public financing of food subsidies create unresolved paradoxes with regard to effects the food has on increasing the public’s need to pay for health care.

Simon (43) estimated that food corporations used fragmented accounting procedures to divert \$3.5 trillion of externalized costs (for health care expenses, unsafe products, and pollution) to be funded by the public while food corporations kept \$822 billion in profits. Fragmented accountability for transparent public messages has created seriously incomplete knowledge that diverts public attention from prevention of disease.

## CONCLUSION

Fifty years of working with molecular medicine let me see the paradox of a continuing need to treat preventable food-related disorders (1). I saw news releases and repeated marketing messages from pharmaceutical firms and food marketers continually inform the public of benefits in purchasing treatments, procedures, and products with no information on how to prevent the need to purchase them.

During that time, the National Library of Medicine has grown to hold more than 22 million scientific reports retrievable by electronic search. Although they no longer are “news,” they have much information not used in messages from pharmaceutical firms and food marketers. Since Hippocrates, food has been a tool for primary prevention. However, lowering intakes of omega-6 nutrients does not fit business plans of large food companies already profiting from the sale of food oils and foods rich in omega-6. Marketers have little interest in advising the public how to prevent the flow of funds for those highly profitable products.

Quantitative competitive metabolism of omega-3 and omega-6 nutrients was shown nearly 40 years ago (16), confirmed 20 years ago (17), and neglected in today’s messages. The need for omega-6 nutrients was shown to be less than 0.5% of daily food energy (1), an old fact neglected by today’s messages that advise eating 10% or more.

Nevertheless, high-oleic (low omega-6) forms of canola, soybean, and sunflower oil could be eaten with foods containing more omega-3 nutrients to prevent the current unintended imbalances in tissue HUFA and unintended health-related financial losses. Continued insertion of high amounts of omega-6 into US foods reflects continued neglect of important old nutrition facts by the food industry.

During the past 30 years, I often told people how balanced intakes of omega-3 and omega-6 nutrients can decrease the severity of many preventable chronic diseases (1, 2, 10, 12, 13, 17, 20, 23–25). I found out that such information did not fit business plans of pharmaceutical companies making and marketing new drugs to treat signs and symptoms of those diseases. Treatments are also important in business plans of doctors, nurses, hospitals, insurers, actuaries, researchers, and many health care professionals, including wellness counselors. All of them gain from a focus on treatments. They have little financial

incentive to discuss old nutrition facts that could prevent the flow of funds for treatments.

As a result, I am discouraged with trying to divert their attention to prevention. My hope now is that the employees and employers who currently pay for treatment of preventable diseases will gain a better quality of life as they put attention on prevention. The tools to do that are ready to use, and the financial savings are large.

## REFERENCES

1. Lands B. A critique of paradoxes in current advice on dietary lipids. *Prog Lipid Res.* 2008;47:77–106.
2. Lands B. Planning primary prevention of coronary disease. *Curr Atheroscler Rep.* 2009;11(4):272–80.
3. Indiana University-Purdue University, Fort Wayne (IPFW) Study, 2006; [http://www.redorbit.com/news/health/788667/new\\_ipfw\\_study\\_finds\\_87\\_of\\_health\\_claims\\_lifestyle\\_related/](http://www.redorbit.com/news/health/788667/new_ipfw_study_finds_87_of_health_claims_lifestyle_related/). Accessed 30 Sept 2012
4. Health Care Statistics. [http://preventdisease.com/worksite\\_wellness/health\\_stats.shtml](http://preventdisease.com/worksite_wellness/health_stats.shtml). Accessed 30 Sept 2011.
5. Mayo Clinic report. <http://www.mayoclinic.com/health/metabolic%20syndrome/DS00522/DSECTION=symptoms>. Accessed 30 Sept 2012.
6. Ross R, Glomset J, Harker L. Response to injury and atherogenesis. *Am J Pathol.* 1977;86(3):675–84.
7. Ross R. Atherosclerosis—an inflammatory disease. *N Engl J Med.* 1999;340(2):115–26. Review. PubMed PMID: 9887164.
8. Ross R. Atherosclerosis is an inflammatory disease. *Am Heart J.* 1999;138(5 Pt 2):S419–20. Review. PubMed PMID: 10539839.
9. Verschuren WM, Jacobs DR, Bloemberg BP, Kromhout D, Menotti A, Aravanis C, et al. Serum total cholesterol and long-term coronary heart disease mortality in different cultures. Twenty-five-year follow-up of the seven countries study. *JAMA.* 1995;274(2):131–6.
10. Lands WEM, Hamazaki T, Yamazaki K, Okuyama H, Sakai K, Goto Y, et al. Changing dietary patterns. *Am J Clin Nutr.* 1990;51:991–3.
11. Ogushi Y, Hamazaki T, Kirihara Y. Blood cholesterol as a good marker of health in Japan. *World Rev. Nutr. Diet.* 2009; 100: 63–70.
12. Lands WEM, Pitt B, Culp BR. Recent concepts on platelet function and dietary lipids in coronary thrombosis, vasospasm and angina. *Herz.* 1980;5:34–41.
13. Lands WEM. Fish and human health. Orlando, FL: Academic; 1986.
14. Omega 3-6 Balance Scores. <http://www.fastlearner.org/Omega3-6Balance.htm>. Accessed 30 Sept 2011.
15. Lands B, Lamoreaux E. Describing essential fatty acid balance as 3 - 6 differences rather than 3/6 ratios. *Nutrition & Metabolism* 2012, 9: 46-54.
16. Mohrhauer H, Holman RT. Effect of linolenic acid upon the metabolism of linoleic acid. *J Nutr.* 1963;81:67–74.
17. Lands WEM, Libelt B, Morris A, Kramer NC, Prewitt TE, Bowen P, et al. Maintenance of lower proportions of n-6 eicosanoid precursors in phospholipids of human plasma in response to added dietary n-3 fatty acids. *Biochim Biophys Acta.* 1992;1180:147–62.
18. Wada M, DeLong CJ, Hong YH, Rieke CJ, Song I, Sidhu RS, et al. Enzymes and receptors of prostaglandin pathways with arachidonic acid-derived versus eicosapentaenoic acid-derived substrates and products. *J Biol Chem.* 2007;282(31):22254–66.
19. Lands B. Measuring blood fatty acids as a surrogate indicator for coronary heart disease. *World Rev Nutr Diet.* 2009;100:22–34.
20. Lands WE. Diets could prevent many diseases. *Lipids.* 2003;38(4):317–21.
21. Siscovick DS, Raghunathan TE, King I, Weinmann S, Wicklund KG, Albright J, et al. Dietary intake and cell membrane levels of long-chain n-3 polyunsaturated fatty acids and the risk of primary cardiac arrest. *JAMA.* 1995;274(17):1363–7.
22. Albert CM, Campos H, Stampfer MJ, Ridker PM, Manson JE, Willett WC, et al. Blood levels of long-chain n-3 fatty acids and the risk of sudden death. *N Engl J Med.* 2002;346(15):1113–8.
23. Hibbeln JR, Nieminen LR, Blasbalg TL, Riggs JA, Lands WE. Healthy intakes of n-3 and n-6 fatty acids: estimations considering worldwide diversity. *Am J Clin Nutr.* 2006;83(6 Suppl):1483S–93.

24. Lands B. Prevent the cause, not just the symptoms. *Prostaglandins Other Lipid Mediat.* 2011;96(1–4):90–3. PubMed PMID: 21827870.
25. Lands B. False profits and silent partners in health care. *Nutr Health.* 2009;20(2):79–89.
26. Loepfke R, Taitel M, Richling D, Parry T, Kessler RC, Hymel P, et al. Health and productivity as a business strategy. *J Occup Environ Med.* 2007;49(7):712–21.
27. De Gruttola VG, Clax P, DeMets DL, Downing GJ, Ellenberg SS, Friedman L, et al. Considerations in the evaluation of surrogate endpoints in clinical trials: summary of a National Institutes of Health Workshop. *Control Clin Trials.* 2001;22:485–502.
28. Kastelein JJ, Akdim F, Stroes ES, Zwiderman AH, Bots ML, Stalenhoef AF, et al. ENHANCE Investigators. Simvastatin with or without ezetimibe in familial hypercholesterolemia. *N Engl J Med.* 2008;358:1431–43.
29. Carey J. Do cholesterol drugs do any good? Available at [http://www.businessweek.com/magazine/content/08\\_04/b4068052092994.htm](http://www.businessweek.com/magazine/content/08_04/b4068052092994.htm). Accessed 30 Sept 2011.
30. Carey J. Heart disease: not about cholesterol? Available at [http://www.businessweek.com/bwdaily/dnflash/content/apr2008/db20080414\\_688906.htm](http://www.businessweek.com/bwdaily/dnflash/content/apr2008/db20080414_688906.htm). Accessed 30 Sept 2011.
31. Couzin J. Cholesterol veers off script. *Science.* 2008;322:220–3.
32. Steinberg D. Thematic review series: the pathogenesis of atherosclerosis. An interpretive history of the cholesterol controversy. Part IV: The 1984 coronary primary prevention trial ends it—almost. *J Lipid Res.* 2006;47(1):1–14.
33. Consensus conference. Lowering blood cholesterol to prevent heart disease. *JAMA.* 1985;253(14):2080–6.
34. Weinstein MC, Coxson PG, Williams LW, Pass TM, Stason WB, Goldman L. Forecasting coronary heart disease incidence, mortality, and cost: the Coronary Heart Disease Policy Model. *Am J Public Health.* 1987;77(11):1417–26.
35. AHA Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2011 update. *Circulation*; 2011. Downloaded from <http://circ.ahajournals.org/> on 3 Sept 2011.
36. ACCORD Study Group, Gerstein HC, Miller ME, Genuth S, Ismail-Beigi F, Buse JB, Goff Jr DC, et al. Long-term effects of intensive glucose lowering on cardiovascular outcomes. *N Engl J Med.* 2011;364(9):818–28.
37. Hoogwerf BJ. Action to Control Cardiovascular Risk in Diabetes Study Group: does intensive therapy of type 2 diabetes help or harm? Seeking accord on ACCORD. *Cleve Clin J Med.* 2008;75:729–37.
38. NICE-SUGAR Study Investigators, Finfer S, Chittock DR, Su SY, Blair D, Foster D, Dhingra V, et al. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med.* 2009;360(13):1283–97. PubMed PMID: 19318384.
39. Ridker PM, Danielson E, Fonseca FA, Genest J, Gotto Jr AM, Kastelein JJ, et al. JUPITER Study Group: rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. *N Engl J Med.* 2008;359:2195–207.
40. Blasbalg TL, Hibbeln JR, Ramsden CE, Majchrzak SF, Rawlings RR. Changes in consumption of omega-3 and omega-6 fatty acids in the United States during the 20th century. *Am J Clin Nutr.* 2011;93(5):950–62.
41. Nestle M. *Food politics: how the food industry influences nutrition and health.* Berkeley: University of California Press; 2007.
42. Imhoff D. *Foodfight: the citizen's guide to a food and farm bill.* Healdsburg, CA: Watershed Media; 2007.
43. Simon M. *Appetite for profit: how the food industry undermines our health and how to fight back.* New York: Nation Books; 2006.



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