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Translating Nutrition Science into Policy as Witness and Actor

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nutrition, science, policy, malnutrition, hunger

Abstract

The sustained effort to witness and participate in the targeted translation of nutritional science and policy forms the structure of this narrative. The memoir starts with an early career-directing experience with nutrition and cholera and proceeds with a long thread of interest in folic acid malabsorption as one of the determinants of nutritional anemia in Asia and the tropics. The thread continues with the relationship of folate and associated vitamins to brain function and aging as a prototype of the study of the interface of aging biology and nutritional biology. My current interest in world hunger and famine and their impact on human security may circle back to studies of the great Bengal famine and the first Bangladesh survey of malnutrition.

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PREFATORY NOTE

This article strives to address the question of how to strike a balance between the concentration needs of the laboratory bench and bedside research and participation in the marketplace of ideas and policy. For most of my career—nearly four decades—I have been privileged to live in an era in which nutrition emerged from the shadows and demanded recognition in the arena of ideas and policies relating to health. If the first half of the twentieth century could be referred to as the era of discovery in nutrition, in which the description and characterization of all the vitamins led to the seminal contribution of nutritional and enzyme/coenzyme biochemists to the creation of the biochemistry enterprise, then the second half of the century might be seen as the era of translation, not only of medical science to policy, but also of diet and nutrition discoveries in nutrition science to infectious disease, heart disease, cancer, and fetal development and aging, among other health concerns. As a peripatetic physician-scientist finding sea legs in the 1960s as far away as Bangladesh, a country rife with malnutrition, cholera, and diarrheal disease, it was not hard to be drawn quickly to the idea that the observations we were making in

the field, in the laboratory, and at the bedside needed application in real time. More specifically, the experience of tending to the patient on an ingenious volume-collecting cholera cot (which titrated life-saving volume replacement) cried out for understanding of the pathogenesis and etiology of this disease as well as for action to limit its impact and lethality. We were aware that in the 1960s, during the very early years of the Southeast Asia Treaty Organization (SEATO)/NIH Cholera Research Laboratory (now the ICDDR/B), our activities smacked somewhat of medical or scientific colonialism. However, it would have been hard to collect information dispassionately about what was a life-threatening disease of dehydration for an infant with cholera or a sight-threatening degree of xerophthalmia in a child with vitamin A deficiency. So we challenged ourselves, often in some frustration, to go beyond documentation and individual treatment to a more ambitious goal of comprehension and prevention. That was my baptism—and I use the term advisedly—and exposure to important nutritional problems and their relation to infection and mortality, and it would have a profound effect on my subsequent orientation in both science and career. This was all in keeping with the emerging zeitgeist that was represented at the time by the “elucidation” of the relationship between diet and heart disease, or the diet/heart hypothesis. This narrative describes some of the places, institutions, and organizations that provided opportunities to me to pursue both science and its translation into policy (**Table 1**).

MALNUTRITION, CHOLERA, AND MALABSORPTION

For those who are interested in how I decided to follow the nutrition science pathway, I begin this narrative in Bangladesh, then East Pakistan, in 1961. It was there that my training in medicine, my hubris about the contribution I could make in the developing world early in the John F. Kennedy era, and my previous interest and experience in science were

Table 1 Glossary of organizations cited

SEATO/NIH CRL	Southeast Asia Treaty Organization/National Institutes of Health, USA, Cholera Research Laboratory
ICDDR/B	International Cholera & Diarrheal Disease Research Laboratory/Bangladesh
ICNND	Interdepartmental Committee on Nutrition for National Defense, USA
USAID	United States Agency for International Development
USDA	United States Department of Agriculture
TML	Thorndike Memorial Laboratory, Boston City Hospital, Harvard Medical Service
CIP/FNB/NAS	Committee on International Programs/Food & Nutrition Board/National Academy of Sciences
SINF/NRC/NAS	Subcommittee on Interactions of Nutrition & Infection/National Research Council/National Academy of Sciences
FNB/NAS	Food & Nutrition Board/National Academy of Sciences
OTC/VMH/FDA	Over-the-Counter/Panel on Vitamins, Minerals & Hematinics/Food & Drug Administration, USA
FAS/FAC/FDA	Folic Acid Subcommittee of the Food Advisory Committee/Food & Drug Administration, USA
UNHCR	United Nations High Commission for Refugees

thrown together by some happenstance into a stewpot called nutrition. The happenstance was that I was assigned to an Interdepartmental Committee on Nutrition for National Defense (ICNND) project (see **Table 1**). I requested an assignment at the National Institutes of Health (NIH) in the developing world rather than the traditional placement for those bent on a career in academic medicine, in a lab studying enzymology or clinical pathogenesis. I assumed my appointment at the NIH in Bethesda in the summer of 1961 after my residency training in internal medicine at Massachusetts General Hospital in Boston, which followed my medical degree at Harvard Medical School. Previous nutritional stimuli included undergraduate research at the University of Wisconsin (a nutritional biochemistry powerhouse) on the urea cycle.

A medical school elective under the tutelage of one of my professors at Harvard, Dr. Jean Mayer, stimulated an interest in eating behavior. My exposure to the growing interest in nutrition and heart disease was advanced by another teacher, Dr. Paul Dudley White, who was also President Dwight Eisenhower's cardiologist. Motivated by the desire to use my medical training to some benefit in the developing world, I requested an international assignment at the NIH even before there was an International Fellow's program. I was offered a posting to a research project on nutritional anemia in

Ghana, which would be carried out in collaboration with my former professor, Jean Mayer. However, when that fell through because of political turmoil in West Africa, I accepted the alternate (then considered a temporary) assignment as clinical advisor to the East Pakistan Nutrition Survey.

This survey would be carried out with the methodology of the ICNND developed in several other countries in the 1950s (a setting that had represented the field experience for several authors of previous *Annual Review of Nutrition* prefatory articles). Off to East Pakistan I went, after I was fortified with two 2-week training experiences in ICNND survey methodology on the Blackfoot Indian Reservation in the United States and in Burma not long before that country closed itself to the world with its new name, Myanmar. Given the "watch one, do one, teach one" conceit of clinical medicine training, I was undaunted by the fact that I was going to a distant country and culture and would train their doctors in carrying out clinical nutrition examinations as part of one of the largest nutrition surveys ever performed. It was soon clear that this would be no short-term assignment in East Pakistan. I would have to change the agreement between the U.S. government and the government of Pakistan in order to make it possible to recruit the doctors and other staff for the survey. It also became evident that I would be needed not just as an instructor, but also as a

field leader if the survey were to be carried out successfully.

As fortune would have it, other contemporary events would contribute to the solidification of the path to both nutrition science and policy quite early in my career. Just after my arrival in Dhaka, two other critical contingents of future collaborators arrived. One was the first small group of clinical scientists to staff the just-developing SEATO Cholera Research Laboratory from the NIH and, almost simultaneously, the second of all Peace Corps contingents (the first having gone to West Africa). Not only was another pair of hands needed to treat and study cholera patients at the Cholera Research Laboratory (CRL), which soon became a dedicated cholera ward with aspirations for research as well as greatly needed life-saving care, but someone was also needed with emerging expertise in local nutritional realities to help examine the relationship between cholera infection and malnutrition. This nexus soon led to great interest in the nutritional risk factors for cholera infection as well as the underlying substrate of intestinal pathology, later called tropical enteritis or enteropathy. The dramatically altered physiology of the cholera patient demanded an understanding of intestinal absorptive and malabsorptive functions as well as fundamental issues of transport of water, salt, and other substances by the intestine. Each of those challenges would lead to subsequent career and research choices. Before elaborating on those choices, I briefly report that prior to assessing and addressing the need or the means to translate emerging knowledge from our survey about the deep problem of malnutrition into some kind of program or policy intervention, I was besieged by well-meaning Peace Corps volunteers who needed changes in their field assignments. In addition, with the Academy of Rural Agriculture, we designed programs in applied nutrition in villages to counteract some of the ravages of prevalent malnutrition, especially in children, that were becoming evident from our nutrition survey in the field.

Returning to the topics of malnutrition, enteritis, and cholera, I briefly summarize a pa-

per entitled "Tropical enteritis: nutritional consequences and connections with the riddle of cholera" (4), which was published as part of a symposium on nutrition and infection in the *Journal of Nutrition* in 2003. With respect to enteritis, or more specifically, tropical enteritis, it was impossible to observe the large prevalence of malnutrition in the countryside without noting the association with highly prevalent diarrheal disease. It was clear to me that if I were to study and understand malnutrition in this kind of setting of poor sanitation and an unclear water supply, I would have to be able to understand more of the role of diarrhea, infection, and malabsorption of nutrients. Just at that time, with the availability of the intestinal capsule biopsy, Dr. Robert Gordon, who was then on loan from the NIH, enlisted collaboration with the U.S. Army Laboratory in Thailand, which was performing biopsies on patients recently recovered from cholera. They found a lesion (demonstrated in **Figure 1**) that was first thought to be the intestinal pathologic expression of the previous cholera infection. It soon became evident that this lesion was not specific to recovered cholera patients, but rather was found in virtually everyone else in that setting, thus initiating the concept of tropical enteritis, a chronic inflammatory condition of the small intestine leading to diarrheal susceptibility as well as to moderate degrees of malabsorption. This finding provided my impetus to study intestinal physiology, not only to understand malabsorption and malnutrition but also to comprehend the dramatic pathogenesis of cholera.

Two stories then merged that would lead me back to the NIH lab in Bethesda followed by a Fellowship in Liver Disease and Nutrition at the Thorndike Laboratory at Harvard Medical School. One story was the exploration of the concept that diarrhea in cholera was due to a poisoning of the sodium pump in the intestinal enterocyte by inhibiting the recently described energy-yielding Na-K-ATPase. At the NIH, we were able to demonstrate that such an NaK-ATPase existed in the intestine (5), as had been previously described in frog skin and

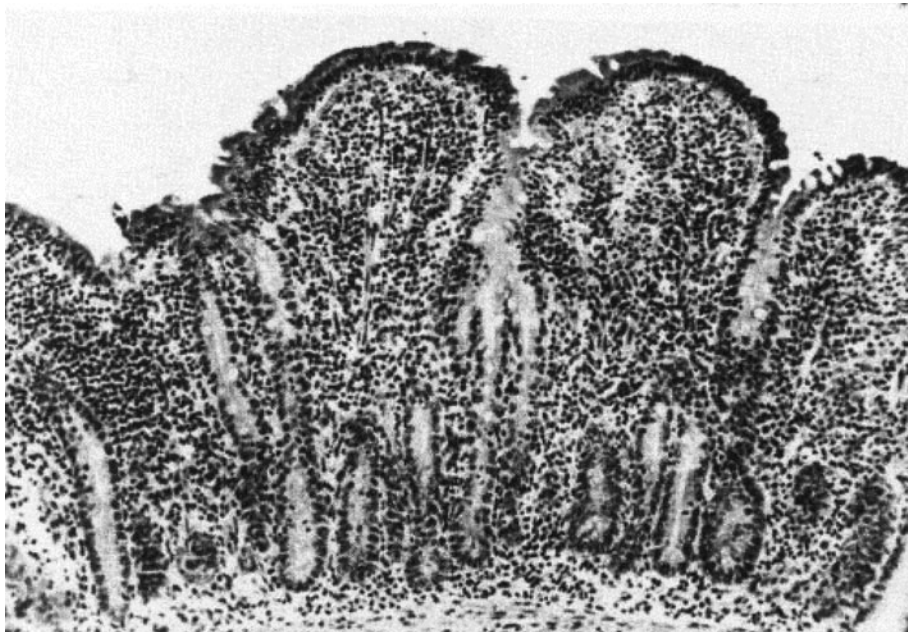


Figure 1

Tropical enteritis (4).

neural tissue, and that crude extracts from vibrio cholera cultures were inhibitory. (It later turned out that this inhibition was not specific for the sodium potassium ATPase.) However, my young colleague at the Thorndike Laboratory, Norbert Hirschhorn, later went to Dhaka intending to perform intestinal biopsies in active cholera patients to measure ATPase and its possible suppression (2). In the process of that work, Hirschhorn and colleagues, perhaps serendipitously, discovered that not only was the sodium pump intact in actively purging cholera patients who were being perfused with a glucose and sodium solution, but also that their hydration improved during the perfusion. This led them to the discovery of oral rehydration solution and the saving of millions of lives to this day by globally adopted oral rehydration therapy. Perhaps I may claim a small asterisk in this history, but the disproval of my hypothesis on the pathogenesis of cholera would mark an end to that pathway of studying intestinal salt and water transport and shift attention to other forms of malabsorption.

During those years, another quest was opened to understand vitamin absorption and malabsorption with a focus on folate, a focus that remains even at present. Vitamin A malnutrition would provide another lesson in politics and policy.

A LESSON IN VITAMIN A, POLITICS, AND POLICY

Before I left the NIH for further training in Boston, I had additional education in the vicissitudes of policy in international health. I share with you one anecdote relating to the survey in Bangladesh and a concurrent one in northeast Brazil. Part of my desk job as deputy to Arnie Schaefer (in the ICNND program at the NIH after I returned from Bangladesh) was to be the referral person at the NIH for questions on international nutrition emanating from the State Department. This was a heavy responsibility for a 27-year-old physician whose total international experience was in ICNND surveys in Burma and East Pakistan. I fielded

questions such as, “Is there really anything to the suggestion that malnutrition in childhood results in mental retardation?” (a topic that I offered to take up at another time) and “How bad an idea is it that some South Asian countries are exporting all their high-protein foods, such as fish and shrimp?” But the one I remember best, which connects to northeast Brazil and the ICNND survey there (3) from March to May 1963, was the question of whether there was any scientific basis for the allegation that skim-milk powder distributed in the poorest part of Brazil under the auspices of the U.S. government and by the U.S. Agency for International Development (USAID) was causing Brazilian children to go blind. The Bahia population was strongly leftist and had good political reasons for wanting to discredit U.S. aid in any form, including surplus skim-milk powder.

My opinion and response was based on experience in East Pakistan and with the northeast Brazil ICNND surveys, in addition to other research. My reply was to acknowledge some of the political motivation for the allegations but to focus on the possible scientific issues. Those included the observations that there was a serious vitamin A deficiency and protein malnutrition in northeast Brazil, as we had observed in East Pakistan, with high numbers of children at risk for keratomalacia and blindness. Part of the problem is the delivery of vitamin A to the peripheral tissues in the face of severely depleted stores and very low plasma levels of retinol-binding protein along with other plasma proteins. There have been reports that vitamin A deficiency becomes the rate-limiting nutrient deficiency and xerophthalmia worsens in the short term when protein malnutrition is treated with a good source of protein—as is skim-milk powder—that totally lacks fat-soluble vitamins. There was at least a theoretical basis for the allegation that feeding these malnourished children skim-milk powder without any sources of vitamin A could actually make their eye condition worse. In the interest of full disclosure, I must confess that my observations in the northeast Brazil case were also related to a plea that we had been making based on the East Pakistan

survey data showing that 50,000 children there were at risk of blindness because of vitamin A deficiency. We were requesting that the skim-milk powder that was distributed there be fortified with vitamin A and possibly D. This was not the responsibility, I was told, of the U.S. Department of Agriculture (USDA), and USAID was not convinced that they needed to do it either. Although it is sad in some ways to say it, the northeast Brazil case probably persuaded USAID finally to fortify skim-milk powder with vitamin A in the face of the potential political fallout from allegations by the northeast Brazil communists at that critical time in the Cold War.

ENTERITIS, TROPICAL SPRUE, FOLATE DEFICIENCY, AND MALABSORPTION

If the logic that chronic intestinal inflammation in the face of persistent environmental exposure contributes along with poverty and dietary insufficiency to malabsorption and malnutrition (a concept that is still underestimated to this day), then the tip of the pyramid rising above this huge base of enteritis and chronic malabsorption would be tropical sprue with severe nutritional anemia and prominent vitamin B12 deficiency. Tropical sprue was prevalent in countries in Asia, the Caribbean, and the Middle East, and I soon discovered that we knew very little about folate absorption and malabsorption that might help to explain the profound nutritional anemia seen in this setting. In the laboratory of Charles Davidson at the Thorndike lab at Harvard Medical School, where I went after the NIH to pursue further training in GI, liver, and nutrition, I was fortunate to also work with William B. Castle, who had discovered the intrinsic factor for vitamin B12 absorption (among many other important contributions that should have won him the Nobel Prize). I had the opportunity to review his experience with nutritional anemias in the Caribbean and their treatment with liver extract. I was quite sure that if we could understand the profound

malabsorption leading to anemia in patients with tropical sprue in their tropical setting, then we would also be able to better understand the importance of malabsorption and enteritis in the problem of malnutrition in the developing world.

With the blessing of both Davidson and Castle, I then embarked on an effort to understand folate malabsorption, but first it would be necessary to understand folate absorption. Folate was then quite a young vitamin, and any studies that were available at that time of absorption of this vitamin had used the synthetic folic acid, whose synthesis had been accomplished in the late 1940s. If we were to understand malabsorption of folate in tropical sprue, we would need to have a much better understanding of the absorption of folate from food, not the synthetic and unreduced vitamin. That led to a quest lasting a couple of decades, in which we would need to isolate natural or polyglutamyl folates from yeast and other foods for intestinal absorption measures and synthesize polyglutamyl folates during a sabbatical at the Weizman Institute in Israel under the direction of a commanding scientist who would become president of that country, Ephraim Katzir. Utilization of both radioactive and stable isotope techniques for intestinal absorption would be required along the way. It was necessary to understand how this negatively charged polyglutamyl folate was digested in the intestine and subsequently how the monoglutamyl folate was transported across the bloodstream and by which kinds of physiologic carriers. The intestinal hydrolase was then purified in birds, rodents, and humans, and the unique intestinal folate transporter that was described (7) was cloned as reported in 2007 (8). It was satisfying to see that some of the physiologic descriptors of the transporter could be perfectly reproduced by the pure expressed protein in frog oocytes.

By this time, my interest in gastrointestinal physiology had channeled me to the field of gastroenterology, where I spent 15 years at the University of Chicago. Also, my interest in folate deficiency and other vitamin and nutrient deficiencies in gastrointestinal

disease would lead to a number of assignments that would have some influence on nutritional policy with respect to vitamins, and also to a focus on these deficiencies as they were important in understanding the functional declines in aging.

In describing this winding and reciprocating path between nutrition science and policy, I refer to some of the organizations and committees that allowed me to participate in policy discussions (**Table 1**) and, at times, to bring my own research findings to the table, along with the observations of others.

My exposure to the problems of malnutrition in Bangladesh and to cholera and diarrheal disease stimulated a dual commitment to the pursuit of nutrition science and to its application. The opportunity to participate in these important advisory committees in my own government and beyond provided an effective vehicle for the expression of that translation. However, I emphasize that the interaction of nutrition science and policy is bidirectional and reciprocal, just as is the relationship between the laboratory bench and bedside in academic medicine, with each end of the shuttle challenging the other with new ideas. As a result of these committee and consultant experiences, my lab and my colleagues were never at a loss for problems of real relevance, even if we fell short on the hoped-for definitive answers.

FOCUS ON FOLATE

As noted above, the challenge of understanding tropical malabsorption led to a strong focus upon folic acid deficiency as one visible manifestation of tropical malabsorption and as potentially a template for a greater understanding as a model of vitamin and nutritional deficiency in developing countries or locations of high environmental contamination and infection. Work in this area identified me with some other concerns of the Committee on International Nutrition Programs, chaired by Dr. Nevin Scrimshaw, which led to my subsequent participation as chair of the Subcommittee on Nutrition and Infections and as Dr. Scrimshaw's

successor as chair of the U.S.-Japan Malnutrition Panel, whose concern was malnutrition in developing countries. The concerns with intestinal absorption and bioavailability of different forms of folic acid or different vitamins of folate led to studies to fill some of the gaps in our knowledge about vitamin absorption more generally along with my growing interest in gastrointestinal disease. As head of a large and active Gastroenterology Department at the University of Chicago, I was confronted with the issue of what my former mentor William B. Castle liked to call “conditioned nutritional deficiency,” i.e., deficiency conditioned by other diseases or abnormalities, such as gastric intrinsic-factor deficiency in the case of pernicious anemia. In the clinical setting, my colleagues and I saw malabsorption of fat-soluble vitamins as well as water-soluble vitamins in gastrointestinal conditions such as celiac and Crohn’s disease. We made the first reports of vitamin D deficiency in Crohn’s and interference with folate absorption by sulfasalazine, which was used widely in the treatment of inflammatory bowel disease.

Soon the commissioner of the Food and Drug Administration invited me to chair a Panel on Over The Counter (OTC) Vitamins, Minerals and Hematinics to provide a rational and science-based set of recommendations for marketing and labeling vitamins and minerals sold over the counter. In the process, we might contribute to the goal of disentangling the battles among Congress, which through the Proxmire amendment was preventing limits on vitamins for safety reasons; the vitamin supplement industry, which was claiming unfettered commercial speech; and the Food and Drug Administration (FDA), which had responsibilities for some oversight of vitamin marketing, both under the terms of its food supplement regulations or lack thereof in the Food Division and the OTC program of the Drug Division.

Here was a case where, in retrospect, hubris at the thought that—with a blue ribbon committee—I might be able to make a contribution to “strike a blow for science” overcame my better judgment that I should turn and run.

Even after accepting the assignment, I should have taken the message when upon entering a health food store one day to purchase some sourdough starter for bread making, I saw a banner that proclaimed, “God gave us vitamins and the FDA is taking them away.” That was before we had even started to meet with the panel and 20 years before we would have a presidential administration in the United States that might have endorsed the sensibilities of that sign. But we were assured by the commissioner that the FDA intended to stay the course in spite of anticipated opposition, and two and a half years later, in 1980, we delivered a report that some have considered to be the best and most scientific compendium on vitamins to that date (1).

The report offered fairly modest recommendations about fair labeling so that consumers could make informed choices, but even before it appeared in the Federal Register, the write-in campaign that was initiated to denounce the report so impressed Congress that they sent warnings to the FDA commissioner (who by that time was no longer the one who appointed us and promised to stay the course). The commissioner decided to file the report without putting out any of its recommendations for public comment. I dwell on this story not to reinforce the cynical conclusion that no good deed goes unpunished, but rather to point out that the best of motivations to contribute to science-based policy can result in disappointment and frustration. However, the lessons learned about vitamins and minerals and their history, along with the science underlying the recommendations for requirements and safety, provided an intensive course of learning for my colleagues on that panel, one that could never have been replaced by any other study method. (Perhaps as a consolation prize, 25 years later I was awarded the FDA Commissioner’s Medal for Service.)

HUMAN NUTRITION AND AGING

The report of the Panel on OTC Vitamins and Minerals, along with work in our own

laboratory on vitamin absorption and even the effect of aging, led to publications that placed me in contention for leadership of the newly emerging Human Nutrition Research Center on Aging (HNRCA) at Tufts University, which was established by Congress under USDA auspices. My acceptance of that appointment in 1986 represented a clear consolidation of my research and scientific interest in nutrition as well as a decline in my involvement as a gastroenterologist with a strong nutrition research orientation. The challenge to lead a research institute that had a physical plant, the institutional commitment by Tufts University with its distinguished nutrition scientist President Jean Mayer, and the core of gifted scientists who had been recruited by the first director, Dr. Hamish Munro, all conspired to challenge a new phase in my own scientific and academic path.

The challenge to better understand the physiological, clinical, and biochemical interface of aging biology and nutritional biology may have focused the most productive phase of my career, scientifically and academically as well as with respect to policy. The demographics that described a profound change in the distribution of elderly among the population, not only in the United States and other industrialized nations but also throughout the world, provided and still provides a strong impetus for understanding the way in which nutritional factors contribute to age-related functional change and potentially for understanding the way in which nutritional interventions, along with physical activity, might influence the trajectory of important functional declines. The declines include cognitive loss, loss of lean body and muscle mass and strength, changes in cardiovascular health and function, diminished immune function leading to higher risk of infection and cancer, bone loss and fracture, and a loss of vision and hearing. The litany could go on, but the message was and is clear enough to guide the agenda of my colleagues at the HNRCA and their explicit contributions to practical advice for the public, which might modify the trajectory of functional decline with

aging and the critical loss of quality of life, not to mention the demand for costly health care services.

These challenges led my own laboratory and my long-time colleague Dr. Jacob Selhub to intensify our efforts to better understand vitamin status, especially folate, in aging humans and to intensify our efforts to describe the transport systems for folate across different membranes, including intestine, kidney, and brain. In the process of seeking a more functional indicator of folate status than the standard method of folate content of plasma or red blood cells, we were drawn to the importance of measuring homocysteine in blood as an indicator of the status of the three vitamins: folate, vitamin B12, and vitamin B6, that contributed to its metabolism.

This led us and our many talented collaborators on another decade-long journey to characterize not only the folate status as it changed with age, but also the importance of B vitamins and their marker or mediator, homocysteine, in the risk of cardiovascular disease and especially of cerebrovascular disease. The pendulum has swung back and forth from observational studies and intervention studies underlying the uncertainty of recommending the policy change of homocysteine measurement and treatment or lowering with B vitamins to achieve cardiovascular and cerebrovascular disease prevention. Suffice it to say that the diet-heart hypothesis can no longer be limited to an investigation of dietary fat and sources of dietary cholesterol, as important as those may be. This scientific path has led us to expand our understanding of the vitamin control and determinants of homocysteine metabolism and to a sharper focus on homocysteine as a marker and likely participant in the progress of cerebrovascular disease, macro- and micro, and the increasingly prevalent age-related cognitive decline and dementia. Here, as with other aspects of the homocysteine story, the literature will speak for itself, but the promise of elucidating the relationship between folate and/or vitamin B12 and brain function, cognition, depression, and brain volume or atrophy provides one of the optimistic

considerations for how improved B vitamin nutrition, along with fatty acids and even now vitamin D, might affect the trajectory of cognitive function in the elderly.

The concern with the surprisingly high prevalence of folate deficiency and hyperhomocysteinemia in the elderly was an unstated consideration in the decision in 1996 by the FDA to mandate the folic acid fortification of flour, which was explicitly directed at the control of neural tube defect births. I served on the Folic Acid Advisory Committee to the FDA, and to this day I stand behind my support for the decision to add folate to flour at the responsible dose of 140 µg/100 g of flour. This dose is twice the replacement for folate lost through milling, and it has had a profound effect on the diminution in neural tube defect births in the United States and Canada. I also stand behind my support for the well-calculated and well-modeled approach to the dose of fortification that was undertaken by the FDA staff, especially Dr. Beth Yetley. I continue to be concerned, as I have expressed in editorials, that we not expose the U.S. population to an excessive amount of folic acid and thus endanger certain populations, including elders with low vitamin B12 status and those prone to colorectal cancer, possibly even as we improve on the risk of neural tube defects.

Once again, as I had learned earlier, the passions surrounding certain recommendations in the vitamin sphere tend to overtake the knowledge that we have accumulated about folic acid nutrition metabolism, bioavailability, and status, especially regarding the differences among vitamins, such as the synthetic folic acid and the main circulating form methyltetrahydrofolate. I hope I have been able to contribute to this debate, which is really about representing all the science fairly to arrive at policy on safe and effective dose and exposure to the large and varied population.

POLICY AND PUBLICATIONS

Additional and important vehicles for the translation of nutrition science in my experience

have been publications. As editor of *Nutrition Reviews* for 15 years (where I still serve as associate editor), I tried to make explicit the functions of that journal in policy discussions by adding a feature with a descriptive logo: science ↔ policy. Founding of the journal *Nutrition in Clinical Care* was a part of my continuing effort to provide medical practitioners with information in the nutrition sphere, which they may use to enhance the quality of their practice. Also, for some 15 years I have served as scientific editor of the *Tufts Health and Nutrition Letter* as part of an effort to communicate to the public what may be the most important advances in nutrition science. Most recently, I accepted the task of editor-in-chief of the *Food and Nutrition Bulletin*, which is the most widely used nutrition journal in the developing world.

FAMINE, NUTRITION, AND HUMAN SECURITY

I am deeply grateful to have my name associated with a professorship at Tufts in Nutrition and Human Security. This professorship, held by Peter Walker, the director of the Feinstein International Center, refers to the essential fusion of human nutrition with human security and recognizes the long commitment of Tufts University to the concept of humanitarian rights to freedom from hunger, starvation, and disease. That history extends from Jean Mayer as champion and voice for the international movement for freedom from hunger to the establishment of the Famine Center, now the Feinstein International Center.

I began this narrative with a reference to Bangladesh, a country with its own relatively recent dramatic history of war and floods as well as the great Bengal famine of 1943. Although hunger, starvation, and famine have existed throughout all of history (even the Joseph story in the Bible narrates the concept of planning for lean years three millennia ago), the notion of human rights to food as an international imperative is really a twentieth-century concept.

The recognition that human security and human nutrition are inextricably linked is even more recent. Jean Mayer was one of the pioneers in advancing the concept that the use of starvation as a weapon is a violation of human rights and a crime against humanity. In 1969, Dr. Mayer led a mission to Biafra, where the consequences of civil war were ravaged food supplies and famine, and he returned and reported to the White House and Congress on the need for action. During that same time, Nevin Scrimshaw (a revered mentor and now a colleague at Tufts) and others familiar with Bangladesh were making a case against starvation and human rights violations in the Bangladesh war for independence. As director for the White Conference on Nutrition in 1969, Mayer emphasized the need to incorporate hunger and famine concerns into the nutrition agenda. As Tufts President a few years later, he was instrumental, along with Stanley Gershoff, in bringing social and political scientists to Tufts in the developing School of Nutrition.

That fusion of outstanding nutrition science and policy embracing cell and molecular biology all the way to famine studies and economic development matured in the 1990s, while I was privileged to be dean of what would soon become the Friedman School. Some of my colleagues were instrumental in redefining hunger in more quantitative terms so that the case for basing international declarations for the right to food and dignity could be made in solid scientific terms.

Hunger and nutrition were redefined here and elsewhere in terms of not only how many tons of food were available, but also by the need for nutritional quality in the human diet. Nutrition for human security would not be accomplished by bread alone. Our own Friedman School Professor Patrick Webb took leave to

establish the office and nutritional standards of the United Nations World Food Program as an expression of this concept.

Jean Mayer's last official act in late 1992, a month before his death, was to lead our Tufts delegation to the International Nutrition Congress in Rome, where he vigorously argued against the continuing use of starvation as a weapon in the Sudan civil war. Sadly, that conflict rages today, 16 years later, in Darfur. In 1995, we built upon this history and formed in the School of Nutrition Science and Policy the first Center for the Study of Famine. We recruited John Hammock, a Fletcher graduate then at Oxfam, to be its first director. This unique Center quickly attracted the attention and commitment of hunger activists as well as philanthropists such as Allen Shawn Feinstein, Henry Leir, and the Mellon Foundation. National and international bodies, such as USAID and the United Nations High Commission for Refugees, came to us for our studies and assessments of needs to counter starvation in many complex emergencies. Early on, our scholar activists, and I emphasized that it is possible and perhaps necessary in this field to be both a scholar and an actor, noted that emergency food aid, though critical, was not sufficient. The preservation of the livelihoods of the afflicted populations was essential for longer-term stability.

My colleagues at Tufts, through research on nutritional biology and the monitoring and evaluations of nutrition interventions, would add newer definitions of the international war on hunger. I am proud to be able to continue my efforts in science and its translation at Tufts, with the University's commitment of President Larry Bacow, its faculty, and its students to be a focus of science and knowledge in the conquest of hunger through nutrition with dignity.

DISCLOSURE STATEMENT

The author is not aware of any biases that might be perceived as affecting the objectivity of this review.

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Errata

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