The Role of Nutrition in Mitigating Environmental Insults: Policy and Ethical Issues

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Research has accumulated which indicates that nutritional factors can significantly modify the host response to environmental toxicants. Correction of malnutrition can clearly mitigate the effects of many toxicants; however, evidence is mounting that supraphysiologic doses of nutrients (nutritional supplements) can further lessen toxicity. The use of dietary supplements, calcium in particular, to blunt the effects of lead is discussed as a case example. The possibility that nutrition could be implemented as a secondary prevention strategy on a public health scale raises important ethical and policy issues. The cost of cleaning up pollution when resources are scarce could favor secondary prevention strategies under a simple utilitarian analysis. This analysis, however, fails to recognize certain inequities such as the plight of those consigned to suffer in polluted conditions. Nutritional strategies can lessen, but not abolish, toxic effects; moreover, they require dissemination and compliance, which are unlikely to be fully effective. These issues demonstrate the need to develop a public health paradigm for the role of nutritional interventions in environmental health.

As our knowledge of environmental hazards grows, the threats they pose to children, in particular, appear formidable. (In this article, our focus is on environmental hazards such as lead paint, organic solvents, and pesticides, the presence of which is dependent on anthropogenic activities. Not included are, for example, infectious agents and plant toxins.) As reviewed by others in this symposium, in addition to biological factors that may increase the susceptibility of children to toxic effects, there are also behavioral factors, such as hand-to-mouth and play activity, that may increase the risk of exposure to toxicants many-fold.

Environmental health entails a continuum from public health to clinical care. Within this continuum there are primary prevention strategies—that prevent the pollution that can lead to disease; secondary prevention strategies—interventions that can abate the effect of pollution on individuals; and clinical care—encompassing treatment for environmentally related diseases. Treatment is the most expedient approach, but is also the least desirable from a public health point of view. Treatment occurs only after the failure of primary and secondary prevention. Primary prevention, on the other hand, while optimal from a public health point of view, is often excruciatingly slow. Regulation and abatement of environmental pollution can take years. Secondary prevention strategies are intermediate in terms of their expediency.

One of the intermediate strategies for mitigating environmental hazards may be the use of nutritional approaches—i.e., nutritional supplementation, substitution, or other forms of dietary manipulation. As such, nutritional approaches would constitute a form of prevention somewhere in between source reduction of pollution and treatment of environmentally related illness. Unlike other forms of treatment (e.g., pharmacotherapy with drugs), nutritional strategies carry the promise of a natural form of therapy that would presumably be cheap and with few to no side effects.

It must be stated that, as yet, no form of nutritional strategy has gained wide acceptance for mitigating any exposure, condition, or illness from environmental hazards as defined above. Much indirect evidence, however, has begun to accumulate on how nutritional factors may modify the host response to environmental exposures, and it is likely that future research will be able to demonstrate the efficacy of such approaches. On the surface the drawbacks would appear to be nonexistent, and rapid implementation of nutritional approaches that seem promising would appear advantageous. A closer inspection of nutritional strategies in the context of public health policy, however, reveals several ethical and philosophical issues of a thorny nature that deserve sober review.

Nutrition as a Possible Prevention Strategy in Environmental Health

Overview

In the chain of events between the introduction of toxicants into our environment and the causation of disease, there are many potential points at which nutritional strategies may be able to mitigate the effects of pollutants. Laboratory and clinical investigations in this area have begun to shed light on this topic. Environmental epidemiological research has also begun to examine the influence of dietary factors by incorporating methods of nutritional epidemiology. In particular, the development...
of biological markers (1) indicating internal dose and early biological effects of toxicants has enhanced our understanding of the intermediate steps required for toxicants to have their effect. In so doing, we can now begin to identify points at which nutritional strategies may be able to inhibit, arrest, or even reverse the chain of events in toxicity, as well as quantitate the magnitude of these effects.

Prevention of access of a toxicant to the body by a nutritional supplement, e.g., inhibition of gastrointestinal absorption of a toxicant, would constitute an example of a nutritional strategy operating early in the causal pathway. Once a toxicant has been absorbed, nutrients may be able alter patterns of its deposition into tissues. In so doing, nutrients may lower (or increase) the amount of a toxicant's dose to organs that are the most critical in terms of toxicity, thereby altering the extent of health effects even though the total amount of toxicant absorbed has not changed.

Further down the causal pathway, nutrients might also arrest the progression of toxicity even after a critical organ dose has been achieved. For example, mineral nutrients may compete with toxicants, particularly metals, for enzymatic binding sites or enhance the activity of detoxifying microsomal enzyme systems. Nutritional factors may be able to block the formation of metabolites that may be the true progenitors of toxicity, such as the free radicals suspected to cause genetic damage and thereby to cause cancer.

The Effect of Malnutrition

In considering the interaction between nutrition and toxic hazards in an individual, much depends on the baseline nutritional status of the individual. Indeed, much of the early scientific work that has been performed on the influence of nutritional factors on toxicity has focussed on deficiencies of selected nutrients.

For example, laboratory experiments on rats have demonstrated that, at levels of dietary protein that are 15% below normal, the toxicity of carbenam and organophosphate pesticides is markedly increased (2–4). Riboflavin and nicotinic acid deficiency have been observed to exacerbate the toxicity of the pesticide dieldrin (5).

Deficiencies of protein and minerals, particularly iron and calcium, have also been found to significantly influence the gastrointestinal absorption of pollutant metals, particularly lead and cadmium (6,7). Generalized fasting also has been found to increase gastrointestinal absorption of metals such as lead (8). There is some evidence that nutritional deficiencies may alter the pattern of soft-tissue deposition of lead and cadmium (9). Deficiencies in dietary calcium also have been noted among children with elevated lead blood in several epidemiological studies (10). The mode of action may not rest entirely on increased intestinal absorption of lead since there is also evidence that calcium deficiency leads to mineral-seeking behavior, such as pica, thereby indirectly increasing lead exposure (11). High blood leads also have been seen among adult women with diets relatively deficient in calcium (12).

The Potential Role of Supraphysiologic Doses of Nutrients

In comparison to malnutrition, the effects on pollutant toxicity of supplementation with nutrients at levels above those that are physiologically required has been studied relatively less, both in animals and humans. With regard to toxic metals, there is some evidence in both animals (13) and human infants (14) that increased dietary calcium will lead to further decreases in lead absorption, even within the range of recommended daily intake of calcium.

One area of toxicology that has directed increased attention to the role of supraphysiologic doses of nutrients is environmental carcinogenesis. An example is vitamin A (i.e., beta-carotene and other carotenoid compounds that can be metabolized to form retinol, the physiologically active form of vitamin A). In a number of animal studies, high doses of vitamin A and synthetic analogues have been found to inhibit the occurrence of induced tumors and even reverse metastatic changes (15,16). Vitamin A has been found to exert an inhibitory effect even when administered after a cancer has been induced (17), raising the possibility that it could be used as a tertiary treatment strategy as well as an agent of secondary prevention. Observational epidemiological studies have tended to support the notion that foods high in vitamin A, such as leafy green vegetables and fruits, confer a strong protective effect from lung cancer and other forms of cancer (18). It has not yet been possible to attribute this effect to vitamin A, as opposed to other nutrient properties of such a diet [and, in fact, a recent study of beta-carotene supplements found an excess, instead of a deficiency, of lung cancer among recipients in comparison with controls (19)]. In the future, it will be important to distinguish whether the beneficial effects of supplementation are derived from foods that are high in multiple nutrients or from purified supplements (for example, in pill form).

Little else can be said with any certainty regarding the ability of nutritional supplementation to mitigate environmental insults. Some papers have begun to surface in the proceedings of symposia regarding nutritional approaches to the treatment of multiple chemical sensitivity syndrome (20,21); however, neither laboratory studies nor well-designed clinical controlled trials exist to back up these assertions, and multiple chemical sensitivity syndrome itself remains a poorly defined disorder with no well-established pathophysiology.

The Case of Nutrition and Lead

As discussed by Maierffey (22), lead has been the most studied toxicant with respect to nutritional influences. Four additional reasons make lead an important case example with which to study the implications of nutritional strategies. First, lead constitutes a ubiquitous toxicant. In this country, multiple pathways of exposure exist, particularly lead in paint, drinking water (from plumbing), house dust, and soil (from deteriorated lead paint and combusted leaded gasoline). Lead is also a worldwide toxicant, exposure to which is increasing in many parts of the world, particularly in areas experiencing urbanization and industrialization. Leaded gasoline, the use and production of which has been declining in developed countries since the 1970s and 1980s, remains the primary fuel of choice for cars in much of the developing world. In addition to being the single largest source of lead emissions into air (23), burned gasoline adds lead to water supplies, soil, food crops, and house dust. Thus, primary prevention of lead exposure requires abatement of lead sources on a massive scale, making secondary prevention efforts such as nutritional strategies an appealing strategy.

Second, toxic effects have been demonstrated for lead at levels of exposure that are being experienced by wide segments of the population. Many recent reviews have summarized the available laboratory and epidemiologic evidence on the neurodevelopmental effects of low-level lead exposure in children (24–26). In general, the weight of the evidence from both prospective studies and metaanalyses of cross-sectional studies indicates that relatively low blood lead levels (as low as 10 μg/dl and below)
are associated with measurable deficits in IQ and other indicators of cognitive performance. Moreover, recent studies of the dose–response relationship have not revealed an apparent threshold for this effect. Thus, any decrease in lead absorption from nutritional supplementation is likely to result in an improved outcome.

Third, in addition to the effect that nutritional factors may have on lead absorption (22), there is mounting evidence that nutritional factors may modify other aspects of lead toxicity. Much research has already indicated that lead is sequestered in bone, with a half-life of years to decades. During pregnancy and lactation, studies on mice and a few studies in humans suggest that mobilization of skeletal calcium reserves is accompanied by release of lead from bone, thereby jeopardizing the fetus from endogenous lead stores even if environmental lead exposure had ceased (27–30). In separate research, an increased calcium diet has been found to prevent bone mineral loss in lactating adolescent mothers (31). Thus, the possibility exists that dietary calcium may be able to prevent the transfer of lead burden from mother to fetus or lactating infant by inhibiting bone mineral loss.

Finally, insufficiency of dietary calcium in terms of bone growth is widely prevalent in some segments of society, thereby creating what may be an opportunity for a dietary intervention with public health benefits in terms of growth and the prevention of lead uptake from the diet and mobilization of skeletal lead. In the United States, for example, typical adult female intakes of dietary calcium are 500 to 600 mg/day (32). In consideration of calcium needs to achieve maximal bone mass, it is also likely that dietary calcium and absorption in children and adolescents are low (33,34). High dietary protein intake, replacement of consumption of dairy products with soft drinks, and limited weight-bearing exercise may all contribute to both the lower dietary calcium intake and the negative overall calcium balance among U.S. females (35,36).

In summary, lead is a widespread and pervasive threat that has been demonstrated to have profound effects on neurobehavioral development. There is growing evidence that due to an effect on gastrointestinal absorption of lead and the demineralization of lead-containing bone, administration of nutritional supplementation, specifically, dietary calcium, to women of reproductive age, infants, and children may constitute a low-cost strategy for mitigating the toxic effects of lead.

**Nutritional Strategies: Policy and Ethical Issues**

Environmental pollution such as the dissemination of lead discussed above poses a threat on a local, regional, and global level. Overviews of pollution on a world scale demonstrate that even as some countries, particularly in the developed world, are striving to reduce pollution, contamination of the environment appears to be escalating elsewhere, particularly in countries experiencing rapid industrial and urban growth (37,38).

Governments have responded to environmental pollution through a variety of mechanisms. In the United States, for instance, we increasingly use criminal law to deter knowing polluters. Tort litigation brought by the victims of environmental pollution serves not only to deter polluting behavior, but also provides reimbursement for injuries. Many have advocated use of market incentive to reduce pollution, for instance, through trading strategies used in the Los Angeles basin for air pollution (39). But by far the greatest government activity takes the form of regulation.

In this country, environmental regulation has tended to be media specific. In addition, we have used permitting and standard-setting strategies generally (although the most prominent environmental statute today, Superfund, or the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, relies primarily on civil litigation techniques). For example, the Clean Air Act focuses on air pollutants and typically uses a standard-setting approach (40). The Federal Water Pollution Control Act relies on permits. Both are enforced through media sampling.

Some hazardous substances, however, tend to cut across several different control strategies. Lead, for instance, is regulated under the Clean Air Act as a criteria pollutant and through various state and federal statutes as an indoor air pollutant. Moreover, lead paint in municipal waste has come under the purview of the Resource Conservation and Recovery Act as well as Superfund. Finally, a certain amount of biological monitoring of lead occurs in those states that have passed serious lead abatement statutes.

The goal of these regulatory efforts is primary prevention. Yet, there is nothing inherent in the way government works that prevents it from making available nutritional supplementation as a further, secondary prevention strategy for mitigating environmental health threats. Indeed, given the above discussion on lead and nutrition, the low cost, and minimal side effects, the favorable risk–benefit appears to favor the institution of some kind of policy advocating the widespread use of nutritional supplementation in societies or segments of society that carry a high risk of environmental lead exposure.

The philosophical and ethical dilemma posed by nutritional supplementation, however, is brought on by the practical considerations of a society that is trying to choose between different approaches towards mitigating the effects of pollution. When resources are scarce, there is an implicit or explicit pressure to direct resources to approaches that cost the least while offering the greatest benefit. Much of regulation is driven by the assumption that simple utilitarianism, the greatest good for the greatest number, governs our policy-making (41,42).

Under the assumption that societies often must choose between approaches for dealing with pollution and that cost-benefit analysis is often the tool used to select choices, a scenario could be envisioned in which a society would advocate channeling resources into nutritional supplements over primary pollution control. Secondary nutritional prevention could be more expedient and cost less than primary prevention.

But this strategy raises equity concerns that do not surface when the goal is primary prevention. Primary prevention benefits all equally, as its goal is reduction of pollution below threshold effect levels. Secondary prevention, however, grants that some populations will face greater hazardous pollution and must be the targets of nutritional strategies. This represents a retreat of sorts from previous commitments to a clean environment and to abatement of hazardous pollutants.

**The Equity of Nutritional Strategies**

Consider our case example of lead exposure in urban America. The primary prevention costs include hazard abatement of multiple sources of exposure. As discussed no doubt elsewhere in this conference, the resources that potentially would be involved are staggering. For example, it has been estimated that there are 57 million housing units in the United States that contain lead paint, the removal of which has been also estimated at $7700 per unit (43). Moreover, removal of a single source of lead would
probably be insufficient; lead exposure sources tend to be associated with each other. For example, both lead paint and lead water mains (44) tend to be found in older homes. These homes, in turn, are often located in older urban centers where soil can be highly contaminated by the past use of leaded gasoline (45).

One could imagine that cost–benefit calculations regarding lead paint exposure reveal that the total cost of cleaning all public housing would be $80 billion, but that the total costs in 1994 dollars of morbidity associated with exposure are only $79 billion. In that case, a cost–benefit analysis would not favor the cleanup. On the other hand, those exposed individuals who suffer neurobehavioral difficulties and subsequent earning loss (not to mention other human suffering) would be quite committed to the cleanup. Of course, not only the costs and benefits matter to most policy-makers, but also who benefits and who bears the costs.

A nutritional strategy of secondary prevention might represent an attractive middle ground in this case. Suppose that a targeted nutritional intervention could abate $50 billion of the costs of pollution at a price of $25 billion. This would be attractive from a cost–benefit perspective. But it would leave in place $29 billion in costs of pollution, borne not by every citizen, but only by those exposed to the pollution for whom nutritional strategies did not work or did not reach.

This paradigm emphasizes the importance of equity concerns in a critique of utilitarianism. The utilitarian tallying up of costs and benefits fails to recognize the plight of those who are consigned to suffer in poor conditions. While efficiency is important in any social program, too is distributive justice.

These issues are now being carefully discussed in a growing literature on the distributive aspects of pollution, or so-called environmental justice. The opposition of some environmentalists to market incentives and commodification of pollution provides an important message for advocates of cost–benefit analysis in a modern liberal state (46–48); once a market in pollution rights is created, so too is a market in human lives. Commodification of the discharge of toxic substances commodifies human lives in that those firms which face high cost rebatement measures may be able to buy rights to pollute and so buy rights to expose individuals to toxic substances. (None of this denies that our society has long made such decisions concerning the value of human lives. Indeed, our regulations evince an incredible variation in the amount we are willing to pay to save human lives (49,50). Explicit recognition of the values we place on life through governmental regulation does at least place our regulations in some perspective.)

Nutritional secondary prevention programs provide a slightly different twist on the environmental equity debate. In this case, the inequitarian issue is that those singled out for a nutritional strategy will still be exposed to hazardous substances. The best approach in terms of avoiding disease outcomes would be to abate the hazard; second best is to intervene with calcium supplements for example. This raises considerable issues in a liberal state that conceives of justice as fairness.

In most descriptions of a modern liberal state, efficiency is important, but it tends to be trumped by individual rights. And increasingly, the right to be free from pollution, or at least to be as free as others, is asserted, especially by those who believe in environmental equity. In this light we would not allow certain inner-city neighborhoods to be heavily polluted with lead simply because a cost–benefit analysis had indicated it was appropriate. Or more to the point, some communities should not have to tolerate pollution exposure that is ameliorated by nutritional supplements.

Exposed individuals are likely to exercise their environmental rights. The concept of environmental rights lacks an explicit constitutional framework, but legal scholars and advocates are amending a series of grounds for asserting it. Some have contended that environmental protection might be based in privacy rights (51). Others envision environmental protection as a First Amendment right to self-fulfillment (52). Still others move environmental rights completely out of a constitutional framework (53). These sorts of analyses in the past barely engendered much further debate, likely because environmentalism tends to involve problems of exposed majorities frustrated by powerful polluting minorities (54). But today, interest is growing as environmental justice comes into the mainstream.

Toxic-exposure injuries suffered by individuals give new substance to the notion of environmental rights. Only a minority of individuals are exposed to significantly toxic levels of environmental pollutants; yet many benefit from the industrial and agricultural enterprises that produce pollution. In a liberal state, especially one with constitutional entrenchment of individual liberties, legal protection from unconsented injuries should be available. This includes those injuries that may be suffered by those who must make do with secondary prevention programs like nutritional supplementation.

Nutritional Strategies: Their Potentially Inequitable Effect

If nutritional strategies were completely efficient in terms of abating the ill effects of pollution, then perhaps there would be no equity problems; but they are unlikely to meet this criteria. Any nutritional strategy will depend on individual compliance, which will bring certain individual costs even if nutritional supplements were made available at little or no cost. Unlike immunization programs, a nutritional strategy will require the creation of new educational programs by health providers, compliance every day, storage of supplies, and frequent visits for refills. Given the subtle nature of impairments caused by pollution and the relatively low attributable fraction of disease, one can expect less than 100% compliance.

Market advocates might retort that, if given the chance to abate health problems associated with pollution, individuals should act as rational players; if they do not comply with nutritional strategies, they are simply making informed decisions. But in public health, we have long assumed an antilibertarian posture. The failure of a nutritional strategy would not be accepted, but we would consider new inducements and advertising strategies to boost compliance. Moreover, public health advocates have experience in the difficulty of voluntary outreach programs to provide essential health services in the face of extreme poverty. Indeed, these arguments generally lead us to favor primary over secondary prevention.

More to the point, even if the nutrition strategy works very well, there will still be some unabated effect of pollution in certain areas, again raising equity concerns. Reducing the effect of pollution around dirty factories or in heavily lead-polluted inner cities by turning to nutritional supplementation does not cure the distributonal problem; it merely ameliorates, and only to the extent that people comply. Why should some have access to pollution-free media while others have to live with pollution and take nutritional supplements?

Some would argue that pollution should be morally stigmatized, and so excluded from market and presumably
cost–benefit analyses. Kelman (55) for instance has opposed market incentives in that they fail to carry out the necessary stigmatization of environmental pollution. He believes that such marking, the moral outrage expressed in terms in term of pollution control, is very important to the society’s consideration of environmental quality. Kelman also argues that it is inapprop-riate to trade some things in the mar-ket. Certain commodities simply cannot be traded, largely because individuals tend to down-value the effect of them and because such markets appear to affirm behavior which society should oppose, that is, creating pollution (56).

Concerns about commodification then create a final barrier to those who would use unrestricted cost–benefit analysis in environmental regulation, and in particular in the use of nutritional strategies as a replacement for primary prevention.

**Conclusion: The Need to Develop a Public Health Paradigm for the Role of Nutritional Interventions**

As stated earlier, there is not yet enough research to identify any particular nutritional type of therapy as a strategy for mitigating environmental health hazards that should be implemented on a public health scale. Evidence is mounting, however, and it is likely that corroborating research will be forthcoming soon.

The promise of providing a low-cost strategy for mitigating the effects of environmental pollution makes nutritional strategies an attractive option. The potential for such strategies to perpetuate or exacerbate considerations of equity, however, should make public health professionals pause before rushing toward implementation.

One could argue that a distinction should be made between strategies that aim to correct malnutrition and strategies that aim to supplement adequate nutrition with supraphysiologial doses. Malnutrition and efforts to correct it have a long and honored tradition within the practice of public health, and it would seem natural for public health professionals to extend the argument for addressing malnutrition as a strategy that will not only directly impact on health, but will also indirectly mitigate the toxicity of environmental hazards.

However, it would be important to not lose sight of the fact that the primary goal of such a strategy is to correct malnutrition as a primary risk factor for disease and developmental delay. Once nutritional strategies are considered as primarily aimed at environmental health hazards, their relationship to primary prevention efforts to control pollution must be understood in the context of environmental equity and environmental rights.

In conclusion, the ethics of public health are fundamentally egalitarian and oriented toward primary prevention. Nutritional strategies aimed at correcting malnutrition are an important component to public health. Nutritional supplement strategies, if found to be effective at mitigating environmental hazards, may be thought of as a possible adjunct to efforts at primary prevention; however, the development of nutritional supplement strategies as a policy must explicitly consider issues of environmental equity and environmental rights.

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