

Obesity and Diabetes in Vulnerable Populations: Reflection on Proximal and Distal Causes

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ABSTRACT

Around the world obesity and diabetes are climbing to epidemic proportion, even in countries previously characterized by scarcity. Likewise, people from low-income and minority communities, as well as immigrants from the developing world, increasingly visit physicians in North America with obesity, metabolic syndrome, or diabetes. Explanations limited to lifestyle factors such as diet and exercise are inadequate to explain the universality of what can be called a *syndemic*, a complex and widespread phenomenon in population health produced by multiple reinforcing conditions. Underlying the problem are complex factors—genetic, physiological, psychological, familial, social, economic, and political—coalescing to overdetermine these conditions. These interacting factors include events occurring during fetal life, maternal physiology and life context, the thrifty genotype, the nutritional transition, health impact of urbanization and immigration, social attributions and cultural perceptions of increased weight, and changes in food costs and availability resulting from globalization. Better appreciation of the complexity of causation underlying the worldwide epidemic of obesity and diabetes can refocus the work of clinicians and researchers to work at multiple levels to address prevention and treatment for these conditions among vulnerable populations.

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INTRODUCTION

Obesity, and its coconspirator, diabetes, increasingly dominate health problems for immigrants, low-income populations, and communities of color. North American physicians who provide primary health care to low-income communities and immigrant populations from the developing world confront such vulnerable patients every day. From Asia, South Asia, the Middle East, Africa, Latin America, and the Caribbean and from indigenous populations, patients seek consultation for a variety of symptoms. Often the initial investigation of the patient, who may be seriously overweight, reveals previously undiagnosed hypertension, diabetes, metabolic syndrome, or dyslipidemia. These conditions appear to relate to personal behaviors based on lifestyle decisions. When these vulnerable patients—be they black, Hispanic, Native American, poor, or immigrant—fail to change their diet or lose weight or exercise, and take their medications erratically owing to cost or misunderstanding or sheer disbelief in the need for daily medication, physicians may attribute the problem to failure of personal responsibility. Epidemiologic studies often focus on individual characteristics and behaviors to explain these problems rather than examine the multiple forces at work.¹ Nevertheless, the epidemic increase in obesity and diabetes around the world suggests that factors far beyond individual behaviors must be at work to explain this recent global process. In this essay, I describe some

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of the emerging understandings about the interacting causes of this worldwide phenomenon with a particular focus on immigrants and members of low-income communities and communities of color within North America.

The Need for Systems Thinking

In the United States and other developed nations, diabetes and obesity are markers for inequalities in health. Although these infirmities affect all segments of the population, minority populations, low-income communities, and immigrants (especially as time passes since arrival) are disproportionately afflicted.²⁻⁵ Around the world emerging nations likewise face this pandemic, but in those cases, the social inequality stands in relation to global economic policy. Internationally it may not be the very poorest populations who face diabetes and obesity, but rather it may be those with some access to the cheapest foodstuffs. The merger of many interacting forces conjoined in creating this global phenomenon calls for explanatory models and ways of thinking that go beyond traditional singular notions of causality. Within the field of social epidemiology, "ecosocial and other emerging multi-level frameworks seek to integrate social and biological reasoning and a dynamic, historical and ecological perspective to develop new insights into determinants of population distributions of disease and social inequalities in health."⁶

To introduce this approach into family medicine, I will describe several of these determinants of the obesity/diabetes epidemic, ranging from the genetic, cellular, biologic, and psychological levels to the social, historical, economic, and political. I will suggest that systems thinking may provide a way to consider such an enormous problem, marked by com-

plexity at every level. Finally, I will offer some areas of action for family medicine in the realms of research, collaboration, and advocacy.

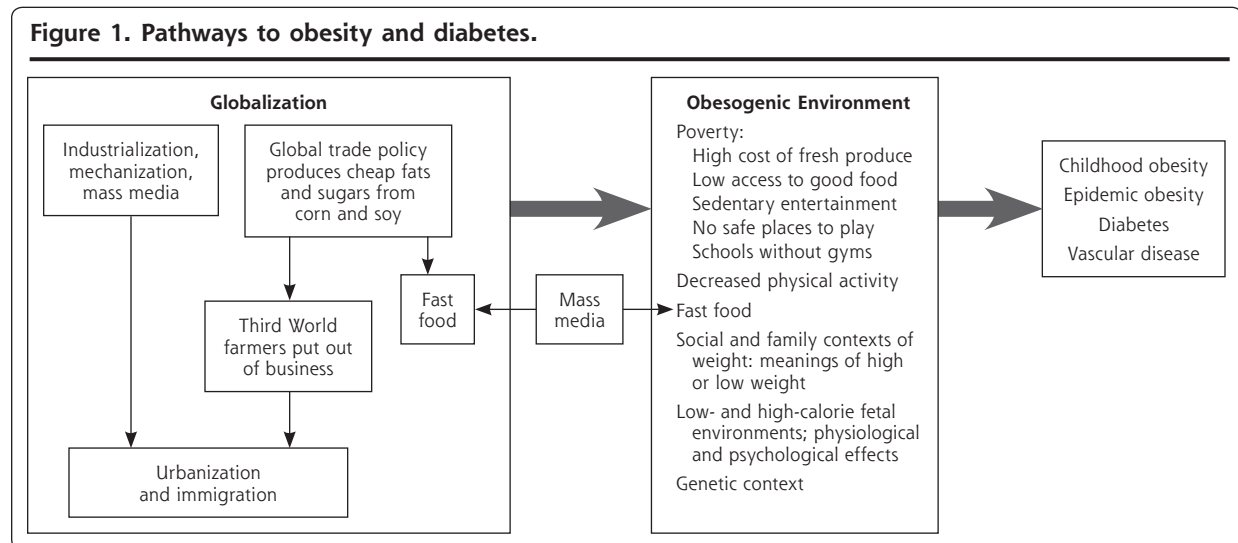
OVERDETERMINATION OF OBESITY, DIABETES, AND RISK OF VASCULAR DISEASE

Although North Americans are familiar with the obesity epidemic, they may be less aware of the truly global extent of the problem. Around the world, especially in developing nations, a constellation of factors conspire to guarantee that obesity, with the high likelihood of ensuing diabetes, will afflict entire populations (Figure 1). The terms used to describe these factors may be unfamiliar to practicing clinicians but are commonly understood among public health experts in nutrition. Recent editorials in major medical journals have begun to bring some of these factors to wider attention.⁷⁻⁹ These factors include the following:

- Fetal and maternal explanations
- The thrifty genotype
- The nutritional transition
- The health impact of urbanization and immigration
- Social attributions and cultural perceptions of increased weight
- The impact of globalization on nutrition

Fetal and Maternal Explanations

Recent research suggests that an adverse intrauterine environment leading to low birth weight is linked with the later development of obesity and the metabolic syndrome.¹⁰⁻¹² This explanation is sometimes called the thrifty phenotype: the calorically deprived fetus and later child responds by hoarding calories as an environmental response. Put another way, the



"fetal origins of disease" hypothesis postulates that early (intrauterine or early postnatal) undernutrition causes an irreversible differentiation of metabolic systems, which may, in turn, increase the risks of certain chronic diseases in adulthood.^{8(p1515)} In one scenario, the fetus permanently switches on genes to maximize energy conservation, causing excess accumulation of energy (and therefore body fat) when the individual gets unrestricted calories later. Physiologically, the hypothesized chain of events is that fetal malnutrition leads to relative tissue resistance to insulin, low birth weight, childhood fat deposition, later childhood and early adult insulin resistance, and ultimately adult diabetes. Another hypothesis derived from animal models is that the maternal organism programs the biologically plastic fetal system to prepare for the caloric environment that it will face. When there is a mismatch between the preparation for scarcity during fetal life with the relative abundance of calories later, the result is a predisposition for caloric hoarding, childhood fat deposition, and so on.¹³⁻¹⁶ (Maternal smoking, strongly associated with fetal deprivation in the form of intrauterine growth retardation, also contributes to the mismatch. Teen offspring of women who smoked during pregnancy are more likely to be overweight or obese than offspring of women who had never smoked or had quit smoking.¹⁷) Thus the intrauterine growth retardation and low birth weight common to babies in developing countries, (but also not infrequent in the United States), where their mothers do not obtain adequate calories, may establish populations of adults susceptible to obesity and the associated conditions.

Supporting this idea of the thrifty phenotype are focused animal studies^{13,18} (not addressed here) and multiple epidemiologic studies. For instance, Dutch investigators found that women who were pregnant during a specific period of severe caloric restriction in the Netherlands during World War II were more at risk to give birth to low birth weight infants who in adulthood had increased risk for conditions leading to diabetes and vascular disease.^{19,20} Likewise, Finnish data of children born between 1934 and 1944 show the later impact of low birth weight. The more that low birth weight babies who were thin up until age 2 years increased their weight in relation to height between ages 2 and 11 years, (ie, their body mass index began to cross the growth curves), the more likely they were to develop insulin resistance and coronary disease as adults.²¹

This dynamic has emerged whenever researchers have followed low birth weight infants in developing countries. These regions are the homes of origin of many immigrants to North America, yet their experience of poor nutrition during the fetal period may be unknown or opaque to the clinicians who encounter

them as adults. For instance, in India—the second leading country of immigrants to the United States in 2005²²—the accelerated increase of the body mass index during childhood (early adiposity rebound) of low birth weight infants correlates with later impaired glucose tolerance and frank diabetes by the age of 26 years. Although such children are not necessarily obese by Western standards, they have more truncal fat and their risk of diabetes is still higher.²³ In Guatemala, severely stunted babies are more likely to become adults with central obesity, especially if their mothers migrated to urban centers.²⁴ In Mexico, when documented early malnutrition among boys was followed by the later development of abdominal fat, insulin resistance was also more likely.²⁵ Likewise, the emerging high rates of glucose intolerance, diabetes, and hypertension in Cambodia may relate to the long periods of deprivation suffered by the Cambodian people during the last 50 years.²⁶

Maternal biological responses, in addition to fetal reactions, may also foster later problems for both mother and child. In some instances, maternal insulin resistance during pregnancy may protect against fetal malnutrition by preferentially driving available calories toward the fetus; such a tendency obviously portends a later risk of diabetes for the mother.¹² In settings of excess calories during pregnancy, so-called maternal overnutrition, typical of urban settings of the late 20th century, maternal insulin resistance has been associated with high birth weight, which itself correlates with later obesity and insulin resistance. This description, the "hefty fetal phenotype"^{27,28} applies to infants born to the growing number of obese women who have insulin resistance but not yet gestational diabetes in pregnancy. Studies from various regions confirm an association between maternal body mass index, birth weight, and later adolescent and adult obesity.²⁹⁻³¹ With two thirds of adult women in the United States overweight, and at least one third of whom are obese (with even higher rates among low-income and women of color), insulin resistance in pregnancy is becoming commonplace. Thus, distinct from any inherited tendency toward diabetes, impaired fetal nutrition itself, mediated through the mother's body, generates tendencies at both ends of the infant weight curve toward obesity and diabetes in adulthood.

The resulting epidemic of diabetes in the next generation of North Americans, children of both immigrants and native-born, looms large. Social and psychological maternal factors, as well as the biological, contribute to their offspring's risk for obesity and diabetes. Mothers who have small thin babies will want to fatten them. Unfortunately, the resulting rapid weight gain for children in infancy and in early childhood is

associated with later obesity.³²⁻³⁴ Mothers, too, may engage in excess eating at those times when they have more access to food, and in the United States food insecurity is associated with overweight, especially for women.^{35,36} The experience of food insecurity and scarcity is likewise a strong stimulus to push calories into children when food becomes available.³⁷ Thus it is not surprising that low socioeconomic status in childhood would be a predictor of obesity for African American women as adults.³⁸ So not only the prenatal environment but also the later socioemotional context in which the perhaps small-for-age child grows and eats—especially if food is not reliably available—promotes the development of later obesity and diabetes.

Thrifty Genotype

This hypothesis proposes that the ability to conserve calories by laying down abdominal fat offers a genetic advantage for selection of this genotype during periods of food scarcity in human history.^{39,40} All humans are likely to have genetically determined mechanisms to survive caloric restriction, but some people may have thrifter genotypes than others, resulting in a higher likelihood of obesity when they are faced with higher caloric loads.⁴¹ With increases in caloric intake and decreases in physical activity brought about by economic and social changes, the genetically driven tendency toward central fat deposition becomes a health hazard. Such visceral fat is directly related to the development of insulin resistance and eventually diabetes. This hypothesis has been criticized for its potential to confuse genes with race and its application of biological determinism in the explanation of diabetes without addressing the social determinants of disease⁴²; however, unique ethnic patterns of abdominal fat deposition resulting from a variety of genetic mutations are demonstrable.¹² Compared with white Europeans, ethnic groups from developing countries—as well as native Americans, native Canadians, Maori, Asian Pacific islanders, and many other native populations⁴³⁻⁴⁵—are more vulnerable to the development of diabetes, possibly because of genetic selection for those who could withstand starvation. Why the rate of diabetes among Europeans is lower despite famines in European history is subject to speculation: perhaps because of the differing patterns of famine, differing ways regions addressed food scarcity, or out-migration of those most at risk for diabetes.⁴⁶

Nutritional Transition

Fetal and genetic explanations do not fully explain why obesity and its consequences have burgeoned so rapidly in the last few decades. Clearly, changes in the environment interact with the other factors in a lethal

combination: “The genetic background loads the gun, but the environment pulls the trigger.”⁴⁷ Even in the developing world, obesity, diabetes, and vascular disease are replacing undernutrition and infectious disease as the main health risks. Whereas previously poverty was linked with low calorie intake, this pattern has shifted with the wide accessibility of cheap fats and the rapid increase in the consumption of high-fructose corn syrup around the world.^{47,48}

The change from a pattern of low calories and low fat in resource-poor settings to higher calories and high fat is termed the *nutritional transition*. For instance, in Brazil the lowest income quartile of women contains the highest prevalence of obesity as well as of underweight women.⁸ In Brazil and India, higher weight and therefore higher risk of diabetes are more prevalent among the lower income groups. Consequently, especially among the poor, rates of cardiovascular disease are already rising precipitously in India, where the projected number of people with diabetes will approach 60 million by 2025.⁷ In many settings in the United States and abroad, poor maternal nutrition and excess calories coexist in the same environment, constituting the dual burden household. In Morocco, where underweight and stunting among children are prevalent, obesity among children and adults, especially women, is also on the rise.⁴⁹ “Cheap energy-dense, nutrient-poor foods may adversely affect the growth of the child but may provide sufficient calories for the adult to gain excess weight.”^{48(p1515)}

Health Impact of Urbanization and Immigration

Globally, people migrate from rural areas to the cities in search of work, more income, and a better life. Practically speaking, they walk less, ride more, watch more television, and eat a diet higher in fat and sugars when they live in the city. They have less access to homegrown and local vegetables and are more likely to eat fast foods, as well as fried foods and sugary drinks widely accessible from vendors on the street. Thus obesity and its sequelae are increasing ominously in cities in the developing world.^{8,50} Recognizing the enormity of this problem in the face of increasing urbanization, the World Health Organization (WHO) issued in a report recommending that fat consumption be limited to 20% to 25% of caloric intake.⁵¹ Inner-city dwellers in North America face similar issues: unavailable or expensive fresh fruits and vegetables, less access to safe settings for exercise, reliance on television for entertainment of adults and children, proliferation of fast-food vendors, and economic pressures limiting time for family meals at home. Children attend urban schools that rely on high fat–high carbohydrate foodstuffs in

lunch programs at the same time that budget cuts often reduce or eliminate gym. Although city dwellers living in mixed-use neighborhoods may walk more than suburbanites, low-income families in the inner-city areas truly live in an obesogenic environment.^{52,53}

Urbanization and migration are worldwide responses to poverty and lack of employment and have similar impacts on people's weight. For instance, as time passes after migration, obesity among the arrivals becomes increasingly likely.^{4,54,55} The prevalence of diabetes increases about 20 years after immigration in multiple ethnic groups entering more high-calorie, low-activity environments.⁴⁶ The children of immigrants may fare even worse—they appear less likely to choose a traditional diet that historically might have contained an array of nutrients (eg, rice and beans lose out to French fries). For example, in the United Kingdom, second-generation offspring of immigrants appeared to adopt British dietary patterns, increasing their fat intake and reducing their consumption of vegetables, fruit and legumes compared with first-generation immigrants.⁵⁶ In the United States, Asian American and Hispanic American adolescents are more than twice as likely to be obese as the first-generation immigrants from their countries of origin.⁵⁰ Thus the children of immigrants may be at even higher risk of obesity and diabetes than their parents.

Social Attributions and Cultural Perceptions of Increased Weight

Culture shapes people's view of weight. In many cultures to be big connotes power, wealth, health, and higher social standing.⁵⁷ "Positive valuations of corpulence" are common outside industrialized societies.⁵⁸ Where scarcity and hunger are still realities, a fat baby is a healthy baby, and a skinny adult or one who is losing weight, is thought likely to die, often from a stigmatized disease like tuberculosis or acquired immunodeficiency syndrome. Even though educated adults with stable food supplies may be aware of modern concerns about obesity, the image of the big, strong, powerful, and healthy adult lingers in the collective imagination. In such cultures, losing weight is contrary to the social construct of health. Moreover, extra weight may be seen as attractive or sexually appealing.⁵⁹ In other words, cultures, with all their biological and historical experience, shape how their members consider weight.⁵⁸ These beliefs translate into practices affecting body weight patterns. For instance, in South Africa, a study of more than 1,000 women from urban and rural sites showed a significant positive association between household income and obesity.⁶⁰ Women with higher incomes and lower physical activity were at the greatest risk of increased body mass index, suggesting that, for now, in

this region obesity and diabetes will track with income.

Among many people of color in the United States, perceptions and attitudes about obesity can also be at variance with the national preoccupation with thinness. African American and Hispanic adults are more likely than white adults to perceive their current overweight status as closer to an ideal body size, and African Americans in general are more likely to perceive obesity as acceptable, desirable, or sexually attractive.⁶¹⁻⁶⁵ Moreover, some of the factors maintaining obesity may have strong cultural determinants related to the role of food.⁶⁶ Although not universal, the positive perceptions of excess weight persist despite the known connection between obesity and the very real health threats of diabetes and vascular disease.

From other viewpoints, some scholars identify the preoccupation with body size as a particularly Western phenomenon,⁵⁸ and others speculate that obesity may not be the top health priority for the black communities.⁶⁷ But such arguments do not address the accelerating accumulation of weight in the most vulnerable populations, particularly in the last 25 years. Regardless of whether an observer thinks that naming large body size as obesity is medicalization, the interaction of those pounds with diabetes, cardiovascular disease, and arthritis is already taking its toll on the health outcomes for Hispanic and African Americans.

Impact of Globalization on Nutrition

International economic forces interact to the detriment of healthful nutrition. Examples abound: Mexican corn farmers are driven out of the market for corn and into the stream of immigrants at risk for obesity because of such economic policies as the US government's price supports to huge conglomerate corn farms.⁶⁸ The resulting low cost of high-fructose corn syrup makes the calories from sweetened beverages extremely cheap: \$1 will buy 875 calories of soda.⁶⁹ Global outsourcing has pushed people into sedentary temporary jobs (such as call centers) with work patterns associated with increased fast-food consumption.⁷⁰ Patterns of food preference are also in flux owing to the success of multinational fast-food vendors and changes in the availability of fats and sugars in developing countries.⁷¹⁻⁷³

Health authorities are familiar with the dangers of fast fatty and sugary foods, but action is difficult. Although in January 2004 WHO recommended tighter regulation of food advertising aimed at children and other strategies aimed at limiting junk food and soft drink consumption internationally,⁷⁴ the United States and other sugar-producing countries opposed the release of the report.^{75,76} Not surprisingly, any serious attempt to limit or channel global food marketing will face enormous political opposition from forces

supported by multinational corporations.^{73,77} The result: global economic forces (ie, world trade policies favoring US corn production, international marketing of fast food, changes in physical activity in work, market-based changes in the availability of domestic products, media promotion of empty calories to children, etc) work synergistically together to produce the same effects—widespread obesity, diabetes, and heart disease in the developing world.

IMPLICATIONS

These complex and interlocking forces are daunting. Some factors, such as the thrifty genotype and a history of fetal malnutrition, cannot be changed; and others, such as urbanization, pressures around immigration, and cultural values around weight, are beyond the immediate reach of doctors and patients and their health care institutions. Some, like food availability and pressures on urbanization and migration, might potentially be amenable to changes in political and economic policies in specific countries; however, the multinational corporations are certain to oppose policies that will work against their interests. Change would need to be at many levels, from many sectors, working synergistically for healthier options, energized by collective will, and informed by excellent information.

In the United States, action at the federal level is already in motion. The Centers for Disease Control and Prevention (CDC) have been documenting for years the impact of overweight on chronic disease and the association of chronic diseases with low-income status. Now, the Federal Bureau of Primary Care and the Institute for Healthcare Improvement (IHI) are engaging community health centers, already committed to the care of the underserved, to adopt the Wagner Chronic Care Model for chronic diseases.⁷⁸ (see <http://www.healthdisparities.net/hdc/html/home.aspx>.) The resulting Health Disparities Collaboratives—initially focused on asthma, diabetes, heart disease, and depression—have fostered improvements in teamwork, development of data systems (registries), adherence to standards of care, design of clinical systems, support for decision making, and encouragement of patients' taking charge of their illnesses. The chronic disease collaboratives can make linkages with community resources to promote health education and lifestyle changes in diet and exercise. Childhood obesity is already a target for this model.⁷⁹ Training patients in adoption of self-management strategies, a prime target of the collaborative, is moving forward.⁸⁰ Community health centers have improved the processes of care although definite proof of improved disease outcomes is still lacking.⁸¹ Even though changes in health care

cannot address all the social and economic factors elaborated above, broad changes in the health care system, as IHI proposes, are initial steps in working for change (see <http://www.ihl.org/ihl>).

How we think about these problems has to change as well. Increasingly public health experts are encouraging clinicians and others concerned about obesity and diabetes to consider a more systemic way of thinking about the enormity of the problem.⁸² Championing such change, the CDC Syndemics Project, proposes that this phenomenon be addressed as a *syndemic* (a complex and widespread phenomenon in population health produced by multiple reinforcing condition).^{83,84} The term syndemic:

... places the responsibility for affliction squarely within the public arena. It acknowledges relationships and signals a commitment to studying population health as a fragile, dynamic state requiring continual effort to maintain and one that is imperiled when social and physical forces operate in harmful ways.⁸⁵

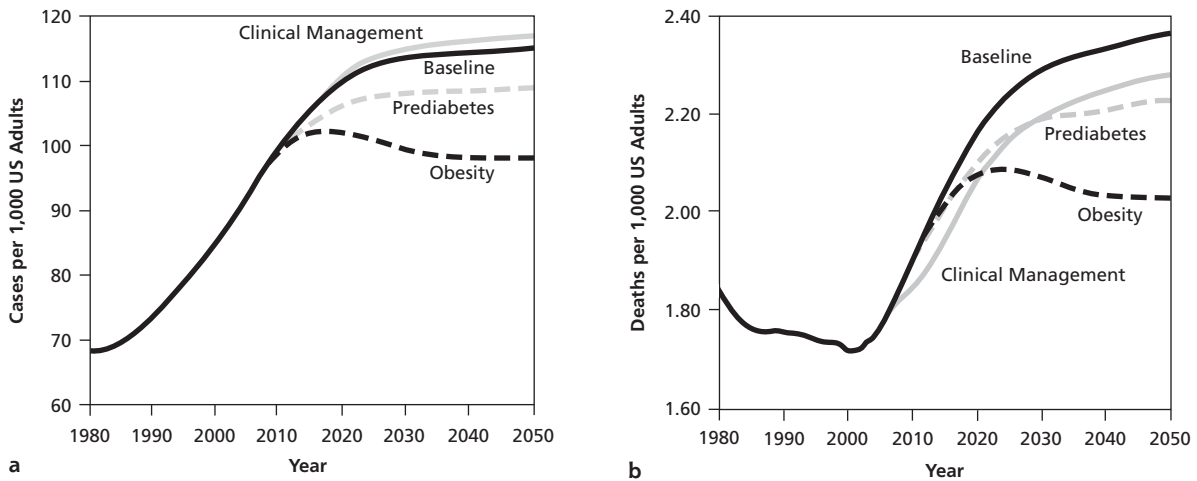
A syndemic orientation sees "dynamic feedback among afflictions, living conditions, and public strength."⁸³ Moving away from disease silos, specialty focus, single-issue organizations, and competition among interest groups for resources, these analysts of obesity and diabetes opt for an activated public health initiative that draws on the energy of all these forces to create change. Indeed, a syndemic approach argues that the same energy that conspires toward the harmful outcome can be harnessed, Jujitsu-style, on behalf of promoting healthful change.

Working for change requires an understanding of what is happening and where it is going. Dynamic modeling is a method to project the extent of a problem going forward in time and to demonstrate the potential impact of single or multiple interventions (Figure 2).^{84,85} These images show graphically that enhanced clinical management of diabetes, as well as recognition and treatment of prediabetes, will have little effect on the magnitude of the epidemic and minimal reduction in deaths secondary to complications of diabetes. The strategy with the most powerful long-term effect in curtailing the escalation is reduction in the prevalence of obesity. Such modeling of complex public health systems incorporates environmental issues, resources, healthy and risky behaviors, delivery systems, and feedback from various interventions; the projections carry the potential to influence policy for the better.⁸⁶

A CALL FOR ACTION

What does all this have to do with the individual clinician and patient? Clinicians, by definition, work within

Figure 2. Model output for 3 intervention scenarios compared with the baseline scenario for diabetes prevalence (a) and complication-related deaths (b).



From: Jones AP, Homer JB, Murphy DL, Essien JD, Milstein B, Seville DA. Understanding diabetes population dynamics through simulation modeling and experimentation. *Am J Public Health*. 2006;96(3):488-494. Reprinted with permission from the American Public Health Association.

the health care system, and can join quality improvement efforts by adopting the chronic care model. This model relies on clinicians and the systems they work in to better address chronic diseases and on patients to undertake management of their illnesses. As the dynamic modeling suggests, however, excellent clinical care will not be enough to change the outcome down the road in 2050. Clinicians need to be involved in change at a broader level.

Clinicians will have to get out of the office, and health care institutions will have to reach out through community collaborations. Economic and social factors may limit patients' abilities to make changes in diet and exercise without a prior intervention at the community level. Clinicians might consider what concrete steps they might take to become involved in local efforts that would make a difference. For instance, health centers can make linkages with local fitness centers to make safe access to exercise more available to patients.⁸⁷ Clinicians in the inner city can open conversations within community groups and leaders about how to support the location of supermarkets in inner-city locations, as the Food Trust did in Philadelphia (see <http://www.thefoodtrust.org/php/programs/super.market.campaign.php>). Clinicians can engage with patients around the community organizations that matter to them: churches, neighborhood associations, radio stations, etc. In these settings clinicians can take their message out of the office and into the community about what is a good weight, what are healthy foods, the need for increased physical activity, and, for immigrants, how work and activity are different in the new land. Ethnic group associations can

promote group education in comfortable settings and suitable language to raise awareness among immigrant families about the health concerns facing them and their children.⁸⁸

Community-based nutritional health and activity interventions have the potential to make a modest public health impact.^{89,90} Many different kinds of communities have had success in fostering community gardens, opening farmers' markets, increasing children's exercise through walk-to-school programs, promoting safe bike and walking paths, and altering the foods available in school lunches and snack machines.⁷³ At the larger political level, consumers and professionals can become involved in national efforts to reduce advertising of foods to children, especially on television, putting taxes on junk foods with revenue to support availability of fresh fruits and vegetables, improving the nutritional quality of foods aimed at children, pushing for a code of conduct for the food industry,^{73,77} or getting involved in politics around the US farm policy.⁶⁸ Taking an active part in working for change in nutrition is likely to work against clinicians' sense of helplessness in the face of the pandemics of obesity and diabetes. At the academic level, clinicians can become involved with the CDC Diabetes Prevention Project, joining forces with others who understand the multifactorial nature of the problems and the necessary solutions.

Primary care researchers as well can play a role. Many questions beg for answers:

- How can we foster healthy eating and activity levels without reinforcing the culture of distorted thinness and its potential for anorexia?

- How can we communicate the urgency of action on healthy activity and eating without further marginalizing and stigmatizing the people who are already overweight and obese?

- What is the best way to engage members of various ethnic groups to take up this challenge?

- Given the long-term ineffectiveness of current individual treatment adult and child obesity, what interventions might work using the chronic care model? Group visits? Multiple family group visits?

- Does role modeling by physicians and other health care workers have any effect on the uptake of healthy behaviors by patients?

Exploring the answers to these and so many more questions needing answers will require qualitative skills and an interest in narrative to gain patients' perspectives. Because these investigations involve vulnerable populations, a participatory research strategy would be the ideal approach.

Practitioners increasingly confront vulnerable patients with obesity, metabolic syndrome, and diabetes, and will soon have to address the huge resultant burden of vascular disease. Although the clinician faces each patient as an individual, or at best, as part of a family, the problems underlying the patient's health risks are not primarily those of individual responsibility, but rather the results of complex factors at work at multiple levels from the cellular to the global. Applying the concept of syndemics will enable the clinician to situate the health problems of vulnerable patients within their genetic, socioeconomic, and migration histories played out in the arena of worldwide economic forces. Whereas individual patients will still require one-on-one and familial interventions, the application of the community linkage aspect of the chronic care model holds the potential for joining with patients and their families to make a difference. Although this work is an uphill battle against prevailing political forces, new tools emerging from syndemic thinking, such as dynamic modeling, point toward more-effective strategies of prevention. Growing collaborations among researchers and clinicians who understand the need for interventions at multiple points can energize those who are committed to reshaping health care and laying the groundwork for lasting prevention of disease among families from vulnerable communities.

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Key words: Obesity; diabetes; poverty; emigration and immigration; minority groups; nutrition; globalization; syndemics; public health practice

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