The CDC's Diabetes Systems Modeling Project:
Developing a New Tool for Chronic Disease Prevention and Control

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The analytic tools presently used for planning and evaluating health policy are not well suited to capture the dynamic complexity of chronic diseases, which involve long delays between causes and health consequences. To explore the potential of system dynamics for addressing chronic disease policy, the U.S. Centers for Disease Control and Prevention (CDC) commissioned a dynamic model of diabetes prevalence and complications that can be used for designing and evaluating intervention strategies. The rapid growth in diabetes in the U.S. and elsewhere has been driven by a similarly rapid growth in obesity, which is a leading risk factor. The model was developed through a series of meetings over several months with participants from the CDC Division of Diabetes Translation and other Divisions with relevant expertise. The CDC and other public health stakeholders may use the model for policy analysis at the national, state, and local levels.

The Public Health Challenge of Chronic Disease

Chronic disease is a growing problem throughout the world, due in large part to increased longevity and the aging of the population—one-third of the chronically ill being over 65—but also due to numerous environmental factors. In the U.S., chronic diseases cause more illness, disability, and death than acute diseases and accidents combined, and are responsible for at least 70% of all health care expenditures (Hoffman et al. 1996, Institute for Health and Aging 1996). More than half of the adult population in the U.S. has at least one chronic disease, and about half of those have more than one such affliction and are responsible for the great majority of the total cost of chronic disease (Institute of Medicine 2001).

Most health planners who work on chronic diseases such as diabetes acknowledge the need for ecological, system-wide interventions. However, their desire to achieve systemic change stands in opposition to what most program staff and researchers are prepared to do. Ingrained in conventional analytic methods and planning frameworks is the idea that each aspect of a complicated disease control strategy can be managed and evaluated separately. Guided by these tools, stakeholders usually act independently even though they depend on one another to achieve comprehensive health goals for the public. In addition, many health problems, particularly chronic diseases, involve long delays between causes and the health events that individuals experience. Diabetes is a good example of this type of problem: it defies quick fixes and demands highly-coordinated, system-wide interventions.

The analytic tools presently used for planning and evaluating health policy—primarily, diagrammatic logic models and epidemiological forecasting models—are not well suited to capture the dynamic complexity of chronic diseases. This analytic gap may be overcome by developing system dynamics simulation models. A system dynamics model can focus on a specific disease process, while incorporating a deeper understanding of how the surrounding health system functions (physically, socially, and organizationally). It can also formalize the collaborative nature of public health practice through group model building, while strengthening the scientific foundation and active policy focus that are the hallmarks of health protection (Homer and Milstein 2003, Homer et al. 2004).

Public health officials are frustrated with approaches that are narrow and focus on just one part of the overall health system—say disease management, or detection, or risk factor reduction—rather than addressing all major parts in their entirety *as a system*. Innovative leaders are searching actively for an integrative, system-wide perspective that avoids this "silo" problem. In this context, public health agencies throughout the U.S. have been encouraged to assess both their internal and collective capacity to deliver the full spectrum of essential services related to disease prevention and control (CDC 2003). As these assessments expose both strengths and gaps in service capacity, questions of priority and strategic direction come to the forefront. Integrative models could help the CDC and other stakeholders to establish priorities in a rational way that effectively promotes health in the short- and long-term.

Growth of Diabetes in the United States and its Causes

Diabetes mellitus is a complex metabolic disorder marked by abnormally high blood glucose levels. If left untreated, the complications of diabetes can be disabling and ultimately fatal. Diabetes affects at least 18 million people in the U.S., a number that has been growing more rapidly than the general population since 1990. The rapid growth has occurred among those who have the non-insulin dependent Type 2 variety of the disease (formerly known as adult onset diabetes), as opposed to among the one million or so who have insulin-dependent Type 1 diabetes (which almost always strikes in childhood). Total costs of diabetes in the U.S. in 2002 were estimated to be $132 billion, with $92 billion of that in direct medical expenditures and the other $40 billion in indirect costs due to disability and premature mortality (NIDDK 2004a).

Figure 1 presents a graph for 1980-2000 of the fraction of the adult population (age 18 and over) already diagnosed with Type 1 or Type 2 diabetes (CDC 2004c). The decade-plus growth trend in prevalence, and its apparent acceleration starting at the end of the 1990s—which by all accounts has continued into the 2000s—has led many observers to conclude that the U.S. is experiencing a diabetes epidemic (Gorman 2003). This perception is supported by a recent Markov modeling study that has forecasted a 120% increase in the fractional prevalence of diagnosed diabetes during 2000 to 2050, assuming no change in rates of diabetes incidence by age, sex, race, and ethnicity (Honeycutt et al. 2003).
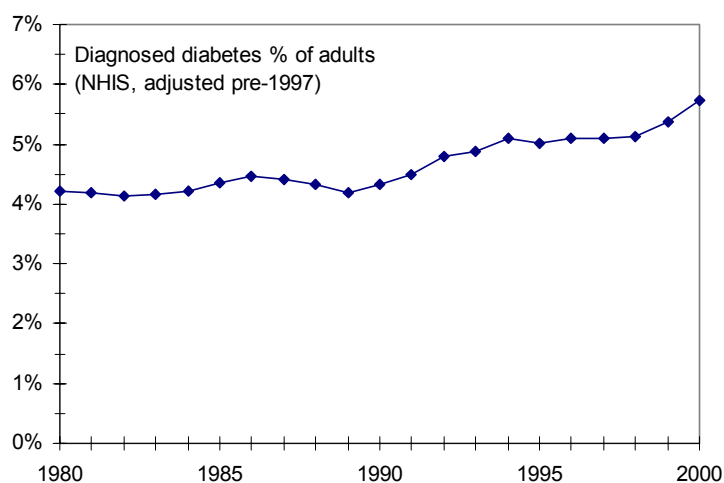

Figure 1. Historical trend of diagnosed diabetes in the U.S.

Concern about growing diabetes prevalence is also based on observed growth in the prevalence of obesity, a leading risk factor for diabetes, heart disease, and several other chronic diseases (NIDDK 2004b). If one considers that it usually takes many years for someone at high risk for diabetes to actually develop the disease, with incidence rates being the highest for the middle aged and the elderly (Narayan et al. 2003), then growth in obesity now may be seen as portending continued growth in diabetes in the future. Figure 2 shows how the obese fraction of the adult U.S. population, which grew only slightly from about 13% to 15% during the 1960s and 1970s, rose increasingly fast during the 1980s and 1990s, and now stands at over 30% (CDC 2004b). (Obesity is defined as having a body mass index of 30 or more; BMI = kilogram weight divided by the square of metric height.) Comparing the two trends presented in this figure, one

may say that the growth in diabetes in the 1990s was foreshadowed by similar growth in obesity in the 1980s. The fact that obesity continued to grow rapidly through the 1990s may suggest similar growth in diabetes in the 2000s.
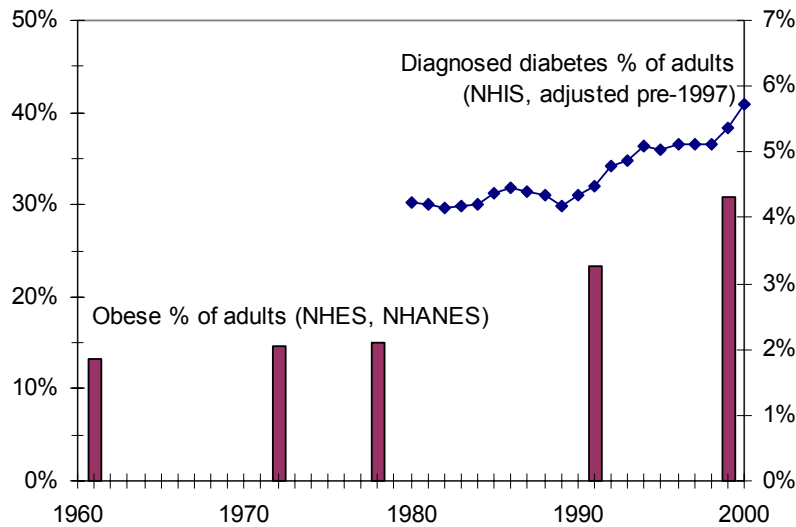

Figure 2. The rise in diabetes has followed a rise in obesity

What has caused the remarkable rise in obesity in the U.S. since 1980? From a thermodynamic standpoint, the documented increase in BMI can only have been the result of caloric intake persistently exceeding caloric expenditures. In regard to caloric expenditures, there are three types: physical activity, resting metabolism, and digestion (Abdel-Hamid 2002). Much has been written about Americans having a more sedentary lifestyle than they did in decades past, but available evidence points to no particular decline in physical activity since 1980. Indeed, the fraction of U.S. adults who say that they get no leisure-time physical activity has been flat or declining since 1990, though still in the 25% range as of 2001-2002 and certainly a continuing cause for concern (CDC 2004a). Neither does available evidence point to reductions in rates of resting metabolism (related to body weight and its fat-lean composition) nor digestion (an essentially fixed 10% of caloric intake).[1]

Rather than a decline in caloric expenditure, what appears to have fueled the increase in BMI and obesity since 1980 is an increase in caloric intake, a conclusion reached by others as well (Cutler et al. 2003). Figure 3 presents data on caloric intake from the National Health and Nutrition Examination Survey (NHANES; see CDC 2004b), representative of the entire U.S. adult population of men and women. According to these data, caloric intake increased by 13% during the 1980s and early 1990s, but has increased rather little since then.[2] The slowdown in the

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[1] An analysis we have done of skinfold measurement data from NHANES (CDC 2004b), applying well-known body density equations of Durnin and Womersley (1974), suggests a significant increase in the fat fraction during the 1970s for both men and women, but since 1980 only a small further increase in the fat fraction for men and a clear (and perhaps surprising) decline in the fat fraction for women.

[2] Among other time-series data we have seen, some support the idea of a very significant 1990s slowdown in the growth of caloric intake, while others are less supportive. Data from the U.S. Department of Agriculture on calories

growth of caloric intake may be a sign of hope that the obesity epidemic will soon reach its peak, though what this means for future diabetes prevalence is less obvious.
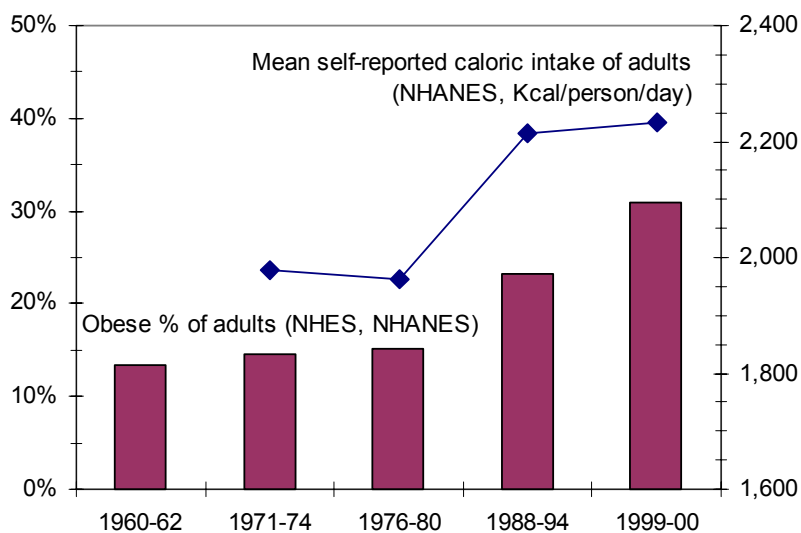

Figure 3. The rise in obesity has followed a rise in caloric intake

The National Diabetes Prevention and Control Program

The U.S. Government responded to the growing burden of diabetes in 1975 by establishing, within the U.S. Centers for Disease Control and Prevention (CDC) a "National Diabetes Prevention and Control Program" (NDPCP). As the emerging science supported new public health responses and as resources became available, the strategy for the NDPCP has evolved through several phases (Murphy et al. 2004).

In the late 1970s, interventions focused on educating diabetes patients and health professionals on the proper care of people with diabetes, aiming towards reducing diabetes-related complications such as blindness, amputations, and heart failure. In the 1980s, research supported the addition of screening for diabetes-related complications, with a particular focus on uninsured people with diabetes. In the 1990s, when two landmark studies established that intensive control of blood sugar could reduce the incidence of complications (DCCT 1993, UKPDS 1998), the strategy shifted again. The NDPCP enhanced its patient and provider education (e.g., incorporating the new disease management practices) but began to minimize individual

available and pounds of food consumed (U.S. Census Bureau 2003, Section 3, Tables 193 and 195, respectively), suggest a slight slowdown only. More supportive, though another step or two removed in causality, are data on cigarette smoking and income inequality. Studies show that smoking cessation leads to greater caloric intake (Flegal et al. 1995). The prevalence of smoking in the U.S. fell rapidly in the 1980s but much less so in the 1990s (NCHS 2003). Income inequality has been shown to be linked to emotional stress and mortality, across social and economic classes (Wilkinson 1996). Stress, in turn, has been implicated as a driver of weight gain, most clearly through a neuroendocrine pathway that involves the impairment of satiety perception and thereby leads to more caloric intake (Björntorp 2001). The commonly used Gini Index of income concentration rose rapidly in the 1980s, but was essentially flat for most of the 1990s (U.S. Census Bureau 2004, Table RDI5).

screening activities. Around the turn of the century, the program began to focus more on population-level or "system-based" interventions and less on direct clinical services to individuals with diabetes. The new focus included more partnering with other organizations and stakeholders. It also included the establishment of national objectives for testing people for diabetes, testing for diabetes-related complications, and preventing complications such as the flu. Finally, as a new landmark study (DPP 2002) demonstrated that effective treatment could reduce diabetes incidence in people with "prediabetes" (that is, elevated blood glucose levels that are not yet in the range of diabetes), NDPCP began to include management of prediabetes as well.

In recent years, public health officials have looked even further "upstream" towards the factors driving the incidence of prediabetes. While age and demographics are important factors in determining an individual's risk for prediabetes and diabetes, the primary changeable risk factor is obesity. Public health officials are now paying significant attention to the obesity epidemic in the U.S., and recognize that diabetes prevalence may grow further than previously forecasted should the prevalence of obesity continue to increase. Such attention led the designers of the NDPCP to consider not just reducing the incidence of diabetes by focusing on those with prediabetes, but also reducing the incidence of prediabetes by controlling obesity.

By 2004, CDC staff in the Division of Diabetes Translation (DDT), which administers the NDPCP, had grown to better understand the influential role that other organization's programs and policies have in focusing on diabetes prevention and achieving diabetes-related goals and objectives. The Division has come to appreciate that efforts to prevent, control, and manage diabetes are tied, in multiple ways, to most other chronic diseases and to a wide array of social and physical processes at work in communities (i.e., food production and distribution, transportation, education, etc.). It has become clear that efforts to address the increasing burden of diabetes will necessarily involve expanding and reconfiguring the kinds of resources that fuel public health work (Murphy et al. 2004).

Today, with the nation's diabetes prevention and control program poised for further transformation, many strategic questions remain unanswered. How can public health officials engage new partners in redirecting the course of the diabetes epidemic? How can these diverse actors build confidence that prevention activities in the present will yield reduced rates of disease in the future? How can policy makers assess the inherent trade-offs among competing strategies? And most importantly, what conditions must exist for people to take action based on their understanding of how a dynamic diabetes system behaves?

The Modeling Project

In order to address the challenge of diabetes growth, a group of CDC leaders decided to employ system dynamics modeling as a tool for enhancing both learning and action. Through a partnership with system modelers at the Sustainability Institute and the Rollins School of Public Health at Emory University, staff from the DDT and several other CDC Divisions worked together to create an interactive, SD model-based "learning lab" that enables multiple stakeholders to design and formally evaluate diabetes-related policies.

The learning lab technique engages multiple stakeholders in a facilitated process that is designed to explore the structural reasons for observed health trends. The process begins by integrating research findings and insights from practical experience into a dynamic hypothesis of the health problem. Next, this hypothesis is translated into a formal simulation model using a system of differential equations and specialized SD modeling software. The model creates a virtual world for learning in which controlled experiments can be conducted as a prelude to introducing policies in the real world. Stakeholders use the model to search for policies that can deliver long-term health impacts, while simultaneously guarding against interventions that lead to unintended effects. Users can also formally compare and evaluate prevention policies that would otherwise have to be tested through trial and error in real populations.

Eventually DDT staff and their state-level colleagues, with training and support from SD practitioners, may begin facilitating model-based learning labs that provide a forum for diverse stakeholders to think together about the dynamics of diabetes strategy.

Developing the Model: Meetings and Participants

Between September 2003 and March 2004, the modeling team constructed a system dynamics model focused on the drivers of increasing diabetes prevalence. The process involved a series four meetings, each taking place over two days.

Meeting 1: Model Conceptualization
After two planning meetings with a team of CDC "project champions", during which a preliminary population stock-and-flow structure was developed, DDT leaders convened a two-day conceptualization meeting in early December 2003. Approximately 45 individuals participated, including CDC staff with a specialty in diabetes prevention and control, as well as representatives from other CDC Divisions that focus on factors within the broader "diabetes system", including nutrition and physical activity, smoking, cardiovascular health, adolescent and school health, adult and community health, and chronic disease in general. The agenda for the first day included time for reviewing the principles of SD methodology; brainstorming dilemmas that arise when confronting diabetes and its many drivers (from obesity to health care access and utilization); and mapping the relationships among those drivers.

That evening, the modeling team simplified what had become a sprawling map of drivers into a tighter, more generalized sector diagram. On the second day, the agenda included time for: discussing the simplified sector diagram; sketching behavior-over-time graphs to explain how various parts of the system have been changing; identifying sources of empirical data; and setting overall priorities for this phase of the project.

Meeting 2: Interacting with a Simulation Model
One line of work undertaken to complement the development of the simulation model involved educating DDT staff in how one can use a system dynamics model to improve strategy and spark effective action. To this end, the modeling team developed a simple user interface for the core stock-and-flow model, and in January 2004 gave staff members an opportunity to experiment with it in teams of two in a computer lab. The intention was not to analyze specific policy

scenarios, but rather to acquaint public health professionals with what a dynamic simulation model can do. On the second day, the modelers also met with several CDC scientists who specialized in various aspects of the diabetes system in order to incorporate their ideas into the model's emerging structure.

Meeting 3: "Pushing on the Model"
A second line of work focused on developing a widely credible simulation model of the diabetes system. This required intense collaborative work, among modelers and subject experts, to articulate theories for the growth in diabetes prevalence; gather data to support or refute those theories; translate those theories into differential equations; test the resulting model; and calibrate the model with reference to historical trends. By February 2004, the emerging model had progressed to the point where it could be critiqued by the project champions group. Again, the focus was not on policy analysis but on potential improvements that would make the model more useful in its goal of supporting effective conversations about diabetes prevention strategy.

Meeting 4: "Letting the Model Push on Us"
The fourth meeting will occur at the end of March 2004 and is designed around a second iteration of the simulation model. On the the first day, we will observe how the model functions in a conversation about diabetes strategy. Approximately 15 CDC staff members, whose jobs entail a variety of roles in the diabetes system, will gather to experience for themselves how an SD learning lab works. Two members of the modeling team will facilitate the event, while a third observes the proceedings. With that experience in mind, the champions group will reconvene on the second day to reflect on the model and the learning lab experience, as well as next steps for widening the dialogue and acting on the main dynamic insights.

Model Structure

As the diabetes system model has evolved, we have continually sought to create a structure that is

(1) generic enough to be adaptable for other chronic diseases;
(2) realistic enough to reproduce national-level historical data on the prevalence of diabetes, prediabetes, and obesity;
(3) comprehensible enough to test practical policies without disaggregating the population into demographic categories of age, sex, race/ethnicity, or other individual attributes;
(4) broad enough to encompass a spectrum of policy measures that are being considered; and
(5) grounded enough in empirical experience that it does not require speculation beyond what the project participants themselves could agree upon or what credible evidence could support.

The preliminary stock-and-flow model, which began the iterative process described above, was based in many of its structural details and even some of its calibration, on a model of diabetes developed for a chronic disease healthcare initiative in Whatcom County, Washington State (Homer et al. 2004). Other system dynamics models have also examined chronic disease care (Hirsch and Wils 1984, Hirsch and Immediato 1999), and they too informed our work.

From the outset, this project recognized a need to reach further "upstream" in the causal chain of disease incidence and progression than the Whatcom County model had done (McKinlay and Marceau 2000, Milstein and Homer 2003). This would mean modeling, in health policy terms, not only disease management and control (also described as secondary and tertiary prevention) for those already afflicted and primary prevention for the at-risk or vulnerable population, but even actions that can protect people from becoming at-risk in the first place. Figure 4 presents the generic view of chronic disease dynamics that came out of these discussions. Healthcare professionals, in their roles as service providers, commonly oversee the downstream activities of secondary and tertiary prevention that are portrayed in this figure. Such professionals may also be involved in primary prevention when their work focuses on risk management or health education—for example, guiding obese individuals into diet and exercise programs and regularly monitoring their progress.

In contrast with these professionally led healthcare services, the upstream activities of health protection focus on changing those living conditions that leave people vulnerable to disease, either in a targeted way for people already at risk, or in a broader way for entire populations. Efforts to improve adverse living conditions are in the natural domain of advocacy and citizen groups, rather than healthcare professionals; they entail a level of organized public action that downstream services do not typically require. For financial and organizational reasons, particularly those stemming from pressures to report near-term success in the form of traditional health outcome measures, upstream efforts typically receive only modest support from public health agencies (see Murphy et al. 2004). The tendency to "lock in" to downstream efforts may be shaken loose only when it finally becomes apparent that such efforts cannot, unto themselves, be effective in protecting the public's health (Milstein and Homer 2003).
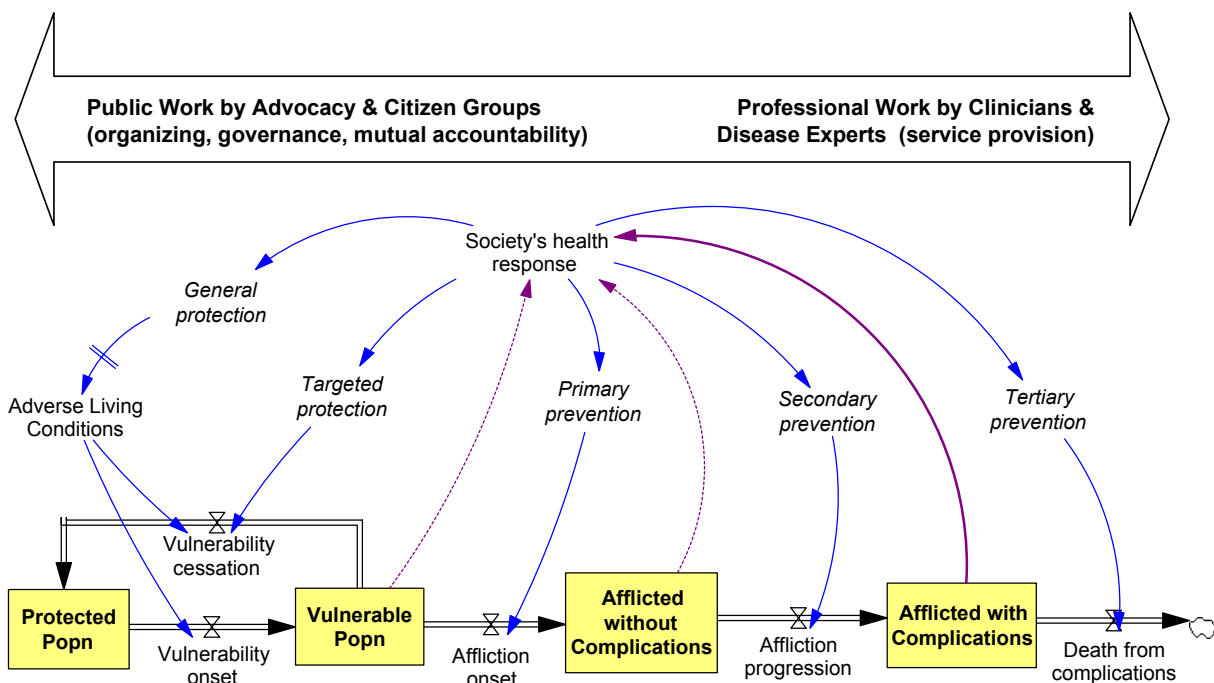

Figure 4. Generic chronic disease population stocks and flows and health system responses

Figure 5 presents the population flow structure that forms the core of the diabetes system model. This structure evolved during the course of the project to reflect the knowledge and policy concerns of project participants. It portrays the movement of people into and out of the following stages:

- Normal blood glucose (normoglycemia);
- Prediabetes, defined as having either impaired glucose tolerance (IGT, non-fasting glucose concentration of 140-199 mg/dl) or impaired fasting glucose (IFG, 100-125 mg/dl) (ADA 2004, Benjamin et al. 2003);
- Uncomplicated diabetes, that is, meeting the testing criteria for diabetes (non-fasting glucose of at least 200 mg/dl) but not yet symptomatic nor showing detectable signs of disease in the eyes, feet, or other organs; and
- Complicated diabetes.

The prediabetes and diabetes (hyperglycemic) stages are further divided into stocks for the undiagnosed and diagnosed population. Diagnosis is important dynamically because it is a prerequisite for proper management and control of hyperglycemia; and control, in turn, strongly reduces the rates of diabetes onset, progression, and death (DCCT 1993, UKPDS 1998, DPP 2002, ADA/NIDDK 2002, Bowman et al. 2003). Because prediabetes and uncomplicated diabetes are not marked by symptoms, their diagnosis generally occurs only as a result of screening that a healthcare professional might do periodically (every three years, according to guidelines) for patients at risk for diabetes by virtue of family history, obesity, age, and other risk factors. While there is currently still some progress to be made in getting providers to test for uncomplicated diabetes, the testing gap for prediabetes is much larger because guidelines for the testing and treatment of prediabetes are relatively new and still somewhat fluid. Complicated diabetes is more readily detected than uncomplicated, because of suspicions aroused by presented symptoms. Even so, a significant fraction of complicated diabetes is not detected rapidly due to issues of provider inattention or the patient's lack of insurance or convenient access to a provider (McKinlay and Marceau 2000).
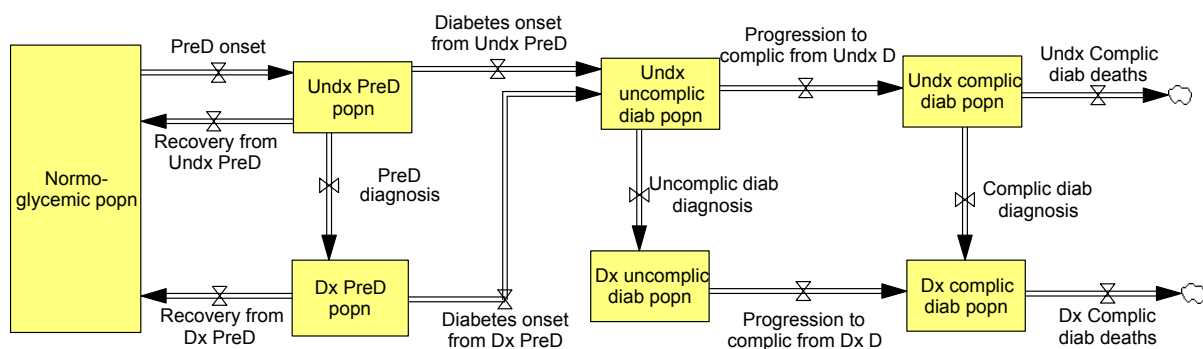

Figure 5. Diabetes and prediabetes population stock and flow structure as modeled
(Undx: undiagnosed; Dx: diagnosed; PreD: prediabetes; D: diabetes.)

This is a simplified view of the full stock and flow structure. The full structure also includes an inflow into the normoglycemic stock for general population growth, and non-diabetes-related death flows out of each population stock.

Figure 6 presents an overview of the full model, which includes the population stock-and-flow structure as well as the main influences that affect the rates of population flow throughout the system. The diagram also highlights (in red) those inputs that are seen as major influences affecting the upstream and downstream flow-rate drivers, and which may be amenable to policy intervention. Upstream, these exogenous policy-related variables include caloric intake and physical activity as drivers of obesity. Downstream, they include testing rates and healthcare access as drivers of disease detection; and clinical management, self-management, and drug affordability as drivers of disease control.
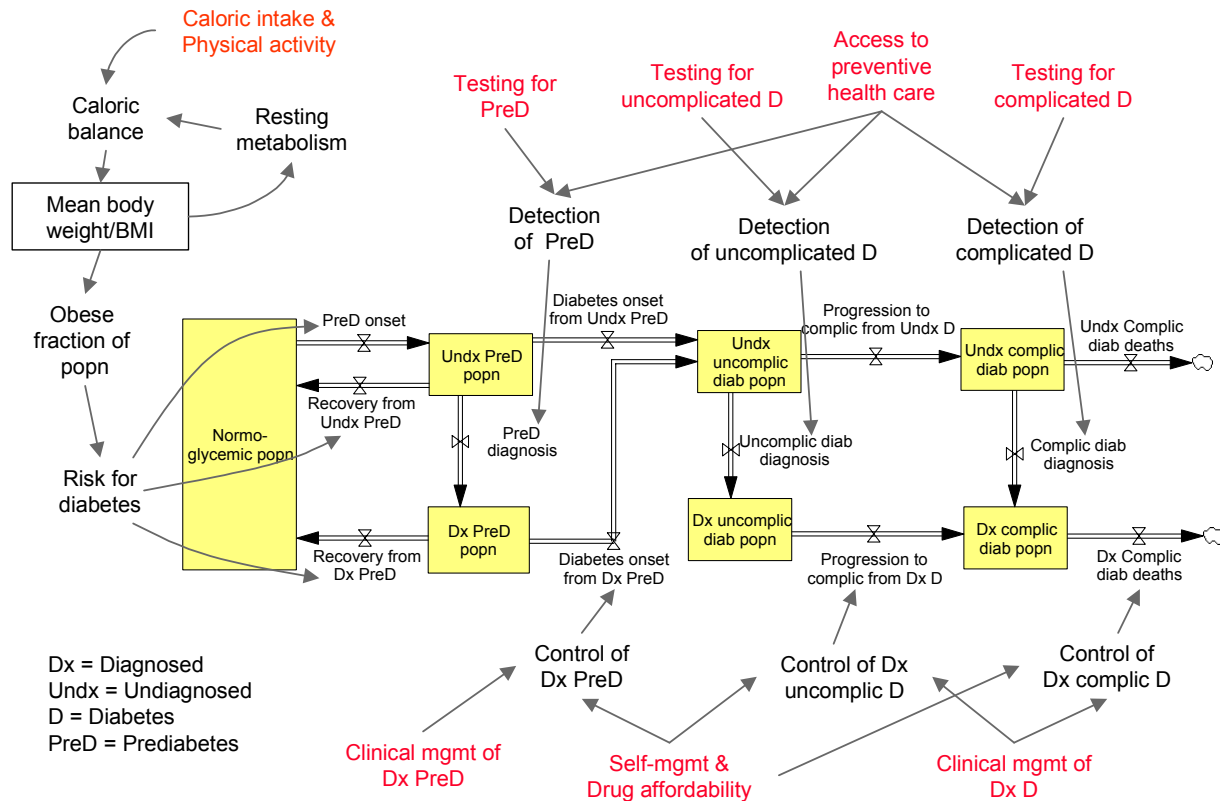
Figure 6. Overview of full model, showing key drivers of population flows
and inputs amenable to policy intervention (in red)

It is notable that the model does not explicitly address the social, economic, and personal determinants behind such factors as caloric intake and the ability to self-manage, but instead leaves such forces outside the model's boundary. Our conceptualization meeting in December 2003 involved extensive discussion of such factors as well as the intricate interactions among them. Despite intense interest in these drivers, the decision to keep them outside the model boundary was made for two reasons.

(1) There was a lack of supporting evidence for hypothesized relationships. Following the December meeting, an effort was made to gather historical evidence and literature on as many of the determinants that had been discussed as possible. In many cases, the available evidence either did not support the hypotheses that had been presented, or was too sparse or full of qualifiers to allow for any firm conclusion. We ultimately concluded that we were delving into areas of speculation that would not enjoy general acceptance among the many stakeholders, both scientists and policymakers, identified as audiences for the initial model. Quantitative modeling of such speculative relationships might be acceptable under some circumstances, but not given the goals and needs of this particular project in its initial phase.

(2) Powerful insights could be gained from the stock-and-flow structure alone. At the December meeting, and then again in January 2004, project participants commented that the core stock-and-flow model by itself revealed counterintuitive dynamics that could have important implications for policy selection. The value to real-world clients of dynamically interesting stock-and-flow structures—perhaps as a first step of analysis before delving into behavioral feedback loops—has been discussed elsewhere, first as a general proposition with examples from various subject domains (Homer 1997), and more recently in connection with the modeling of diabetes for Whatcom County (Homer et al. 2004).

Baseline Model Behavior

The model has been calibrated based on historical data available for the U.S. adult population, as well as estimates from the scientific literature, and from diabetes clinicians who were interviewed as part of the Whatcom County study.[3] These data address the:

- Prevalence of diagnosed diabetes, prediabetes, and obesity;
- Fraction of diabetes undiagnosed;
- Complicated fractions of undiagnosed and diagnosed diabetes;
- Population growth rate, and death rates from diabetes complications and from all other causes;
- Effects of control (glucose, lipids, blood pressure) on rates of diabetes onset, progression, and death;
- Caloric intake;
- Mean body weight, height, and BMI;
- Fat fraction of body weight;
- Fraction of population with health insurance;
- Fraction of people with diagnosed diabetes receiving annual dilated eye exam;
- Fraction of people with diagnosed diabetes doing daily glucose self-monitoring; and
- Fraction of people who can afford diabetes medications.

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[3] Many of the data were obtained from online sources, including websites of the CDC (2004a, 2004b, 2004c, 2004d), the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK 2001, 2004a, 2004b), and the U.S. Census Bureau (2003, 2004). Data and empirical equations were also culled from the following published articles: Abdel-Hamid 2002, Benjamin et al. 2003, Durnin and Womersley 1974, and Honeycutt et al. 2003. Many thanks also to the CDC's Edward Gregg for extracting summary statistics from the NHANES database, spanning the 1960-2000 period, on weight, height, BMI, and skinfold measurements.

As described previously, the model contains a number of exogenous time series for which future assumptions are required for policy testing. In order to establish a baseline case against which alternative policies could be compared, we started by asking for project participants' best estimates of what a "status quo" future would look like, in which the NDPCP was frozen in place as it stands currently and no new initiatives were introduced. Interestingly, the participants felt this task was confusing and subjective, and ultimately infeasible. So, we suggested a different sort of baseline case, in which all exogenous time series were simply held constant at their assumed 2004 values. Despite the likely lack of realism of such a baseline for some variables (especially in the case of prediabetes testing and clinical management, which only got their start in the last few years and are still early in their growth path), the participants agreed to such a baseline because of its simplicity and clarity.

Figures 7 and 8 demonstrate the model's ability to reproduce the 1980-2000 data for diagnosed diabetes prevalence and obesity prevalence, respectively, as well as the model's baseline projections for these variables through 2010. Figure 9 takes the baseline projection out to the year 2050, illustrating the growth in the prevalence of hyperglycemia and its three major components: complicated diabetes, uncomplicated diabetes, and prediabetes.
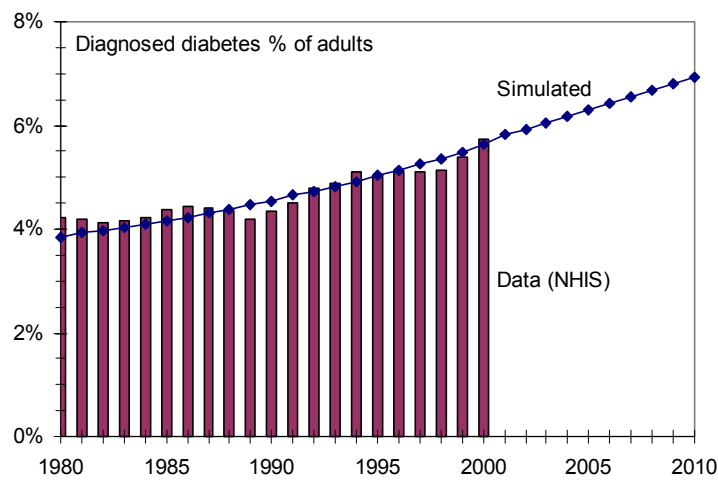

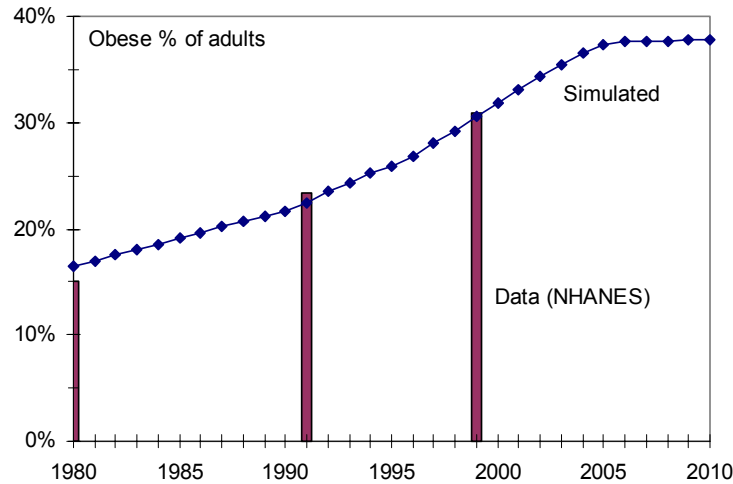Figure 7. Simulated diagnosed diabetes prevalence compared with historical data

Figure 8. Simulated obesity prevalence compared with historical data
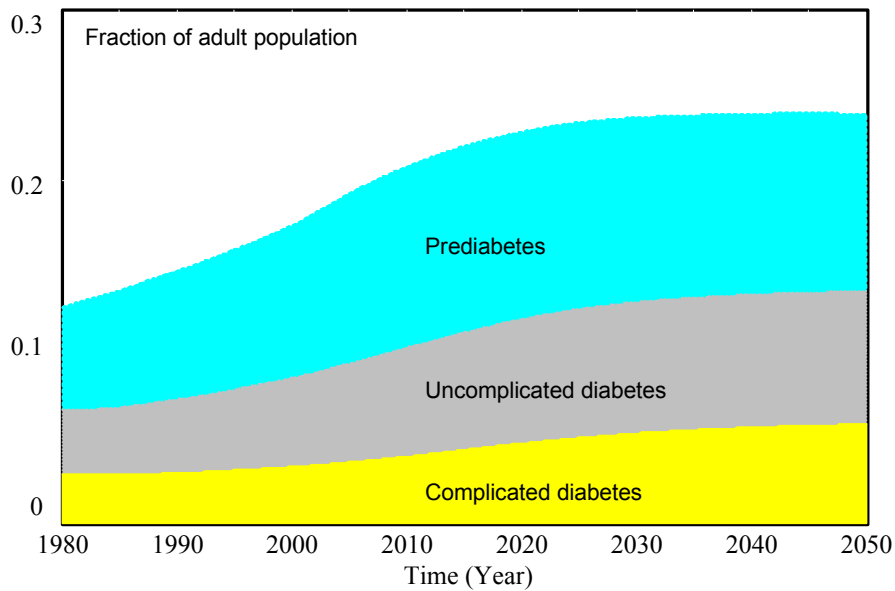

Figure 9. Baseline growth of diabetes and prediabetes prevalence through 2050

The story told by these graphs is as follows: About two years after the assumed cessation in the growth of caloric intake as of 2004, obesity prevalence soon ceases to grow as well. This goal-seeking behavior reflects the negative feedback loop seen in Figure 6, wherein higher body weight leads to a higher resting metabolism. Significant growth in hyperglycemia (diabetes and prediabetes combined) continues, in decelerating fashion, for another 25 years, reflecting the long delays in the system connecting obesity as a risk factor to prediabetes and then to diabetes. The prevalence of prediabetes peaks and then actually declines somewhat after 2020, due to the fact that the normoglycemic fraction of the population (1 – the hyperglycemic fraction), from which prediabetes draws, is itself on the decline. The growth of uncomplicated diabetes prevalence continues until about 2035, while the growth of complicated diabetes prevalence

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continues right through 2050. From 2000 to 2050, the prevalence of diabetes (diagnosed and undiagnosed) increases from 8.4% to 13.5%, while the prevalence of diagnosed diabetes per se increases from 5.6% to 9.5%.[4]

Illustrative Policy Tests

Project participants will soon have an opportunity to do policy testing of the model in a workshop-type environment. They will be asked first to describe a policy strategy that they feel is feasible, and second, to specify the plausible impacts of that strategy in terms of the time-series inputs identified in Figure 6. We expect that three broad strategy types may be explored: a pure downstream approach, a pure upstream approach, and a balanced approach of both upstream and downstream interventions.

We have created sample simulations of these three scenarios and compared them with the base run in Figures 10 to 14. The runs are as follows:

- *Base*　　　　　No changes after 2004
- *Mgmt90*　　　　A purely downstream approach, with clinical management of diagnosed diabetes and prediabetes ramped up to 90% during 2005-15. (Base case assumption for diabetes management = 70%; for prediabetes management = 50%.)
- *Cal3*　　　　　A purely upstream approach, with caloric intake ramped down by 3% during 2005-15.
- *Mgmt85Cal2*　　A balanced approach, with clinical management ramped up to 85% and caloric intake ramped down by 2% during 2005-2015.

Figures 10 and 11 illustrate graphically the assumptions we have made for the four runs.[5] It　is interesting to note, in Figure 11, that the 3% reduction of caloric intake in *Cal3* represents a mean reduction of "only" 74 kcal/person/day, and the 2% reduction in *Mgmt85Cal2* only 49 kcal/person/day, reductions that may sound too modest to have much discernible effect on body

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[4] As with most SD models, specific output values from this model are intended to be used only for understanding the dynamic implications of assumptions and comparing the effects of simulated policies. They do not correspond numerically to the values that are generated from predictive models that are designed to yield specific point estimates of diabetes prevalence, such as the Markov model by Honeycutt et al. 2003. Various effects of population aging, for example, have been excluded from our model in an effort to improve its transparency and therefore its value as a tool for learning about how dynamic complexity affects intervention policies in the overall diabetes system. In future phases, after users have developed a greater understanding of how and why the dynamic model works the way it does, we may incorporate aging and other factors that would increase the predictive accuracy of the SD model.

[5] Note that the balanced approach, *Mgmt85Cal2*, achieves 3/4 of the full downstream impact of *Mgmt90*, and 2/3 of the full upstream impact of *Cal3*. Let us call these fractions the downstream and upstream impact fractions, respectively, for the balanced approach. Now, think of the three intervention approaches as alternative ways of allocating a fixed public health budget for diabetes. One might then suppose that the sum of the two impact fractions for the balanced approach should be 1, rather than a value greater than 1 as in our example (3/4 + 2/3 = 17/12 > 1). The two impact fractions would, in fact, sum to 1 if the downstream and upstream policies had impacts that were strictly proportional to their budgets; in other words, if they had constant returns to scale. But if the returns to scale are diminishing, as is true of many (and, beyond some point, all) policies, then the sum of the impact fractions for a balanced approach would be greater than one, as in our example.

weight. But other analysts have concluded in a similar vein that further weight gain in the U.S. could be eliminated by reducing the mean caloric balance (intake less expenditure) by 100 kcal/person/day or less, which could be achieved by eating just a few bites less at each meal (Hill et al. 2003). Such a task sounds easy enough for a select group of individuals in an experimental study, but to reduce the mean daily intake by 50-100 kcal/person/day across a population of more than 200 million adults is, in fact, a considerable challenge, and would require a society-wide reaction against those cumulative social forces and adverse living conditions that have caused caloric intake to increase rather than decrease for the past 20-plus years.

Public health officials want to be able to anticipate the endpoint results of their policy decisions in terms of reduced morbidity, mortality, and healthcare costs. One of the things our project participants—and also we—have come to realize with respect to chronic diseases is that it may be just as important for these officials to understand the intermediate effects by which they may gauge progress during the long years that often separate policy intervention and endpoint results. Figures 12 and 13 represent two examples of such intermediate effects. They show policy impacts on prediabetes prevalence and complicated diabetes prevalence, respectively. These graphs illustrate the value of the upstream approach for reducing prevalence, but also the fact that it will take many years for the full benefits of prevention to be felt at the symptomatic end of the disease chain.
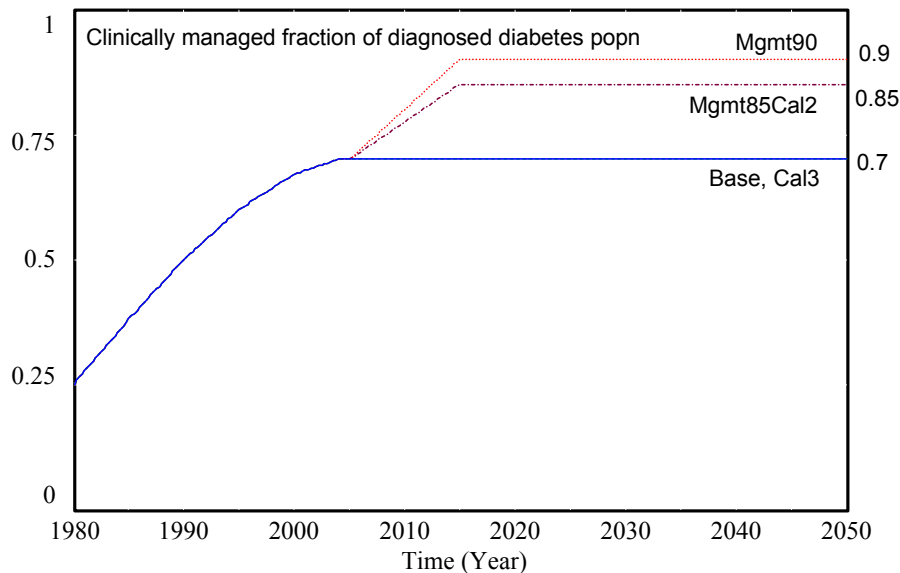

Figure 10. Assumed clinical management fraction, four policy tests
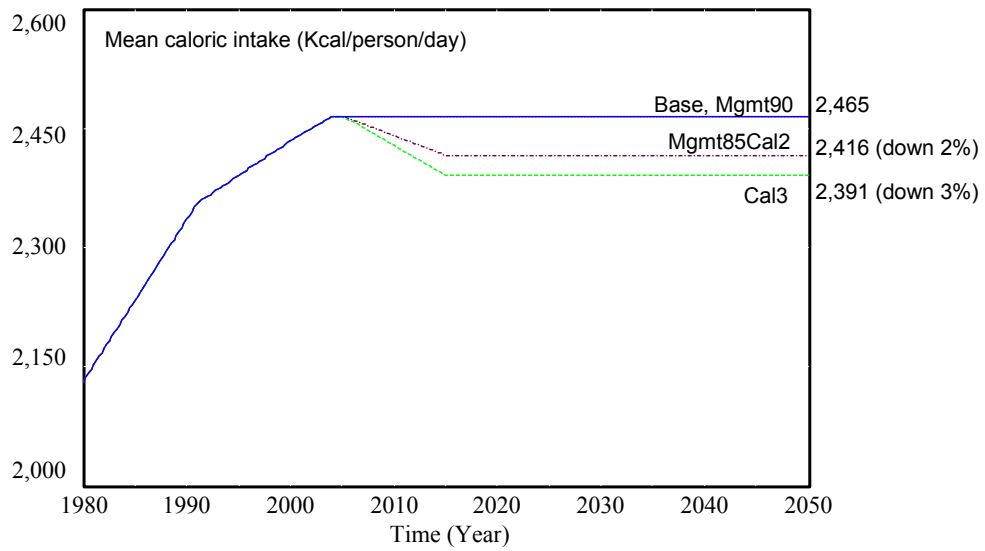
Figure 11. Assumed caloric intake, four policy tests
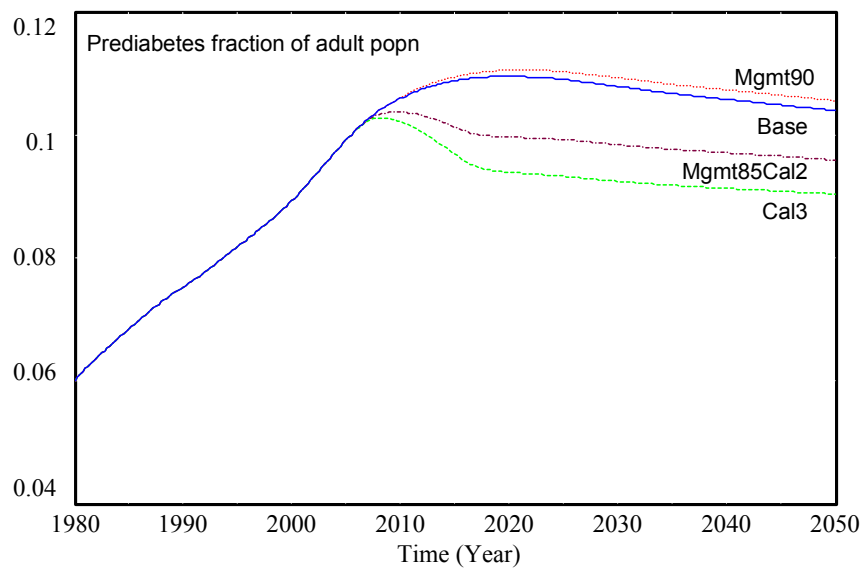

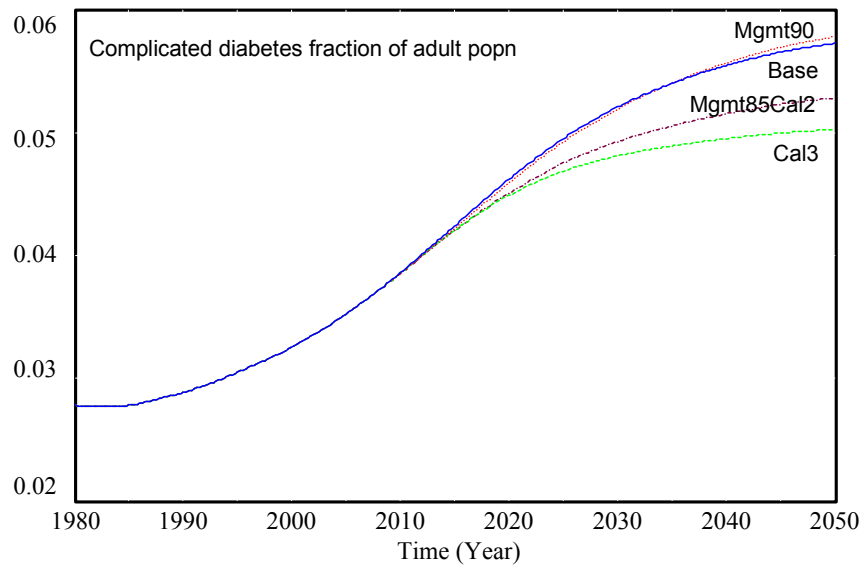Figure 12. Simulated prediabetes prevalence, four policy tests

Figure 13. Simulated complicated diabetes prevalence, four policy tests

Figure 14 shows the death rate from complications of diabetes, expressed as an annual fraction of the total adult population. This death rate represents the product of

(1) the prevalence of complicated diabetes, as seen in Figure 13, and
(2) the death rate among those with complicated diabetes, which improved clinical management—see Figure 10—can quickly reduce.

A comparison of the policy impacts in Figure 14 shows that the purely downstream scenario has the virtue of acting quickly, but does nothing to prevent a subsequent growth in the death rate along a trajectory parallel to that of the base run. The purely upstream scenario has the virtue of significantly reducing the growth trajectory of death, but does so only after a delay of 10 to 15 years. The balanced approach succeeds in both the short term and the long term, in effect paying attention to both those people who have the misfortune of progressing to complicated diabetes, but also to those who might otherwise ultimately progress to that state.
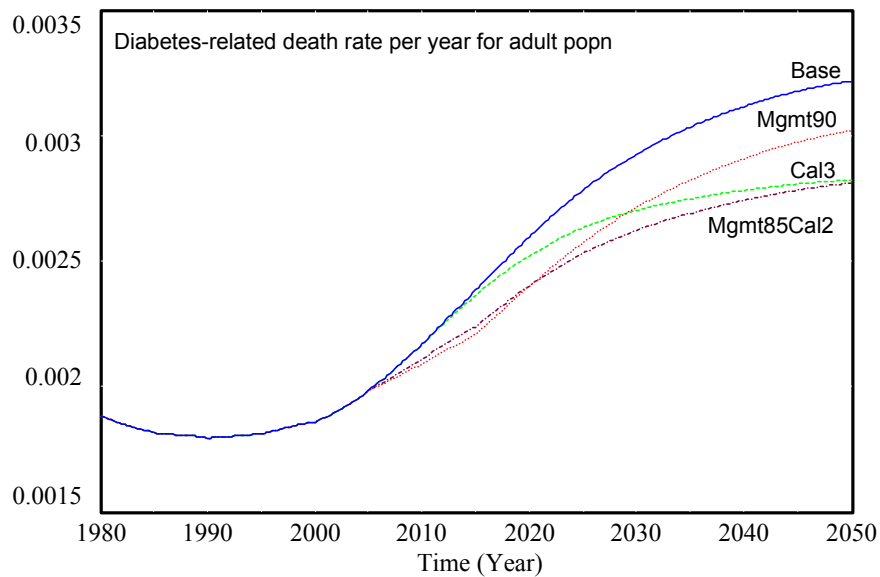
Figure 14. Simulated diabetes-related death rate, four policy tests

Making It Useful

The model is intended to help improve strategies for addressing the burden of diabetes at two levels: national and state/regional.

At the national level, the DDT may incorporate insights from this analytical work into its own strategy and its interaction with the other Divisions within the National Center for Chronic Disease Prevention and Health Promotion. This will likely happen through facilitated meetings that use the model as a framework for discussion and a tool for experimentation. The DDT will also consider opportunities to use the model as a catalyst for meetings with other national organizations working on diabetes prevention and control, such as the American Diabetes Association.

At the state/regional level, the current plan is for a small team of leaders within the DDT to learn to facilitate a model-based workshop we are calling the "Diabetes Action Lab." Participants in these one-to-two day workshops may include state and local public health officials along with a diverse group of stakeholders from non-governmental organizations such as healthcare professionals, leaders of not-for-profit agencies, people living with diabetes, and many others. The goal, after studying the short- and long-term dynamics of diabetes policy options, would be to improve the overall strategic direction for redirecting the course of the diabetes epidemic, and to spark more determined and coordinated action towards meeting explicit performance objectives.

For the Diabetes Action Lab to be effective in a wide range of settings for diverse participants, it must be embedded in a carefully designed learning environment, one that incorporates facilitated presentations, small group discussions, strategy exercises, and other supplemental learning techniques. It may also be necessary to adjust the model's calibration so that it reflects the

unique context of the health challenge in different states, based on local demographics, diabetes programs already in place, and other factors.

Acknowledgments

References

Abdel-Hamid TK. 2002. Modeling the dynamics of human energy regulation and its implications for obesity treatment. *System Dynamics Review* 18(4): 431-471.

American Diabetes Association (ADA). 2004. How to Tell if You Have Pre-Diabetes. Accessed at: http://www.diabetes.org/pre-diabetes.jsp.

American Diabetes Association and National Institute of Diabetes and Digestive and Kidney Diseases (ADA/NIDDK). 2002. The prevention or delay of Type 2 diabetes. *Diabetes Care* 25:742-749.

Benjamin SM, Valdez R, Geiss LS, Rolka DB, Narayan KMV. 2003. Estimated number of adults with prediabetes in the U.S. in 2000: Opportunities for prevention. *Diabetes Care* 26(3): 645-9.

Björntorp P. 2001. Do stress reactions cause abdominal obesity and comorbidities? *Obesity Reviews* 2: 73-86.

Bowman BA, Gregg EW, Williams DE, Engelgau MM, Jack, L Jr. 2003. Translating the science of primary, secondary, and tertiary prevention to inform the public health response to diabetes. *J Public Health Mgmt Practice* November(Suppl): S8-S14.

Centers for Disease Control and Prevention (CDC). 2003. *The Public Health Competency Handbook: Optimizing Individual and Organizational Performance for the Public's Health*. Atlanta, GA.

Centers for Disease Control and Prevention (CDC). 2004a. Behavioral Risk Factor Surveillance System (BRFSS). Accessed at: http://www.cdc.gov/brfss/.

Centers for Disease Control and Prevention (CDC). 2004b. National Health and Nutrition Examination Survey (NHANES, created in 1960 as NHES). Accessed at: http://www.cdc.gov/nchs/nhanes.htm.

Centers for Disease Control and Prevention (CDC). 2004c. National Health Interview Survey (NHIS). Accessed at: http://www.cdc.gov/nchs/nhis.htm.

Centers for Disease Control and Prevention (CDC). 2004d. Diabetes Surveillance System. Accessed at: http://www.cdc.gov/diabetes/statistics/.

Cutler DM, Glaeser EL, Shapiro JM. 2003. Why have Americans become more obese? National Bureau of Economic Research, Working Paper 9446. Cambridge, MA. Accessed at: http://www.nber.org/papers/w9446.

Diabetes Control and Complications Trial Research Group (DCCT). 1993. The effect of intensive treatment of diabetes on the development and progression of long term complications in insulin dependent diabetes mellitus. *New England Journal of Medicine* 329: 977-986.

Diabetes Prevention Program Research Group (DPP). 2002. Reduction in the incidence of Type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine* 346: 393-403.

Durnin JVGA, Womersley J. 1974. Body fat assessed from total body density and its estimation from skinfold thickness: Measurements on 481 men and women aged from 16 to 72 years. *British Journal of Nutrition* 32: 77-91.

Flegal KM, Troiano RP, Pamuk ER, Kuczmarski RJ, Campbell SM. 1995. The influence of smoking cessation on the prevalence of overweight in the United States. *New England Journal of Medicine* 333(18): 1165-70.

Gorman C. 2003. Why so many of us are getting diabetes. *TIME* (8 December): 58-69.

Hill JO, Wyatt HR, Reed GW, Peters JC. 2003. Obesity and the environment: Where do we go from here? *Science* 299 (7 February): 853-855.

Hirsch GB, Immediato CS. 1999. Microworlds and generic structures as resources for integrating care and improving health. *System Dynamics Review* 15(3):315-330.

Hirsch GB, Wils W. 1984. Cardiovascular disease in the Dutch population: A model-based approach to scenarios. *Ministry of Health: Conference on Health Care Scenarios*. The Hague, Netherlands.

Hoffman C. Rice DP, Sung H-Y. 1996. Persons with chronic conditions: Their prevalence and costs. *JAMA* 276(18):1473-9.

Homer JB. 1997. Structure, data, and compelling conclusions: Notes from the field. *System Dynamics Review* 13(4):293-309.

Homer J, Hirsch G, Minniti M, Pierson P. 2004. Models for collaboration: How system dynamics helped a community organize cost-effective care for chronic illness. *System Dynamics Review* (in press).

Homer J, Milstein B. 2003. Planning comprehensive public health programs: A promising opportunity for SD. Presentation to System Dynamics Winter Conference. January 9, 2003: Austin, TX.

Honeycutt AA, Boyle JP, Broglio KR, Thompson TJ, Hoerger TJ, Geiss LS, Narayan KMV. 2003. A dynamic Markov model for forecasting diabetes prevalence in the United States through 2050. *Health Care Mgmt Science* 6: 155-164.

Institute for Health and Aging, University of California, San Francisco. 1996. *Chronic Care in America: A 21st Century Challenge*. Robert Wood Johnson Foundation: Princeton, New Jersey. Accessed at: http://www.rwjf.org/publications/publicationsPdfs/Chronic_Care_in_America.pdf.

Institute of Medicine Committee on Quality of Health Care in America. 2001. *Crossing the Quality Chasm: A New Health System for the 21st Century*. National Academy Press: Washington, DC.

McKinlay J, Marceau L. 2000. U.S. public health and the 21st century: Diabetes mellitus. *The Lancet* 356(26 August): 757-761.

Milstein B, Homer J. 2003. The dynamics of upstream and downstream: Why is it so hard for the health system to work upstream and what can be done about it? Presentation to CDC Health Futures Workgroup. November 25, 2003: Atlanta, GA.

Murphy D, Chapel T, Clark C. 2004. Moving diabetes care from science to practice: The evolution of the National Diabetes Prevention and Control Program. *Annals of Internal Medicine* (in press).

Narayan KMV, Boyle JP, Thompson TJ, Sorensen SW, Williamson DF. 2003. Lifetime risk for diabetes mellitus in the United States. *JAMA* 290: 1884-90.

National Center for Health Statistics (NCHS). 2003. *Health, United States 2002* (published annually). Data on smoking prevalence accessed at: http://www.infoplease.com/ipa/A0762370.html.

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). 2001. Diabetes Control and Complications Trial. Accessed at: http://www.niddk.nih.gov/health/diabetes/pubs/dcct1/dcct.htm.

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). 2004a. National Diabetes Statistics. Accessed at: http://diabetes.niddk.nih.gov/dm/pubs/statistics/index.htm.

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). 2004b. Statistics related to overweight and obesity. Accessed at: http://www.niddk.nih.gov/health/nutrit/pubs/statobes.htm#preval.

U.K. Prospective Diabetes Study Group (UKPDS). 1998. Tight blood pressure control and risk of macrovascular and microvascular complications in Type 2 diabetes. *The Lancet* 352: 703-713.

U.S. Census Bureau. 2003. *Statistical Abstract of the United States: 2002*. Accessed at: http://www.census.gov/prod/www/statistical-abstract-02.html.

U.S. Census Bureau. 2004. Historical Income Tables. Accessed at: http://www.census.gov/hhes/income/histinc.

Wilkinson RG. 1996. *Unhealthy Societies: The Afflictions of Inequality*. London: Routledge.