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## **Accommodating Complexity and Human Behaviors in Decision Analysis LDRD Final Report**

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# Accommodating Complexity and Human Behaviors in Decision Analysis

## LDRD Final Report

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### ***Abstract***

This is the final report for a LDRD effort to address human behavior in decision support systems. One sister LDRD effort reports the extension of this work to include actual human choices and additional simulation analyses. Another provides the background for this effort and the programmatic directions for future work. This specific effort considered the feasibility of five aspects of model development required for analysis viability. To avoid the use of classified information, healthcare decisions and the system embedding them became the illustrative example for assessment.



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## Chapter 1: Introduction

In the age of globalization, it is becoming increasingly apparent that future security issues could be dominated more by behavioral considerations than engineering ones. As such, SNL requires an expansive, high-confidence, behavioral/social simulation capability for its mission. This effort evaluates a computational approach for decision support that included behavioral-response simulation in an attempt to further the development of a broad SNL capability useful for evaluation of national/international policy implications.

As noted in Strip 2007, healthcare is itself a national security issue that threatens the U.S. economy. To avoid the use of classified information, US healthcare dynamics will act as the example problem. The modeling will examine the impacts of policy options on economy, demographics, and costs. It will utilize system dynamics and agent-based methodologies to simulate the feedback dynamics for evolving impacts.

The health system contains many complex interactions and is prone to counterintuitive outcomes. Figure 1.1 indicates the range of considerations a complete model of the healthcare system would need to include.

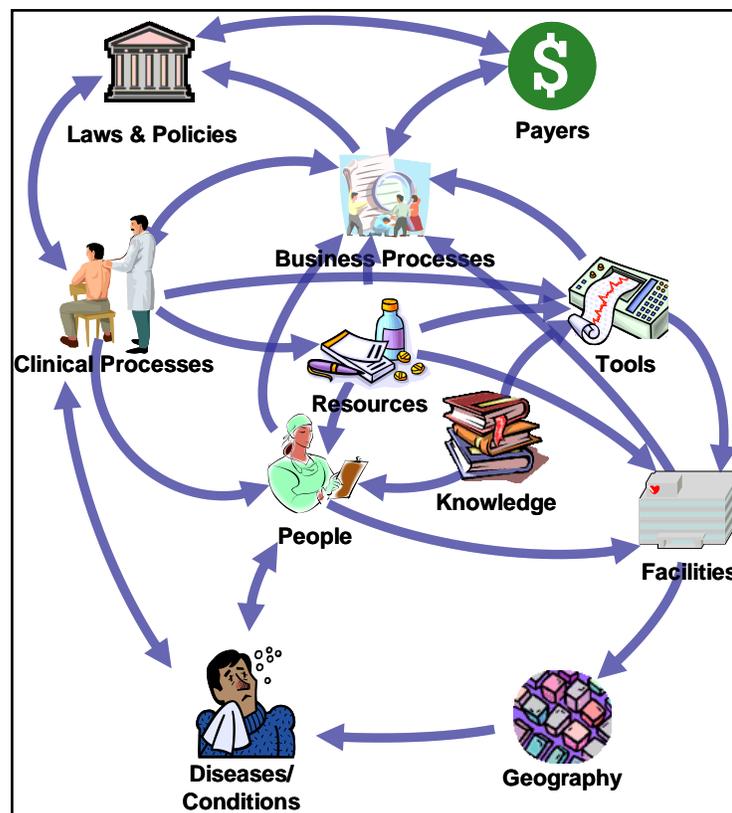


Figure 1.1 The Healthcare System.

The purpose of this effort is to determine the feasibility of developing a framework to address the healthcare issues as needed to support discussions related to national healthcare policy.

Strip (2007) reports the architectural and programmatic considerations for furthering this work. We have partnered with The Leonard Davis Institute (LDI) at The University of Pennsylvania and various institutes and Schools within the University of Texas System. LDI has offered to produce textbooks if the effort moves forward and the LBJ School of Public Policy has offered to create a curriculum. In all cases, our research partners act as collaborative researchers, reviewers, and subject matter experts. Under the auspices of Senator Bingaman's office, UTS convened a NAS-style Blue ribbon panel of experts to prioritize the needs and requirements for the modeling effort initiated here. The encouraging results of that workshop are provided in Appendix B.

Separately, Linda Bilheimer, Director, Office of Analysis and Epidemiology at the National Center for Health Statistics, CDC/HHS and formerly with The Robert Wood Johnson Foundation and Congressional Budget Office, provided her reasons for supporting the SNL efforts as noted below:

*Existing models either do not address or address only weakly:*

1. *Impact on health care providers and the interactive effects of their responses on model outcomes*
2. *Impact on the public health system*
3. *Macroeconomic impacts both short-term and long-term*
  - a. *Those impacts include the implications of alternative financing options, such as general revenues, payroll taxes or cuts in other federal or state spending*
4. *Winners and losers by socio-demographic characteristics (for individuals) and firm size (for employers)*
5. *Winners and losers among the states*
6. *Effectiveness of cost-containment strategies. (Modelers make assumptions about effectiveness, but they're often not much more than speculation, given lack of data.)*
7. *Complexities of implementation*
8. *Measures of uncertainty are largely lacking. Given the difficulty of estimating standard errors in large microsimulation models, alternative approaches using sensitivity analyses need to be more extensively used and the degree of uncertainty needs to be clearly spelled out for policymakers.*

## **Chapter 2: Modeling Approach**

A variety of computational modeling approaches have been used to model healthcare systems. The least computationally intensive modeling aggregates the various players into a relatively small number of components whose interactions are expressed by closed form mathematical equations. The equations are then solved using a variety of techniques, in the simplest cases using a spreadsheet program like Excel. The attractiveness of this approach is the simplicity of the computational model. Unfortunately, the cost of this simplicity leads to the loss of detail inherent in aggregate models. Often the actions of individuals lead to composite behaviors that are not anticipated and therefore cannot be expressed by such models.

Systems dynamics provides a higher level of modeling fidelity and requires a commensurate increase in the skills required to develop a model and interpret its output. One of the great strengths of systems dynamics models is their ability to represent feedback mechanisms. Examples might include pricing impacts on demand, or substitution effects that arise from availability or rationing. An additional benefit is that software for developing systems dynamics models is very affordable, requires only a moderately configured PC, and often provides a graphical programming interface, reducing the entry-level knowledge required to start developing models. Systems dynamics suffers from two main weaknesses. The first is that it only represents mean behavior – it does not produce a distribution of behaviors that would enable an analyst to understand the range of consequences of a policy. The second weakness is the need to aggregate the individual entities comprising the healthcare system into a manageable small number of representative classes, lest the model become unmanageable.

Micro-simulation is probably the currently dominant technique used in healthcare policy modeling. These models are used primarily in health finance and health economics models which seek to predict levels of insurance coverage, distribution of costs, and similar kinds of measures. Typically micro-simulation models do not predict health outcomes measures as a consequence of policy change. In contrast to systems dynamics models, micro-simulation models are sample across a broad range of population representatives, providing a means to estimate the distribution of effects, and hence provide visibility into “tail” behavior. Unlike systems dynamics models, micro-simulations generally are quite weak in representation of feedback effects, which can be critical to predictions that involve more than a few time periods of interest.

The computational requirements of micro-simulation models are generally compatible with a desktop PC. Although this might suggest accessibility of this class of models to a broad community of users, we are not aware of any generally available healthcare micro-simulation models or tools for developing one. One reason lies, no doubt, in the amount of data required to populate a meaningful model; even if you could develop the model in the first place, accumulating, formatting, and managing the data is beyond the scope of anyone other than a dedicated user. A small number of micro-simulation models underlie most of the published studies that rely on this class of model. As far as we have been able to tell, these models are treated as extremely proprietary – virtually nothing is

published about the structure, assumptions, or other factors needed to determine one's level of trust in the outputs of the models. As a consequence it is impossible to compare, let alone reconcile, inconsistent predictions produced by competing models. Informed debate on competing policies is impossible when the policies are evaluated with different models.

Advances in computing, especially the advent of massively parallel systems, enables an alternative approach called *intelligent agent-based modeling*. In this approach we model each individual decision-maker in a system, capturing their behavior and interactions with other decision-makers. By instantiating thousands (or even millions) of these intelligent agents in one (HPC-enabled) model, a realistic simulation of a large-scale system can be achieved. The interactions between the intelligent agents are automatically captured by the model, which provides the many feedback loops present in such a large and complex system. The principal advantage of this modeling approach is the ability to define and analyze individual decision-makers' behavior and its effects on the entire system. These models are being used at Sandia to analyze military logistics of globally deployed weapons systems (Schoenwald 2005), regional and national economic performance in the face of infrastructure disruptions (Barton 2000, 2002; Schoenwald 2004; Sprigg 2004a), consumer and corporate economic confidence in the face of terrorist acts (Hand 2005; Sprigg 2004b, 2004c), and economic impacts of global climate change (Backus 2002).

Although the anticipated larger effort of implementing the techniques developed here depend on utilizing and even advancing the state-of-the-art in modeling, it is fundamentally driven by the requirement that as a nation we develop alternatives to our currently unsustainable system of healthcare. There will be no shortage of alternatives proposed; what we hope to provide is a means to reliably predict and debate the consequences of any given policy. The proposed follow-on efforts would engender a policy analysis tool unlike any in common use on any policy issue in the country. Hence, our research partners view training a community of users as an essential component of follow-on programs.

Working in partnership with academic institutions, they have offered will develop teaching curricula and associated materials for use at a variety of levels. At the most fundamental level, they anticipate a graduate level course on the use of computational models in policy analysis. The course would cover the range of factors such as understanding limitations of models, the role of uncertainty, basic statistics, etc., the impact the use of models in policy analysis. A topic specific course will be developed to focus on the large scale healthcare model we would potential develop, based on the foundation described in this report. This course will concentrate on the mechanics of performing analysis with this model – developing input data, representing policy, and analyzing input.

Our advocates also propose to develop an executive short-course for policy-makers and legislators who will be the consumers of analyses. The short-course would enable the actual decision makers to understand the limitations of the data they are being provided

and teach them the underlying concepts that will enable them to appropriately question, guide, and challenge the analyses provided to them by those who have their hands directly on the knobs and levers that control the simulation tools.

The modeling of the U.S. health care system is a complicated endeavor but one in which Sandia National Laboratories has the expertise to develop such a model by leveraging its experience in high performance computing, agent-based modeling, human behavior, and economics.



### Chapter 3: Five Feasibility Assessments

The purpose of this LDRD effort was to determine the feasibility of the five components shown in Figure 3.1.

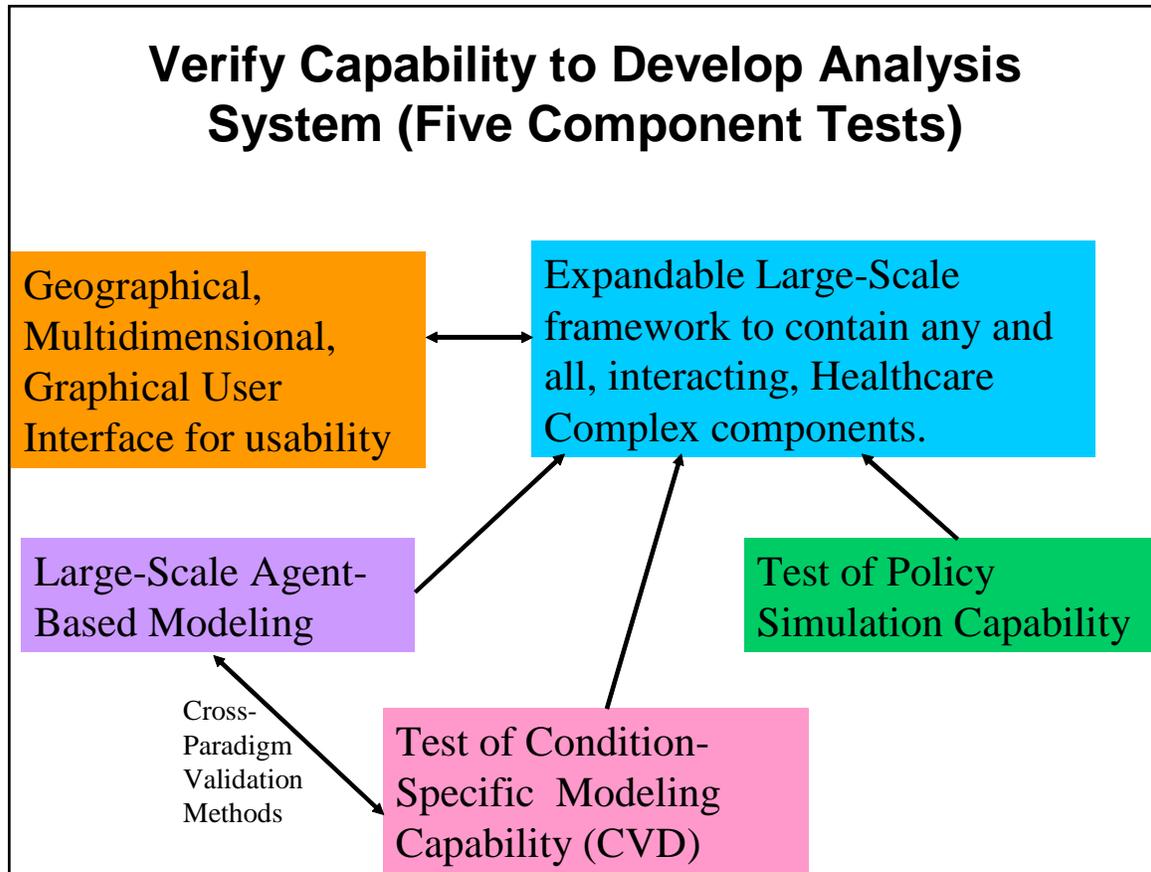


Figure 2 Five Components of Feasibility.

The first need is for a metaphor to present information to the user. The development of a Graphical User Interface is presented in the next section. The modeling feasibility has several components. This effort included the development of a disease-specific (Cardiovascular Disease CVD) to verify that ability to simulate the dynamics with available data sets, within a bounded framework, and using currently understood processes.

Model implementation took the form of using both agent-based and system dynamics paradigms. This approach allowed a novel method for verification and validation, and it resolved the need to use both methods for any large-scale implementation -- where agent-based methods could simulate distributional and stochastic phenomena, with system dynamics capturing aggregate institutional/macro-economic processes.

A separate policy model, calibrated and parameterized to national conditions, permitted the testing of policy options where physical behavioral dynamics affected outcomes.

Lastly, a minor effort readily showed that proper project design could ensure a computationally tractable approach for an expandable, multi-dimensional framework. The sections that follow, discuss the development and testing of each component.

## Chapter 4: User Interface for Decision-Maker Assessment

Because healthcare is as much a political as it is a financial and technical problem, and “because all politics are local,” the interface for this effort is a map metaphor. As designed, users can highlight a region and expand out on any areas. A common database framework (Apache Derby) drives the interface. Figure 4.1 starts out with a global color-coded map, here illustratively depicting a hypothetical flu epidemic initiated in China.

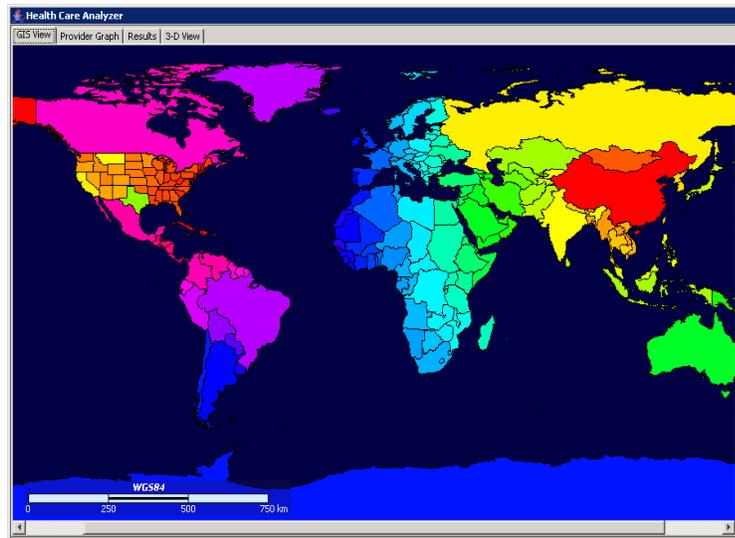


Figure 4.1 Global, Colorized Map Interface.

Figure 4.2 then highlights a U.S. continental expansion. The icon entities used here represent airports (but could they be hospitals or anything else of the user’s choice), and each can be “clicked” to expose more detailed information.

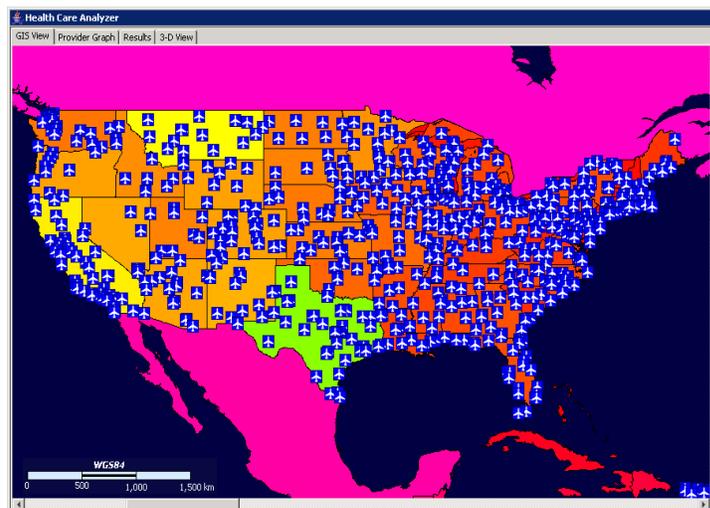
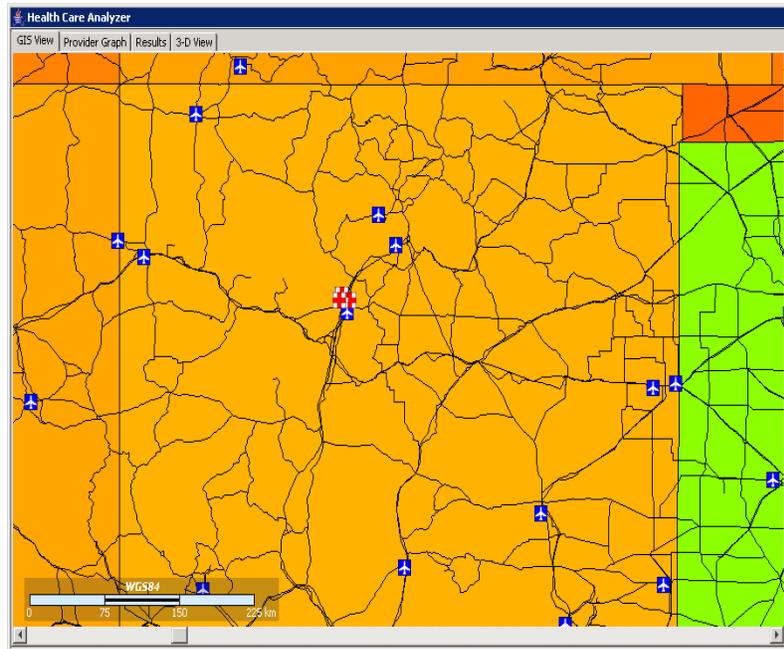


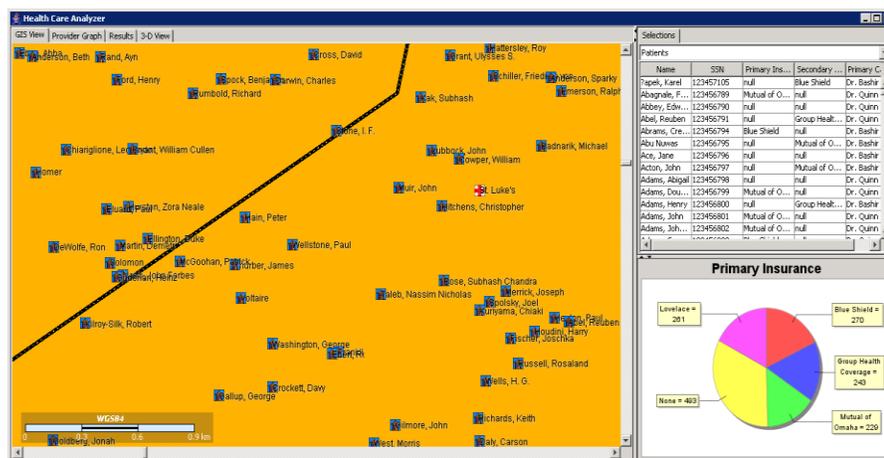
Figure 4.2 U.S. National Drill Down with First Level of “Entity” Detail.

Figure 4.3 then highlights and expands the map to show New Mexico. At this level, the interface adds hospital icons (three shown illustratively for Albuquerque).



**Figure 4.3 New Mexico Detail with Hospital Icons.**

Lastly, this examples drills down to Albuquerque and shows individual names (patients). The names of famous personalities are shown for illustrative purposes. To the right of the figure are graphics and tables that, in this case, show the insurance statistics for the group of individuals noted. The use of a “mouse” to select an area of the map will cause only those entities and their summary/aggregate statistics to show on the left pane. Clicking an individual will highlight that information (with a finer level of detail).



**Figure 4.4 Entity Level Drill-Down.**

Review indicated that this approach could provide the information in a format useable by decision makes. Analysts would use the OMEGA-SIM platform/interface to produce forecasts for the interface or to populate the interface with analysis results.

OMEGA –SIM is a combined agent-based, system dynamics, discrete-event simulation framework whose parallelization would appear to be effective for the follow-on efforts anticipated from this work (Siirola 2007). OMEGA will be discussed further in later chapters.



## Chapter 5: Design for Demonstration Policy Model

(by Gary B. Hirsch <http://garybhirsch.com/> and Jack Homer <http://www.angelfire.com/biz2/HomerConsulting/> )

The Demonstration Policy Model illustrates some basic ideas about the impacts over time of changes in the US health care system. It focuses on the prevention and treatment of cardiovascular diseases such as heart disease and stroke, which are principal causes of morbidity, mortality, and disability, and contributors to high health care costs in the US. More specifically, the model demonstrates the potential consequences for mortality and cost of changes in resource allocations among (1) treatment of complications, (2) disease management to prevent complications, and (3) risk management to prevent the onset of cardiovascular disease. The model may also serve as a rudimentary framework for assessing the potential impacts of certain other interventions that have been suggested to improve the performance of the US health care system. Figure 5.1 depicts an overall perspective on healthcare interactions.

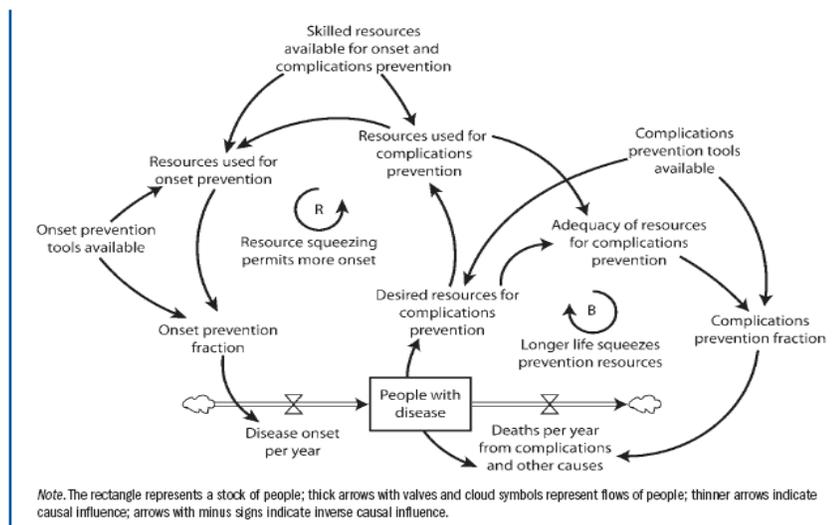


Figure 5.1 Health care Interactions (Hirsch 2006).

Figures 5.2 through Figure 5.5 display the model's structure. This structure is based on our thinking at this time and may change as further studies evolve, as we gain a better idea of what data are available for quantifying the model, and in response to feedback from reviewers.

Figure 5.2 focuses on the stock-and-flow structure reflecting how people develop and experience cardiovascular disease (CVD), including heart disease and cerebrovascular (stroke) disease. CVD should be reflected as a two-stage process in which people first develop subacute illness in which they may or may not experience symptoms, but do not require an inpatient hospital stay. Some fraction of the people in this group would suffer an acute episode such as a myocardial infarction or stroke requiring hospitalization. If they survive, they would move into the next (post-acute) stage in which they are likely to require more care and more likely to suffer recurrent acute attacks including fatal ones.

The rate of onset of CVD depends on

- the size and composition of the population,
- its risk profile (e.g., prevalence of high cholesterol levels and blood pressure),
- onset rate in the absence of preventive programs, and
- presence of risk management programs that help to reduce prevalence of those risks.

The rate at which people in the subacute group develop acute complications depends on a rate at which these complications would occur in the absence of disease management programs, the presence of these programs, and their potential impact in reducing the rate of acute complications. Once people are in the post-acute group, there is also a characteristic (higher) rate at which they will develop complications in the absence of disease management and potential impact of disease management programs. Some fraction of these acute episodes will be fatal. People in both groups may also die of causes other than CVD. The rate of acute episodes for both groups and resources committed to risk and disease management together determine total health care costs.

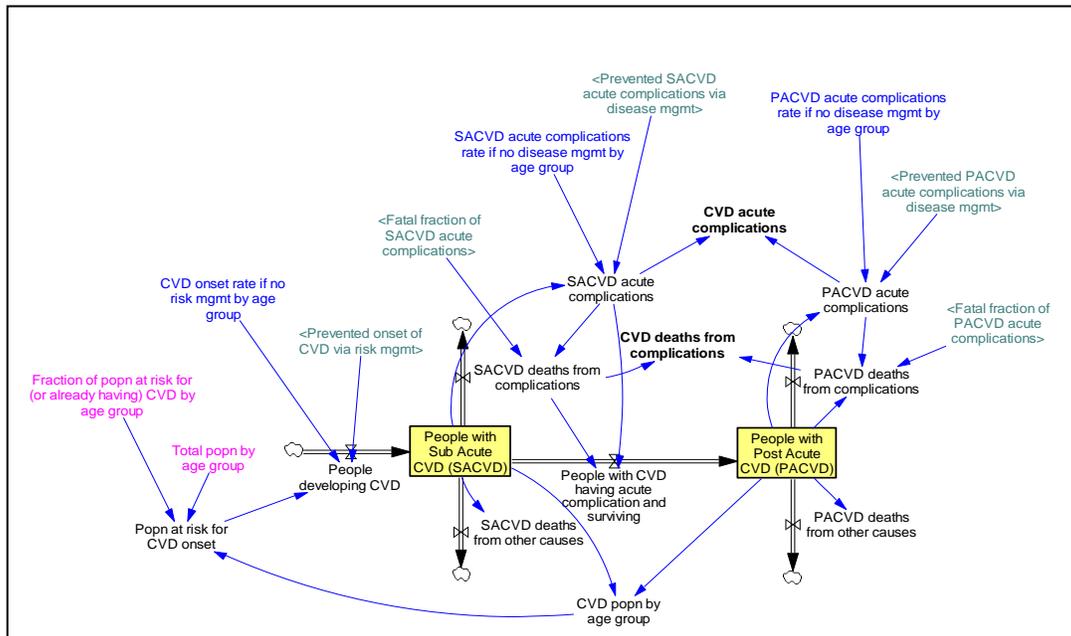
The structure shown in Figure 5.2 could be replicated for two age groups, those under 65 and those over 65, who have quite different rates of disease and death. One could also disaggregate the model to differentiate patients with heart and cerebrovascular disease. The separation of heart and cerebrovascular disease and the subacute and post-acute stages would depend on whether data are available to support such separations. Many of the available data do not make these distinctions. If there are sufficient data to make such separations, this could increase the model's accuracy with regard to deaths and costs. For example, although the post-acute group is only a small percentage of the population, it consumes a considerable fraction of the nation's health care resources.

Another question is whether to include people who are undiagnosed or asymptomatic among those whom are counted as having CVD. These people do not show up in the published prevalence data and may or may not be an important group to identify separate from those who are at risk for CVD. It may be possible that screening for CVD is a practical option and the identification of undiagnosed CVD patients via screening could lead to their being managed differently (more intensively) than those with risk factors alone. If the data indicate that this is so, modeling this subpopulation explicitly would be useful in that it allows us to project the impact of the screening programs. This is currently a research question.

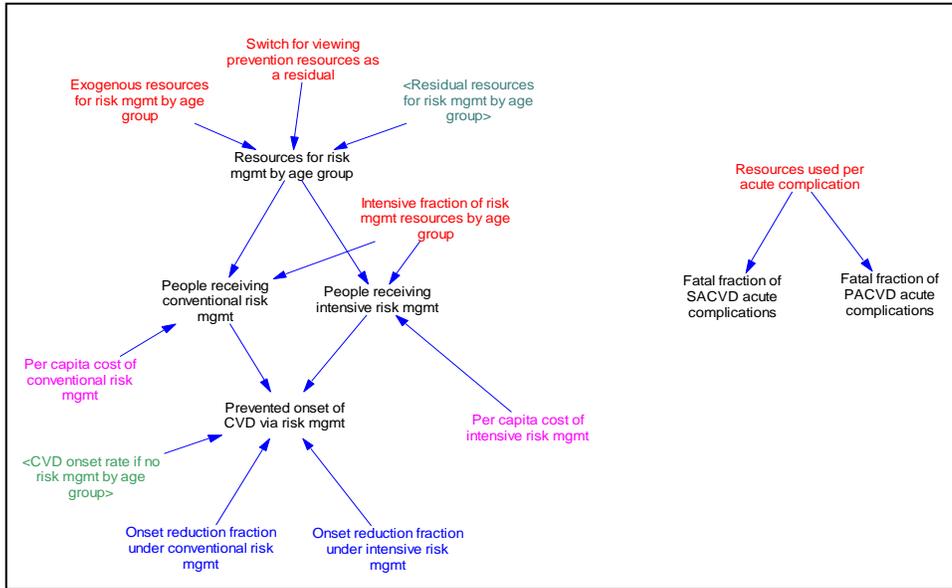
Figure 5.3 presents a causal framework for the allocation of resources to risk management and the resulting prevention of disease onset. As the diagram suggests, the model could contain an option to determine this resource allocation exogenously or to make it endogenous and a function of what's left after resources have been utilized for treatment of complications and disease management. It is also possible to differentiate between conventional risk management (e.g., education about diet and exercise) and more intensive programs that include use of medications to lower blood pressure and cholesterol levels.

Figures 5.4 and 5.5 show a similar set of mechanisms for allocating resources to disease management and making a further allocation between disease management programs for the subacute and post-acute groups. There would again be an option to set this allocation exogenously or to make it a function of what's left over after treating complications. The model could also differentiate between conventional and intensive disease management programs. Figure 4 simply shows how the residual resources available for disease management would be calculated.

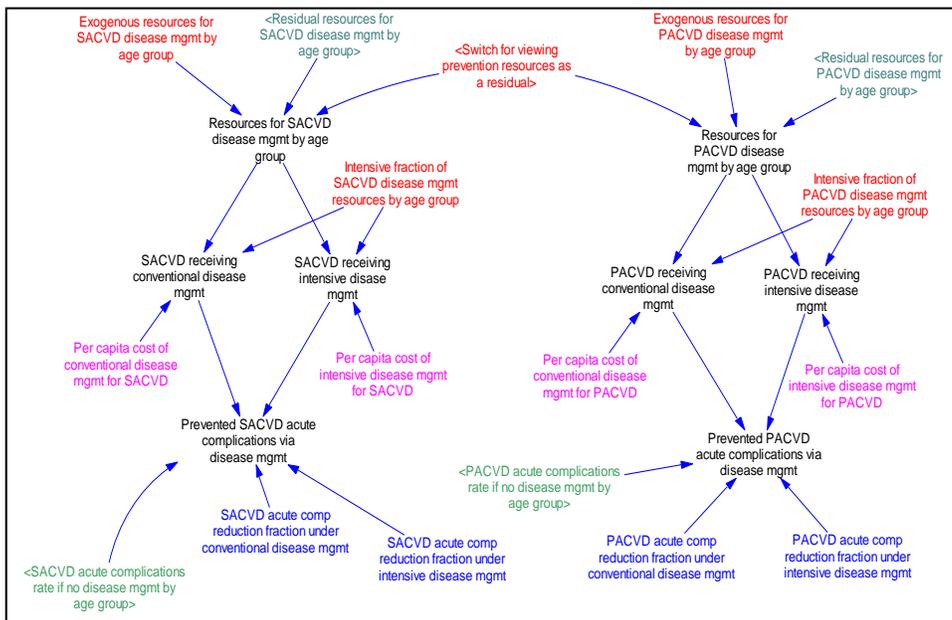
As indicated earlier, this model can serve as a rudimentary framework in which to assess various policy options that have been suggested. For example, improved use of Electronic Medical Record technology might increase the fraction of people receiving effective disease management who would then experience fewer acute complications. Better reimbursement for risk management might increase the fractions of patients seeing primary care providers who receive those services and who receive the more intensive services. Similarly, standards of practice backed up by “pay for performance” programs implemented by insurers could also increase the fraction receiving effective risk management. On the other hand, adoption of “catastrophic” health insurance policies (that don't pay for preventive care) by employers trying to save money may reduce the fraction receiving risk management services and end up costing more in the long run.



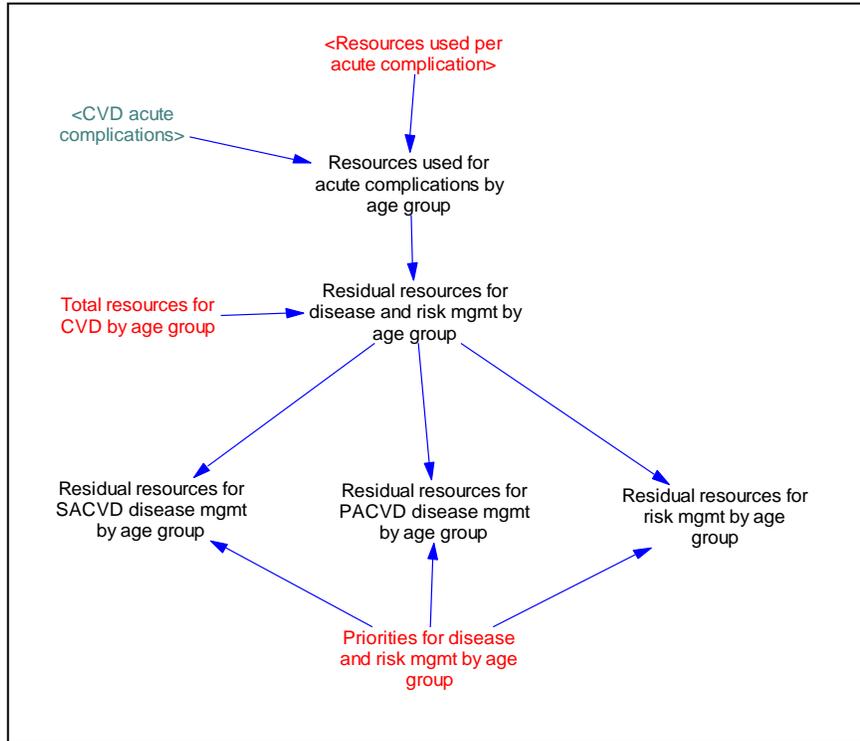
**Figure 5.2 Stock and Flow Structure for Cardiovascular Disease.**



**Figure 5.3 Allocation of Resources to Risk Management.**



**Figure 5.4 Allocation of Resources to Disease Management.**



**Figure 5.5 Calculation of Resources Available for Risk and Disease Management.**

Appendix A presents the equations (in VENSIM Syntax)<sup>1</sup> associated with this model. Its construct is then used for the Verification and Validation (V&V) and agent-based modeling described in the next chapter.

<sup>1</sup> <http://www.vensim.com/documentation.html>



## Chapter 6: Multiple Paradigm approaches to modeling and V&V

The System Dynamics (SD) model of the previous chapter was re-coded into OMEGA-SIM and verified with the VENSIM version. It was then further transformed into an agent-based (AB) model. The CVD ( Cardiovascular Disease ) model considers the dynamics of: Risk Levels; Asymptomatic, Sub-acute, Post Acute Population; CVD Death Rates; Non-CVD Death Rates; Disease Treatment, Management, Screening, Prevention; Resource Expenditure And Allocation; Care Prioritization; Attack and Symptom Dynamics; Sudden Death, non-sudden Death, and Attack Survival Treatment. This model was developed originally by Jack Homer and Gary Hirsch, and was modified for the purposes here.

A sister effort expanded the model further and is reported in a separate document, The SD model represents the population as a continuous flow from one condition to the next. In AB models, the population is a represented as interacting individuals (in this example over 100,000 entities were used for demonstration purposes). Figure 6.1 shows the diagram of the initial Homer-Hirsch model. Figure 6.2 shows the OMEGA agent-based version of it.

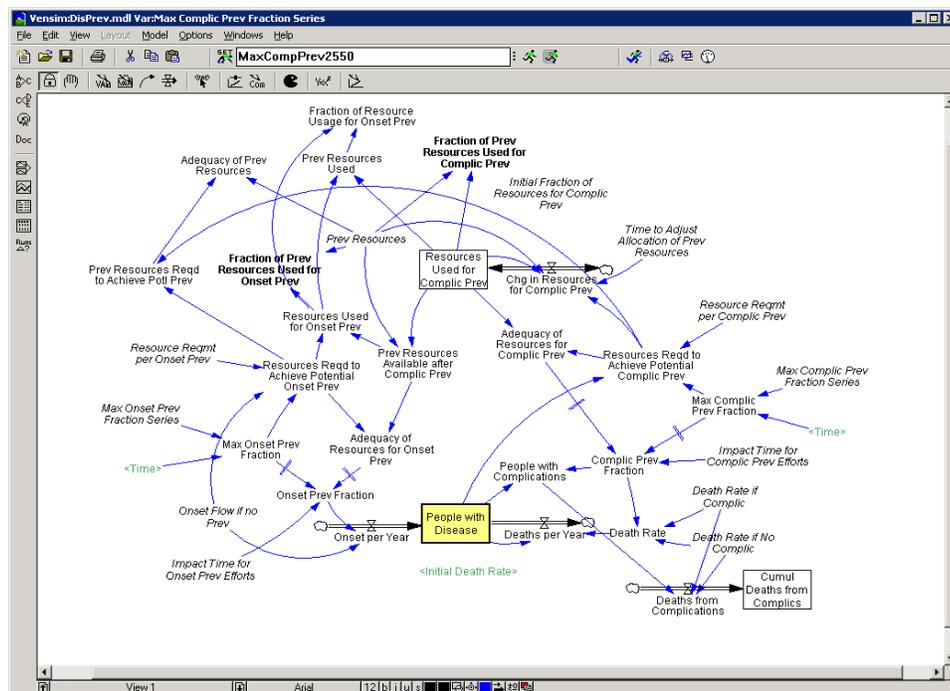
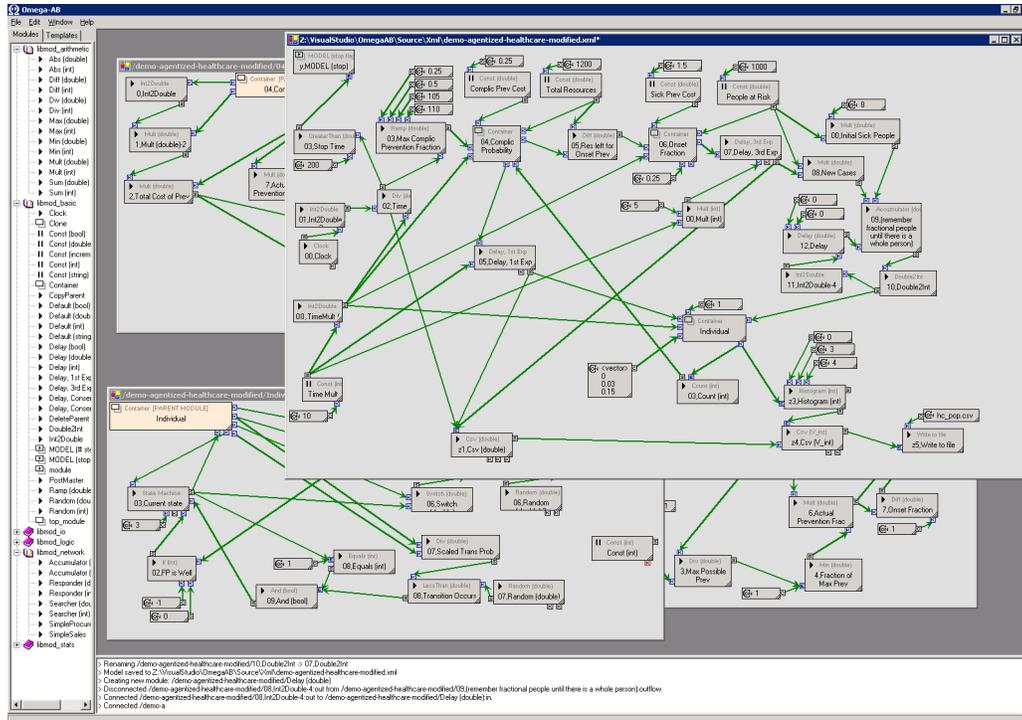


Figure 6.1 SD Version of H-H Model.



**Figure 6.2 Agent-based Version of the H-H Model.**

Figure 6.3 shows the initial comparison of the original, OMEGA SD and OMEGA AB models. Using the identical data and the perfect translation of the equations, the AB and SD models give two different sets of results. The use of two paradigms to model the same identical system clarifies a V&V oversight in both. Historical data records deaths by cause. If 60 out of 100 total deaths out of a population 100,000 (with 40,000 diagnosed with chronic disease) are defined as chronic-disease-caused deaths, then the chronic death rate 60/40000 and the non-chronic is 40/60000. Plug this in the SD model, run it through time, and you get the right historic answers. Despite real data, real numbers, and corroborated results, there is an obvious flaw in logic. The recorded deaths actually denote a frequency expressed as a fraction. An actual death is a probability. A chronically-ill person can die from a car accident. Saving a person from a car accident still may result in their death a few days later from a heart attack. Modeled deaths must be recorded as simply occurring as the result of a conditional probability. Only a post analysis can indicate the probabilistic break down for cause of death for each person. When this correction is made, both models give the same answer for the same reason. Policies that would affect chronic disease intervention in the “obvious death by disease case” would give different results from those assuming the more unintuitive conditional probability perspective.

This example usage indicates that the routine usage of multiple paradigms in analysis may improve the V&V of policy models.

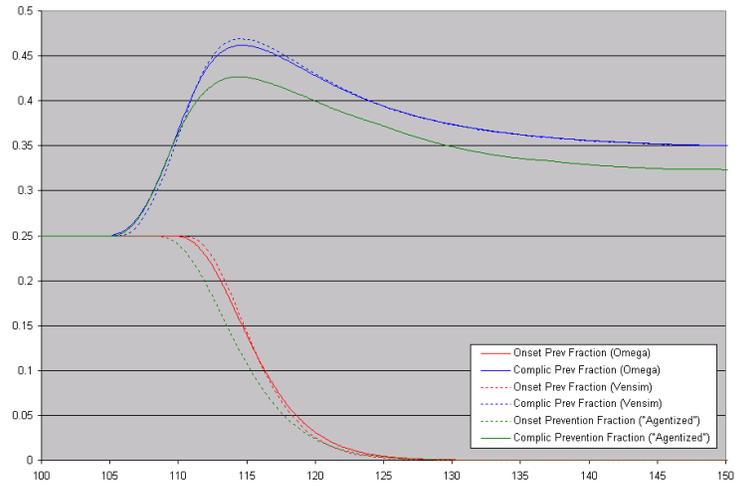


Figure 6.3 "Agentized" Homer-Hirsch Model in Omega vs. SD Model in Vensim.



## Chapter 7: Policy Simulation Feasibility

This section describes a relatively simple national demographics framework that focuses on chronic illness, and medical costs. Its purpose is to determine the feasibility of 1) policy testing, 2) detailed morbidity simulation, and 3) fusing of disparate data sources into a self-consistent framework. The model uses national-level data and represents national dynamics, but the equations are easily extendable to multi-region simulations and multiple afflictions. The equations represent both integral and differential equations, but the data is only available on an annual basis (if that). The integrating equations use 2003 for the initialization year.

This policy-testing prototype model includes endogenous variable dynamics for: Population, Birth, Deaths, Chronic Condition, Chronic Death Rates, Chronic Incidence Rates, Non-Chronic Death Rates, Drug Costs, Maintenance Costs, Acute-Care Costs, Impact on GDP, Income, and Participation Choices. The policy testing includes: Information Technology, Prevention, Patient Participation (insurance coverage), High Technology Diagnostics/Procedures, Standards of Practice (evidence-based medicine), Drug Improvements, and Life-style impacts.

### Model Equations

The equations provide full demographics representation, including chronic and non-chronic medical conditions along with the associated costs. The equations are described below:

$$P_{a,t} = P_{a-1,t-1} - D_{a,t} + I_{a,t} + \delta_{a,0} * TB_t$$

The current population (P), by age (a) and at time (t) is the previous population, reduced by deaths (D), increased by net immigration (I) and, and, for age group 0, increased by Total Births (TB).  $\delta_{ij}$  is the Kronecker-delta function. The population is based on the US Health/Census data.<sup>2</sup> (HHS 2006)

$$TB_t = \sum_a B_{a,t}$$

Total births (TB) are the sum of births (B) for that year over all ages of the population having births.

$$B_{a,t} = P_{a,t} * BR_{a,t} * BP_{a,t}$$

Births (B) are calculated using the total population (P) and the Birth Rate (BR). BR assumes a homogenous population that implies constant female to male ratio. The BR

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<sup>2</sup> <http://www.cdc.gov/nchs/data/hus/hus05.pdf>

per person by age is based on historical values<sup>3</sup> but can be modified by a Birth Rate Policy multiplier (BP).

$$I_{a,t} = I_{a,t-1} * (1.0 * IGR_{a,t})$$

Net immigration simply grows the initial recoded immigration by a forecasted immigration growth rate (IGR), based on DHS data.<sup>4</sup> (IGR has a default value of 3%/yr.)

$$I_{a,o} = RI_{a,o} * TLR_a$$

The initial immigration takes the raw immigration and corrects it by total immigration to legal immigration ratio. (TLR= 1.478 in 2003.)

$$D_{a,t} = CD_{a,t} + ND_{a,t}$$

The Deaths are the sum of the Chronic Deaths (CD) and Non-chronic Deaths (ND).<sup>5</sup>

$$CP_{a,0} = CP_{min} + CP_{max} / (1.0 + (a / \tau)^{\sigma})$$

The initial chronic population by age is a GEV logistic<sup>6</sup> fit of the data<sup>7</sup>, scaled to the total and with the unit parameterization shown in Table 1.

**Table 1. Chronic Prevalence Initialization by Age.**

CP <sub>min</sub>	0.065
CP <sub>max</sub>	1.17
τn	57
σ	-4.9

$$CP_{a,t} = CP_{a-1,t-1} + CI_{a,t} - CE_{a,t}$$

The current Chronic Population (CP) is the previous CP increased by new incidences (CI) and decreased by “exits” (CE). “Exits” are used because the deaths need not be only due to the chronic illness.

<sup>3</sup> <http://www.cdc.gov/nchs/>

<sup>4</sup> <http://www.dhs.gov/ximgtm/statistics/>

<sup>5</sup> <http://www.cdc.gov/nchs/products/pubs/pubd/hestats/finaldeaths03/finaldeaths03.htm> and [http://www.cdc.gov/nchs/data/nvsr/nvsr54/nvsr54\\_13.pdf](http://www.cdc.gov/nchs/data/nvsr/nvsr54/nvsr54_13.pdf)

<sup>6</sup> GEV implies a Generalized Extreme Value distribution and a logistic is used to limit the fraction of the population to an asymptote.

<sup>7</sup> <http://www.americanheart.org/downloadable/heart/1136308648540Statupdate2006.pdf> and

[http://www.cdc.gov/diabetes/pubs/pdf/ndfs\\_2005.pdf](http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2005.pdf) and

[http://www.agingstats.gov/agingstatsdotnet/main\\_site/default.aspx](http://www.agingstats.gov/agingstatsdotnet/main_site/default.aspx) and

<http://www.cancer.org/downloads/STT/CAFF2006PWSecured.pdf>

$$CE_{a,t} = (1.0 - (1.0 - NDR_{a,t}) * (1.0 - CDR_{a,t})) * CP_{a-1,t-1}$$

The CE is a conditional probability based on the CP and the Non-chronic death rate (NDR) as well as the Chronic Death Rate (CDR). It is implicitly corrected for the non-chronic deaths within the entire population as noted below.

$$CI_{a,t} = (P_{a-1,t-1} - CP_{a-1,t-1}) * CIR_{a,t}$$

The Chronic Incidence Rate (CIR) only affects the population that is not yet chronically ill.

$$CIR_{a,t} = \text{Min}(CIR_{\text{min}}, CIR_{\text{max}} * Sm_t * \exp(\text{Max}(0, (a - m - \tau_0)) * (\lambda * \lambda m_t)))$$

The incident rate is an exponential fit of the data using the data noted in the sources for CP above. Sm is a scale multiplier derived from policy impacts, as will be discussed below.  $\tau_0$  is an aging offset caused by assumed (Sm) policies that improve health.  $\lambda m$  modifies the time constant of the exponential function due to Sm and  $\tau_0$ .

The parameterization is shown in table 2.

**Table 2. Incidence Parameterization.**

$CIR_{\text{min}}$	0.01
$CIR_{\text{max}}$	1.0
$\tau n$	40
$\lambda$	0.145

$$CD_{a,t} = CP_{a-1,t-1} * CDR_{a,t}$$

Chronic deaths come from the Chronically-ill Population (CP) and the Chronic Death Rate (CDR).

$$CDR_{a,t} = \text{Min}(CDR_{\text{min}}, CDR_{\text{max}} * Sm_t * \exp(\text{Max}(0, (a - m - \tau_0)) * (\lambda * \lambda m_t)))$$

CDR is an exponential fit like the CIR. Its parameterization is shown in Table 3.

**Table 3. Chronic Death Rate Parameterization.**

$CDR_{\text{min}}$	0.0027
$CDR_{\text{max}}$	1.0
$\tau n$	40
$\lambda$	0.074

$$ND_{a,t} = P_{a-1,t-1} * NDR_{a,t}$$

Non-Chronic Deaths (ND) follow the same logic as Chronic Deaths except that they are based on the entire population.

$$NDR_{a,t} = \text{Min}(NDR_{\text{min}}, NDR_{\text{max}} * Sm_t * \exp(\text{Max}(0, (a - m - \tau 0_t)) * (\lambda * \lambda m_t)))$$

The Non-Chronic Death Rate (NDR) follows the same logic as the CDR, and has the estimated parameterization shown in Table 4.

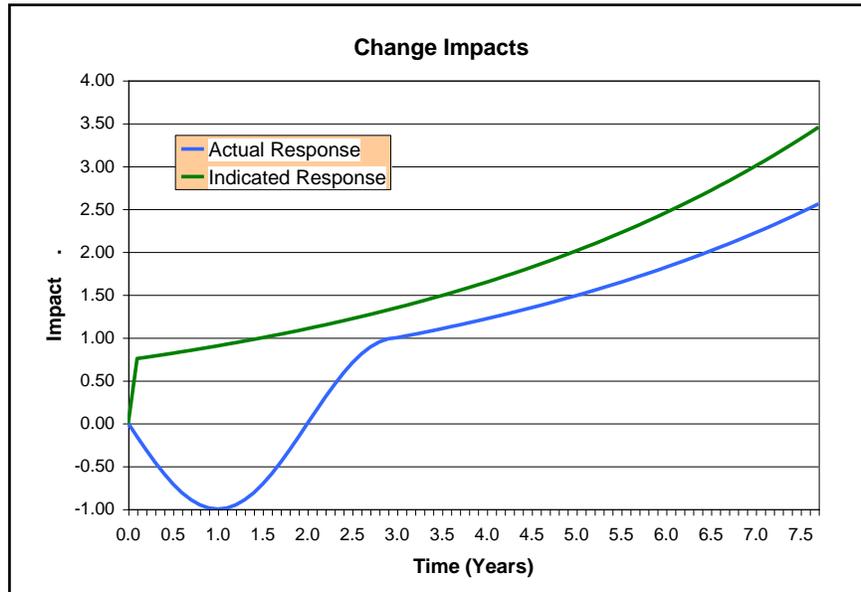
**Table 4. Non-Chronic Death Rate Parameterization.**

CDR <sub>min</sub>	0.0003
CDR <sub>max</sub>	1.0
τn	30
λ	0.0752

$$Sm_t = (STS_t / Cm_t)^{RE} * LTS_t$$

The Scale multiplier (Sm) is the net dynamics impact of combined policy interventions (Cm). The STS captures the short term (possibly negative start up impacts) and the LTS captures the long term impacts.

Figure 7.1 shows the dynamics that the Sm equation typically produces to an exponential input (such as with new technology).



**Figure 7.1 Short-term/Long-term response interactions.**

$$STS_t = STS_{t-1} + (Cm_t - STS_{t-1}) / SAT$$

The Short-Term response Status (STS) is captured by an exponential smoothing function that also represents the Erlang distribution typically used to capture failure modes. The Short-term Averaging Time (SAT) controls the smoothing function.

$$LTS_t = LTS_{t-1} + (Cm_t - LTS_{t-1}) / LAT$$

The Long-Term response Status (LTS) is also captured by an exponential smoothing function. The Long-term Averaging Time (LAT) controls the smoothing function.

$$Cm_t = (1.0 - (1.0 - Im_t) * PP_t)^{(1-se)} * \prod_p IMC_{p,t}$$

The Combined impact multiplier (Cm) uses a Substitution Elasticity (SE) approach performed through three calculations. The SE approach ensures the diminishing returns from additional policy implementations. The Prevention Participation (PP) notes that for many interventions the patient must cooperate, such as in the taking of medicines or following doctor's orders.

$$Im_t = Min(p)(Pm_p)$$

The policy (p) with the maximum impact is the Policy multiplier (Pm) with the minimum value. (All policies are defined as multipliers.) For chronic incidence, chronic populations, and non-chronic populations, there are currently eight intervention policies: Prevention, Information Technology, Standard of Practice, High Tech, Improved Drugs, Early Diagnosis, Bad Life Style, and Good Lifestyle. Prevention reduces the age-dependent incidence of disease; Information Technology reduces costs by improving efficiency and communications; Standards of Practice improve procedural efficiency and diagnosis; High Tech improves diagnosis and the physical viability of interventions; Improved Drugs reduce the frequency of acute conditions; Early Diagnosis recognizes a condition before it reaches acute levels; a Bad Lifestyle include obesity, drug abuse or other activities the damage health; and a Good Lifestyle includes exercises and food regimens that improve health. Note that all these can only delay death and they often compete with each other for resources. All policy interventions are exogenous to the model

$$IMC_{p,t} = (1.0 - (1.0 - Pm_p) * PP_t)^{SE}$$

The Indicated Multiplier Component (IMC) is the weighting of each individual policy impact (as affected by participant participation) for later use, to determine the overall combined impact of interventions.

$$\tau 0_t = \tau 0_{t-1} + (IE_t - \tau 0_{t-1}) / (LAT + IE_t)$$

The aging offset ( $\tau_0$ ) is a gradual increase in life expectancy due to an assumed policy Indicated-Extension (IE) intervention. (Note again that the purpose of this study is to assess the feasibility to test such interventions and not to validate their actual efficacy.)

$$IE_t = \sum_p PLE_{p,t} * PP_t / \sum_p ITW_{p,t}$$

An indicated life extension could be the consequence of several interacting Policy Life-Extension (PLE) measures, as affected by patient participation and the indicated time weight (ITW) for that implementation to show impact).

$$ITW_{p,t} = PLE_{p,t} / (PLE_{p,t} + \varepsilon)$$

The ITW is simply a zero or one weighting on the existence of the intervention policy. (The  $\varepsilon$  adder avoids divide by zero calculations.)

$$\lambda m_t = \ln(AR / (Sm_t * XDR_{min})) / (\lambda * (\tau_{\infty_t} - m - \tau_0_t))$$

$$\tau_{\infty_t} = \text{Min}(MLT, \ln(1 / XDR_{min}) / \lambda + m - \tau_0_t)$$

The time constant multiplier  $\lambda m$  and the asymptotic time  $\tau_{\infty}$  of CDR or NDR (noted as XDR) renormalizes the impacts to ensure the time to death does not exceed the Maximum Life expectancy Time (MLT) [MLT is assumed not to exceed 120 years.]

$$AR = \text{Min}(XDR_{min} * \exp((MLT - m) / \lambda), 1.0)$$

Similarly the Asymptotic Rate (AR) renormalizes the slope of the death rate curves when changed under policy interventions.

$$TC_t = \sum_q CC_{q,t}$$

The total medical costs (TC) are the sum of the medical cost components (CC) over the categories “q.” The categories are: Chronic maintenance medical costs, Acute chronic medical costs, Chronic Drug costs, Non-chronic maintenance medical costs, Non-chronic acute medical costs, and Non-chronic drug costs.<sup>8</sup>

$$CC_{q,t} = UC_q * XD_t * XDR_t * Sm_{q,t}$$

The intensity of medical conditions within the population (assuming fixed proportions of diseases) is proportional to the chronic or non-chronic death rate (XDR) of that population (P) compared to the normal (XDRN). The costs increase with acuteness of disease and are therefore all proportional to XDR. [Again, the “X” is meant to be a placeholder that designates a variation in name such as CDR versus NDR.] The cost is

<sup>8</sup> All economic information is in real 2003\$. Thus, stated cost growth is above inflation.

then proportional to the Deaths involved. The XDRN can be subsumed in the Unit Cost Constant (UC). The UC is derived from existing costs data.<sup>9</sup>

$$Sm_{q,t} = TM_{q,t} * \prod_p Pm_{p,q,t}$$

For costs, the scale multiplier is the combined policy interventions and the technology multiplier (TM).

$$TM_{q,t} = TM_{q,0} + \int_0^t TM_{q,t} * TMR_{q,t} * dt$$

The technology multiplier compounds with exogenously specified growth rates. The default values are noted below. No data were found to distinguish chronic from non-chronic medical costs growth.

**Table 5: Autonomous Cost Growth Rates.**

Cost category	Cost growth rates (%/yr)
Non Chronic Maintenance	5.0
Non Chronic Acute	5.0
Non Chronic Drugs	3.0
Chronic Maintenance	5.0
Chronic Acute	5.0
Chronic Drugs	3.0

$$GDP_t = ((P_t - CP_t) * GPP * Min(1, (GPP * exp(TG * t) - MPC_t)) / MI + CP_t * GPP * PAI * Min(1, (GPP * PAI * exp(TG * t) - MPC_t) / MI) * exp(TG * t))$$

The Gross Domestic Product (GDP) calculation uses a Cobb-Douglas production-function formulation that contains a constant capital-labor ratio and two labor types (chronic and non-chronic). Technological growth (TG) also improves GDP.

Data indicate chronic patients have only half the productivity (defined as on-average GDP per capital -- GPP) of more healthy individuals and thus, often the equivalent reduced income (PAI). Medical costs Per Capita (MPC) and work issues further lead to reduced income. As medical cost erode disposable income compared to the poverty (Minimum) levels of Income (MI), productivity further erodes to unemployment or homelessness. This formulation attempts to provide a first-order approximation to increasing medical costs on the economy.

$$MPC_t = TC_t / P_t$$

<sup>9</sup> <http://www.cms.hhs.gov/NationalHealthExpendData/>  
<http://www.meps.ahrq.gov/mepsweb/WhatIsMEPS/Overview.HTM>

Medical cost per Capita is simply total costs divided by population. A homogenous value is used to reflect the convention equalitarian approach to medical costs payments and to avoid ethical components to this analysis.

$$TG_t = TG_0 + \int_0^t TG_t * TGR_t * dt$$

Technology growth is just the compounding integral of the exogenously specified technology growth rate (TGR).

$$PP_t = 1.0 / (1.0 + (MPC_t * (1 - PC_t) / (AI_t * PAI * OPF))^{PVF})$$

The Patient Participation fraction (PP) is here assumed to be an economic decision that compares the uncovered costs to the fraction of Average Income (AI) that is deemed usable for Out-of-Pocket Payments (OPF). The uncovered costs are the MPC less any reduction through government aid (participation coverage fraction - PC). OPF and PC are policy variables. The participation variance factor (PVF) is set to 2 (equivalent to a unity elasticity that implies a change in a \$ used for medical needs will be offset by a \$ previously used elsewhere.) A sister study will use actual SNL employee data to estimate the value for a test population.

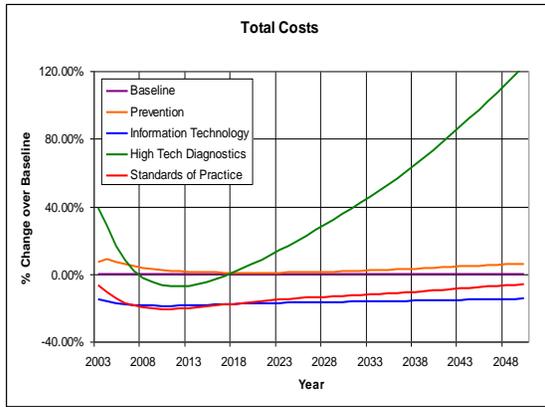
$$AI_t = GDP_t / P_t$$

The metric for average income (AI) is simply defined here as the GDP divided by the population (P).

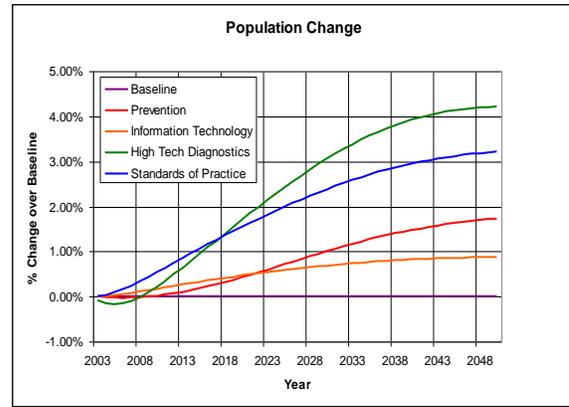
## Analysis

A series of model tests looked at prevention (assuming 100% participation), information technology, Standards of Practice, and High Tech solutions, where the interventions corresponded to an assumed 20-30% improvement over historical values. The results indicate ready feasibility and are shown in Figures 7.2 and 7.3 below.

With Prevention and High Tech solutions, there are initial added costs before benefits occur. For Information Technology and Standards of Practice, there are immediate declines in cost (assuming efficient implementation), but there is still a delay to achieve full response. In all cases, the long term response again leads to a rise in costs. This is due to the positive impact of causing a population increase and the added associated cost with the larger, older population.



**Figure 7.2 Cost Impacts.**



**Figure 7.3 Population Impacts.**

In many instances, the population initially declines with the beginning of new intervention. This occurs because of limited resources and start-up glitches that reduce services for acute patients. This is especially true for the high costs of high-tech solutions. The long-term does produce significant benefits, but at a high initial cost in dollars and conventional life-saving. In the very long term, ever more impressive technologies marginally increase life spans, but with diminishing returns. (The model assumes the maximum life span is 120 years. A longer maximum life span would lead to yet large costs due to a much large, medically-dependent population.)



## **Chapter 8: Scalability and Dimensional Mapping**

While the OMEGA-SIM system is on an evolutionary path that should allow the national scale required for a full implementation of the efforts begun here, the organization of the computation framework needed consideration. This effort used the PROMULA<sup>10</sup> system that is an advancement over FORTRAN 95 for large array-based systems of equations. Using the characteristics of individual and institutions within the healthcare system as array dimensions would ease the mathematical complexity of a full scale system. The dimensions of key interest are:

*Entity: Index for each individual agent (100,000 used in this test)*

*Interval Time: 70 year historical and future time increments for results.*

*Diagnosis: Type of health conditions (only two used for testing)*

*Condition: Level of health condition intensity: asymptomatic, sub-acute, or post-acute' (3 used for testing)*

*Status: Characterization of agent by age, gender, (e.g. state), locale (e.g. urban vs.. rural), ethnicity, wealth, education, household position (e.g., head of household), group (e.g., adult vs.. child), weight, employer, occupation, medical condition and other to be determined (20 used for testing)*

*Characteristic: Type of institution (e.g. insurance companies, hospital)' (1 use for testing)*

*Institution: Index for institutions (1 use for testing)*

*Region: Index for Regions (53 used for testing)*

*Gender: Gender Index (2 used for testing)*

*Ethnicity: Ethnic Index (4 used for testing)*

*Wealth: Wealth Index (Quintile used for testing)*

*InsOpt: Insurance Options Index (6 used for testing)*

*Regime: Medical Procedure or Drugs Index (2 used for testing)*

PROMULA type methods appear to have all the flexibility needed for efficient computation mapping of dimensionality across algorithmic domains. An example of prototype equations and the organizational framework is provided in Appendix D

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<sup>10</sup> [www.promula.com](http://www.promula.com) , <http://www.greatmigrations.com/>



## ***Chapter 9: Summary***

Future security assessment will need to focus on the behavioral impacts of interventions. This study uses the national healthcare system as a vehicle to explore the methods and approaches available for including behavior within an analysis framework. The work here indicates that all the required components to successful analysis are feasible.



## References

- Backus, G. A., et al, 2002, *Climate Change Plan for Canada*, [http://climatechange.gc.ca/english/publications/plan\\_for\\_canada/](http://climatechange.gc.ca/english/publications/plan_for_canada/)
- Barton, D. C., and G. A. Backus, 2002, *An Example of Infrastructure Interdependency Analysis: Local, Regional, and National Economic Impacts*. Sandia Report SAND2002-0911, Sandia National Laboratories, Albuquerque, NM.
- Barton, D. C., E. D. Eidson, D. A. Schoenwald, R. G. Cox, and R. K. Reinert, January 2004, *Simulating Economic Effects of Disruptions in the Telecommunications Infrastructure*. Sandia Report SAND2004-0101, Sandia National Laboratories, Albuquerque, NM.
- Barton, D. C., E. D. Eidson, D. A. Schoenwald, K. L. Stamber, and R. K. Reinert, December 2000, *Aspen-EE: An Agent-Based Model of Infrastructure Interdependency*. SAND Report, Sandia National Laboratories, Albuquerque, NM.
- Hand, M. S., P. J. Paez, and J. A. Sprigg Jr., April 2005, *On the Need and Use of Models to Explore the Role of Economic Confidence: A Survey*. SAND Report, Sandia National Laboratories, Albuquerque, NM.
- Hirsch 2006: J. Homer J., and G. B. Hirsch, 2006, System Dynamics Modeling for Public Health: Background and Opportunities. *American Journal of Public Health*, vol. 96:3, pp. 452-458.
- Hirsch, G. B., July 2004, *Modeling the consequences of major incidents for health care system*. 22<sup>nd</sup> International Conference of the System Dynamics Society, July 25-29, 2004, Oxford, England. Available at <http://cgi.albany.edu/~sdsweb/sdsweb.cgi?P121> .
- Hirsch, G. B., and J. Homer, (forthcoming 2007). System Dynamics Applications to Health Care in the United States. (to be published in *Encyclopedia of Complexity and System Science*), R. Meyers, ed. Springer-Verlag: Berlin, Germany.
- Hirsch, G. B., J. Homer, G. McDonnell, and B. Milstein, July 2005, *Achieving health care reform in the United States: toward a whole-system understanding*. 23<sup>rd</sup> International Conference of the System Dynamics Society, July 17-21, 2005, Boston, MA. Available at <http://www.systemdynamics.org/conf2005/proceed/papers/HIRSC406.pdf>
- Hoard, M., J. Homer, W. Manley, P. Furbee, A. Haque, and J. Helmkamp, 2005, Systems Modeling in Support of Evidence-Based Disaster Planning for Rural Areas. *International Journal of Hygiene and Environmental Health*, vol. 208, pp. 117-125.

Homer J., G. B. Hirsch, M. Minniti, and M. Pierson, 2004, Models for Collaboration: How System Dynamics Helped a Community Organize Cost-Effective Care for Chronic Illness. *System Dynamics Review*, vol. 20(3), pp. 199-222.

Jones A., J. Homer, D. Murphy, J. Essien, B. Milstein, and D. Seville, D., 2006, Understanding Diabetes Population Dynamics through Simulation Modeling and Experimentation. *American Journal of Public Health*, vol. 96(3), pp. 488-494.

McFadden, D., and K. Train, 2000, Mixed MBL Models for Discrete Response. *Journal of Applied Economics*, vol. 15, pp. 447-470.

Milstein, B., A. Jones, J. Homer, D. Murphy, J. Essien, and D. Seville, July 2007, Charting Plausible Futures for Diabetes Prevalence in the United States: A Role for System Dynamics Simulation Modeling. *Preventing Chronic Disease*, vol. 4(3). Available at: [http://www.cdc.gov/pcd/issues/2007/jul/06\\_0070.htm](http://www.cdc.gov/pcd/issues/2007/jul/06_0070.htm).

National Center for Health Statistics, 2005, Health, United States. Department of Health and Human Services, Hyattsville, MD.

Office of Immigration Statistics, January 2006, *2004 Yearbook of Immigration Statistics*. U.S. Department Of Homeland Security, Washington DC.

Schoenwald, D. A., and R. M. Cranwell, September 2005, "Development of an Enterprise-Scale Agent-Based Autonomic Logistics Simulation Model," *Laboratory Directed Research & Development (LDRD) FY2005 Annual Report*. Sandia National Laboratories, Albuquerque, NM.

Schoenwald, D. A., D. C. Barton, and M. A. Ehlen, M. A., July 2004, "An agent-based simulation laboratory for economics and infrastructure interdependency," *Proceedings of the 2004 American Control Conference*. Boston, MA (also Sandia Report SAND2004-2591C).

Siirola, J. D., August 2007, *Agent-oriented Software Engineering for Simulation and Optimization*. Sandia Report SAND 2007-5021P, Sandia National Laboratories, Albuquerque, NM.

Sprigg Jr., J. A., November 2004, *Market Disruption, Cascading Effects, and Economic Recovery: A Life-Cycle Hypothesis Model*. SAND Report, Sandia National Laboratories, Albuquerque, NM.

Sprigg Jr., J. A., C. R. Jorgensen, and R. J. Pryor, August 2004, *Approach and Development Strategy for an Agent-Based Model of Economic Confidence*. SAND Report, Sandia National Laboratories, Albuquerque, NM.

Sprigg, J. A., and M. A. Ehlen, November 2004, *Full Employment and Competition in the Aspen Economic Model: Implications for Modeling Acts of Terrorism*. SAND Report, Sandia National Laboratories, Albuquerque, NM.

Strip, D. R., and G. A. Backus, September 2007, *Architectural Considerations for Agent-Based National Scale Policy Models, LDRD Final Report*. Sandia Report SAND2007-5847, Sandia National Laboratories, Albuquerque, NM.



## **Appendix A: Data Sources and Analysis for Calibration of Cardiovascular Disease Population Model**

### **View 1: population by risk class and mortality rates**

#### **Distribution of US adults by number of risk factors (RF's):**

<i>RF's</i>	<i>%</i>	<i>Risk Class</i>
0	.38	RC1 (Low Risk)
1	.34	RC2a (Intermediate Risk, would screen negative for CVD)
2	.19	RC2b (Intermediate Risk, would screen positive for CVD)
3+	.09	RC3 (High Risk)

Breakdown for number of RF's from Greenlund, et al (2004.) We assume a direct correspondence between number of RF's and Risk Class; the latter determines eligibility for risk management.

#### **US adult population**

201 million people age 20 and above from 2000 Census.

[http://www.censusscope.org/us/chart\\_age.html](http://www.censusscope.org/us/chart_age.html)

#### **Adult population death rate if no CVD attack deaths, and Extent to which CVD attack deaths increase total deaths**

Start with age distribution of people with CVD from NHIS series 10, number 200, P.82

Age	% of adult popn
18-44	.01
45-64	.36
65-74	.24
75+	.38

and apply mortality rates by age from NCHS at

[http://www.cdc.gov/nchs/data/nvs.r/nvs.r54/nvs.r54\\_19.pdf](http://www.cdc.gov/nchs/data/nvs.r/nvs.r54/nvs.r54_19.pdf)

to get weighted average mortality rate for population with same age distribution as CVD population. This calculation yields an overall mortality of .036 and implies 853K deaths from the *symptomatic CVD population of 23 million*.

Of these 853K deaths in the symptomatic, some are due to acute attacks (for attack and fatality rates, see further below):

Subacute: 11m popn x 3.6% attack rate x 47% fatality rate = 186K attack deaths;

Post-acute: 12m popn x 5.8% attack rate x 47% fatality rate = 327K attack deaths;

Total symptomatic attack deaths = 513K.

The difference is the number of deaths of symptomatic due to reasons other than CVD attack:  $853\text{K} - 513\text{K} = 340\text{K}$ ;  $340\text{K}/23\text{M} = 1.48\%$  per year rate.

This 1.48% rate tells us the rate of dying due to non-CVD causes, but it does not tell us what the death rate *would be* in the absence of CVD attacks. In the extreme, imagine that the entire CVD population died from acute attacks, leaving none to die from other causes. The apparent non-CVD death rate would then be 0%. Now imagine that CVD deaths were instantaneously and universally eliminated. That would not reduce the death rate to zero! It would only reduce it by the *extent to which CVD attack deaths increase total deaths*.

Thus, we see that the base rate (*Adult popn death rate if no CVD attack deaths*) must be something greater than 1.48%. To know how much greater, we must know the *Extent to which CVD attack deaths increase total deaths*. To pick a starting point, let us assume that parameter = **0.5**, and see where that gets us with regard to the symptomatic population statistics cited above.

Overall deaths = (*Adult popn death rate if no CVD attack deaths*\*Popn) + (Attack deaths \* *Extent to which CVD attack deaths increase total deaths*)

$853\text{K} = (\text{Adult popn death rate if no CVD attack deaths} * 23\text{M}) + (513\text{K} * 0.5)$

implies

*Adult popn death rate if no CVD attack deaths* = 2.6%

Is this 2.6% a reasonable figure? The average age of adults in the U.S. is 46 years. The inverse of 2.6% implies additional life expectancy of 38 years, giving a total life expectancy for adults of 84 years. This seems like a reasonable estimate of life expectancy for an adult without CVD.

Note that with the inclusion of attack deaths, the death rate of the symptomatic is  $(853\text{K}/23\text{M}) = 3.7\%$ , the inverse of which is 27 years, giving a total life expectancy for symptomatic CVD adults of 73 (=46+27) years at present. So, given our assumptions, the elimination of attack deaths could add 11 (=84-73) years to the life expectancy of Americans with CVD.

### ***View 3: Attack Rates and Fatal Fractions***

#### ***Attack rates for Asymptomatic population***

Average annual attack rates for asymptomatic people by risk class were based on calculations using Framingham data for different numbers of risk factors. The NHLBI cardiovascular risk calculator available at <http://hp2010.nhlbihin.net/atp/iii/calculator.asp> gives the following:

#### ***Attack rates by number of risk factors:***

*RF's*    *Average attack rate/yr.*

0	.0028
1	.005
2	.0084
3+	.014

When these average attack rates are applied to the 178M asymptomatic population (201M – 23M = 178M asymptomatic), one gets 1.13M acute attacks among the asymptomatic, rather than the 800K expected from AHA statistics (see below). Therefore, we assume that the risk calculator has overestimated attacks, and multiply each of the attack rates above by a factor of 0.71 (= 800/1130) to get: .0020, .0036, .0060, and .0099.

Risk classes 2b (RF 2) and 3 (RF 3+) are eligible for risk management. We estimate (see below) that 20% of RC3 patients receive intensive risk management and 0% of RC2b patients (because only a negligible number today receive screening.)

Various studies have found 23-42% reduction in cardiac events by having patients on statins. The UKPDS found a 32% reduction in diabetes-related endpoints. The CDC Diabetes Cost-Effectiveness Group (CDC, 2002) brings these impact estimates together; they estimate the impact of conventional and intensive treatment as follows:

- For glycemic control, they estimate a 25% reduction in HbA1c levels for intensive treatment.
- For intensive hypertension control, they use a 21% reduction for coronary heart disease and 44% reduction for stroke which together produce a weighted average 29% reduction in CVD.
- Serum cholesterol reduction using Pravastatin is assumed to produce a 31% risk reduction for patients without CHD and a 25% reduction for patients who already have CHD.

We estimate that half of the high-risk group requires glycemic control; so, the average effect of management on glycemic control is 12.5%. Multiplying the three (.875 x .71 x .69) to get a joint effect produces a multiplier for the three together of **.43**, a reduction of 57% in attack rate for the highest risk group. Thus:

$$\begin{aligned} \text{Avg attack rate for RC3} &= (\text{Zero-mgmt rate})(80\%) + (\text{Max-mgmt rate})(20\%) \\ &= (\text{Zero-mgmt rate})(80\% + (0.43)(20\%)) \\ &= (\text{Zero-mgmt rate})(.886) \end{aligned}$$

Avg rate for RC3 (RF=3+) from the above table = .0099, so

$$\text{Zero-mgmt attack rate for RC3} = .0099 / .886 = .0112$$

$$\text{Max-mgmt attack rate for RC3} = .0112 * .43 = .0048$$

We assume that the Max-mgmt rate for RC2b is mid-way between that of RC2a and RC3.

This gives the following table:

***Attack rates/yr. by Risk Class and Risk Management***

<i>RC</i>	<i>Zero mgmt</i>	<i>Max mgmt (% Reduction for Max- vs.. Zero-mgmt)</i>
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RC1	.0020	.0020	(N/A)
RC2a	.0036	.0036	(N/A)
RC2b	.0060	.0042	30%
RC3	.0112	.0048	57%

***Symptoms onset rates for Asymptomatic population***

Annual incidence of 400K new cases of angina and approximately another 100K cases of new TIA based on AHA statistics: ***symptoms onset of 500K per yr.*** Compare this to 700K new heart attacks and 500K new strokes of which about two-thirds (67%) are in people who were not previously symptomatic:  $1200K * 2/3 = 800K$  ***attacks among the Asymptomatic per yr.*** Thus, the ratio of symptoms onset to attacks in the Asymptomatic is  $500K/800K = 0.625$ . This yields annual symptoms incidence rate that are  $500K/(1200K * 0.67)$  or 0.55 times the acute attack incidence by risk class for asymptomatic people.

The ***fraction of attacks without previous symptoms (67%)*** comes from an estimate that 50-60% of new (non-recurrent) heart attacks are in people who previously had no new symptoms, and that 85% of people with new strokes had no previous symptoms, only 15% of strokes were heralded by a TIA as indicated in the AHA heart and stroke statistics (Bechar et al, 1992; and Pierard et al. 1988). 67% is a weighted average of the fractions for heart disease (55%) and stroke (85%).

When one assumes that the ratio of symptoms onset to attack onset is 62.5% for all risk classes based on the above, the model produces steady-state prevalence of the Subacute which is smaller than the 11M that it should be based on AHA statistics. To get the 11M, one must instead assume that the ratio of symptoms onset to attack onset is 85% rather than 62.5%. This produces the following table:

***Symptoms onset rates/yr. by Risk Class and Risk Management***

RC	Zero mgmt	Max mgmt
RC1	.0017	.0017 (N/A)
RC2a	.0031	.0031 (N/A)
RC2b	.0051	.0036
RC3	.0095	.0041

***Attack rates for Subacute patients***

If two-thirds of new attacks are in the Asymptomatic, then one-third are in the Subacute:  $1/3$  of  $1200K = 400K$  ***attacks among the Subacute per yr.*** Divided by an ***estimated Subacute population of 11M***, this gives an average attack rate of 3.6% per year.

Let us assume that the reduction in acute attack rates with maximum disease management is 50% for the subacute, the same as for the post-acute (see below). Let us also assume that the managed fraction of the subacute is 33% (see below). Thus:

$$\begin{aligned} \text{Avg attack rate for subacute} &= (\text{Zero-mgmt rate})(67\%) + (\text{Max-mgmt rate})(33\%) \\ &= (\text{Zero-mgmt rate})(67\% + (0.50)(33\%)) \\ &= (\text{Zero-mgmt rate})(.835) \end{aligned}$$

Avg rate = .036 (above), so

$$\text{Zero-mgmt attack rate for subacute} = .036 / .835 = .0431$$

$$\text{Max-mgmt attack rate for subacute} = .0431 * .50 = .0216.$$

### ***Recurrent attack rates for Post-acute patients***

AHA statistics (Heart Disease and Stroke Statistics: 2006 Update) indicate 500K recurrent heart attacks (based on ARIC data) and 200K recurrent strokes (based on Greater Cincinnati/Northern Kentucky Stroke Study) for a total of **700K recurrent attacks per yr.** In a **Post-acute population of about 12 million**, this implies a rate of 5.8% per year.

The literature suggests 25% reductions in recurrent attacks and death with each of beta-blocker usage and statin usage (Goldman et al. 1988; and Sacks et al. 1996). Assuming a combined program of these and other interventions (glycemic control, weight loss, smoking cessation) might suggest a 50% overall reduction in recurrence with maximum disease management.

Let us also assume that the managed fraction of the post-acute is 50% (see below). Thus:

$$\begin{aligned} \text{Avg attack rate for post-acute} &= (\text{Zero-mgmt rate})(50\%) + (\text{Max-mgmt rate})(50\%) \\ &= (\text{Zero-mgmt rate})(50\% + (0.50)(50\%)) \\ &= (\text{Zero-mgmt rate})(.75) \end{aligned}$$

Avg rate = .058 (above), so

$$\text{Zero-mgmt attack rate for post-acute} = .058 / .75 = .0773$$

$$\text{Max-mgmt attack rate for post-acute} = .0773 * .50 = .0387.$$

### ***Fatal fractions for non-sudden death acute attacks, and***

#### ***Sudden death fraction of attacks***

AHA reports **900K CVD deaths from 1.9M acute attacks**: a fatality rate for attacks of 47%. Data don't make it easy to distinguish between mortality rates for new vs. recurrent attacks; absent other data, we'll assume 47% for both.

The data suggest limited ability to reduce the fatality of attacks. According to one article, 63% of cardiac deaths are sudden and occur before the patient even gets to the hospital (Zheng, et al., 2001.) A study in Oregon found that only 8% of cardiac arrest patients were successfully resuscitated before getting to the hospital (Chugh, et al., 2004.) The sudden death fraction for strokes is probably less than that for heart attacks. We therefore assume that 55% of acute attack deaths overall are sudden. This would make the sudden

death fraction of attacks  $[55\% \times 47\%] = 26\%$ . Non-sudden-death attacks account for the remainder, 74%, of all attacks, and their deaths account for  $[47\% - 26\%] = 21\%$  of all attacks. Thus, the average death rate for non-sudden death attacks is  $(21\%/74\%) = 28\%$ .

Once patients are in the hospital, case fatality rates have fallen as a result of better treatment and average around 10%. More aggressive treatment (e.g., angioplasty) might reduce that more substantially, by as much as two-thirds (Zahn et al., 2000).

We assume that, today, 70% of non-sudden death attacks are receiving aggressive intervention (see below).

Thus:

Avg fatality rate for non-sudden death attacks = (Zero-mgmt rate)(30%) + (Max-mgmt rate)(70%)

$$= (\text{Zero-mgmt rate})(30\% + (0.33)(70\%))$$

$$= (\text{Zero-mgmt rate})(.53)$$

Avg non-sudden death fatality rate = .28 (above), so

Zero-mgmt non-sudden-death attack fatality rate =  $.28/.53 = .53$

Max-mgmt non-sudden-death attack fatality rate =  $.53 * .33 = .175$ .

#### ***View 4: Risk Screening and Management***

##### ***Resources required per RC2 screening***

Several sources suggested that screening costs for Risk Class 2 (intermediate risk) patients would be about \$350 for one of the more sophisticated tests or \$140 for one that is less sophisticated, but still more expensive than simply calculating the ratio between brachial and femoral blood pressures. An article <http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=hstat6.section.622> indicates that EBCT and exercise stress tests have a similar cost of about \$350-400.

Hayashino, et al. (2004), show exercise electrocardiography to be quite inexpensive (\$140), but that exercise echocardiography is actually the most cost-effective method at a cost of \$334 per test in patients with diabetes plus other risk factors.

<b><i>Screening costs (2003 \$)</i></b>	Baseline	Low	High
Exercise electrocardiography	140	98	182
Exercise echocardiography	334	234	434
Exercise SPECT	730	511	949
Coronary angiography	6,035	4,225	7,846

We assume that RC2 screening would be done by exercise echocardiography at a cost of **\$350** per screening.

***Screening Interval***

There is no clear guidance in the literature. Screening is mostly discussed as a one-time event. Five years seems a reasonable interval given the rate at which CVD might develop in an at-risk population. This is the same interval recommended for some other similarly priced mass-population screening procedures, such as colonoscopy.

***Resources required per patient for maximum asymptomatic risk management***

The following annual costs are from the CDC diabetes cost-effectiveness study (CDC 2002).

Intensive glycemic control	\$1531	(\$538 for conventional)
Intensive hypertension control	667	(\$301 for conventional)
Pravastatin for serum cholesterol	1398	

The following costs are from Hayashino et al. (2004).

<b>Risk management costs</b>	Baseline	Low	High
Conventional diabetes care	1,113	779	1,447
Simvastatin	1,293	905	1,680
Aspirin	16	11	21

***We assume that 50% of patients eligible for CVD risk management (RC3 and RC2b) would require treatment for hyperglycemia.*** With inclusion of anti-hypertensive treatment at a cost of \$667 and aspirin at \$16, the average total cost for comprehensive treatment based on CDC would be  $[1398+667+16+ 0.5*1531] = \$2846.50$ ; and based on Hayashino would be  $[1293+667+16+ 0.5*1113] =\$2532.50$ . We assume a cost of \$2690, midway between these estimates.

***View 5: Disease Management and Attack Treatment***

***Resources required per subacute patient for maximum disease management, and  
Resources required per post-acute patient for maximum disease management***

In addition to the costs listed above, Hayashino (2004) lists the following additional annual costs for symptomatic CVD patients, due to more frequent monitoring and testing, plus increased episodic visits to the physician due to symptoms flare-up:

<b>Additional costs for symptomatic CVD</b>	Baseline	Low	High
Symptomatic myocardial ischemia	1,224	857	1,591
History of MI	1,431	1,002	1,860

For subacute patients, we add the first of these costs, \$1224, to the \$2690 risk management costs cited previously, or \$3914 in total. For post-acute patients, we add the second of these costs, \$1431, to the \$2690 risk management costs, or \$4121.

***Resources required per subacute symptoms onset for maximum disease management***

Hayashino (2004) mentions a one-time cost for onset of ischemic symptoms of \$2992 (see table below). This is likely a cost for imaging and other diagnostic studies when a patient first becomes symptomatic.

***Resources used per sudden death attack***

We assume \$1000 per sudden death attack for EMT services and post-mortem procedures at the hospital.

***Resources required per non-sudden death acute attack for maximum treatment***

Hayashino (2004) shows a cost in the table below of \$21,161 for treating a surviving MI patient.

<b>One-time acute attack costs</b>	Baseline	Low	High
Symptomatic myocardial ischemia	2,992	2,094	3,889
MI death	23,843	16,690	30,996
MI survival	21,161	14,813	27,509

Hayashino (2004) also outlines the potential risk reduction and cost of PTCA (angioplasty) and CABG (bypass surgery) for preventing recurrent attacks.

<b><i>Risk reduction for revascularization after MI</i></b>	Baseline	Low	High
PTCA	17%	0	22%
CABG	42%	29%	55%

<b>Cost for revascularization after MI</b>	Baseline	Low	High
PTCA	15,884	11,119	20,650
CABG	42,125	29,487	54,762

It's not clear what fraction of patients is expected to benefit from these procedures. According to the *2002 NCHS Hospital Discharge Survey*, there were 515K CABG's, 1328K cardiac catheterizations, and 1204K removal of coronary obstructions and insertions of stents. Because there are often several procedures performed on the same patient, a better indicator may be the numbers of discharges with one of these procedures: **653K with PTCA and insertion of stents, and 306K with CABG**. This would suggest that PTCA and stent insertion are performed in 653K/2153K or 30% of CHD admissions and CABG is performed in 306K/2153K or 14% of CHD admissions. Applying these fractions yields a cost of  $[21,161 + .30 \times 15,884 + .14 \times 42,125]$  or \$31,913 or about \$32,000 per non-sudden death acute attack patient.

### ***View 6: Initial and Total Resources by Type***

#### ***Asymptomatic risk management vs. maximum***

AHA statistics, quoting 1999-2000 NHANES indicate that only 33% of hypertension is controlled in whites and smaller percentages in Hispanics and blacks. Regarding serum cholesterol, less than half of persons who qualify for any kind of lipid-modifying treatment for CHD risk reduction are receiving it. Less than half of even the highest-risk persons, those who have symptomatic CHD, are receiving lipid-lowering treatment. Only about a third of treated patients are achieving their LDL goal; less than 20 percent of CHD patients are at their LDL goal. These statistics would suggest that 0.2 is a good initial estimate.

#### **Subacute disease management vs. maximum, and**

***Post-acute disease management vs. maximum***

There is a lot written about failure of physicians to prescribe basic things such as beta blockers for post-MI patients, but no quantification. The fractions of 0.33 for subacute and 0.5 for post-acute seem reasonable and may even be overly optimistic. One article indicated that even when patients are getting beta blockers, they get less than the optimal dose.

***Non-sudden-death acute attack treatment vs. maximum***

Treatment for acute attacks appears to be more aggressive and 0.7 seems reasonable for this parameter.

***View 7: Quality Adjusted Life-Years and Unhealthy Days***

The model also includes a measure of Quality Adjusted Life Years (QALY's) that is useful for comparing simulations. This measure calculates the numbers of years lived in a population over time and larger numbers reflect greater effectiveness of preventive or treatment strategies in keeping more people alive. The model also calculates the cost per additional QALY compared to a baseline simulation in order to show the cost-effectiveness of different strategies. The QALY measure includes an adjustment for lower quality of life (in terms of number of unhealthy days per month) for people with subacute or post-acute CVD and differentiates between people receiving effective disease management and those who are not. Unhealthy days are the broadest measure of reduced quality of life due to illness and we therefore thought they represented an appropriate basis for adjustment. The following numbers of unhealthy days per month are used in the model for each of these groups.

	No Disease Management	With Disease Management
Asymptomatic (and General non-CVD Population)	6	N/A
Subacute	9	7.5
Post-Acute	13.6	9.8

Data for these numbers came from a monograph called Measuring Healthy Days (<http://www.cdc.gov/hrqol/monograph.htm>) (see Table 2) published by the CDC's National Center for Chronic Disease Prevention and Health Promotion and were adjusted to reflect effects of disease management based on experience with diabetes modeling. The monograph (Table 2) also contains numbers of limited activity days for people with CVD. Limited activity days could also be incorporated into the model in the future if there seems to be value in doing so. While a narrower measure, limited activity days

could be more easily linked to disability costs and used as part of an overall cost of disease measure.

## ***References***

Bechar S., Reicher-Reiss H., Abinader, E., et al (1992). The prognostic significance of angina pectoris preceding the occurrence of a first acute myocardial infarction in 4166 consecutive hospitalized patients. *American Heart Journal*, 123, 1481-6.

CDC Diabetes Cost-Effectiveness Group (2002). Cost-effectiveness of intensive glycemic control, intensified hypertension control, and serum cholesterol level reduction for Type 2 diabetes. *Journal of the American Medical Association*, 287(19), 2542-2551.

Chugh, S. S., et al (2004). Current burden of sudden cardiac death: Multiple source surveillance versus retrospective death certificate-based review in a large U.S. community. *Journal of the American College of Cardiology*, 44, 1268-1275.

Goldman, L., Sia, S. T., Cook, E. F., Rutherford, J.D., Weinstein, M. C. (1988). Costs and effectiveness of routine therapy with long-term beta-adrenergic antagonists after acute myocardial infarction. *New England Journal of Medicine*, 319, 152-157.

Greenlund, et al (2004). Trends in self-reported multiple cardiovascular disease risk factors among adults in the United States 1991-1999. *Archives of Internal Medicine*, 164, 181-187.

Hayashino, Y., Nagata-Kobayashi, S., Morimoto, T., et al (2004). Cost-effectiveness of screening for coronary artery disease in asymptomatic patients with Type 2 diabetes and additional atherogenic risk factors. *Journal of General Internal Medicine*, 19(12), 1181-1191.

Pierard, L. A., et al (1988). Prognostic significance of angina pectoris before first acute myocardial infarction. *American Journal of Cardiology*, 61, 984-7.

Sacks, F. M., et al., for the Recurrent Events Trial Investigators (1996). The effect of Pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. *New England Journal of Medicine*, 335(14), 1001-1009.

Zahn, R., et al., for the Maximal Individual Therapy in Acute Myocardial Infarction (MITRA) and the Myocardial Infarction Registry (MIR) Study Groups (2000). Decreasing hospital mortality between 1994 and 1998 in patients with acute myocardial infarction treated with primary angioplasty but not in patients treated with intravenous thrombolysis: Results from the pooled data of the maximal individual therapy in acute myocardial infarction (MITRA) registry and the myocardial infarction registry (MIR). *Journal of the American College of Cardiology*, 36, 2064-2071.

Zheng, Z-J., Croft, J.B., Giles, W.H., and Mensah, G.A. (2001). Sudden cardiac death in the United States, 1989 to 1998. *Circulation*, 104, 2158.

## ***Appendix B: Summary of the Workshop***

### **Modeling Healthcare: The Sandia Initiative**

**September 18-19, 2007**

**Houston, Texas**

At the request of David R. Strip of the Sandia National Laboratory, Dr. Kenneth Shine, Executive Vice Chancellor for Health Affairs in The University of Texas System convened a workshop of experts to discuss the potential for modeling American healthcare phenomena. The participants, names and titles are listed in the accompanying material. They represented broad areas of expertise including health economics, health policy, hospital administration, academic health center administration, computational mathematics, computer simulation and modeling, health services researchers, the director for the Agency for Healthcare Research and Quality, state health policy, information technology and electronic health records. Public health was represented by a member of the group and by a written statement from the Texas Commissioner of Health, Dr. Eduardo Sanchez. Letters were also received from Larry Lewin, founder of the Lewin Consultation Group and Linda Bilheimer of the Center for Health Statistics.

The agenda of the meeting is enclosed. It included a series of formal presentations by several of the participants and a wide range of discussion among the group. The overall conclusions and recommendations are summarized as follows:

- A sensitive, accurate, and comprehensive computer simulation of American healthcare and health outcomes would be of inestimable value to the nation, to policy makers at the state, local and national levels, to researchers in health policy and many other participants in healthcare delivery systems.
- The long term vision for this exercise would be a model which allows prediction of outcomes from changes in financing, public health, quality of care, organization of healthcare services, access, and cost. The relationships between domestic and international developments in healthcare delivery, public health, training, and

workforce issues would also be part of such a model. The participants concluded that such a model is feasible, but would take a number of years to develop and would have to be developed incrementally.

- The participants recommended the development of models through a series of modules which focus on a particular element in healthcare, but have the capacity to communicate with other such modules. For example, a module might be focused on financing and/or insurance coverage in the United States and the various states. Ultimately these specialized modules would have to be connected to the inputs of and predict the outcomes related to the institutions which provide care, workforce needs, and health outcomes for the various populations affected. This concept was expressed variously as modular development, incremental development, and a multi-layer process which would take place over an extended period of time. It would have a strong basis in multi-scale analysis so that short term, intermediate and long term outcomes could be measured, impacts on populations are measured in aggregate, as well as for specific age, ethnicity, geographic, socio-economic and other subgroups, as well as similar treatment of many other parameters.

The attributes of such a system would include:

- Potent predictive capacity with emphasis on validation.
- Modeling of possible solutions as well as of problems, with an emphasis on predictive models
- Using health outcomes including health status, and functional status as well as morbidity, mortality, and longevity outcomes.
- Sub-analysis of special populations identified by ethnicity, socioeconomic class and in relation to health disparities.
- The model should be transparent a number of ways.
  - a. The assumptions underlying each portion of the model would be explicitly stated.
  - b. The source code would be available.
  - c. The methodology and language used would be provided in terms

understandable to broad variety of users.

- d. The methods for selecting questions to be asked and used in analysis would be available to all users.
  - e. Early in the development of the model a user's manual would be developed not only to guide those developing the program but to create a broad community of users of the system. (A textbook based upon the model, its use and applications was a topic of discussion).
- In the long term the simulation should be designed to test a wide variety of concepts and ideas, be they original or well-known, evolutionary, or revolutionary, creative or mundane. However, in the short term modules should be developed that can demonstrate early success and evidence that they can, in fact, inform the various users in an effective way.
  - Considerable concern was expressed about the ability to use existing data sets to inform a useful model going forward. While initial modules may be driven by such data, it is likely that substantial effort will have to be invested in additional data development, particularly as gaps in the data become obvious in the course of creating the model.
  - Substantial effort will have to be placed on the validation of model, quantification of the uncertainty of both data and of predictions, sensitivity analysis, particularly in determining the extent to which a variation in the outcome would actually affect policy. Success will be dependent on clear identification of the quantities of interest and a model which is adaptive, flexible and subject to modification as the healthcare system changes.
  - In a discussion led by Professor Tinsley Oden, three computational models of abstract systems were identified:
    1. System dynamics models.
    2. Multi-agent systems (agent-based models).

### 3. Discrete event models.

- There was consensus that agent-based models are better matched to the broad policy community's cognitive model and a consequently more transparent to the intended users. This would be particularly important in the healthcare system where there will be critical challenges in evaluating the choices which various agents make.
- Professor Oden cautioned that maintenance costs for the simulation could be a multiple of development costs up to 10x or greater. Consequently any plan should account for ongoing maintenance of both the software and required data sources.
- There was considerable discussion of roadmaps and/or planning documents which could be used to outline both the approach of the overall model and steps to be taken in order to create a proper model and computer code. The workshop participants believed that a white paper describing some detail of the overall approach to the model, with a specific set of early priorities, would be timely now.
- There was extensive discussion about the subjects which might be the initial modules to be developed in the process. There was some agreement that a module built around healthcare financing with special attention to employer-based health insurance would be timely and useful. This module would include an analysis of the various funding sources for healthcare; attempts to model cost; subsidization of care; identification of interactions between various forms of financing; consideration of "crowding out phenomena"; and the inter-relationship between the cost of care of caring for the uninsured, employer costs and other financing sources.
- The second area for modeling would be an area of current disease management, for example, diabetes, which one could analyze the organizational, structural workforce, educational and cost issues associated with improving care of diabetics and decreasing complication rates. The relationship between diabetes care before and after Medicare eligibility would be of particular interest since much of the costs for

complications are paid for by the Medicare system. This module would provide good opportunities to examine the interaction between public and private insurance systems, as well as the interaction between Medicare and Medicaid.

- A third module focused upon prevention intervention in the system assessing the various elements required for such a preventative activity, the cost and potential savings associated with prevention and the impact on health outcomes. A variety of such subjects were considered including prevention of colon cancer, decreased cigarette smoking and improved detection of hypertension.

The workshop participants recommended that the Sandia organization form a board of advisors who could provide guidance with regard to the overall direction of the effort and could identify expert consultants who might assist the Sandia personnel in developing modules and in identifying, evaluating or adding additional data. The board of advisors would oversee and perhaps endorse some uses of the model over others. It would help define the scope of the model and help to avoid uses based on unsupportable assumptions. Although once developed, portions of the model could be available for additional users, input by graduate students and academic faculty, the participants believed that the initial development of the model would require substantial Sandia investment in collaboration with appropriate experts from the outside community.

- The users of this system would range from academics attempting to do research in various aspects of the healthcare system to policy makers at the national, state and local level who wish to have timely answers to proposed changes in the healthcare system. Professional organizations, health provider organizations, insurance companies and others might also be important users of this model as it develops.
- There was general agreement, though not unanimous, that the models should be both nationally and state-based. While there was a general agreement that national models would be very important, the wide variation in policies and programs in the various states suggested to a number of participants that modeling states such as New Mexico

and Texas would provide a template which could then be used by other states as they assess the potential implications of the model for their own citizens. This will be an important and complex challenge to the project developers but should be kept in mind as these models develop.

The workshop participants expressed both support, and in many cases enthusiasm, for the development of this project. Almost all of the participants indicated they would be available to consult with Sandia staff as this project develops. Sandia staff was urged to consult further with state and local legislative staff to identify the most compelling policy issues for the next several years. Recommendations were also made for consultation with sources of statistical information including AHRQ, the National Center for Health Statistics, the Congressional Budget Office, the Office of Management of Budget, the AMC, the AMA, the University Hospital Consortium and the American Hospital Association.

Respectfully submitted

Kenneth Shine, M.D.

Executive Vice Chancellor for Health Affairs

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**Health Care Modeling Meeting**  
**September 18, 2006 to September 19, 2006**  
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## Appendix C: Initial CVD Model Equations (VENSIM Version)

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.Active

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### Active Equations

- (002) Acute attacks = Asympto popn acute attacks + Subacute popn acute attacks + Post-acute recurrent attacks
- (003) Acute attacks per thousand adult popn = Acute attacks / Adult popn \* 1000
- (004) Additional resources = 1e+009 \* Additional resources billions series ( Time )
- (005) Additional resources billions series ( [(0,0)-(50,80)],(0,0),(5,0),(10,0),(20,0),(30,0),(40,0),(50,0) )
- (006) Additional resources for asympto risk mgmt = Additional resources provided by intervention type [RiskMgmt]
- (007) Additional resources for nonsudden death acute attack treatment = Additional resources provided by intervention type [AttackTx]
- (008) Additional resources for post-acute disease mgmt = Additional resources provided by intervention type [PostDisMgmt]
- (009) Additional resources for subacute disease mgmt = Additional resources provided by intervention type [SubDisMgmt]
- (010) Additional resources provided = SUM (Additional resources provided by intervention type [InterventionType!])
- (011) Additional resources provided by intervention type [InterventionType] = ALLOCATE BY PRIORITY (Additional resources requested by intervention type [InterventionType], Priority of intervention by type for additional resources [InterventionType], 5, 10, Additional resources)
- (012) Additional resources provided vs. requested = ZIDZ (Additional resources provided, Additional resources requested)
- (013) Additional resources provided vs. requested by intervention type [InterventionType] = ZIDZ (Additional resources provided by intervention type [InterventionType], Additional resources requested by intervention type [InterventionType])
- (014) Additional resources requested = SUM (Additional resources requested by intervention type [InterventionType!])
- (015) Additional resources requested by intervention type [InterventionType] = SMOOTHI (Additional resources required for max intervention by type [InterventionType], Time to reassess resource needs, 0)
- (016) Additional resources required for max acute attack treatment = MAX (0, Resources required for max treatment of nonsudden death acute attacks - Initial resources for nonsudden death acute attack treatment)

- (017) Additional resources required for max asympto risk mgmt = MAX (0, Resources required for max asympto risk mgmt - Initial resources for asympto risk mgmt)
- (018) Additional resources required for max intervention by type [Screen] = Resources required for max RC2 screenings  
 Additional resources required for max intervention by type [RiskMgmt] = Additional resources required for max asympto risk mgmt  
 Additional resources required for max intervention by type [SubDisMgmt] = Additional resources required for max subacute disease mgmt  
 Additional resources required for max intervention by type [PostDisMgmt] = Additional resources required for max post-acute disease mgmt  
 Additional resources required for max intervention by type [AttackTx] = Additional resources required for max acute attack treatment
- (019) Additional resources required for max post-acute disease mgmt = MAX (0, Resources required for max post-acute disease mgmt - Initial resources for post-acute disease mgmt)
- (020) Additional resources required for max subacute disease mgmt = MAX (0, Resources required for max subacute disease mgmt - Initial resources for subacute disease mgmt)
- (021) Adult deaths = SUM (Adult deaths by RC[RiskClass!])
- (022) Adult deaths by RC[RiskClass] = Adult deaths from acute attack by RC[RiskClass] + Adult nonCVD deaths by RC[RiskClass]
- (023) Adult deaths by RC initial[RiskClass] = INITIAL (Adult deaths by RC[RiskClass])
- (024) Adult deaths from acute attack by RC[RiskClass] = Asympto deaths from acute attack by RC[RiskClass] + Subacute deaths from acute attack by RC[RiskClass] + Post-acute deaths from recurrent attack by RC[RiskClass]
- (025) Adult deaths per thousand adult popn = Adult deaths / Adult popn \* 1000
- (026) Adult nonCVD deaths by RC[RiskClass] = Asympto nonCVD deaths by RC[RiskClass] + Subacute nonCVD deaths by RC[RiskClass] + Post-acute nonCVD deaths by RC[RiskClass]
- (027) Adult popn = SUM (Adult popn by risk class[RiskClass!])
- (028) Adult popn by risk class[RiskClass] = Asymptomatic popn by risk class[RiskClass] + Symptomatic popn by risk class[RiskClass]
- (029) Adult popn by risk class initial[RiskClass] = 1e+006 \* Adult popn millions initial \* Fraction of adult popn by risk class[RiskClass]
- (030) Adult popn inflow by RC[RiskClass] = Adult deaths by RC initial[RiskClass]
- (031) Adult popn millions initial = 214
- (032) Asympto attack deaths as fraction of total = ZIDZ (Asympto deaths from acute attack, Deaths from acute attacks)
- (033) Asympto attack rate if max risk mgmt[RiskClass] = 0.0022, 0.004, 0.0047, 0.0054
- (034) Asympto attack rate if zero Risk Mgmt[RiskClass] = 0.0022, 0.004, 0.0067, 0.0126
- (035) Asympto attacks as fraction of total = ZIDZ (Asympto popn acute attacks, Acute attacks)

- (036) Asympto deaths from acute attack = SUM (Asympto deaths from acute attack by RC[RiskClass!])
- (037) Asympto deaths from acute attack by RC[RiskClass] = Asympto popn acute attacks by RC[RiskClass] \* Fatal fraction of acute attacks
- (038) Asympto nonCVD deaths by RC[RiskClass] = Asymptomatic popn by risk class [RiskClass] \* NonCVD death rate for asympto popn
- (039) Asympto popn acute attack rate [RC1] = Asympto attack rate if zero Risk Mgmt [RC1]  
 Asympto popn acute attack rate [RC2a] = Asympto attack rate if zero Risk Mgmt [RC2a]  
 Asympto popn acute attack rate [RC2b] = Asympto attack rate if zero Risk Mgmt [RC2b] - (Asympto attack rate if zero Risk Mgmt [RC2b] - Asympto attack rate if max risk mgmt [RC2b]) \* Asymptomatic risk mgmt vs. max \* Screened fraction of asympto RC2 popn  
 Asympto popn acute attack rate [RC3] = Asympto attack rate if zero Risk Mgmt [RC3] - (Asympto attack rate if zero Risk Mgmt [RC3] - Asympto attack rate if max risk mgmt [RC3]) \* Asymptomatic risk mgmt vs. max
- (040) Asympto popn acute attack rate initial [RC1] = Asympto attack rate if zero Risk Mgmt [RC1]  
 Asympto popn acute attack rate initial [RC2a] = Asympto attack rate if zero Risk Mgmt [RC2a]  
 Asympto popn acute attack rate initial [RC2b] = Asympto attack rate if zero Risk Mgmt [RC2b]  
 Asympto popn acute attack rate initial [RC3] = Asympto attack rate if zero Risk Mgmt [RC3] - (Asympto attack rate if zero Risk Mgmt [RC3] - Asympto attack rate if max risk mgmt [RC3]) \* Asympto risk mgmt vs. max initial
- (041) Asympto popn acute attacks = SUM (Asympto popn acute attacks by RC[RiskClass!])
- (042) Asympto popn acute attacks by RC[RiskClass] = Asymptomatic popn by risk class[RiskClass] \* Asympto popn acute attack rate[RiskClass]
- (043) Asympto popn symptoms onset rate [RC1] = Asympto symptoms onset rate if zero risk mgmt [RC1]  
 Asympto popn symptoms onset rate [RC2a] = Asympto symptoms onset rate if zero risk mgmt [RC2a]  
 Asympto popn symptoms onset rate [RC2b] = Asympto symptoms onset rate if zero risk mgmt [RC2b] - (Asympto symptoms onset rate if zero risk mgmt [RC2b] - Asympto symptoms onset rate if max risk mgmt [RC2b]) \* Asymptomatic risk mgmt vs. max \* Screened fraction of asympto RC2 popn  
 Asympto popn symptoms onset rate [RC3] = Asympto symptoms onset rate if zero risk mgmt [RC3] - (Asympto symptoms onset rate if zero risk mgmt [RC3] - Asympto symptoms onset rate if max risk mgmt [RC3]) \* Asymptomatic risk mgmt vs. max
- (044) Asympto popn symptoms onset rate initial [RC1] = Asympto symptoms onset rate if zero risk mgmt [RC1]  
 Asympto popn symptoms onset rate initial [RC2a] = Asympto symptoms onset rate if zero risk mgmt [RC2a]  
 Asympto popn symptoms onset rate initial [RC2b] = Asympto symptoms onset rate if zero risk mgmt [RC2b]  
 Asympto popn symptoms onset rate initial [RC3] = Asympto symptoms onset rate if zero risk mgmt [RC3] - (Asympto symptoms onset rate if zero risk mgmt [RC3] - Asympto symptoms onset rate if max risk mgmt [RC3]) \* Asympto risk mgmt vs. max initial
- (045) Asympto risk mgmt vs. max initial = 0.2
- (046) Asympto surviving acute attack by RC[RiskClass] = Asympto popn acute attacks by RC[RiskClass] - Asympto deaths from acute attack by RC[RiskClass]

- (047) Asympto symptoms onset rate if max risk mgmt[RiskClass] = 0.0018, 0.0031, 0.0037, 0.0043
- (048) Asympto symptoms onset rate if zero risk mgmt[RiskClass] = 0.0018, 0.0031, 0.0053, 0.01
- (049) Asymptomatic popn = SUM (Asymptomatic popn by risk class[RiskClass!])
- (050) Asymptomatic popn by risk class[RiskClass] = INTEG (Adult popn inflow by RC[RiskClass] - Onset of subacute symptoms by RC[RiskClass] - Asympto deaths from acute attack by RC[RiskClass] - Asympto surviving acute attack by RC[RiskClass] - Asympto nonCVD deaths by RC[RiskClass] , Adult popn by risk class initial[RiskClass] - Subacute popn by risk class[RiskClass] - Post-acute popn by risk class[RiskClass] )
- (051) Asymptomatic RC2 popn = Asymptomatic popn by risk class [RC2a] + Asymptomatic popn by risk class [RC2b]
- (052) Asymptomatic risk mgmt vs. max = MIN (1, ZIDZ (Resources used for asympto risk mgmt, Resources required for max asympto risk mgmt))
- (053) Combined intervention resources = Resources used for RC2 screening + Resources used for asympto risk mgmt + Resources used for subacute disease mgmt + Resources used for post-acute disease mgmt + Resources used for acute attacks
- (054) Combined resources required for max intervention = Resources required for max RC2 screenings + Resources required for max asympto risk mgmt + Resources required for max subacute disease mgmt + Resources required for max post-acute disease mgmt + Resources required for max treatment of nonsudden death acute attacks
- (055) Deaths from acute attack per thousand adult popn = Deaths from acute attacks / Adult popn \* 1000
- (056) Deaths from acute attacks = Asympto deaths from acute attack + Subacute deaths from acute attack + Post-acute deaths from recurrent attack
- (057) Fatal fraction of acute attacks = Sudden death fraction of attacks \* 1 + (1 - Sudden death fraction of attacks) \* Fatal fraction of nonsudden death acute attacks
- (058) Fatal fraction of acute attacks initial = Sudden death fraction of attacks \* 1 + (1 - Sudden death fraction of attacks) \* Fatal fraction of nonsudden death acute attacks initial
- (059) Fatal fraction of nonsudden death acute attacks = Fatal fraction of nonsudden death attacks if zero treatment - (Fatal fraction of nonsudden death attacks if zero treatment - Fatal fraction of nonsudden death attacks if max treatment) \* Nonsudden death acute attack treatment vs. max
- (060) Fatal fraction of nonsudden death acute attacks initial = Fatal fraction of nonsudden death attacks if zero treatment - (Fatal fraction of nonsudden death attacks if zero treatment - Fatal fraction of nonsudden death attacks if max treatment) \* Nonsudden death acute attack treatment vs. max initial
- (061) Fatal fraction of nonsudden death attacks if max treatment = 0.175
- (062) Fatal fraction of nonsudden death attacks if zero treatment = 0.53
- (063) First time acute attacks = Asympto popn acute attacks + Subacute popn acute attacks
- (064) Fraction of adult popn by risk class[RiskClass] = 0.38, 0.34, 0.19, 0.09

- (065) Fraction of resources used by intervention type [Screen] = Fraction of resources used for RC2 screening  
 Fraction of resources used by intervention type [RiskMgmt] = Fraction of resources used for asymptomatic risk mgmt  
 Fraction of resources used by intervention type [SubDisMgmt] = Fraction of resources used for subacute disease mgmt  
 Fraction of resources used by intervention type [PostDisMgmt] = Fraction of resources used for post-acute disease mgmt  
 Fraction of resources used by intervention type [AttackTx] = Fraction of resources used for acute attack treatment
- (066) Fraction of resources used for acute attack treatment = Resources used for acute attacks / Combined intervention resources
- (067) Fraction of resources used for asymptomatic risk mgmt = Resources used for asymptomatic risk mgmt / Combined intervention resources
- (068) Fraction of resources used for post-acute disease mgmt = Resources used for post-acute disease mgmt / Combined intervention resources
- (069) Fraction of resources used for RC2 screening = Resources used for RC2 screening / Combined intervention resources
- (070) Fraction of resources used for subacute disease mgmt = Resources used for subacute disease mgmt / Combined intervention resources
- (071) Initial resources for asymptomatic risk mgmt = Initial resources required for max asymptomatic risk mgmt \* Asymptomatic risk mgmt vs. max initial
- (072) Initial resources for nonsudden death acute attack treatment = Initial resources required for max treatment of nonsudden death acute attacks \* Nonsudden death acute attack treatment vs. max initial
- (073) Initial resources for post-acute disease mgmt = Initial resources required for max post-acute disease mgmt \* Post-acute disease mgmt vs. max initial
- (074) Initial resources for subacute disease mgmt = Initial resources required for max subacute disease mgmt \* Subacute disease mgmt vs. max initial
- (075) Initial resources required for max asymptomatic risk mgmt = INITIAL (Resources required for max asymptomatic risk mgmt)
- (076) Initial resources required for max post-acute disease mgmt = INITIAL (Resources required for max post-acute disease mgmt)
- (077) Initial resources required for max subacute disease mgmt = INITIAL (Resources required for max subacute disease mgmt)
- (078) Initial resources required for max treatment of nonsudden death acute attacks = INITIAL (Resources required for max treatment of nonsudden death acute attacks)
- (079) Interventions vs. max by type [Screen] = Screened fraction of asymptomatic RC2 popn  
 Interventions vs. max by type [RiskMgmt] = Asymptomatic risk mgmt vs. max  
 Interventions vs. max by type [SubDisMgmt] = Subacute disease mgmt vs. max  
 Interventions vs. max by type [PostDisMgmt] = Post-acute disease mgmt vs. max

- Interventions vs. max by type [AttackTx] = Nonsudden death acute attack treatment vs. max
- (080) Max RC2 first time screenings = (Asymptomatic RC2 popn - Screened asympto RC2 popn) / 1
- (081) NonCVD death rate for asympto popn = 0.0071
- (082) NonCVD death rate for symptomatic popn = 0.022
- (083) Nonsudden death acute attack treatment vs. max = MIN (1, ZIDZ (Resources used for nonsudden death acute attack treatment, Resources required for max treatment of nonsudden death acute attacks))
- (084) Nonsudden death acute attack treatment vs. max initial = 0.7
- (085) Nonsudden death acute attacks = Acute attacks \* (1 - Sudden death fraction of attacks)
- (086) Onset of subacute symptoms = SUM (Onset of subacute symptoms by RC[RiskClass!])
- (087) Onset of subacute symptoms by RC[RiskClass] = Asymptomatic popn by risk class[RiskClass] \* Asympto popn symptoms onset rate[RiskClass]
- (088) Outflow of screened asympto RC2 popn = (Asympto popn acute attacks by RC [RC2a] + Asympto popn acute attacks by RC [RC2b] + Onset of subacute symptoms by RC [RC2a] + Onset of subacute symptoms by RC [RC2b]) \* Screened fraction of asympto RC2 popn + (Screened asympto RC2 popn \* NonCVD death rate for asympto popn)
- (089) Outflow rate of screened asympto RC2 popn = ZIDZ (Outflow of screened asympto RC2 popn, Screened asympto RC2 popn)
- (090) People surviving acute attacks = Acute attacks - Deaths from acute attacks
- (091) Post-acute attack deaths as fraction of total = ZIDZ (Post-acute deaths from recurrent attack, Deaths from acute attacks)
- (092) Post-acute attack rate if max disease mgmt = 0.0387
- (093) Post-acute attack rate if zero disease mgmt = 0.0773
- (094) Post-acute attacks as fraction of total = ZIDZ (Post-acute recurrent attacks, Acute attacks)
- (095) Post-acute deaths from recurrent attack = SUM (Post-acute deaths from recurrent attack by RC[RiskClass!])
- (096) Post-acute deaths from recurrent attack by RC[RiskClass] = Post-acute popn recurrent attacks by RC[RiskClass] \* Fatal fraction of acute attacks
- (097) Post-acute disease mgmt vs. max = MIN (1, ZIDZ (Resources used for post-acute disease mgmt, Resources required for max post-acute disease mgmt))
- (098) Post-acute disease mgmt vs. max initial = 0.5
- (099) Post-acute fraction of symptomatic = Post-acute popn / Symptomatic popn
- (100) Post-acute nonCVD deaths by RC[RiskClass] = Post-acute popn by risk class[RiskClass] \* NonCVD death rate for symptomatic popn

- (101) Post-acute popn = SUM (Post-acute popn by risk class[RiskClass!])
- (102) Post-acute popn acute attack rate = Post-acute attack rate if zero disease mgmt - (Post-acute attack rate if zero disease mgmt - Post-acute attack rate if max disease mgmt) \* Post-acute disease mgmt vs. max
- (103) Post-acute popn acute attack rate initial = Post-acute attack rate if zero disease mgmt - (Post-acute attack rate if zero disease mgmt - Post-acute attack rate if max disease mgmt) \* Post-acute disease mgmt vs. max initial
- (104) Post-acute popn by risk class[RiskClass] = INTEG( Asympto surviving acute attack by RC[RiskClass] + Subacute surviving acute attack by RC[RiskClass] - Post-acute nonCVD deaths by RC[RiskClass] - Post-acute deaths from recurrent attack by RC[RiskClass] , Post-acute popn by risk class initial[RiskClass] )
- (105) Post-acute popn by risk class initial[RiskClass] = Adult popn by risk class initial[RiskClass] \* Post-acute popn fraction by risk class initial[RiskClass]
- (106) Post-acute popn fraction by risk class initial[RiskClass] = ZIDZ ((1 - Subacute fraction of Not Post-acute by risk class initial[RiskClass]) \* Asympto popn acute attack rate initial[RiskClass] \* (1 - Fatal fraction of acute attacks initial) + Subacute fraction of Not Post-acute by risk class initial[RiskClass] \* Subacute popn acute attack rate initial \* (1 - Fatal fraction of acute attacks initial), (1 - Subacute fraction of Not Post-acute by risk class initial[RiskClass]) \* Asympto popn acute attack rate initial[RiskClass] \* (1 - Fatal fraction of acute attacks initial) + Post-acute popn acute attack rate initial \* Fatal fraction of acute attacks initial + NonCVD death rate for symptomatic popn + Subacute fraction of Not Post-acute by risk class initial[RiskClass] \* Subacute popn acute attack rate initial \* (1 - Fatal fraction of acute attacks initial))
- (107) Post-acute popn prevalence = Post-acute popn / Adult popn
- (108) Post-acute popn recurrent attacks by RC[RiskClass] = Post-acute popn by risk class [RiskClass] \* Post-acute popn acute attack rate
- (109) Post-acute popn surviving recurrent attack by RC[RiskClass] = Post-acute popn recurrent attacks by RC[RiskClass] - Post-acute deaths from recurrent attack by RC[RiskClass]
- (110) Post-acute recurrent attacks = SUM (Post-acute popn recurrent attacks by RC[RiskClass!])
- (111) Priority of intervention by type for additional resources [InterventionType] = 8, 8.5, 9, 9.5, 10
- (112) RC2 first time screenings = Max RC2 first time screenings \* RC2 first time screenings vs. max
- (113) RC2 first time screenings vs. max = MIN (1, ZIDZ (Resources used for RC2 first time screenings, Resources required for max RC2 first time screenings))
- (114) RC2 rescreenings = Screened asympto RC2 popn / Screening interval
- (115) Resources required for max asympto risk mgmt = Resources required per patient for max asympto risk mgmt \* (Asymptomatic popn by risk class [RC3] + Asymptomatic popn by risk class [RC2b] \* Screened fraction of asympto RC2 popn)
- (116) Resources required for max post-acute disease mgmt = Post-acute popn \* Resources required per post-acute patient for max disease mgmt
- (117) Resources required for max RC2 first time screenings = Max RC2 first time screenings \* Resources required per RC2 screening

- (118) Resources required for max RC2 screenings = Resources required for max RC2 first time screenings + Resources used for RC2 rescreenings
- (119) Resources required for max subacute disease mgmt = Resources required for max subacute disease mgmt ongoing + Resources required for max subacute disease mgmt from onset
- (120) Resources required for max subacute disease mgmt from onset = Onset of subacute symptoms \* Resources required per subacute symptoms onset for max disease mgmt
- (121) Resources required for max subacute disease mgmt ongoing = Subacute popn \* Resources required per subacute patient for max disease mgmt
- (122) Resources required for max treatment of nonsudden death acute attacks = Nonsudden death acute attacks \* Resources required per nonsudden death acute attack for max treatment
- (123) Resources required per nonsudden death acute attack for max treatment = 32000
- (124) Resources required per patient for max asympto risk mgmt = 2690
- (125) Resources required per post-acute patient for max disease mgmt = 4121
- (126) Resources required per RC2 screening = 350
- (127) Resources required per subacute patient for max disease mgmt = 3914
- (128) Resources required per subacute symptoms onset for max disease mgmt = 2992
- (129) Resources used for acute attacks = Resources used for nonsudden death acute attack treatment + Resources used for sudden death attacks
- (130) Resources used for asympto risk mgmt = Additional resources for asympto risk mgmt + Initial resources for asympto risk mgmt
- (131) Resources used for nonsudden death acute attack treatment = Additional resources for nonsudden death acute attack treatment + Initial resources for nonsudden death acute attack treatment
- (132) Resources used for post-acute disease mgmt = Additional resources for post-acute disease mgmt + Initial resources for post-acute disease mgmt
- (133) Resources used for RC2 first time screenings = Resources used for RC2 screening - Resources used for RC2 rescreenings
- (134) Resources used for RC2 rescreenings = RC2 rescreenings \* Resources required per RC2 screening
- (135) Resources used for RC2 screening = Additional resources provided by intervention type [Screen]
- (136) Resources used for subacute disease mgmt = Additional resources for subacute disease mgmt + Initial resources for subacute disease mgmt
- (137) Resources used for sudden death attacks = Sudden death acute attacks \* Resources used per sudden death attack
- (138) Resources used per sudden death attack = 1000

- (139) Screened asymptomatic RC2 population = INTEG (RC2 first time screenings - Outflow of screened asymptomatic RC2 population, 0)
- (140) Screened fraction of asymptomatic RC2 population = ZIDZ (Screened asymptomatic RC2 population, Asymptomatic RC2 population)
- (141) Screening interval = 5
- (142) Subacute attack deaths as fraction of total = ZIDZ (Subacute deaths from acute attack, Deaths from acute attacks)
- (143) Subacute attack rate if maximum disease management = 0.0216
- (144) Subacute attack rate if zero disease management = 0.0431
- (145) Subacute attacks as fraction of total = ZIDZ (Subacute population acute attacks, Acute attacks)
- (146) Subacute deaths from acute attack = SUM (Subacute deaths from acute attack by RC[RiskClass!])
- (147) Subacute deaths from acute attack by RC[RiskClass] = Subacute population acute attacks by RC[RiskClass] \* Fatal fraction of acute attacks
- (148) Subacute disease management vs. maximum = MIN (1, ZIDZ (Resources used for subacute disease management, Resources required for maximum subacute disease management))
- (149) Subacute disease management vs. maximum initial = 0.33
- (150) Subacute fraction of Not Post-acute by risk class initial[RiskClass] = ZIDZ (Asymptomatic population symptoms onset rate initial[RiskClass], Asymptomatic population symptoms onset rate initial[RiskClass] + Subacute population acute attack rate initial + NonCVD death rate for symptomatic population)
- (151) Subacute nonCVD deaths by RC[RiskClass] = Subacute population by risk class[RiskClass] \* NonCVD death rate for symptomatic population
- (152) Subacute population = SUM (Subacute population by risk class[RiskClass!])
- (153) Subacute population acute attack rate = Subacute attack rate if zero disease management - (Subacute attack rate if zero disease management - Subacute attack rate if maximum disease management) \* Subacute disease management vs. maximum
- (154) Subacute population acute attack rate initial = Subacute attack rate if zero disease management - (Subacute attack rate if zero disease management - Subacute attack rate if maximum disease management) \* Subacute disease management vs. maximum initial
- (155) Subacute population acute attacks = SUM (Subacute population acute attacks by RC[RiskClass!])
- (156) Subacute population acute attacks by RC[RiskClass] = Subacute population by risk class[RiskClass] \* Subacute population acute attack rate
- (157) Subacute population by risk class[RiskClass] = INTEG (Onset of subacute symptoms by RC[RiskClass] - Subacute deaths from acute attack by RC[RiskClass] - Subacute nonCVD deaths by RC[RiskClass] - Subacute surviving acute attack by RC[RiskClass], Subacute population by risk class initial[RiskClass])

- (158) Subacute popn by risk class initial[RiskClass] = (Adult popn by risk class initial[RiskClass] - Post-acute popn by risk class initial[RiskClass]) \* Subacute fraction of Not Post-acute by risk class initial[RiskClass]
- (159) Subacute popn prevalence = Subacute popn / Adult popn
- (160) Subacute surviving acute attack by RC[RiskClass] = Subacute popn acute attacks by RC[RiskClass] - Subacute deaths from acute attack by RC[RiskClass]
- (161) Sudden death acute attacks = Acute attacks \* Sudden death fraction of attacks
- (162) Sudden death fraction of attacks = 0.26
- (163) Symptomatic popn = SUM (Symptomatic popn by risk class[RiskClass!])
- (164) Symptomatic popn by risk class[RiskClass] = Subacute popn by risk class[RiskClass] + Post-acute popn by risk class[RiskClass]
- (165) Symptomatic popn prevalence = Symptomatic popn / Adult popn
- (166) Time to reassess resource needs = 2

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.Array

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Subscribed Arrays

(168) InterventionType: Screen,RiskMgmt,SubDisMgmt,PostDisMgmt,AttackTx

(169) RiskClass: RC1,RC2a,RC2b,RC3

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.Control

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Simulation Control Parameters

(171) FINAL TIME = 50

(172) INITIAL TIME = 0

(173) SAVEPER = TIME STEP

(174) TIME STEP = 0.25



## **Appendix D: Scope Testing Framework (PROMULA Version)**

\*Index Definitions:

### **Define SET**

Entity(1E5) 'Index for agents (1 to N)'

\* If have 10,000 entities active to represent US, this is an aggregation of 30,000 individuals  
\* for each represented entity]

Interval(70) 'Time from 1965-12035 ... by Year for now'

Diagnosis(2) '1:cvd.... n:misc. (Only two to start)'

Condition(3) '1: asyptomatic, 2:sub-acute, 3:post-acute'

Status(12) 'Status of agent'

\*s: Age = age (-99 = non-existent/dead)

\*s: Location= Location (NM, US as keys)

\*s: Locale = Rural/Urban

\*s: Ethnicity (Will be Caucasian, Hispanic, Black, Asian, or some variant in the future)

\*s: Wealth = income in \$/year for now

\*s: Education

\*s: Household Position

\*s: Adult/Child

\*s: Weight

\*s: Employer

\*s: Occupation

\*s:## ... any other demographic descriptor

\*s:CVD = CVD status (0=No CVD, 1=subacute, 2=post-acute)

\*s:## ...any other medical condition

\*s:Misc = Other disease (0=None, 1=subacute, 2=post-acute)

Characteristic(1) 'Characteristics of institutions (e.g. insurance companies, hospital)'

Institution(1) 'Index for institutions'

Region(53) 'Regions'

Gender(2) 'Gender'

Ethnicity(1) 'Ethnic background'

Wealth(5) 'Wealth Quintile'

InsOpt(6) Insurance Options

Regime(2) 'Medical Procedure or Drugs'

### **End Define Set**

\*

\* Model Variables/Parameters

### **Variable Definitions:**

QAge 'Age Index', Value=1

QLocation 'Location Index', Value=2

QEthnicity 'Ethnicity Index', Value=3

QWealth 'Wealth Index', Value=4

QEducation 'Education Index', Value=5

QHousehold 'If part of Household Index', Value=6

QStage 'Adult/Child status index', Value=7

QWeight 'Weight Index', Value=8

QEmployer 'Employer Index', Value=9

QOccupation 'Occupation Index', Value=10

\* Next two 'Values' need to change if Disease or Status set changes.

QCVD 'CVD Index', Value=11

QMisc 'Index of last catch-all disease', Value=12

\*

AF 'annualization factor (.1 for now)'

BirthRate(Age, Location, Ethnicity, Wealth, Interval) 'Exogenous Birthrate'

CAT 'Change Averaging Time (Years)', Value=3  
 CC(Condition, Diagnosis, Entity) 'cost coverage by insurance, state, or Medicaid'  
 CIE Change Impact Exponent  
 CIE Change Impact Exponent  
 COM(Condition, Disease, Entity, Interval) 'Choice Impact Occurrence Multiplier'  
 COM(Condition, Disease, Entity, Interval) 'Choice Impact Occurrence Multiplier'  
 Copay(InsOpt)  
 CostCap(InsOpt)  
 DDCM(Condition, Disease, Entity, Interval) 'Drug Death Change Multiplier'  
 DDE(Condition, Disease) 'Drug Death Exponent'  
 DDM(Condition, Disease, Entity, Interval) 'Drug Impact Death Multiplier'  
 DDR(Condition, Disease, Interval) 'Drug Resources (\$)  
 DDRN(Condition, Disease) 'Normal Drug Resources (\$)  
 DOCM(Condition, Disease, Entity, Interval) 'Drug Occurrence Change Multiplier'  
 DOE(Condition, Disease) 'Drug Occurrence Exponent'  
 DOffset 'Status offset for Disease sub-vector', Value=10  
 DOM(Condition, Disease, Entity, Interval) 'Drug Impact Occurrence Multiplier'  
 DOR(Condition, Disease, Interval) 'Drug Resources (\$)  
 DORN(Condition, Disease) 'Normal Drug Resources (\$)  
 ECMC(Wealth) Expected Cost of Medical Care (\$)  
 ECPD(Wealth) Expected Cost of Prescription Drugs (\$)  
 EDDM(Condition, Disease, Entity, Interval) 'Expected Impact of Drugs'  
 EDOM(Condition, Disease, Entity, Interval) 'Expected Impact of Drugs'  
 EF(Condition, Diagnosis, Entity) 'Expense Fraction (max fraction of income allocated to medical costs)'  
 EIDM(Condition, Disease, Entity, Interval) 'Expected Impact of Intensified IT'  
 EIOM(Condition, Disease, Entity, Interval) 'Expected Impact of Intensified IT'  
 EOMC(Wealth) Expected Occurrences of Medical Care (number)  
 EOPD(Wealth) Expected Occurrences of Prescription Drugs (number)  
 EQDM(Condition, Disease, Entity, Interval) 'Expected Impact of Standards of Practice'  
 EQOM(Condition, Disease, Entity, Interval) 'Expected Impact of Standards of Practice'  
 ESDM(Condition, Disease, Entity, Interval) 'Expected Impact of Intensified Standard Practices'  
 ESOM(Condition, Disease, Entity, Interval) 'Expected Impact of Intensified Standard Practices'  
 ETDM(Condition, Disease, Entity, Interval) 'Expected Impact of Technology'  
 ETOM(Condition, Disease, Entity, Interval) 'Expected Impact of Technology'  
 F(Characteristic, Institution, Interval) 'Institutions interacting with population'  
 FDrugCopay(InsOpt) Drug Copay as Fixed Cost  
 FMedCopay(InsOpt) Medical Procedure Copay as Fixed Cost  
 I(status, Entity, Interval) 'Individuals/Entities within population'  
 IDCM(Condition, Disease, Entity, Interval) 'Intensified IT Death Change Multiplier'  
 IDDM(Condition, Disease, Entity, Interval) 'Indicated Impact of Drugs'  
 IDE(Condition, Disease) 'IT Death Exponent'  
 IDM(Condition, Disease, Entity, Interval) 'Intensified IT Impact Death Multiplier'  
 IDOM(Condition, Disease, Entity, Interval) 'Indicated Impact of Drugs'  
 IDR(Condition, Disease, Interval) 'Intensified IT Resources (\$)  
 IDRN(Condition, Disease) 'Normal IT Resources (\$)  
 IFInc "Acceptable Fraction of Income for Insurance"  
 IIDM(Condition, Disease, Entity, Interval) 'Indicated Impact of Intensified IT'  
 IIOM(Condition, Disease, Entity, Interval) 'Indicated Impact of Intensified IT'  
 InsVF Insurance Variance Factor  
 IOCM(Condition, Disease, Entity, Interval) 'Intensified IT Occurrence Change Multiplier'  
 IOE(Condition, Disease) 'IT Occurrence Exponent'  
 IOM(Condition, Disease, Entity, Interval) 'Intensified IT Impact Occurrence Multiplier'  
 IOR(Condition, Disease, Interval) 'Intensified IT Resources (\$)  
 IORN(Condition, Disease) 'Normal IT Resources (\$)  
 IQDM(Condition, Disease, Entity, Interval) 'Indicated Impact of Standards of Practice'

IQOM(Condition, Disease, Entity, Interval) 'Indicated Impact of Standards of Practice'  
 IRVF "Information Receptivity Variance Factor"  
 ISDM(Condition, Disease, Entity, Interval) 'Indicated Impact of Intensified Standard Practices'  
 ISOM(Condition, Disease, Entity, Interval) 'Indicated Impact of Intensified Standard Practices'  
 ITDM(Condition, Disease, Entity, Interval) 'Indicated Impact of Technology'  
 ITOM(Condition, Disease, Entity, Interval) 'Indicated Impact of Technology'  
 IVF 'Information Variance Factor', Value=-5  
 LCondition 'Local Value of Condition Index'  
 LDisease "Local Value of Disease Index"  
 MaxFertilityAge 'Age of Fertility End'  
 MI(Condition, Diagnosis, Entity) 'Medical Information index (knowledge of medical trade-offs)'  
 MinFertilityAge 'Age of Fertility Onset'  
 NActive 'Number of Active Entities'  
 NonExist "Entity is not Alive", Value=-99  
 NPrevious 'Previous interval number of Active Entities'  
 OC(Condition, Disease, Entity) Occurrence of Condition  
 OC(Condition, Disease, Entity, Interval) 'Occurrence of Condition'  
 OD(Condition, Disease, Entity) Occurrence of Death  
 PCost(InsOpt) Insurance Premiums (\$/Year)  
 PCostMin(InsOpt) 'Minimum Premium Cost'  
 PD(Condition, Disease, Entity, Interval) 'Probability of Death'  
 PDM(Condition, Disease, Entity, Interval) 'Probability of Death Multiplier'  
 PDN(Condition, Disease, Entity, Interval) 'Natural Probability of Death'  
 PDrugCopay(InsOpt) Drug Copay as Percent  
 PMedCopay(InsOpt) Medical Procedure Copay as Percent  
 PO(Condition, Disease, Entity, Interval) 'Probability of Occurrence'  
 POM(Condition, Disease, Entity, Interval) 'Probability of Occurrence Multiplier'  
 PON(Condition, Disease, Entity, Interval) 'Natural Probability of Occurrence'  
 POP(Age,Location,Ethnicity,Gender,Wealth,Interval) 'Population Statistics'  
 Problns(Entity) Probabililty of getting Insurance  
 QDCM(Condition, Disease, Entity, Interval) 'Standards of Practice Death Change Multiplier'  
 QDE(Condition, Disease) 'Standards of Practice Death Exponent'  
 QDM(Condition, Disease, Entity, Interval) 'Standards of Practice Impact Death Multiplier'  
 QDR(Condition, Disease, Interval) 'Standards of Practice Resources (\$)'  
 QDRN(Condition, Disease) 'Normal Standards of Practice Resources (\$)'  
 QOCM(Condition, Disease, Entity, Interval) 'Standards of Practice Occurrence Change Multiplier'  
 QOE(Condition, Disease) 'Standards of Practice Occurrence Exponent'  
 QOM(Condition, Disease, Entity, Interval) 'Standards of Practice Impact Occurrence Multiplier'  
 QOR(Condition, Disease, Interval) 'Standards of Practice Resources (\$)'  
 QORN(Condition, Disease) 'Normal Standards of Practice Resources (\$)'  
 RIncome "Reference Income"  
 RIT 'Resource Implementation Time (Years)', Value=3  
 SDCM(Condition, Disease, Entity, Interval) 'Intensified Standard Practice Death Change Multiplier'  
 SDE(Condition, Disease) 'Standard Practice Death Exponent'  
 SDM(Condition, Disease, Entity, Interval) 'Intensified Standard Practice Impact Death Multiplier'  
 SDR(Condition, Disease, Interval) 'Intensified Standard Practice Resources (\$)'  
 SDRN(Condition, Disease) 'Normal Standard Practice Resources (\$)'  
 SF 'Scaling factor'  
 SOCM(Condition, Disease, Entity, Interval) 'Intensified Standard Practice Occurrence Change Multiplier'  
 SOE(Condition, Disease) 'Standard Practice Occurrence Exponent'  
 SOM(Condition, Disease, Entity, Interval) 'Intensified Standard Practice Impact Occurrence Multiplier'  
 SOR(Condition, Disease, Interval) 'Intensified Standard Practice Resources (\$)'  
 SORN(Condition, Disease) 'Normal Standard Practice Resources (\$)'

TDCM(Condition, Disease, Entity, Interval) 'Technology Death Change Multiplier'  
TDE(Condition, Disease) 'Technology Death Exponent'  
TDM(Condition, Disease, Entity, Interval) 'Technology Impact Death Multiplier'  
TDR(Condition, Disease, Interval) 'Technology Resources (\$)'  
TDRN(Condition, Disease) 'Normal Technology Resources (\$)'  
TOCM(Condition, Disease, Entity, Interval) 'Technology Occurrence Change Multiplier'  
TOE(Condition, Disease) 'Technology Occurrence Exponent'  
TOM(Condition, Disease, Entity, Interval) 'Technology Impact Occurrence Multiplier'  
TOR(Condition, Disease, Interval) 'Technology Resources (\$)'  
TORN(Condition, Disease) 'Normal Technology Resources (\$)'  
UCost(Condition, Diagnosis, Entity) 'Expected Unit cost of care for condition'  
XPOP(Age,Location,Ethnicity,Gender,Wealth,Interval) 'Historical Population'

**End Define Variable**

\*

\* Variable Keys

**Define Variable**

StrRegion 'State Descriptor', Type=String(21)

KeyRegion 'State Key', Type=String(3)

KeyGender 'Gender Key', Type=String(6)

KeyEthnicity 'Ethnicity Key' Type=String(6)

**End Define Variable**

\*

**Define Relation**

Key(Region,KeyRegion)

Row(Region,StrRegion)

Key(Gender,KeyGender)

Key(Ethnicity,KeyEthnicity)

**End Define Relation**

\*

**\* Read data for keys and descriptor**

Read KeyDiagnosis

CVD

MISC

Read KeyRegime

Medical

Drug

Read KeyCondition

Asymptomatic

SubAcute

Post-acute

Read KeyGender

FEMALE

MALE

Read KeyEthnicity

HUMAN

Read StrRegion, KeyRegion

/State, Abbreviation

ALABAMA, AL

ALASKA, AK

ARIZONA , AZ

ARKANSAS, AR

CALIFORNIA , CA

COLORADO , CO

CONNECTICUT, CT

DELAWARE, DE

DISTRICT OF COLUMBIA, DC

FLORIDA, FL

GEORGIA, GA

HAWAII, HI

IDAHO, ID

ILLINOIS, IL

INDIANA, IN

IOWA, IA

KANSAS, KS

KENTUCKY, KY

LOUISIANA, LA

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WASHINGTON, WA  
WEST VIRGINIA, WV  
WISCONSIN, WI  
WYOMING, WY  
Rest-Of-World, ROW

**\*Equations:**

\*[Automatic looping around all non-fixed indices unless constrained by DO loop, interval SELECTION]

**\*Initialization:**

\*

\*\*\*\*\*

Define Procedure Initialize

\*\*\*\*\*

Select Interval(1),Entity\*

NActive=10000

SF=sum(a,l,e,g,w)(XPOP(a,l,e,g,w,t))/NActive

I(Age,n,t)=NonExist

Select Status(CVD-MISC)

I(Status,N,t)=ASYMPTOMATIC

Select Status\*

End Procedure Initialize

```

*
* Determine Probability Adjustment to Occurrence
*
*****
Define Procedure Occurrence
*****
*
Define Parameter
E(Condition, Disease) 'Exponent'
IM(Condition, Disease) 'Indicated Multiplier'
R(Condition, Disease) 'Resource'
RN(Condition, Disease) 'Normal Resource'
EM(Condition, Disease) 'Expected Multiplier'
CM(Condition, Disease) 'Change Multiplier'
M(Condition, Disease) 'Multiplier'
End Define Parameter
*
* Indicated Multiplier
  IM(c,d)=exp(-E(c,d)*(R(c,d)/RN(c,d)-1))
* Expected Impact after ramp up time
  EM(c,d)=Delay3(IM(c,d), RIT)
* Startup Problems from Change
  CM(c,d)=(Smooth(EM(c,d),CAT)/IM(c,d))*CIE
* Net Impact
  M(c,d)= EM(c,d)*CM(c,d)
End Procedure Occurrence

*
* Impact of Individual choice of cooperation with, or participation, in healthcare
*
*****
Define Procedure Participate
*****
*
Define Parameter
CM(Condition, Disease, Entity) 'Choice Multiplier'
PCM(Condition, Disease, Entity) 'Probability of Choice Multiplier'
UnitCost((Condition, Disease) 'Unit Cost'
CostCov((Condition, Disease, Entity) 'Cost Coverage'
CF(Condition, Diagnosis, Entity) 'Cost Fraction'
IPI(condition, Disease,Entity) Information Program Impact
IP(Condition, Disease) Information Program
End Define Parameter
*
* Receptivity of Information
  IPI(c,d,n)=IP(c,d)/(1+(I(Income,n,t)/RIncome)**IRVF)
* Acceptable Economic Fraction of $ to Healthcare
  EF(c,d,n)=1/(1+IPI(c,d,n)**IVF)
* Probability of making Choosing to participate.
  PCM(c,d,n)=1/(1+(UnitCost(c,d)*(1-CostCov(c,d,n))/(I(QIncome,n,t)*(1-EF(c,d,n))))**IVF)
* Choice to participate (Want 0.0 value if participate)
  CM(c,d,n)=(PCM(c,d,n) LT Random)
End Procedure Participate

```

```

*
* Population Growth
*
*****
Define Procedure PopBirths
*****
*
* Will worry about families (single, married divorced, children) later.
* New Population
SELECT Entity IF (I(g,entity,t) EQ FEMALE) AND
                (I(Age,n,t) GE MinFertilityAge) AND I(Age,n,t) LE MaxFertilityAge)
DO N
* Use Individuals array as index
SELECT
AGE(I(QAge,n,t)),Ethnicity(I(QEthnicity,n,t)),Location(I(QLocation,t)),Wealth(I(QWealth,n,t))
  Do If Random LE BirthRate(a,l,e,w,t)
* Add new 'baby' agent to population
  NActive+1
* Inherits mother's characteristics for now (Including educational status of home).
  I(c,NActive,t)=I(c,n,t)
* Initialize as healthy but update within year
  I(d+dOffset)=c
  I(AGE,NActive,t)=0
* Leave as sample to consider future 'population planning' efforts or other countries.
  Do If Random LE MaleFraction
  I(Gender,N,t)=MALE
  ELSE
  I(Gender,N,t)=FEMALE
  END DO IF MaleFraction
END DO IF BirthRate
END DO N
* Agent age over time
SELECT N(1-NActive)
* Eventually add family and household change here.
I(c,n,t)=I(c,n,t-1)
I(Age,n,t)=I(Age,n,t-1)+1
*
* Migration ( Neglect for now). Will be function of income and possibly health care services
* (as they affect cost to maintain perceived quality of life).
END Procedure PopBirths

```

```

*
* Health Condition
*
*****
Define Procedure PopCondition
*****
*
*   Delivery Policies
*   (Need to resource logic to only include population affected)
*   (Assumes Staff are available after delay. May need to model train/hire/fire dynamics later.)
*   Impact of Intensified Standard Practice
*   Occurrence(ISOM(c,d),SOR(c,d),SORN(c,d),ESOM(c,d),SOCM(c,d),SOM(c,d))
*   Impact of Technology
*   Occurrence(ITOM(c,d),TOR(c,d),TORN(c,d),ETOM(c,d),TOCM(c,d),TOM(c,d))
*   Impact of Information Technology
*   Occurrence(IOIM(c,d),IOR(c,d),IORN(c,d),EIOM(c,d),IOCM(c,d),IOM(c,d))
*   Impact of Standards of Practice (or Information Technology)
*   Occurrence(IQOM(c,d),QOR(c,d),QORN(c,d),EQOM(c,d),QOCM(c,d),QOM(c,d))
*   Impact of Drug Regimen
*   Occurrence(IDOM(c,d),DOR(c,d),DORN(c,d),EDOM(c,d),DOCM(c,d),DOM(c,d))
*
SELECT ENTITY*
SELECT ENTITY IF I(Age,N,Current) NE NonExist
DO Entity
*   Impact of Individual choice of cooperation with, or participation, in healthcare
*   Participate(EF(c,d,n),PCOM(c,d,n),UCost(c,d),CC(c,d,n),EF(c,d,n),COM(c,d,n))
*   Occurrence of Medical Condition
*   Prob. Of Occurrence Multiplier
*    $POM(c,d,n) = \text{MAX}(SOM(c,d,n)*TOM(c,d,n)*DOM(c,d,n)*QOM(c,d,n)*IOM(c,d,n), COM(c,d,n))$ 
*   Probability if Occurrence
*    $PO(c,d,n) = \text{PON}(c,d,I(Age,n,t))*POM(c,d,n)*(I(d+dOffset,n) \text{ EQ } c:s)$ 
*   Update Entity Status
*   Can have acute multiple times. All are conditional on sequencing,
  DO Disease
    LCondition=Condition:M-1
    LDisease=Disease:s
    SELECT Condition(LCondition-1)
  DO Condition
*
    Occurrence of Condition
     $OC(c,d,n) = (PO(c,d,n) \text{ GE } \text{Random}) * (I(LDisease+dOffset,n) \text{ EQ } \text{Condition:s})$ 
  DO IF OC(c,d,n)
    I(LDisease+dOffset,n)= Condition:s
  End DO IF
  END DO Disease
  END Do Condition
END DO Entity
*
End Procedure PopCondition

```

```

*
* Death Rate
*
*****
Define Procedure PopDeath
*****
*
*   Delivery Policies
*   (Need to resource logic to only include population affected)
*   (Assumes Staff are available after delay. May need to model train/hire/fire
*   dynamics later.)
*   Impact of Intensified Standard Practice
*   Occurrence(ISDM(c,d),SDR(c,d),SDRN(c,d),ESDM(c,d),SDCM(c,d),SDM(c,d))
*   Impact of Technology
*   Occurrence(ITDM(c,d),TDR(c,d),TDRN(c,d),ETDM(c,d),TDCM(c,d),TDM(c,d))
*   Impact of Information Technology
*   Occurrence(IDIM(c,d),IDR(c,d),IDRN(c,d),EIDM(c,d),IDCM(c,d),IDM(c,d))
*   Impact of Standards of Practice (or Information Technology)
*   Occurrence(IQDM(c,d),QDR(c,d),QDRN(c,d),EQDM(c,d),QDCM(c,d),QDM(c,d))
*   Impact of Drug Regimen
*   Occurrence(IDDM(c,d),DDR(c,d),DDRN(c,d),EDDM(c,d),DDCM(c,d),DDM(c,d))
*
SELECT ENTITY*
SELECT ENTITY IF I(Age,N,Current) NE NonExist
DO Entity
* Occurrence of Death
* Prob. Of Death Multiplier
  PDM(c,d,n)=SDM(c,d,n)*TDM(c,d,n)*DDM(c,d,n)*QDM(c,d,n)*IDM(c,d,n)
* Probability of Death
  PD(c,d,n)=PDN(c,d,I(Age,n,t))*PDM(c,d,n) *(I(d+dOffset,n) EQ c:s)
* Occurrence of Death
  OD(c,n)=(SUM(d)(PD(c,d,n)) GE Random)
* Agent dies by age going to -99
  I(Age,n,t)=I(Age,n,t)*(1-SUM(c)OD(c,n)*100)
* Determine recorded cause of death by looping PD
END DO Entity
End Procedure PopDeath

```

```

*
* Insurance Decision
*
*****
Procedure PopInsure
*****
*
* Is the Individual (Family) Insured?
PCostMin=MIN(InsOpt)(PCost(InsOpt))
Problns(n)=1/(1+(PCostMin/(IFInc*I(Income,n,t)))^InsVF)
Insured(n)=(Problns(n) GE Random)
* What Insurance Option will they select
* ECMC,ECPD,EOMC,EOPD are an exo.function of wealth (The more the more)
Copay(InsOpt,n)= Min(PDrugCopay(InsOpt)*ECPD(I(wealth,n,t) +
                    FDrugCopay(InsOpt))*EOPD(I(wealth,n,t)+
                    FMedCopay(InsOpt)*ECMC(I(wealth,n,t)+
                    FMedCopay(InsOpt) *EOMC(I(wealth,n,t),
                    CoastCap(InsOpt))+Pcost(InsOpt)
MOptUtiliy(InsOpt,n)=InsPref(InsOpt)*Copay(InsOpt,n)^InsVf
* XinsOpt is a dummy summing variable
PInsOpt(InsOpt,n)=MOptUtility(InsOpt)/SUM(XInsOpt,n)( MOptUtiliy(InsOpt,n))
* “:M” syntax designate maximum value of index
SELECT InsOpt(2-InsOpt:M)
PInsOpt(InsOpt)= PInsOpt(InsOpt)+ PInsOpt(InsOpt-1)
*Find the Choice
HoldChoice=Random
Select InsOpt if HoldChoice GE PInsOpt(InsOpt)
Select InsOpt if HoldChoice LT PInsOpt(InsOpt+1)
* Was there a choice
InsChoice(n)=Problns(n) GT Random
* What will the choice mean to health cost coverage.
CC(n)= Min(PDrugCopay(InsOpt)*ECPD(I(wealth,n,t) +
            FDrugCopay(InsOpt))*EOPD(I(wealth,n,t)+
            FMedCopay(InsOpt)*ECMC(I(wealth,n,t)+
            FMedCopay(InsOpt) *EOMC(I(wealth,n,t),
            CoastCap(InsOpt))+Pcost(InsOpt)/( ECMC(I(wealth,n,t)+ ECPD(I(wealth,n,t)))*
            InsChoice(n)

* What was the Choice?
InsChoice(n)=InsChoice(n)*InsOpt:N
End Procedure PopInsure

```

```

*
*****
Define Procedure MedAllocate.
*****
*
*Placeholder for now
*
*Determine and Allocate resources. Limit usage from staff constraints.
*Prioritize by condition but allow over-ride.
*
*Need Costs (Call CostOccurrence, CostCondition, CostPrevention)
*Need state, Fed, Personal, and Insurance funding
*
End Procedure MedAllocate

```

```

*
*****
Procedure CostOccurrence
*****
*
*Placeholder for now
*
* Determine Cost of Occurrence
* (Need to add inflation issues)
*
*Patient Out-of-Pocket Cost Paid
*Insurance Costs Paid
*Medicaid Costs Paid
*
*Costs=f(Standard Practice, Standards of Practice, IT, Drugs, Technology, Insurance)
*
*TOA: Technology Assets for Occurrences (TOA)
*TAL: Technology Asset Life TAL
*TCost: Technology Annual Cost
*
*TOA(c,d,I,Current)=TOA(c,d,I,Prior)+dt*(TOR(c,d,I,Current)-TOA(c,d,I,Prior)/TAL)
*TCost=TOA*AF
*
End Procedure CostOccurrence

```

\*  
\*\*\*\*\*  
Procedure CostCondition  
\*\*\*\*\*  
\*  
\* Placeholder for now  
\*  
\* Determine Cost of Condition (Maintaining status)  
\* (Need to add inflation issues)  
\*Patient Out-of-Pocket Cost Paid  
\*Insurance Costs Paid  
\*Medicaid Costs Paid  
\*  
\*Costs=f(Standard Practice, Standards of Practice, IT, Drugs, Technology, Insurance)  
\*  
Procedure CostCondition

\*  
\*\*\*\*\*  
Procedure CostPrevention  
\*\*\*\*\*  
\*  
\* Placeholder for now  
\*  
\* Determine Cost of Prevention  
\* (Need to add inflation issues)  
\*  
\*Patient Out-of-Pocket Cost Paid  
\*Insurance Costs Paid  
\*Medicaid Costs Paid  
\*  
\*Costs=f(Standard Practice, Standards of Practice, IT, Drugs, Technology, Insurance)  
\*  
End Procedure CostPrevention

```

*
*Example of Post Processing
*
*****
Define Procedure Post
*****
* This can be post processed
* Summary Population Statistics (POP)
Do Age
  Do Location
    Do Ethnicity
      Do Gender
        QMaxWealth=0
*      Put Wealth/Income into quintiles by $ not population.
          Do Wealth
            MaxWealth =MAX(N)(I(income,N,t)
              QMinWealth=QMaxWealth
              QMaxwealth=QWealth+MaxWealth/5
              Select N(1-NActive)
              Select N If I(Age,N,t)=VAge(Age) And
                I(Gender,N,t)=VGender(Gender) And
                I(Location:s,N,t)=VLocation(Location)) And
                I(Ethnicity,n,t)=VEthnicity(Ethnicity) And
                I(Wealth,N,t) GE QMinWealth) And I(Wealth,N,t) LT
            QMaxWealth)
*          The ":N" syntax tell the number of indices active (in
          PROMULA)
            POP(age,location,ethnicity,gender,wealth,Current)=Entity:N
            Select N(1-NActive)
          End Do Wealth
        End do Gender
      End Do Ethnicity
    End Do Location
  End Do Age
End Procedure Post

```

```

*Actual Model
*
*****
Define Procedure Model
*****
*
Initialize
Do Interval
  Current=Interval:s
  Previous=MAX(Interval:s-1,1)
  Next=MIN(interval:s+1,MaxInterval)
  PopBirths
  PopInsure
  PopCondition
  MedAllocate
  PopDeath
*   Below are just place holders for now
*   HealthProvider
*   HealthInsuranceCompany
*   Govt_State
*   Govt_Fed
*   Govt_Local
*   PopEmployer
*   PopEmployment
*   PopIncome
End Do Interval
End Do Model

```

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