Type 2 Diabetes: A Controllable Epidemic

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1 INTRODUCTION

Type 2 Diabetes has become a pandemic in all societies. It leads to kidney failure, blindness, nerve damage, vascular damage, heart attacks, and a vast panoply of other tertiary diseases. It has started to creep down in age in incidence. It consumes almost 10-12% of the total health care costs in the United States alone. It also is an almost totally preventable disease. It is in almost all cases a disease of life style. It presents a case study for handling the delivery of health care in the United States. For we argue herein that this disease, which in many ways is akin to the disorders related to smoking, is a totally preventable disorder. It is a disease which can be modulated and reduced by economic means.

Type 2 Diabetes is a disease of insulin production[1]. The pancreas no longer produces adequate insulin to transport the glucose produced by sugar and carbohydrate consumption and the net result is an excessive rise of glucose in the blood stream. In simple terms this excess glucose catalyzes many reactions which in turn cause the circulatory, renal, optic, and neurological damage. The excess of glucose in simple terms wears down these systems in rapid order.

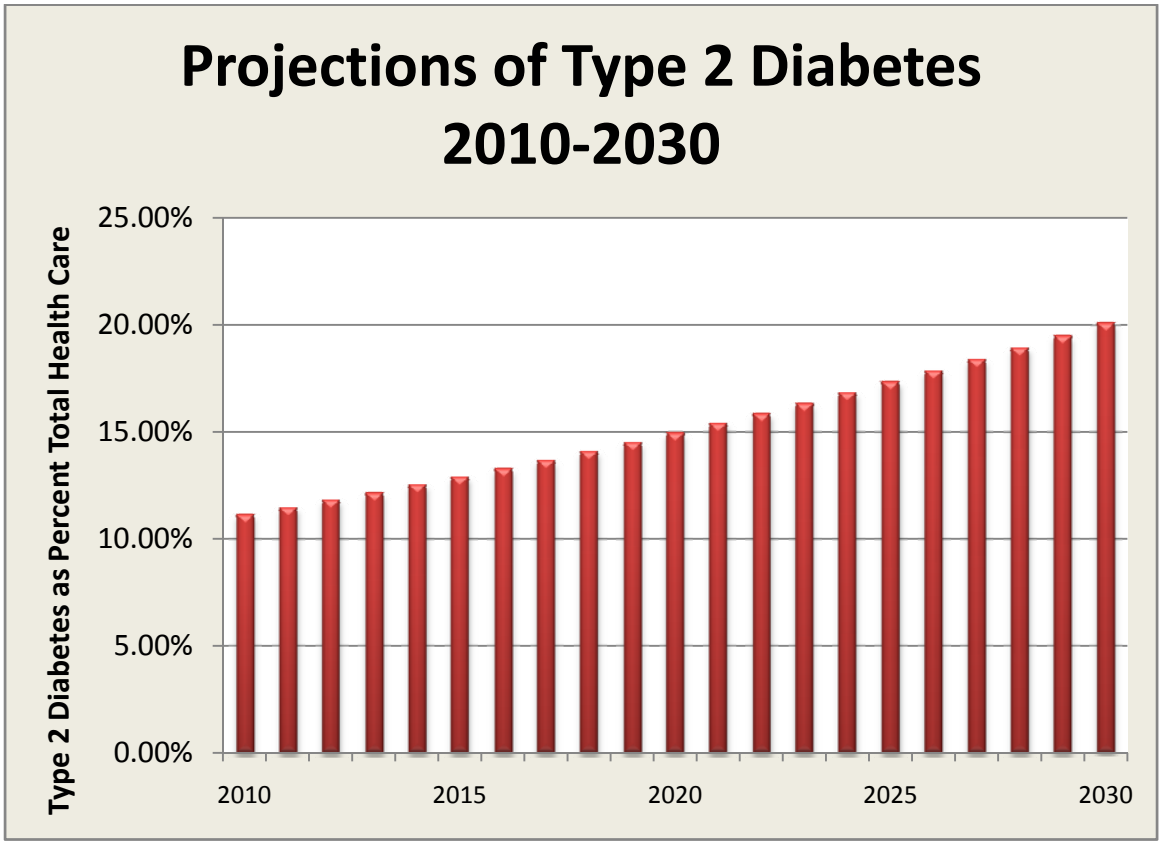
The primary drive of this process leading to Type 2 Diabetes is excess consumption of carbohydrates and generally this excess is directly exhibited in overweight individuals. Weight impact on health is measure by the Body Mass Index calculation. If the BMI is less than 25 and greater than 20 then this is a healthy range. Overweight is defined as BMI greater than 25 and less than 30. Obese is defined as anything greater than 30. Thus a 6'3" male of 250 lbs is obese, where as if he is greater than 210 he is overweight. The most predisposing factor, and many believe causative factor of Type 2 Diabetes if being overweight, not just being obese. This now applies for any age, children included.

In 1997 about 4.7% of people were Type 2 Diabetics. In 2008 that has increased to 5.9%. By 2020 the number is anticipated to increase to 15%. The total expenditures for Type 2 Diabetes in 2008 are in excess of $250 billion, about 12% of all health care expenditures. This combination results in an expenditure of $12,500 per Type 2 Diabetic per annum.

However the problem is explosive. With health care growing at 6% per annum in excess of inflation and Type 2 Diabetes prevalence growing at 3% per annum, and the overall population growing at 1% per annum, the net result is costs growing at in excess of 10% per annum! That exceeds rate of the health care costs alone. It is conceivable that Type 2 Diabetes will reach 20% of all health care costs by 2030.

We present this growth picture in the following Figure.

[1] See Porte et al and LeRoith et al.


Projections of Type 2 Diabetes 2010-2030
Type 2 Diabetes as Percent Total Health Care
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
2010
2015
2020
2025
2030

2 EPIDEMIOLOGY

This section presents a brief overview of the epidemiological factors associated with Type 2 Diabetes, and reviews the causes and the incidence and prevalence.

2.1 CAUSES

The primary cause of Type 2 Diabetes is excess weight[2]. It commences as a breakdown of the pancreas' β cells to produce adequate amounts of insulin which in turn results in excess glucose in the blood stream[3]. It appears that when the body has excess weight that pancreas cannot generate the insulin fast enough and has a "run down" result. It just stops generating insulin totally. There is however a social stigma to telling a Type 2 Diabetes patient that they are the cause of their disease due solely to their weight and their inability to control it. This is the distinct opposite to what had happened in cigarette smoking. Any attempt to modulate behavior related to caloric consumption is viewed as politically incorrect in today's environment which we believe is the main reason that we shall see an epidemic of Type 2 Diabetes especially in the young.

There is a strong relationship between Type 2 Diabetes and chronic inflammation, stresses that wear out the body's cells. As Lazar states:

"*The close relationship between inflammation and diabetes is supported by the observation that stimulation of the innate immune response [by bacterial endotoxin during sepsis, for example] results in insulin resistance that contributes to the high mortality of critical illness. The interaction between inflammation and insulin signaling is also suggested by the ability of aspirin to improve insulin resistance, in part by preventing the antagonistic effects of fatty acids and cytokines.*"

Lazar continues:

"Why is obesity an inflammatory state and why does inflammation cause diabetes? The search for answers to these questions takes us again to evolutionary considerations. Perhaps the response to infection is more effective when glucose is shunted from muscle to the inflammatory cells involved in the immune response and tissue repair. A potentially unifying view is that the body's ability to survive major stress, including infection and starvation, is enhanced by peripheral insulin resistance that preserves the

[2] See the paper by Lazar. This is an exceptionally good summary of the obesity argument placed within a genetic and evolutionary context.

[3] See the paper by Marx for a recent summary of the status of the causes of Diabetes.

brain's glucose supply. This hypothesis might explain why cortisol, the major stress hormone, causes insulin resistance and stimulates the innate immune response, even though chronic cortisol exposure is anti-inflammatory because of down-modulation of the acquired immune response. The stress connection may extend to individual cells, as it has recently been shown that intracellular stress induces insulin resistance in a manner that is exacerbated by obesity, potentially through adipocyte-secreted factors. Moreover, chronic metabolic stress impairs the ability of pancreatic beta cells to secrete sufficient insulin to overcome insulin resistance, which is a hallmark of type 2 diabetes."

Marx states:

"Although insulin resistance and the resulting impairment in glucose tolerance are early signs of diabetes, malfunction or even death of the insulin-producing β cells also contributes to the disease. Ultimately about a third of diabetes 2 patients end up having to take insulin. Several factors seem to be involved in β-cell dysfunction, including some of the same culprits implicated in insulin resistance. For example, in experiments performed on the Zucker rat, a rodent model of obesity and diabetes, Unger's group at UT Southwestern has found that fatty acids can trigger a form of cell death called apoptosis in β cells. The fatty acids work indirectly, the UT Southwestern team found: They are first converted in β cells to toxic compounds known as ceramides.

That suggests to Unger that the β-cell loss can be prevented. "If we block that [ceramide-producing] pathway, we can block apoptosis," he says. Unger also suggests that this fatty acid toxicity may result from the body's insensitivity to leptin. In his view, that hormone's job is to keep fatty acids from accumulating in cells that aren't designed to handle them, such as β cells and muscle. But β cells don't have to die to contribute to diabetes 2 pathology: They can simply fail to secrete the insulin needed to handle all the glucose the body takes in.

At least in mouse models, researchers can duplicate that type of malfunction. For example, a team led by Ronald Kahn of the Joslin Diabetes Center in Boston and Mark Magnuson of Vanderbilt University School of Medicine in Nashville, Tennessee, found that they could prevent the increase in insulin secretion that normally occurs in response to glucose ingestion by specifically inactivating the insulin receptor in the β cells of mice. As a result of the consequent block in insulin activity, glucose can't get inside the cells to trigger release of the hormone."

There are several studies which link certain genetic markers to Type 2 Diabetes. Several of these are shown below are from Marx.

SOME CANDIDATE DIABETES 2 GENES

Mutated Gene	*Function*	*Effect*	*Linked to*
HNF-4-α, HNF-1-β Transcription IPF-1, NeuroD1	Insulin→ factors	MODY (human) secretion	
HNF-1-α factor	Transcription secretion	Insulin→ Oji-Cree diabetes	MODY
Glucokinase metabolism	Glucose secretion	Insulin→	MODY
Calpain-10	Protease	Unknown Mexican and African Americans	Diabetes 2 in
PPAR-γ factor	Transcription sensitivity	Insulin→	Diabetes 2
Insulin receptor insulin signals into cell	Transmits sensitivity and secretion	Insulin→ (rare); mouse models	Human diabetes
IRS1 and -2 signaling	Insulin sensitivity	Insulin→	Mouse models
Akt2 signaling	Insulin sensitivity	Insulin→	Mouse models
11-β-HSD synthesis	Glucocorticoid lipids,	Blood → insulin sensitivity	Mouse models
UCP2	ATP → synthesis	Insulin→ secretion	Mouse models
Resistin	Fat cell "hormone"	Insulin→ sensitivity →	Mouse studies
Adiponectin	Fat cell "hormone"	Insulin sensitivity	Mouse, human studies

The current approach to treating Type 2 Diabetes is all too often the use of medications[4].

2.2 INCIDENCE AND PREVALENCE

The incidence and prevalence of Type 2 Diabetes is dramatically increasing. We briefly review the data on these two factors.

2.2.1 Incidence

[4] See the paper by DeFronzo and the one by Padwal. The DeFronzo paper is exceptionally good as a review of the various types of medications and their effective means of reducing insulin overload and maintaining proper insulin production.

The following is a summary of the NIH review of Type 2 Diabetes. The incidence of Diagnosed Diabetes in People Younger than 20 Years of Age, United States, 2002 to 2003 is as follows:

Based on 2002 to 2003 data, 15,000 youth in the United States were newly diagnosed with type 1 diabetes annually, and about 3,700 youth were newly diagnosed with type 2 diabetes annually.

The rate of new cases among youth was 19.0 per 100,000 each year for type 1 diabetes and 5.3 per 100,000 for type 2 diabetes.
Non-Hispanic white youth had the highest rate of new cases of type 1 diabetes.

Type 2 diabetes was extremely rare among youth aged <10 years. While still infrequent, rates were greater among youth aged 10 to 19 years compared with younger children, with higher rates among U.S. minority populations compared with non-Hispanic whites.

Among non-Hispanic white youth aged 10 to 19 years, the rate of new cases of type 1 diabetes was higher than for type 2 diabetes.

For Asian/Pacific Islander and American Indian youth aged 10 to 19 years, the opposite was true—the rate of new cases of type 2 was greater than the rate for type 1 diabetes.

Among African American and Hispanic youth aged 10 to 19 years, the rates of new cases of type 1 and type 2 diabetes were similar.

2.2.2 Prevalence

Prevalence of Diagnosed Diabetes in People Younger than 20 Years of Age, United States, 2007. About 186,300 people younger than 20 years have diabetes—type 1 or type 2. This represents 0.2 percent of all people in this age group. Estimates of undiagnosed diabetes are unavailable for this age group.

Prevalence of Diagnosed and Undiagnosed Diabetes in the United States, All Ages, 2007

Total: 23.6 million people—7.8 percent of the population—have diabetes.
Diagnosed: 17.9 million people
Undiagnosed: 5.7 million people

Prevalence of Diagnosed and Undiagnosed Diabetes among People Aged 20 Years or Older, United States, 2007

Age 20 years or older: 23.5 million, or 10.7 percent, of all people in this age group have diabetes.

Age 60 years or older: 12.2 million, or 23.1 percent, of all people in this age group have diabetes.

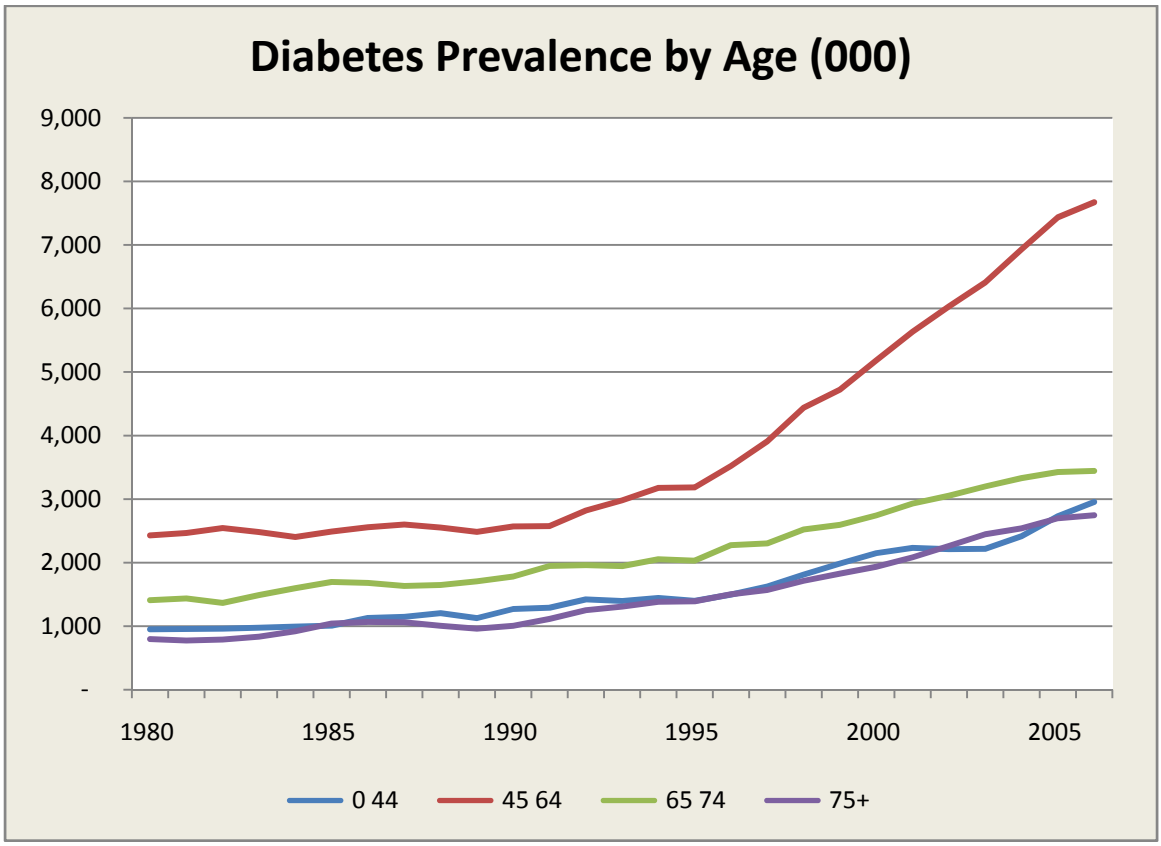
Men: 12.0 million, or 11.2 percent, of all men aged 20 years or older have diabetes.

Women: 11.5 million, or 10.2 percent, of all women aged 20 years or older have diabetes.

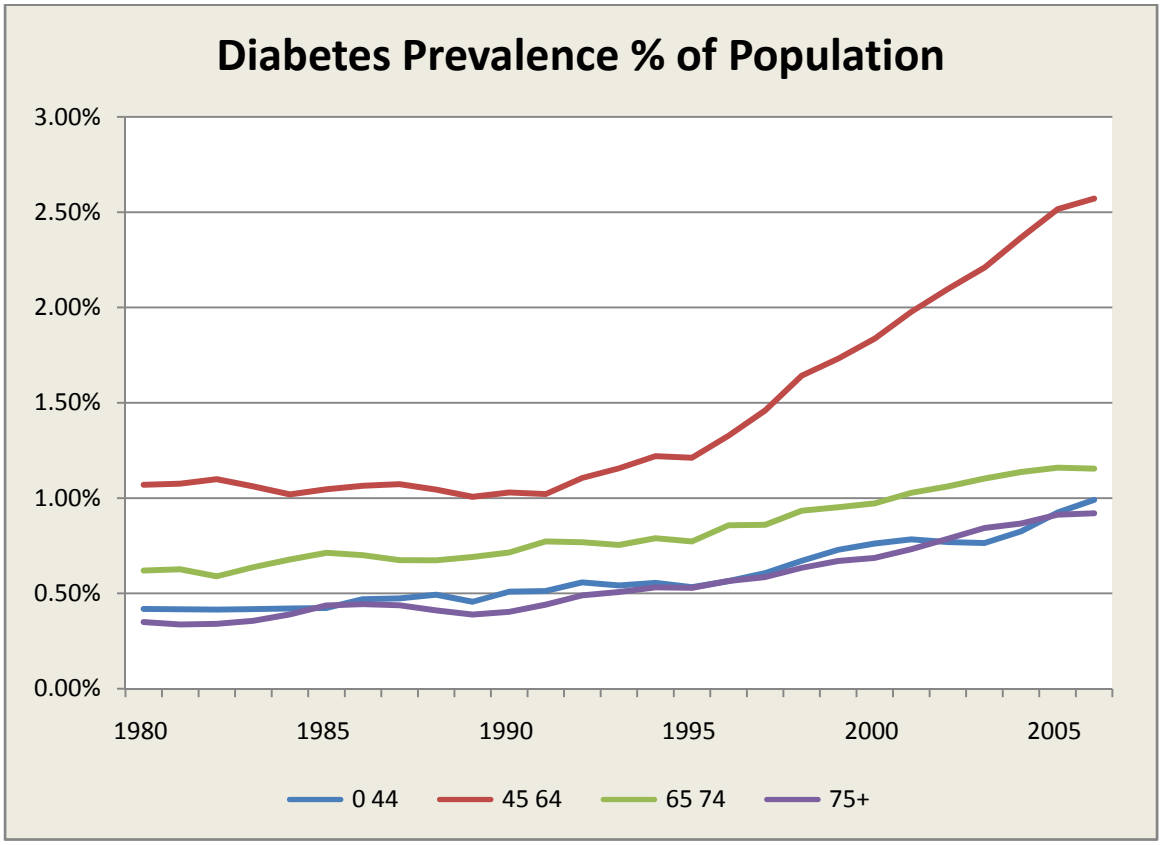
Non-Hispanic whites: 14.9 million, or 9.8 percent, of all non-Hispanic whites aged 20 years or older have diabetes.

Non-Hispanic blacks: 3.7 million, or 14.7 percent, of all non-Hispanic blacks aged 20 years or older have diabetes.

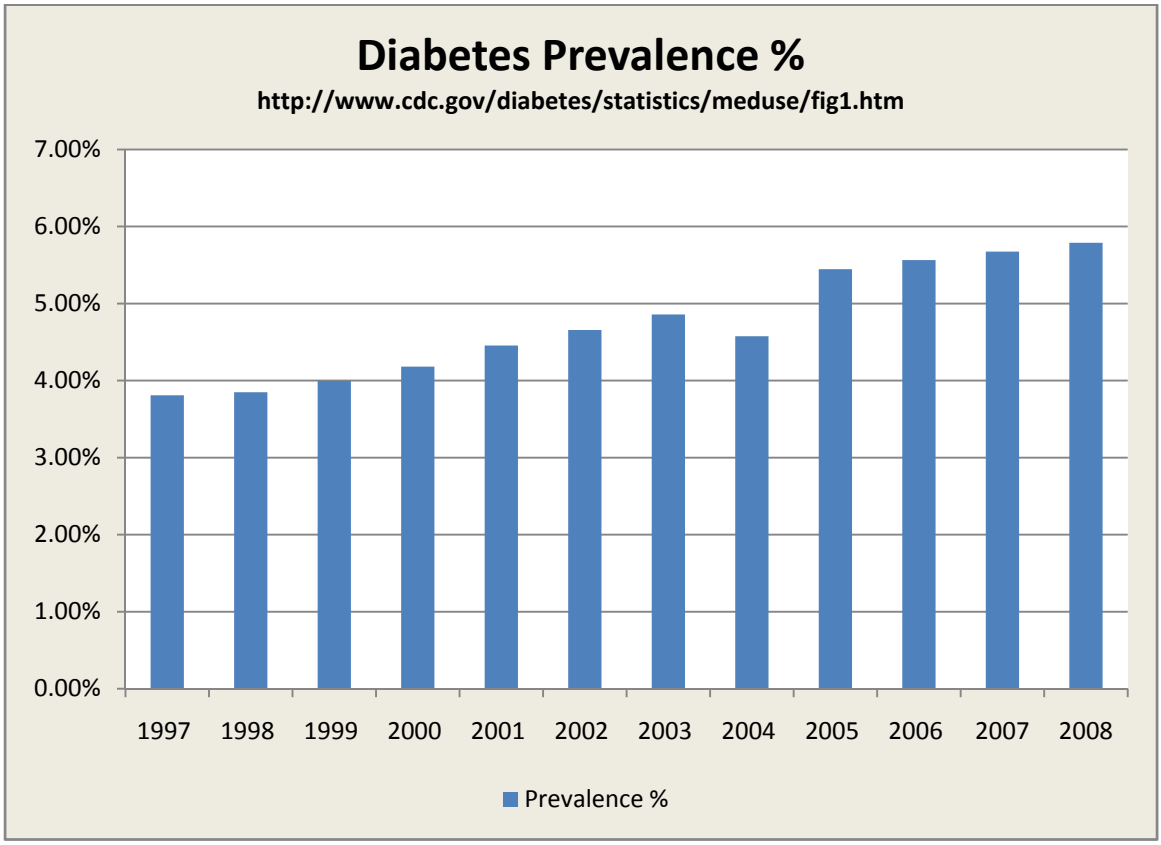
The following Figures depict prevalence over the past 25 years broken out by age group. There is an explosive growth amongst the 45-64 age group. This will of course spread as they age and create significant chronic health care costs.



The following shows the same data but now as percent of the population. Note that as before the 45-64 age group grows from 1% to over 2.5% of the total population. This is truly a worrisome factor. These are the baby boomers. They are



The overall combined prevalence is shown in the following Figure. This is a summary of the previous Figure but it rolls up all of the age categories.



2.3 COMPLICATIONS

The following is from the NIH data source[5].

Heart Disease and Stroke

- In 2004, heart disease was noted on 68 percent of diabetes-related death certificates among people aged 65 years or older.
- In 2004, stroke was noted on 16 percent of diabetes-related death certificates among people aged 65 years or older.
- Adults with diabetes have heart disease death rates about two to four times higher than adults without diabetes.
- The risk for stroke is two to four times higher among people with diabetes.

High Blood Pressure

- In 2003 to 2004, 75 percent of adults with self-reported diabetes had blood pressure greater than or equal to 130/80 millimeters of mercury (mm Hg) or used prescription medications for hypertension.

[5] See http://diabetes.niddk.nih.gov/dm/pubs/statistics/index.htm#allages

Blindness

- Diabetes is the leading cause of new cases of blindness among adults aged 20 to 74 years.
- Diabetic retinopathy causes 12,000 to 24,000 new cases of blindness each year.

Kidney Disease

- Diabetes is the leading cause of kidney failure, accounting for 44 percent of new cases in 2005.
- In 2005, 46,739 people with diabetes began treatment for end-stage kidney disease in the United States and Puerto Rico.
- In 2005, a total of 178,689 people with end-stage kidney disease due to diabetes were living on chronic dialysis or with a kidney transplant in the United States and Puerto Rico.

Nervous System Disease

- About 60 to 70 percent of people with diabetes have mild to severe forms of nervous system damage. The results of such damage include impaired sensation or pain in the feet or hands, slowed digestion of food in the stomach, carpal tunnel syndrome, erectile dysfunction, or other nerve problems.
- Almost 30 percent of people with diabetes aged 40 years or older have impaired sensation in the feet—for example, at least one area that lacks feeling.
- Severe forms of diabetic nerve disease are a major contributing cause of lower-extremity amputations.

Amputations

- More than 60 percent of nontraumatic lower-limb amputations occur in people with diabetes.
- In 2004, about 71,000 nontraumatic lower-limb amputations were performed in people with diabetes.

Dental Disease

- Periodontal, or gum, disease is more common in people with diabetes. Among young adults, those with diabetes have about twice the risk of those without diabetes.
- Persons with poorly controlled diabetes (A1C > 9 percent) were nearly three times more likely to have severe periodontitis than those without diabetes.
- Almost one-third of people with diabetes have severe periodontal disease with loss of attachment of the gums to the teeth measuring 5 millimeters or more.

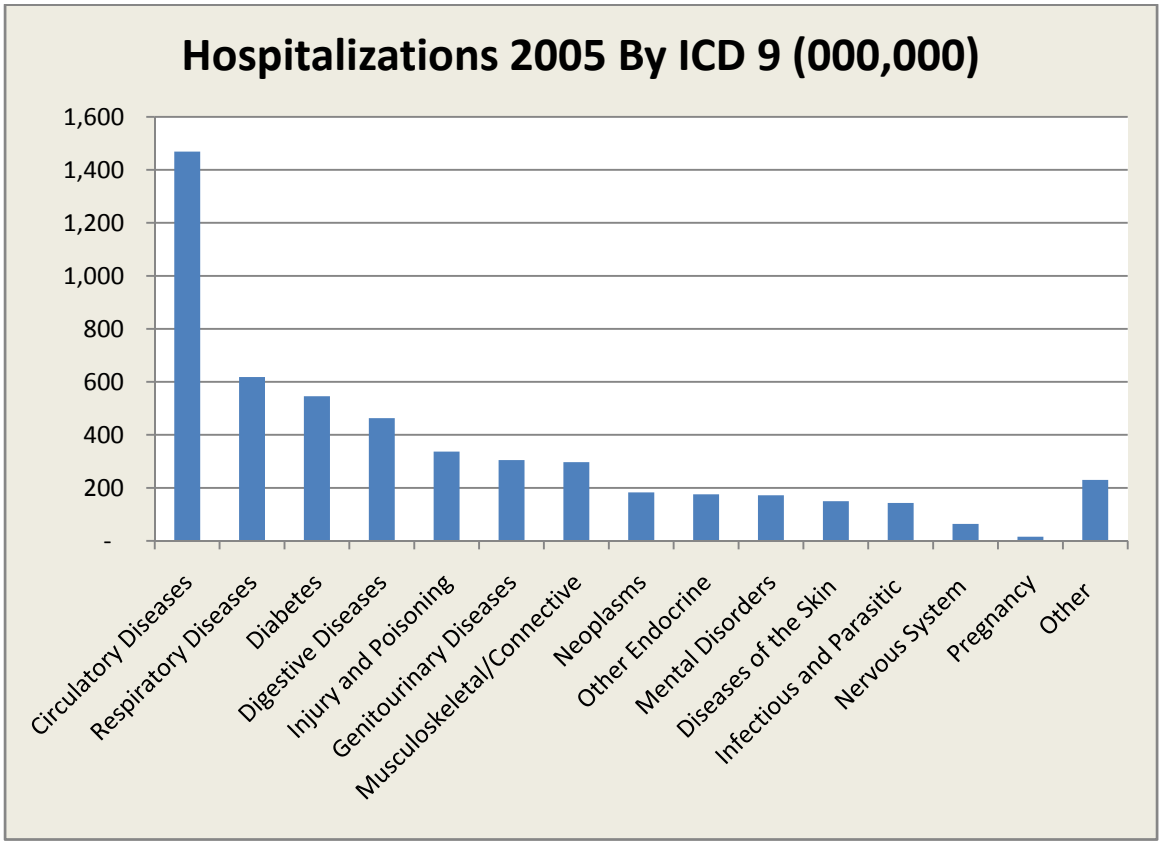
Complications of Pregnancy

- Poorly controlled diabetes before conception and during the first trimester of pregnancy among women with type 1 diabetes can cause major birth defects in 5 to 10 percent of pregnancies and spontaneous abortions in 15 to 20 percent of pregnancies.
- Poorly controlled diabetes during the second and third trimesters of pregnancy can result in excessively large babies, posing a risk to both mother and child.

Other Complications

- Uncontrolled diabetes often leads to biochemical imbalances that can cause acute life-threatening events, such as diabetic ketoacidosis and hyperosmolar, or nonketotic, coma.
- People with diabetes are more susceptible to many other illnesses and, once they acquire these illnesses, often have worse prognoses. For example, they are more likely to die with pneumonia or influenza than people who do not have diabetes.
- Persons with diabetes aged 60 years or older are two to three times more likely to report an inability to walk a quarter of a mile, climb stairs, do housework, or use a mobility aid compared with persons without diabetes in the same age group.

Hospitalizations resulting from Type 2 Diabetes are shown in the Figure below:



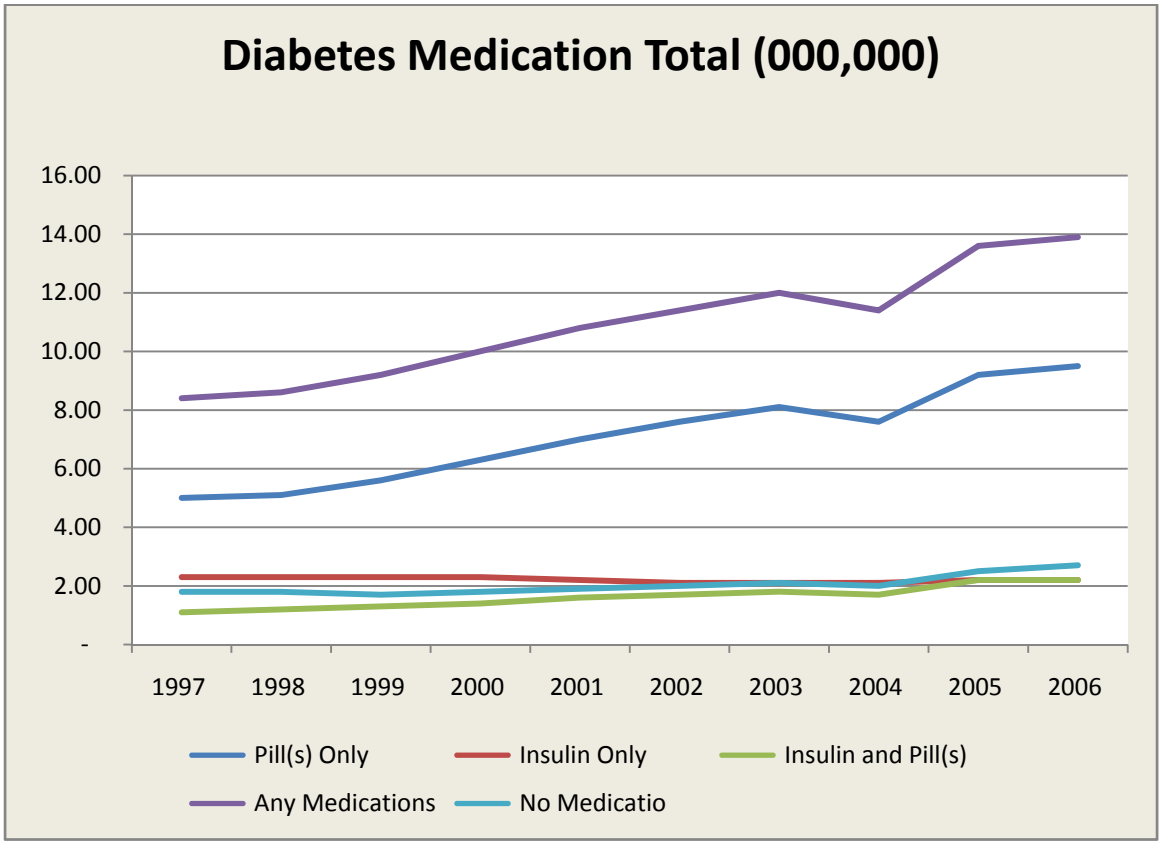
Note that in the above we find that the cardiovascular complications are the dominant factor causing a hospitalization. Most other treatments as we shall see later are chronic in nature. Type 2 Diabetes is from the perspective of treatment thus predominantly a chronic disorder which requires a great deal of complex medical treatment.

2.4 TREATMENTS

The main treatment for Type 2 Diabetes is either weight reduction combined with exercise or medication.

Drug (Trade Name, Manufacturer)	*Usual Daily*	*Dosage Cost*
	mg	*$*
First-generation sulfonylureas		
Acetohexamide	500–750	$14.18
Chlorpropamide (Diabenase, Pfizer)	250–375	$1.19
Tolazamide	250–500	$2.81
Tolbutamide	1000–2000	$2.75
Second-generation sulfonylureas		
Glimepiride (Amaryl, Hoechst Marion Roussel)	1–4	$7.06
Glipizide	10–20	$3.38
(Glucotrol, Pfizer)		$21.38
(Glucotrol XL, Pfizer)		$10.07
Glyburide	5–20	$14.95
(DiaBeta, Hoechst Marion Roussel)		$20.43
(Micronase, Pharmacia & Upjohn)		$22.93
(Glynase, Pharmacia & Upjohn)		$20.10
Nonsulfonylureas		
Acarbose (Precose, Bayer)	150–300	$41.05
Metformin (Glucophage, Bristol-Myers Squibb)	1500–2550†	$48.38
Repaglinide (Prandin, Novo Nordisk)	1–4	$57.12
Troglitazone (Rezulin, Parke-Davis)	400–600	$147.20

The growth of these medications is shown in the Graph below.


Diabetes Medication Total (000,000)
16.00
14.00
12.00
10.00
8.00
6.00
4.00
2.00
-
1997
1998
1999
2000
2001
2002
2003
2004
2005
2006
Pill(s) Only
Insulin Only
Insulin and Pill(s)
Any Medications
No Medicatio

3 COSTS

We can now assess the costs associated with Type 2 Diabetes. We rely upon a study performed by Michael Brandle, MD et al entitled The Direct Medical Cost of Type 2 Diabetes, from the 1Division of Endocrinology & Metabolism, Department of Internal Medicine, University of Michigan, Ann Arbor, Michigan. This is an interesting study of benchmarking prevalence and costs by treatment type. We rely upon this data and we have updated it from 2000 cost numbers to 2008 cost numbers as well as incorporating a total US prevalence. It should be noted that certain procedures have not been costed in the model so that the actual costs are estimated to be 10-15% higher than calculated herein.

3.1 SECONDARY DISORDERS

Type 2 Diabetes leads to many secondary disorders. The key one we have briefly discussed before but they are as follows:

1. Retinopathy status
    a. Nonproliferative retinopathy
    b. Proliferative retinopathy
    c. Macular edema
2. Nephropathy status
    a. Microalbuminuria
    b. Proteinuria
    c. ESRD with dialysis
3. Neuropathy status
    a. Neuropathy
    b. Amputation
4. Cerebrovascular disease
5. Cardiovascular status
    a. Angina
    b. MI
6. Peripheral vascular disease
7. High blood pressure BP 140/90 mmHg

In a study by Brandle the team determined the prevalence of each of these and then went and determined the costs of treatment. We shall rely on and modify the Brandle work herein as a basis for estimating the current and projected costs. The following Table presents the statistics of the study group and the prevalence of the above common secondary effects.

Factor	*Range*	*Mean*	*Percent*
Sample Size *N*	1,364	1,364	100.0%
Age (years)	66 (54–72)	66	4.8%
Diabetes duration (years)	8 (4–16)	8	0.6%
Sex			
Male	681 (50)	681	49.9%
Race			
White	1,005 (74)	1005	73.7%
African American	176 (13)	176	12.9%
BMI (kg/m2)	30.7 (27.1–36.1)	31	2.3%
HbA1c (%)	7.1 (6.3–8.2)	7.00	0.5%
Education			
Not a high school graduate	244 (18)	244	17.9%
High school graduate	397 (29)	397	29.1%
Some college	383 (28)	383	28.1%
College graduate	133 (10)	133	9.8%
Any postgraduate work	153 (11)	153	11.2%
Missing*	54 (4)	54	4.0%
Household income			0.0%
$40,000	903 (66)	903	66.2%
$40,000–69,999	289 (21)	289	21.2%
$70,000	172 (13)	172	12.6%
Diabetes treatment			0.0%
Diet or exercise only	69 (5)	69	5.1%
Oral antidiabetic medication	870 (64)	870	63.8%
Insulin	425 (31)	425	31.2%
Retinopathy status			0.0%
Nonproliferative retinopathy	170 (12)	170	12.5%
Proliferative retinopathy	41 (3)	41	3.0%
Macular edema	29 (2)	29	2.1%
Missing	248 (18)	248	18.2%
Nephropathy status			
Microalbuminuria	99 (2)	99	7.3%
Proteinuria	207 (15)	207	15.2%
ESRD with dialysis	6 (0.4)	6	0.4%
Missing	248 (18)	248	18.2%
Neuropathy status			
Neuropathy	544 (40)	544	39.9%
History of amputation	25 (2)	25	1.8%
Cerebrovascular disease			
Cerebrovascular disease	199 (15)	199	14.6%
Missing	217 (16)	217	15.9%
Cardiovascular status			0.0%
Angina	58 (4)	58	4.3%
History of MI	363 (27)	363	26.6%
Missing	205 (15)	205	15.0%
Peripheral vascular disease			0.0%
Peripheral vascular disease	538 (39)	538	39.4%
Missing	248 (18)	248	18.2%
High blood pressure status			0.0%
BP 140/90 mmHg without treatment	481 (35)	481	35.3%

Factor	*Range*	*Mean*	*Percent*
Treated with medication	416 (31)	416	30.5%
Missing	253 (19)	253	18.5%
Cholesterol status			0.0%
LDL 100 mg/dl without treatment	386 (28)	386	28.3%
Treated with medication	404 (30)	404	29.6%
Missing	248 (18)	248	18.2%
Cigarette smoking			0.0%
Current smoker	233 (17)	233	17.1%
Missing	17 (1)	17	1.2%
Total cost	3,715 (1,894–7,719)	$3,715	656847.1%

This above Table will be used again in the costing process.

3.2 PROCEDURE AND COSTS

Using the Brandle data we again take the secondary factors and the incorporate the Brandle data using the 2000 year costs and the prevalence and then update to 2008 populations and escalated health care costs.

The following Table details the analysis of providing the 2008 costs. These costs can then be projected forward in time using the same process of scaling.

	Baseline cost	*$1,684*	$2,977
Disease status	*Increment SE in $\log_{10}$ scale*	*Multiplier (2000)*	*Amount (2008)*
Sex			
Female	0.095 0.025	1.25	$3,722
Age	‡		$0
Race			
African American	0.088 0.036	0.82	$2,442
Duration			
Every 1 year after onset	‡		$0
BMI (kg/m2)			
Every unit 30 kg/m2	0.004 0.002	1.01	$3,007
Diabetes intervention			
Oral antidiabetic medication	0.040 0.056	1.10	$3,275
Insulin	0.200 0.058	1.59	$4,734
High blood pressure			
Treated blood pressure	0.092 0.028	1.24	$3,692
Retinopathy			
Nonproliferative retinopathy	‡		$0
Proliferative retinopathy	‡		$0
Macular edema	‡		$0
Nephropathy			
Microalbuminuria	0.067 0.048	1.17	$3,484
Proteinuria	0.113 0.036	1.30	$3,871
ESRD with dialysis	1.023 0.183	10.53	$31,353
Neuropathy			
Clinical neuropathy	‡		$0
History of amputation	‡		$0
Cerebrovascular disease	0.113 0.035	1.30	$3,871
Cardiovascular disease			
Angina	0.239 0.061	1.73	$5,151
History of MI	0.278 0.029	1.90	$5,657
Peripheral vascular disease	0.116 0.028	1.31	$3,900

Finally there are three major events that Brandle notes and they are shown below, each requiring hospitalization. We have also updated their costs and their prevalence.

	Subjects who survived first year (n)	*Total costs for 1 year after onset of acute event for subject who survived first year (2000 Dollars)*	*Costs in 2008 Dollars*	*Incidence*
Acute event			1.77	
Stroke	88	$26,600 (15,400–44,900)	$47,031	6.45%
Acute MI	84	$24,500 (15,000–50,000)	$43,318	6.16%
Amputation	47	$37,600 (23,300–62,200)	$66,480	3.45%

3.3 TOTAL ANNUAL COSTS

Now taking the above data we can then combine the result to estimate total costs of Type 2 Diabetes in 2008. This is accomplished in the following Table. Here we have provided the data for only the secondary factors.

Disease status	*Prevalence %*	*Total Prevalence*	*Unit Cost*	*Total Cost ($000,000)*
Diabetes intervention			$0	
Oral antidiabetic medication	3.32%	584,622	$3,275	$1,915
Insulin	1.45%	254,687	$4,734	$1,206
High blood pressure				
Treated blood pressure	35.26%	6,206,452	$3,692	$22,915
Retinopathy				
Nonproliferative retinopathy	12.46%	2,193,548	$0	$0
Proliferative retinopathy	3.01%	529,032	$0	$0
Macular edema	2.13%	374,194	$0	$0
Nephropathy				
Microalbuminuria	7.26%	1,277,419	$3,484	$4,450
Proteinuria	15.18%	2,670,968	$3,871	$10,339
ESRD with dialysis	0.44%	77,419	$31,353	$2,427
Neuropathy				
Clinical neuropathy	39.88%	7,019,355	$0	$0
History of amputation	1.83%	322,581	$0	$0
Cerebrovascular disease	14.59%	2,567,742	$3,871	$9,939
Cardiovascular disease				
Angina	4.25%	748,387	$5,151	$3,855
History of MI	26.61%	4,683,871	$5,657	$26,498
Peripheral vascular disease	39.44%	6,941,935	$3,900	$27,077
Acute Illnesses				
Stroke	6.45%	1,135,484	$47,031	$53,403
Acute MI	6.16%	1,083,871	$43,318	$46,951
Amputation	3.45%	606,452	$66,480	$40,317
Total		39,278,018		$251,291

The result is that we estimate that Type 2 Diabetes costs $251 billion in 2008. This does not include the retinopathy costs which were lacking. We estimate that they add an additional 10%, raising the total to $275 billion, exceeding 12% of all 2008 health care expenditures.

4 ECONOMICS OF CONTROL

Having developed a model for costs we now consider the economics of management and control of Type 2 Diabetes. Since we have argued that by managing weight we can control if not cure Type 2 Diabetes we can argue that this is no longer a Medical problem but an economics and social problem. We have managed to deal with cigarette smoking and have reduced male mortality by 50% over the past 25 years and thus we argue that the same can be done for Type 2 Diabetes in even less a time period. Clearly obesity is the problem, the cause of obesity is clear. Each pound of body weight result from the consumption of 3,500 calories (Kcal) more than the sustaining level. For most people the sustaining level is 2,000 calories per day. Thus two cans of Coke, at 180 cal each, 360 cal for the two, will add a pound every 10 days, and will add 36 pounds in a year! There is no way of getting around this. The basic law of physics is:

Input-Output = Net Accumulation

4.1 THE ECONOMIC IMPLICATIONS

From the NIH data source we have the Estimated Diabetes Costs in the United States in 2007:

1. **Total—direct and indirect:** $174 billion. Note that we have calculated a greater number for 2008 using the model developed herein.

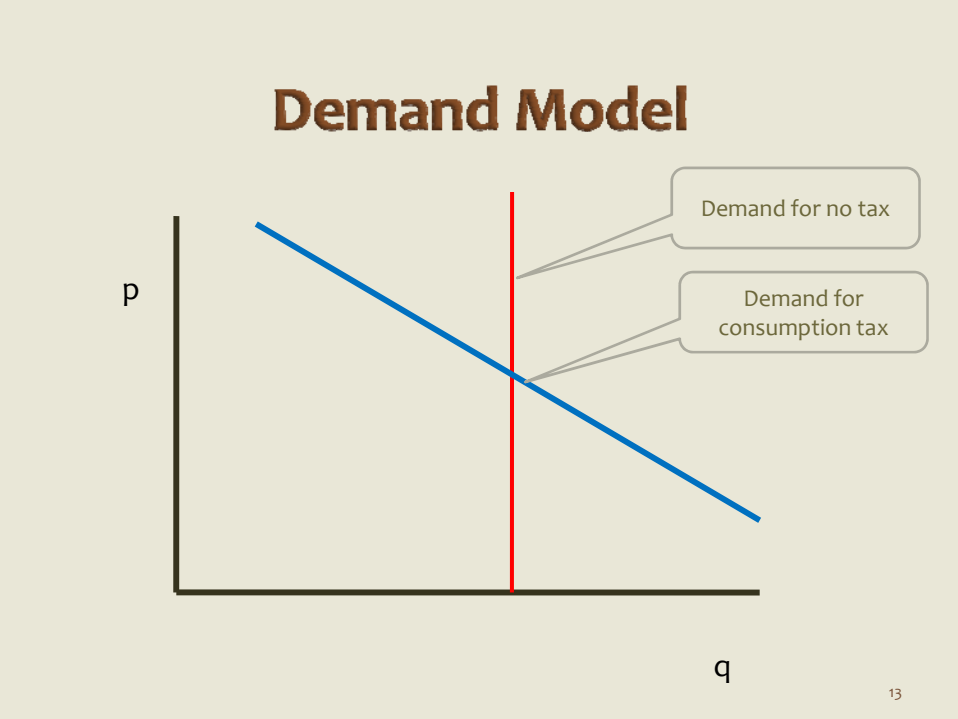
2. **Direct medical costs:** $116 billion: After adjusting for population age and sex differences, average medical expenditures among people with diagnosed diabetes were 2.3 times higher than what expenditures would be in the absence of diabetes.

3. **Indirect costs:** $58 billion—disability, work loss, premature mortality

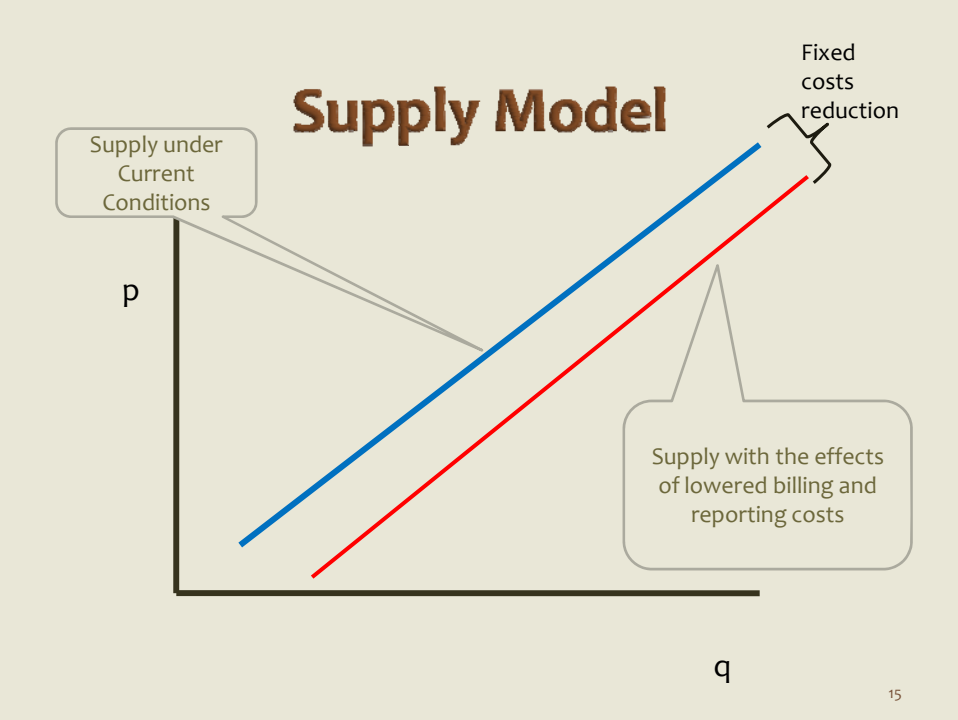
We can now examine in simple economic terms what the impact of the proposal will be. We proceed through five steps; demand, supply (three steps) and market stability.

Consider first the demand. Here we plot demand on a price, p, and quantity, q, and diagram common to all economics. Currently demand is independent of price. The current demand is a vertical line that is fixed and independent to any costs. This is more than simplistic since we generally accept anyone into the ER and in states like California illegal immigrants are provided care independent of any status. Now if we apply to the system some "tax" for bad foods or behavior and also provide costs incentives for

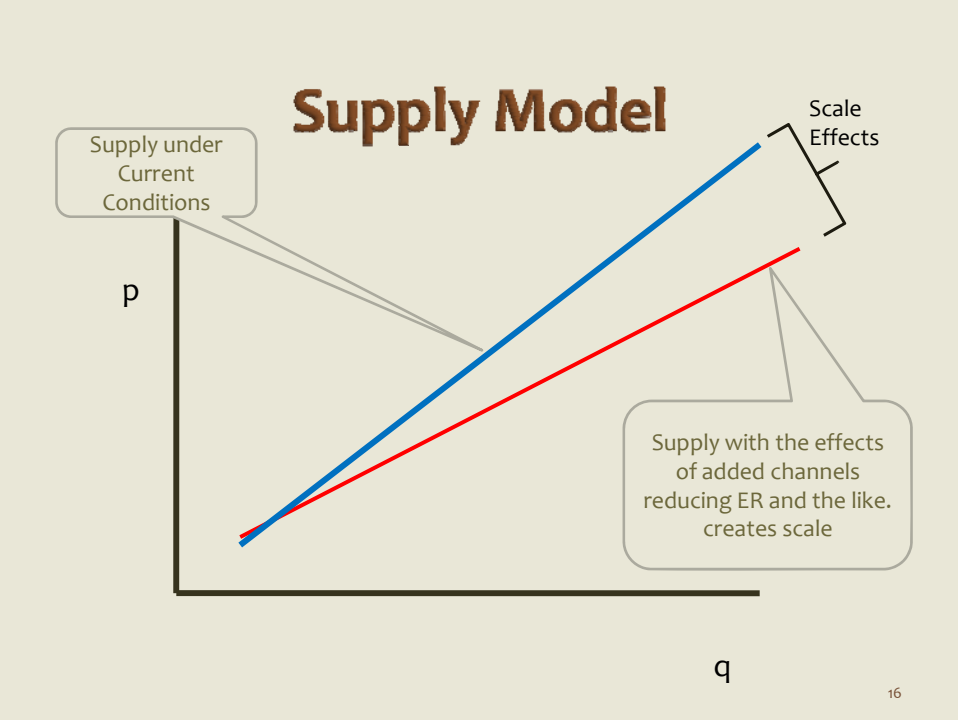
excessive use then we get a more normal demand curve, namely price or cost sensitivity.



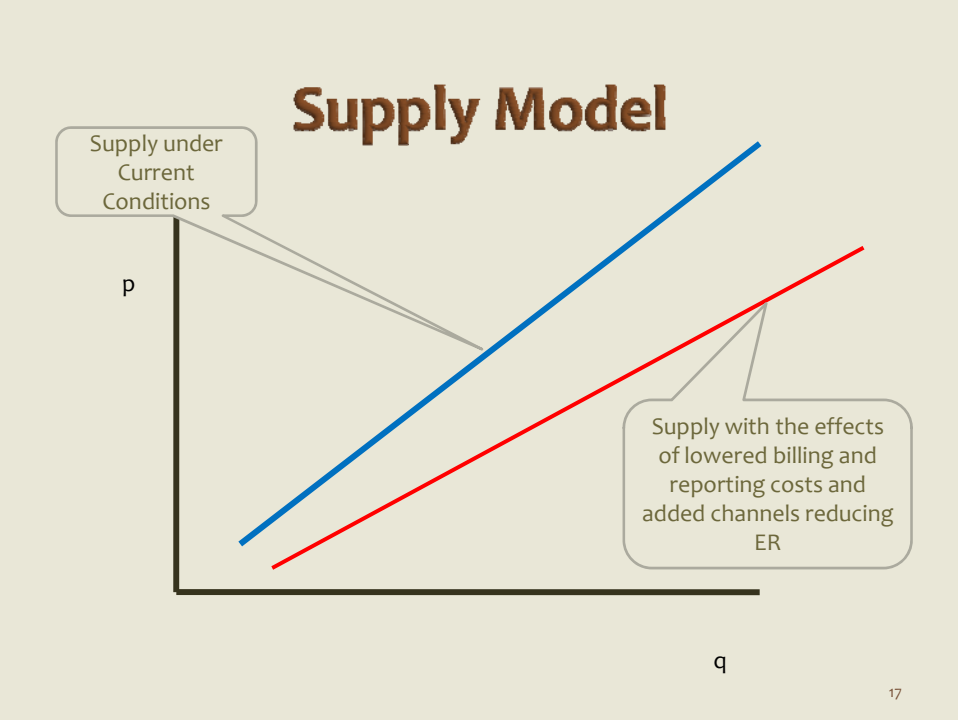
Now the first step in the supply curve will be to drive out costs which are overhead costs. Thus is we reduce the cost of billing and that of report management on a per patient basis this would represent a shift in the supply curve downward as shown in the following. This is the first step in cost reduction.



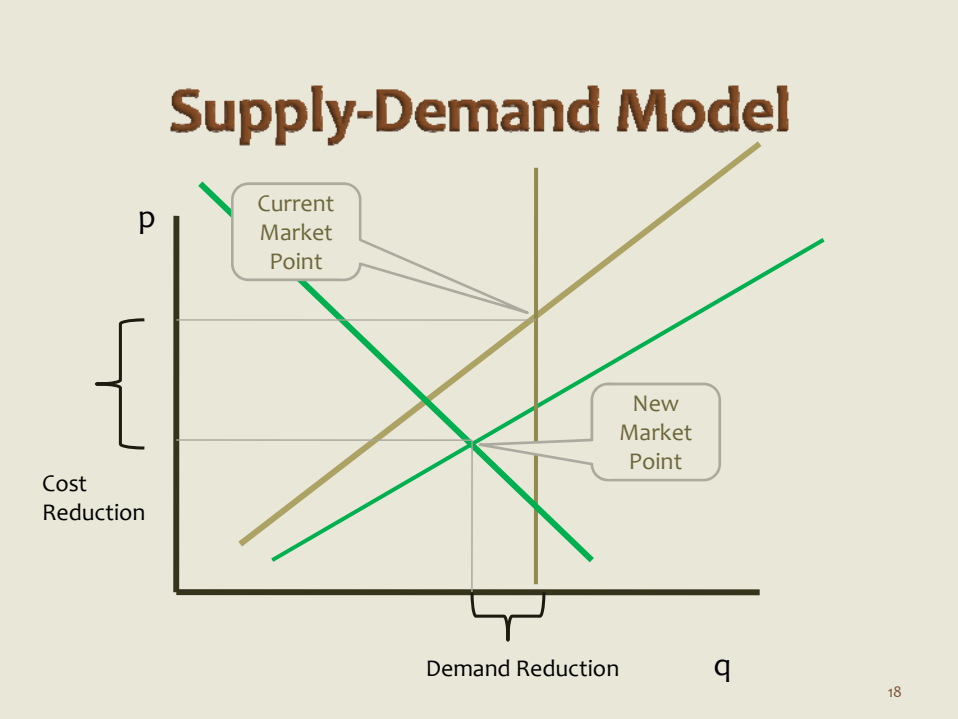
The alternative would be to create scale economies in the delivery, namely making it less costly the more service that are delivered. We argue herein that using a Public Health delivery system would do so by alleviating other more costly means such as the ER. There are many more examples of such an approach. The following Figure depicts what would happen in this event. Namely we see a decline of the supply curve the larger the demand becomes, clear scale effects.



We can then combine the two effects of reducing overhead and achieving scale to create a compound new supply curve as shown in the following Figure



Finally we can combine the Demand and the Supply curves to show what the total effect would be. This is done in the following Figure.



We note that we reduce the costs significantly while have a small but measurable decrease in the supply by means or reallocation while keeping the overall quality high. This above graphic is in essence what we propose in the plan.

4.2 METHODS OF REMEDIATION

Remediation of Type 2 Diabetes is readily accomplished by pricing the offending entity, in this case carbohydrates, at a point where the cost become prohibitive. This is the tax at the source approach. A second approach is tax at the result approach where we tax a person based upon BMI. Thus at BMI less than 25 we have no tax and from there upwards we tax at an aggressively increasing rate. This of course would be quite difficult to achieve. However the tax at source approach would be readily achieved.

5 CONCLUSIONS

This White Paper demonstrates several key conclusions:

1. Type 2 Diabetes is a disease caused by the person having it. It is a self induced disease for almost all. A very few have a massive as yet identified genetic flaw, but that is de minimis. It is obesity as the main and almost sole cause.

2. The prevalence of Type 2 Diabetes is growing exponentially and the term of epidemic is not unjustly employed. It is an epidemic resulting from childhood lack of food control, lack of exercise, and lack of recognition that the problem is moving downward in age and becoming explosive as that population itself ages.

3. The current means of managing Type 2 Diabetes with drugs such as metformin, insulin, sulfonylureas, and others mask the true problems and in many ways exacerbate the secondary disease states.

4. Type 2 Diabetes is now at 12% of the total health care costs and with its current growth rate combined with costs and population may reach 25% of the total health care budget. This disease has the sole potential for collapsing the health care system.

5. Type 2 Diabetes is a disease which can be controlled and possibly eliminated by purely open market means. By pricing carbohydrates at a point that exceed the propensity to buy, then there will be a reduction in carbohydrate driven obesity. This is akin to taxing the use of tobacco in cigarettes.

6. The use of combine drugs such as statins and the existing insulin modulating drugs oftentimes create additional tertiary health problems that add to the overall burden while also costing a great deal in the use of the drugs themselves.

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