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UNDERSTANDING RECENT TRENDS IN DISEASE TREATMENT RATES: ARE WE GETTING SICKER OR GETTING SCREENED?

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Abstract: The proportion of the population treated for major medical conditions, including diabetes, cancer, and mental illness, increased rapidly during the 1990s. We document the magnitude of these increases and calculate the impact on condition-specific health care spending. We then seek to explain the trend, focusing on the contribution of the obesity epidemic, increases in life expectancy, and more aggressive screening and detection practices. We find that increases in obesity levels explain a large proportion of the increase in treatment rates for conditions closely linked to obesity, like diabetes. The residual appears to be driven by a shift towards the detection and treatment of mildly symptomatic or asymptomatic cases. The shift towards early detection defies easy explanation. For some conditions detection patterns have changed in response to the availability of new treatment technology, but this is not always case.

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The concept of morbidity has been enlarged to cover prognosticated risks.

– Ivan Illich, Medical Nemesis: The Expropriation of Health, 1982.

It's no longer a question of staying healthy. It's a question of finding a sickness you like.

– Jackie Mason

1. INTRODUCTION

Health care spending is projected to reach 20% of Gross Domestic Product by 2015 (Borger et al. 2006). The consensus among health economists is that while rising incomes and the spread of insurance contribute to cost growth, technological innovation is the major factor driving increased spending (Fuchs 1996). New technology leads to higher costs via increases in the cost per treated case and increases in the number of treated cases (i.e. "treatment expansion") (Cutler and McClellan 2001). Most of the economic literature on health care spending has emphasized the impact of technology on costs per treated case and, in an attempt to gauge the benefits of more intensive treatment, investigators have purposefully restricted attention to medical conditions (for example, heart attack and low birthweight infants) where the number of patients has been relatively stable over time. The literature has acknowledged the role of treatment expansion in passing, but it has not been a focus. Perhaps this is because the potential for treatment expansion is bounded. As Cutler (2005) writes: "As more people are treated with existing technologies, the share of untreated people in the population will fall. The number of people left to expand treatment to would thus naturally diminish." However, there are important reasons to examine treatment expansion as a distinct phenomena. As we have shown in previous work (Thorpe et al. 2005; Thorpe and Howard 2006), expansions in recent years have been large in magnitude and are responsible for a non-trivial share of cost growth. The development of ever more powerful diagnostic devices and the use of tests based on genetic markers for disease will likely ensure that treatment rates continue to rise. Also, treatment expansion is often linked to new technology, but in for some conditions the contribution of

technology in driving treatment expansion is unclear. Indeed there is an extensive literature on treatment expansion in sociology that downplays the "technological determinism" of health economics. To the extent that expansion is driven by factors other than technology, and to the extent that newly treated patients differ in fundamental respects from existing patients, then studies on the marginal effects of medical technology tell us little about the welfare implications of treatment expansion.

In this paper we seek to document and explain increases in treatment rates for major medical conditions. A number of studies in the medical literature have addressed increases in treatment rates on a disease-by-disease basis, but we are interested in determining whether there are factors that underlie increases across diseases. We present evidence suggesting that increases in treatment rates are driven by more aggressive detection and treatment of patients with mildly symptomatic and asymptomatic cases. In seeking to explain shifting treatment patterns, we discuss the contribution of treatment technology, diagnostic technology, pharmaceutical marketing practices, and "medicalization".

2. TRENDS IN TREATED PREVALENCE

"Treated prevalence" refers to the proportion of the population receiving treatment for a specific medical condition. (Here we use the term interchangeably with "treatment rate") It differs from the standard epidemiological concept of prevalence in two respects. It excludes persons who have the disease in question but do not receive medical treatment for it. It includes persons who do not have the disease but are receiving treatment in response to a false positive test result. Theoretically, treated prevalence could be higher or lower than the actual, clinical prevalence of disease, though in most cases it will be lower.

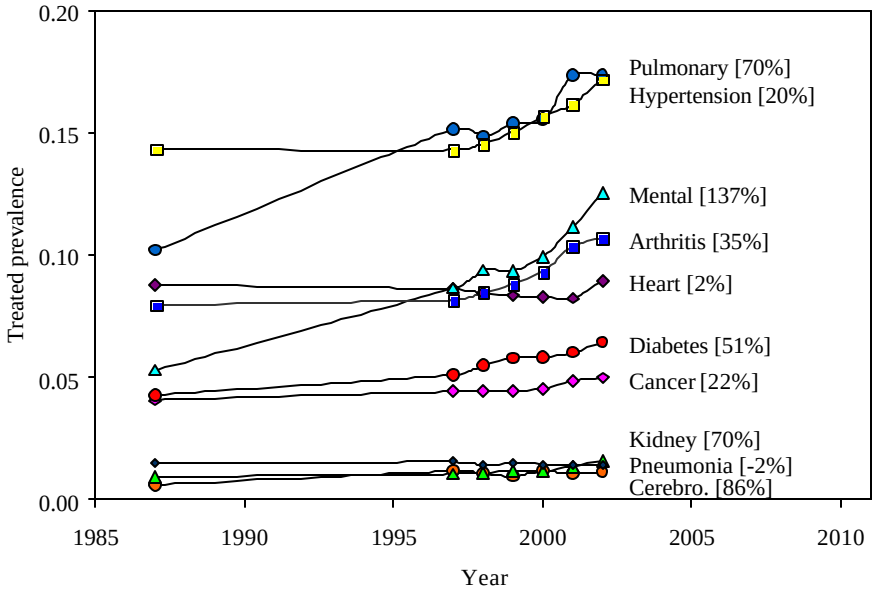
We begin by examining trends in treated prevalence using the 1987 National Medical Expenditure Survey (NMES) and the 1997-2002 Medical Expenditure Panel Surveys (MEPS). We focus on the 10 most costly diseases in terms of total spending: heart disease, cancer, mental disorders, pulmonary conditions (which includes asthma and emphysema), diabetes, hypertension (i.e. high blood pressure), cerebrovascular

disease (i.e. stroke), arthritis, pneumonia, and kidney disease (Cohen and Krauss 2003).[1] NMES and MEPS are household level surveys that record information on the medical spending and treatment patterns of the U.S. civilian, non-institutionalized population. Both are administered by the Census Bureau. Survey respondents are asked by an in-person interviewer whether they have used medical care or were diagnosed with a medical condition in the period since the last survey (MEPS is a panel survey) or a fixed interval before the survey. For survey respondents who report use of medical care, Census Bureau interviewers contact the respondent's medical providers to determine reimbursement amounts, treatments, and diagnoses. Information about the frequency of treated conditions is based on both individual and provider responses.

Figure 1 displays the treated prevalence of the ten medical conditions for the years 1987 through 2002. The yearly sample size ranges between 16,847 and 27,574 respondents. An individual is counted as receiving treatment for a disease if he received inpatient, outpatient, or physician office care or a prescription drug associated with the condition. These and all subsequent results using the NMES/MEPS data are weighted to produce nationally representative estimates. The graph shows that treatment rates have risen for all diseases except pneumonia, which declined slightly. The numbers in the brackets show the percentage increase from 1987 to 2002.

[1] Cohen and Krauss (2003) also include trauma and back problems in their list of the ten most costly medical conditions. We exclude trauma because the factors that determine treatment rates (such as seat belt use) makes it unique among the conditions we examine. We exclude "back problems" because it really more of a symptom than a disease.

Figure 1: Treated prevalence among NMES/MEPS respondents 18+, standardized by age and sex to 2002



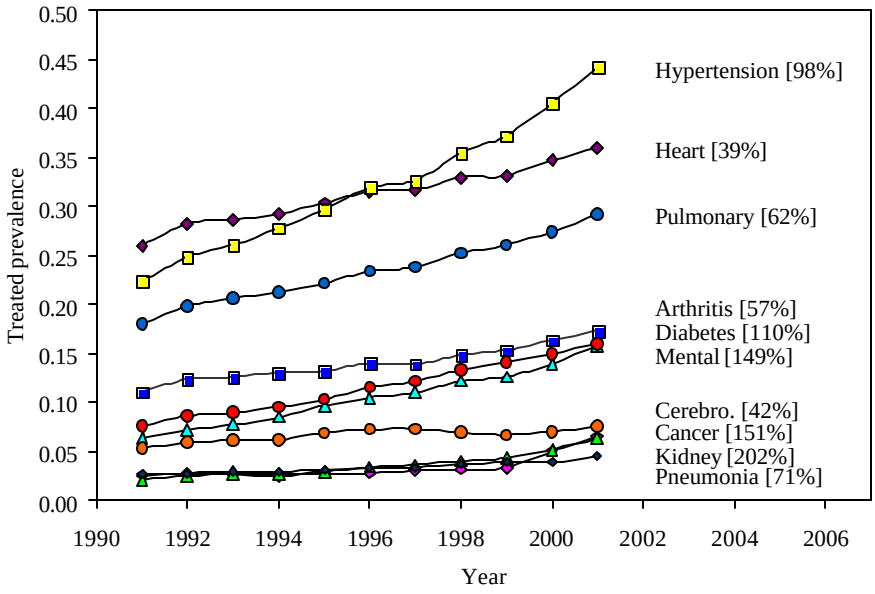
Interestingly, the increase in treated prevalence for most conditions appears to be much steeper in the period 1997 to 2002 versus 1987-1997. It is unclear why this is the case. The MEPS was designed to be comparable to the NMES.

To confirm that increases in treated prevalence are not an artifact of changes in NMES/MEPS data collection methods or other data-specific factors, we also examined trends in treatment rates in Medicare claims data. The data consist of a random 5% sample of Medicare beneficiaries residing in one of the nine cities and states that reported data to the Surveillance Epidemiology and End Results cancer tumor registry in 1991. The sample includes beneficiaries with cancer as well as beneficiaries never diagnosed with cancer. We limit the analysis to beneficiaries age 65 and up. The yearly sample size ranges between 109,104 and 116,369 beneficiaries. Conditions are identified based on the diagnosis codes on outpatient, physician office, and inpatient claims. Results are display in Figure 2. Increases in treated prevalence are larger than those from NMES/MEPS.[2] Treated prevalence levels between the Medicare data and NMES/MEPS are not directly comparable because the Medicare sample consists of

[2] The observed pattern is consistent with previous work showing that the number of physician office visits by Medicare beneficiaries rose steeply during the 1990s (Beeuwkes Buntin et al. 2004).

persons 65+ (as opposed to 18+ in NMES/MEPS). Also, Medicare claims contain information on only those services billed to and covered by the Medicare program.

Figure 2: Treated prevalence among Medicare beneficiaries 65+, standardized by age and sex to 2002



Based on the analysis of NMES/MEPS and Medicare claims data, we conclude that the proportion of persons receiving treatment for one or more major medical conditions increase substantially over the 1990s. The fact that similar trends are observed in both datasets should alleviate concerns that increases in treated prevalence are the artifact of dataset-specific changes in survey methodology (in the case of NMES/MEPS) or billing practices (in the case of Medicare). Also, because the Medicare population consists of retirees with stable health insurance coverage, we can discount short run macroeconomic fluctuations and changes in insurance coverage and structure as a factor behind increases in treated prevalence.

We chose to focus on the ten most costly medical conditions because of their economic significance and high baseline prevalence (so there will be enough cases in a population-level database). It's worth noting, however, that treatment rates for many low prevalence conditions are on the rise as well. Two examples are autism and Crohn's disease.

3. IMPLICATIONS FOR HEALTHCARE SPENDING

To better understand the implications of changes in treatment rates for health care costs, we determine the proportion of condition-specific spending attributable to increases in treated prevalence (as opposed to increases in costs per treated case). The change in per capita costs, ΔPCC, from 1987 to 2002 for a condition can be written as:

$$\Delta PCC = \Delta CPC \times TP_{87} + \Delta TP \times CPC_{87} + \Delta CPC \times \Delta TP, \qquad [1]$$

where CPC is the change in costs per case and TP represents treated prevalence. One method of determining how much of the increase in per capita costs is attributable to the increase in treated prevalence is to set the change in costs per case equal to zero ($\Delta CPC = 0$) and compute the counterfactual increase in costs based on the change in treated prevalence alone: $\Delta TP \times CPC_{87}$. An alternative, equally-valid method is to set the change in treated prevalence equal to zero ($\Delta TP = 0$), compute the counterfactual increase in costs based on the change in costs per case alone, and take the difference from the actual increase: $\Delta PCC - \Delta CPC \times TP_{87}$. The second method will produce higher estimates than the first because the seconde includes the final term in [1]: $\Delta CPC \times \Delta TP$. We compute estimates using both methods, treating the first as a lower bound and the second as an upper bound.

We use the 1987 NMES and 2002 MEPS data for this analysis. NMES/MEPS records actual annual health care spending for each respondent for all sources of payment (for example, out-of-pocket, employer sponsored insurance, public insurance). We inflated spending levels in the 1987 NMES to 2002 dollars using the GDP deflator. We restrict the analysis to non-elderly adults (age 18+). Because many adults have multiple conditions, we cannot simple take the mean spending level of persons with a specific condition in 1987 and 2002 and call the difference the change in costs per case. Instead, we use ordinary least squares regression to estimate annual health care spending as a function of categorical age dummy variables (10 year age groups starting with 25-34), sex, year (2002 versus 1987), condition-specific dummy variables, and

interactions between the condition variables and year. The coefficients on the condition variables yield the cost per treated case in 1987 and the sum of the coefficients on the condition variables and condition-year interactions yield the cost per treated case in 2002.

Results are displayed in Table 1. The first set of columns displays estimates of the cost per case in 1987 and 2002 (in 2002 dollars) from the ordinary least squares regression. All of the coefficients on the condition variables and condition-year interactions were significant at the 5% level with the exception of the the level coefficient on high blood pressure and the condition-year interaction coefficient for cerebrovascular disease. Increases in costs per case as a percent of 1987 levels range between 70% and 140% for five of the conditions. Increases for three conditions fall below this range and increases for the remaining two are above it.

Table 1: The contribution of increases in treated prevalence to increases incondition-specific per capita spending

	Costs per case ($)			Treated prevalence			Per capita spending ($)			Prevalence-attributable
Condition	1987	2002	Change	1987	2002	Change	1987	2002	Change	increase
Heart disease	2,781	4,720	1,939	0.081	0.089	0.008	225	421	197	12% - 20%
Cancer	3,001	5,130	2,129	0.038	0.050	0.012	114	255	141	25% - 42%
Mental disorder	1,255	2,671	1,416	0.051	0.125	0.074	64	333	269	34% - 73%
Pulmonary conditions	559	1,340	781	0.099	0.172	0.073	55	231	176	23% - 56%
Diabetes	2,107	2,658	552	0.040	0.064	0.024	84	171	87	59% - 75%
Hypertension	48	958	910	0.132	0.172	0.040	6	164	158	1% - 24%
Cerebrovascular disease	5,491	6,567	1,076	0.006	0.011	0.006	31	74	43	72% - 86%
Arthritis	624	2,375	1,751	0.073	0.106	0.034	45	252	207	10% - 39%
Pneumonia	3,260	4,794	1,535	0.014	0.014	0.000	46	69	23	3% - 4%
Kidney disease	4,050	7,231	3,181	0.009	0.016	0.007	36	115	79	36% - 65%

All costs are in real 2002 dollars.
Treated prevlaence estimates are standardized by age and sex.

The second set of columns shows treated prevalence levels in 1987 and 2002 from NMES/MEPS, standardized based on age and sex. Increases as a percent of 1987 levels range between 1% for pneumonia to 145% for mental disorders. The third set of columns shows per capita spending by condition in 1987 and 2002. These figures were

calculated by multiplying costs per case by treated prevalence in each year. Increases as a percent of 1987 levels range from 49% for pneumonia to 2,499% for hypertension.

The final column displays the range of the total increase in per capita costs attributable to the increase in treated prevalence (i.e., $\Delta TP \times CPC_{87}$ to $\Delta PCC - \Delta CPC \times TP_{87}$). For five of the ten conditions, the interval includes 50%. For seven of the ten conditions, the interval includes 33%. Increases in treated prevalence appear to contribute the most to increases in per capita spending for mental disorders, diabetes, and cerebrovascular disease, and the least to pneumonia, hypertension, and heart disease. In many cases the intervals are wide, reflecting the contribution of the third term in [1], $\Delta CPC \times \Delta TP$, to the change in per capita costs.

To determine the sensitivity of results to the specification of the model used to determine costs per case, we also estimated a more standard two part regression model, using a generalized linear model with a log link and gamma variance specification for the second stage (Manning and Mullahy 2001). Using the estimated coefficients, we predicted spending levels for the entire sample for each condition to compute costs per case in each year. The ranges of the treated prevalence-attributable increases in per capita spending were larger in most cases than those based on the ordinary-least squares regression. Seven of the ten ranges included 50%. Yet another method of calculating costs per case – estimating condition specific spending based on the diagnosis codes associated with specific health care events – yields qualitatively similar results (Thorpe et al. 2005).

To sum up, estimates of the proportion of the increase in spending attributable to increases in treated prevalence vary somewhat based on specification. Whether the exact contribution is 25% or 50% or even 75% is not particularly important. The main point to take away from this analysis is that increases in treated prevalence are responsible for a non-trivial share of increases in inflation-adjusted per capita spending for the ten most costly medical conditions. If treated prevalence were constant, total spending would still be increasing, but by a much lower amount.

One caveat to this analysis is that our assumption that increases in costs per case and increases in treated prevalence are independent is probably incorrect. We argue below that the composition of patients treated for a particular condition has changed

over time. In particular, patients with milder, and potentially less costly, cases of disease are more likely to receive treatment, implying that changes in treated prevalence influence changes in costs per case. By the same token, treated prevalence may be related to costs per case in the sense that improvements in medical technology, which are costly, lead more patients to seek care. For some conditions, technological advances have been aimed specifically at patients with mild cases, suggesting that costs per case have risen more rapidly for this group. Without measures of disease severity and the relationship between severity and spending, it is difficult to say how our estimates would change if we took these factors into account.

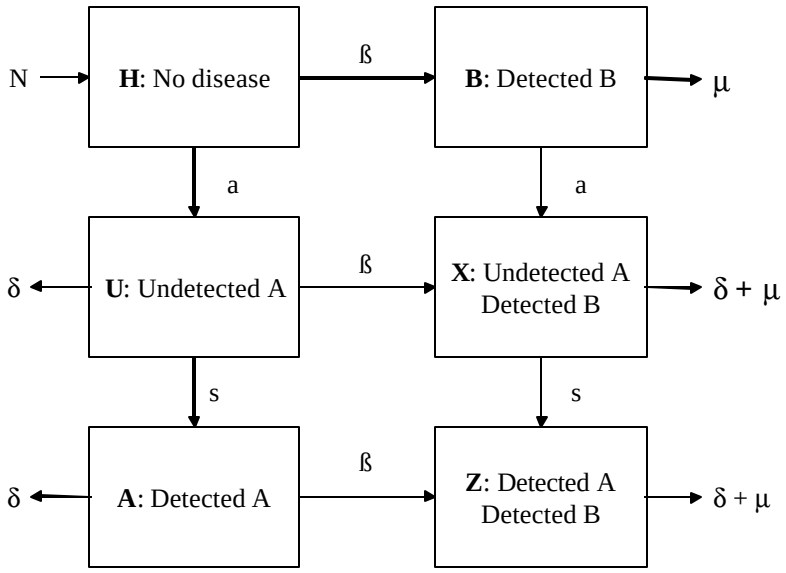
We have not attempted to project the impact of treated prevalence on lifetime spending, as others have done (Joyce et al. 2005). The impact will depend to some extent on the factors behind the increase in treated prevalence. For example, if treated prevalence is increasing because persons with chronic conditions are treated earlier in the course of their illness, then lifetime costs will increase as these persons incur higher costs from the date of diagnosis onward. The remainder of the paper focuses on trying to understand why treated prevalence increased so rapidly during the 1990s.

4. THE EPIDEMIOLOGY OF TREATED PREVALENCE

4.1 A model of treated prevalence

Before discussing behaviorally- and sociologically-based explanations for increases in treated disease prevalence, a mechanistic, epidemiological model of treated prevalence is useful for building intuition and identifying factors that could be responsible for observed increases in treated disease prevalence. The model is depicted graphically in Figure 3. Each box represents a segment of the population, or a state, and arrows represent transitions between population groups.

Figure 3: A model of treated prevalence



This model is meant to capture the dynamics of a chronic disease for which there is no cure (for example, diabetes) or a disease for which there is a long latent, symptom-free period (for example, prostate cancer). It is not representative of episodic diseases (for example, cerebrovascular disease) or infectious diseases (for example, pneumonia). Nevertheless, we believe it serves as a useful starting point for discussing the factors responsible for increases in treated prevalence.

There are six states and two diseases, A and B. Disease A is of primary interest, disease B represents a "competing risk" of death. Persons in states U and UD have disease A, but it is asymptomatic and undetected. The disease is detected, either via screening or once symptoms develop, at rate s. For simplicity, we assume that disease B, which develops at rate b, is always symptomatic is thus always detected. Disease specific mortality rates are respresented by d and m. N represents the number of births into the no disease, healthy state, H. In equation form, the model is described by six equations, one for each state.

$$
\begin{aligned}
\dot{H} &= N - (a + b)H \\
\dot{B} &= bH - (a + m)B \\
\dot{U} &= aH - (s + b + d)U \\
\dot{A} &= sU - (b + d)A \\
\dot{X} &= aB + bU - (s + d + m)X \\
\dot{Z} &= bA + sX - (d + m)Z
\end{aligned}
\qquad [2]
$$

The right hand size of each equation describes the period-to-period change in the population of the state.

The size of the population with treated and detected disease A is A + Z and treated prevalence is this quantity divided by the total population size

$$TP = \frac{A + Z}{H + U + A + B + X + Z} \qquad [3]$$

The equilibrium, steady state values of the H,…,Z are characterized by the following conditions: $H_{t+1} = H_t$, $U_{t+1} = U_t$, and so on. We calculate the steady state value of treated prevalence by plugging the equilibrium values into equation 3: $TP = TP(a, b, s, d, m)$. We can use this expression to identify the possible factors responsible for increases in treated disease prevalence: $TP_a > 0$, $TP_b > 0$, $TP_s > 0$, $TP_d < 0$, $TP_m < 0$. (Note that treated prevalence does not depend on N: $TP_N = 0$). Treated prevalence will increase if the true incidence rates of the disease or the competing risk increase (a and b), if the detection rate increases (s), or if the cause-specific or competing risks of death decline (d and m). We address each of these below.

4.2 Changes in risk factors

Increases in incidence rates will reflect changes in the risk factors underlying each condition. Given that treated prevalence has increased by more than 30 percent for eight of the ten conditions we examine, it is unlikely that simultaneous changes in idiosyncratic, condition-specific risk factors can account for increases in treated prevalence. To the extent that changes in incidence rates underlie the increase in treated prevalence, it will be because there has been a change in a risk factor that is common to multiple conditions. Obesity immediately comes to mind. Obesity has been linked in the medical literature to increased rates of diabetes, heart disease, arthritis, kidney disease, and cancer. According to NMES/MEPS, the proportion of the adult population considered overweight based on respondent self-report has increased from 27.3% to 34.4% and the proportion considered obese has increased from 11.7% to 23.5%.

To gauge the impact of changes in obesity levels, as defined by body mass index, and other observable, individual characteristics on treated prevalence, we used the NMES/MEPS data to estimate a probit regression for each condition where the dependent variable is equal to one if the individual is receiving treatment for the condition and independent variables include age group (18-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+), sex, race (Hispanic, black, other), census region, educational attainment (less than 12 years, high school graduate, some college, college graduate), smoking status (current smoker versus not a current smoker), and body mass index group (<18.5, 18.5-24.9, 25-29.9, 30-34.9, >35). Persons with a body mass index in excess of 30 are considered obese and persons with a body mass index in excess of 35 are considered severely obese. For the sake of comparison, we also estimated a model where the independent variables include age and sex only. We estimated separate regressions for the 1987 NMES and 2002 MEPS data, and we applied the Blinder-Oaxaca decomposition, modified for non-linear models (Fairlie 2003), to the parameter estimates to calculate the portion of the total change in treated prevalence that is explained by changes in observable characteristics. We used the coefficient estimates from 2002 MEPS data to calculate the decomposition. Results based on coefficients from the 1987 NMES data were qualitatively similar. As above, all estimates are

weighted so that they are representative of the U.S. civilian, non-institutionalized population.

Results are display in Table 2. (Coefficient estimates from the probit regressions are available from the authors upon request.) The first three columns display treated prevalence in 1987, treated prevalence in 2002, and the change in treated prevalence. The fourth column shows the marginal effect of severe obesity (body mass index >35) on the likelihood of receiving treatment for the condition. Being severely obese significantly raises the likelihood of receiving treatment for all conditions except cancer and cerebrovascular disease. Being obese increases the likelihood of receiving treatment for seven of the ten conditions (results not shown). Most of these results are consistent with the findings in the medical literature, though it is somewhat puzzling why obesity is not more strongly related to the occurrence of kidney disease. Kidney disease is often precipitated by diabetes, which is strongly linked to obesity.

Table 2: Impact of obesity and other observable characteristics on increases in treated prevalence

	Treated prevalence			Impact of	% of change explained by	
	1987	2002	Change	BMI >35	Age+sex	All variables
Heart disease	0.081	0.089	0.008	0.047 *	89%	87%
Cancer	0.038	0.050	0.012	-0.008 *	28%	32%
Mental disorder	0.051	0.125	0.074	0.085 *	8%	4%
Pulmonary conditions	0.099	0.172	0.073	0.118 *	4%	19%
Diabetes	0.040	0.064	0.024	0.116 *	17%	52%
Hypertension	0.132	0.172	0.040	0.225 *	36%	75%
Cerebrovascular disease	0.006	0.011	0.006	-0.001	22%	<1%
Osteoarthritis	0.073	0.106	0.034	0.097 *	23%	38%
Pneumonia	0.014	0.014	0.000	0.006 *	N/A	N/A
Kidney disease	0.009	0.016	0.007	0.007 *	12%	10%

*p<0.05.
BMI: body mass index

The fifth column shows the proportion of the increases in treated prevalence explained by age and sex, and the sixth and final column shows the proportion

explained by all the observable variables. The proportion of increases in treated prevalence explained by age and sex ranges from 4% to 89%, and the proportion explained by all variables ranges from <1% to 87%. For some conditions, like mental disorders, the proportion of the increase explained by age and sex actually exceeds the proportion explained by all variables. The reason is that there are some factors that are negatively related to the likelihood of having one or more of the conditions that have increased between 1987 and 2002. In the case of mental disorders, Hispanics are 6% less likely to have mental disorders compared to whites and Asians and the proportion of Hispanics has increased from 7% to 12%. Similarly, the likelihood of receiving treatment for cerebrovascular disease is negatively related to educational attainment, and so the 6.2 percentage point increase in the proportion of the population having a college degree is enough to reduce the proportion of the increase (which is very small to begin with) explained from 23% with age and sex to less than 1% with all variables. For four of the conditions – pulmonary conditions, diabetes, hypertension, and osteoarthritis – the proportion of the increase explained by all variables is over 10 percentage points larger than the proportion explained by age and sex alone. Not coincidentally, the marginal effects of obesity on the likelihood of having each of these four conditions are the largest among the ten conditions we examine.

These results must be interpreted cautiously because weight is measured at the time of the survey. Since disease often leads to weight loss, coefficients are biased by endogeneity. Ideally, we would like to have a pre-illness measure of weight, not to mention measures of exercise and caloric consumption. Results are also biased by non-random measurement error. According to data from the National Health and Nutrition Examination Surveys, the actual prevalence of obesity in the 20-74 year old population has increased from 23% in 1988-1994 to 31% in 1999-2000 (Flegal et al. 2002). The comparable figures from the 1987 NMES and 2002 MEPS, which are based on respondent self-report, are 12% and 25%. Thus, weight levels are under-reported, biasing estimates of the marginal impact of obesity on treated prevalence towards zero, but the increase in weight levels over time appears to be somewhat over-stated, biasing estimates of the proportion of the increase in treated prevalence attributable to changes in obesity levels upwards. To correct for self-report bias, we estimated models where

body mass index was imputed to study subjects based on the validation data estimates of the relationships between true height and weight and self-reported height and weight (Cawley and Burkhauser 2006).[3] Results were very similar to those from models where body mass index was calculated using only the unadjusted self-report measures.

Data from Gregg et al. (2005) present additional evidence on the degree to which observed increases in treatment rates reflect changes in clinical incidence rates. Gregg et al. used data from the National Health and Nutrition Examination Survey to calculate the proportion of persons meeting the clinical criteria for high cholesterol, hypertension, and diabetes (the survey includes extensive physiological and laboratory measurements of subjects) and the proportion of subjects reporting having received treatment for each of the conditions. The sum is a measure of the true prevalence of the condition. The results of their analysis, displayed in Table 3, show that the prevalence of high cholesterol and hypertension have declined over time, even as the proportion of the population receiving prescription drugs for these conditions has increased. For diabetes, the data suggest that both the true prevalence and the treated prevalence have increased (the survey asks respondents whether they have ever been diagnosed with diabetes). The data show, for example, that 5.3% of subjects met the clinical criteria for diabetes or reported having been diagnosed with diabetes in 1976-1980. By 1999-2000, the figure had risen to 8.1%.

[3] The 2002 MEPS includes body mass index for all respondents, but self-reported height and weight is available only for a subsample of respondents. Thus, we report only the full-sample estimates based on the MEPS body mass index variable, which is calculated using self-reported height and weight.

Table 3: Trends in the prevalence and treated prevalence of three conditions

	1960-1962	1971-1975	1976-1980	1988-1994	1999-2000
High cholesterol					
Treated or clinical criteria	0.336	0.282	0.272	0.208	0.225
Medication use				0.030	0.074
Treated/Total				0.144	0.329
High blood pressure					
Treated or clinical criteria	0.322	0.351	0.308	0.208	0.242
Medication use	0.067	0.087	0.113	0.112	0.155
Treated/Total	0.208	0.248	0.367	0.538	0.640
Diabetes					
Diagnosed+undiagnosed			0.053	0.074	0.081
Diagnosed	0.018	0.034	0.035	0.046	0.050
Diagnosed/Total			0.660	0.622	0.617

Source: Gregg et al. JAMA 2005.

The conclusion we draw from the data presented in Tables 2 and 3 is that the "obesity epidemic" is responsible for a sizable share of increases in treated prevalence, especially for conditions clinically linked to obesity like diabetes. However, trends in obesity levels alone are not sufficient to explain increases in treated prevalence.

4.3 Changes in mortality rates

Another factor that could explain increases in treatment prevalence according to the model is a reduction in the mortality rate. If the mortality rate for heart disease declines, then persons with heart disease will live longer and comprise a larger segment of the population. Between 1990 and 2002, life expectancy increased from 75.4 to 77.3, an increase of almost two years, and life expectancy at age 65 increased from 17.2 to 18.2 years, an increase of one and one half years (National Center for Health Statistics 2005). It is difficult to precisely identify the sources of longevity increases, but research suggests that declines in the mortality rate for heart disease have played an important

role (Cutler and Meara 2001). Treated prevalence for a condition will increase in response to declines in either cause-specific mortality or mortality rates for competing risks of death. The role of mortality for competing risks is especially important to consider in light of the fact that many persons have multiple conditions (Thorpe and Howard 2006). So, for example, it is plausible that a reduction in the death rate for heart disease could lead to an increase in the treated prevalence of diabetes.

We can use the model like a Markov model to characterize treated prevalence rates by age: $TP(a) = TP(a \mid a, b, s, d, m)$. A cohort is "born" in state U and transitions between states each period. Treated prevalence for the cohort at age *a* is the proportion of the cohort in states A and Z divided by the number of cohort members surviving until age *a*. The model predicts that if the cause-specific mortality rate for disease A declines, the proportional increase in the treated prevalence of disease A will be positively related to age.

$$\frac{\partial TP(a+1)}{\partial d} > \frac{\partial TP(a)}{\partial d} \qquad [4]$$

The same is true of a decline in the mortality rate for a competing risk of death (disease B). Based on these predictions, we examine increases in treated prevalence by age group to assess the contribution of changes in mortality rates to the growth of treated prevalence. Data from the 1987 NMES and 2002 MEPS, in the first three columns, and 1991 and 2001 SEER-Medicare claims, in the last three columns, are shown in Table 4. The NMES/MEPS data show that for most conditions, treated prevalence has increased at similar rates among the non-elderly and elderly. The SEER-Medicare data suggest that among the elderly, treated prevalence has increased at similar rates among those ages 65-84 and >85. These findings suggest that declines in mortality rates are not responsible for increases in treated prevalence.

Table 4: Annualized growth rates in treated prevalence by age group, standardized by age and sex

Condition	NMES/MEPS 1987-2002			Medicare 1991-2001		
	18-64	65+	All	65-84	85+	All
Heart disease	-0.2%	0.3%	0.1%	3.5%	2.7%	3.3%
Cancer	1.2%	1.5%	1.4%	9.6%	10.0%	9.6%
Mental disorder	5.8%	6.3%	5.9%	10.0%	8.4%	9.5%
Pulmonary conditions	3.9%	2.7%	3.6%	4.9%	5.0%	4.9%
Diabetes	2.9%	2.7%	2.8%	7.8%	6.9%	7.7%
Hypertension	0.8%	1.8%	1.2%	7.0%	7.4%	7.1%
Cerebrovascular disease	7.0%	3.1%	4.2%	4.1%	1.9%	3.6%
Arthritis	2.4%	1.5%	2.0%	4.8%	4.1%	4.6%
Pneumonia	-1.4%	2.3%	-0.2%	5.5%	5.4%	5.5%
Kidney disease	4.2%	2.3%	3.6%	11.6%	12.2%	11.7%

The annualized growth rate is the annual percent increase in treated prevalence.

4.4 Changes in the screening and detection rate

The third and final factor that could lead to increases in treated prevalence, according to the model, is an increase in the screening and detection rate, s. The model predicts that an increase in s will lead to a proportional increase in treated prevalence by age group. This prediction is consistent with the results in Table 4.

We can also find evidence on the degree to which changes in the detection rate have led to changes in treated prevalence by examining trends in health status by condition. If detection rates have increased substantially, we would expect that incident cases (i.e. newly treated patients) have less severe cases of disease today than in the past. This trend may show up in prevalent cases as an improvement in the average health status of patients receiving treatment for a condition. We examine trends in self-reported health status using the 1997 and 2002 MEPS. Self-reported health status, a widely used measure of health, is measured using a five item Likert scale.[4] For the entire sample and each condition, we estimate probit regressions of the likelihood of

[4] The 1987 NMES survey also measured health status, but on a four point scale, so it is difficult to compare responses with MEPS data.

reporting fair or poor health status as a function of year (2002 versus 1997), age group, and sex. Table 5 reports sample means by year and by condition (which are not age or sex adjusted) and the marginal effect of year from the probit regressions. The results show that self-reported health status has not changed between 1997 and 2002 in the general population (13% in both years), but there have been fairly sizable reductions (>2 percentage points) in the likelihood of reporting fair or poor health status for eight of the ten conditions. Similar findings are reported by Freedman and Martin, who found that while the prevalence of self-reported conditions among the elderly has increased uniformly, persons with conditions were less likely to report functional limitations in 1995 compared to 1984 (Freedman and Martin 2000).[5]

Table 5: Changes in self-reported health status by condition

	% with fair or poor SRHS		
	1997	2002	Change
Population	0.13	0.13	0.00
Heart disease	0.38	0.36	-0.02
Cancer	0.28	0.26	-0.01
Mental disorder	0.31	0.32	0.01
Pulmonary conditions	0.24	0.22	-0.03 *
Diabetes	0.46	0.43	-0.03
Hypertension	0.29	0.27	-0.02 *
Cerebrovascular disease	0.57	0.48	-0.10 *
Arthritis	0.35	0.32	-0.04 *
Pneumonia	0.41	0.34	-0.05
Kidney disease	0.43	0.38	-0.04

*$p<0.05$.

These results are consistent with the hypothesis that there has been a change in the types of patients treated over time. In particular, patients with less severe cases are

5 There is some evidence, however, that disability rates are rising among the non-elderly population (Lakdawalla et al. 2004; Autor and Duggan 2003), perhaps due to an increase in obesity rates.

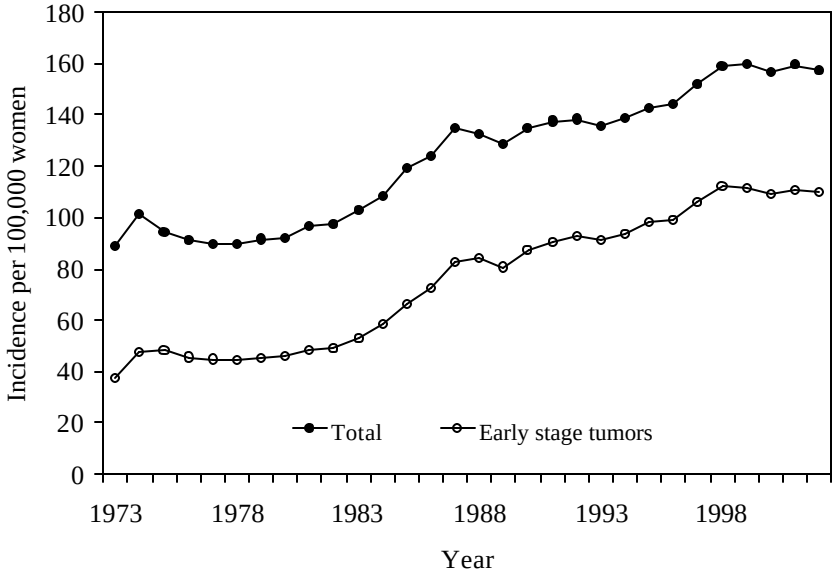
more likely to receive treatment. Results are also consistent with an alternative explanation: improvements in treatment technology have reduced the burden of disease. Interestingly, there has been a large improvement in self-reported health status among patients with pneumonia, even through treated prevalence is unchanged and there have been no major identifiable changes in treatment technology. Bear in mind that many persons have multiple conditions and self-reported health status is a summary measure of health. Changes in self-reported health status for persons with pneumonia may reflect improvements in the treatment of heart disease or other prevalent conditions.

To document the shift towards earlier diagnosis, we would ideally like to have measures of health status for newly diagnosed patients (i.e. incident cases) prior to treatment. Unfortunately, there is no comprehensive data source that records such information consistently across diseases and across time. Instead, there are several disease-specific registries that collect data on new cases. These are the Surveillance Epidemiology and End Results (SEER) registry for cancer and the United States Renal Data Registry (USRDS) for kidney failure. SEER collects data on all patients newly diagnosed with cancer in large regions of the U.S. (including Atlanta, GA; Los Angeles, CA; Connecticut, Utah). We present data on women newly diagnosed with breast cancer for the years 1972 to 2002. We use cancer stage as a measure of health status and disease severity at diagnosis. Early stage tumors are confined to the breast tissue and are usually detected via screening. Most women with early stage breast cancer are not symptomatic. Late stage tumors, by contrast, are usually diagnosed symptomatically.

Figure 4 shows trends in total incidence and the incidence of early stage tumors. Over the period 1973 to 2002, the incidence of breast cancer in women increased from less than 100 per 100,000 women to over 150 per 100,000 women. The increase in the incidence of early stage tumors mirrors the increase in the overall incidence rate. These data suggest that changes in screening and detection practices have played a role in the rise of treated breast cancer prevalence and the rise of the treated prevalence for cancer overall (trends are similar for other types of tumors like colon and prostate). The fact that the increase in the incidence of early stage tumors has not been accompanied by a decline in the incidence of late stage tumors suggests that there is substantial

overdetection; women are being diagnosed with early stage tumors that would never have become clinically manifest if left untreated (Berry et al. 2005).

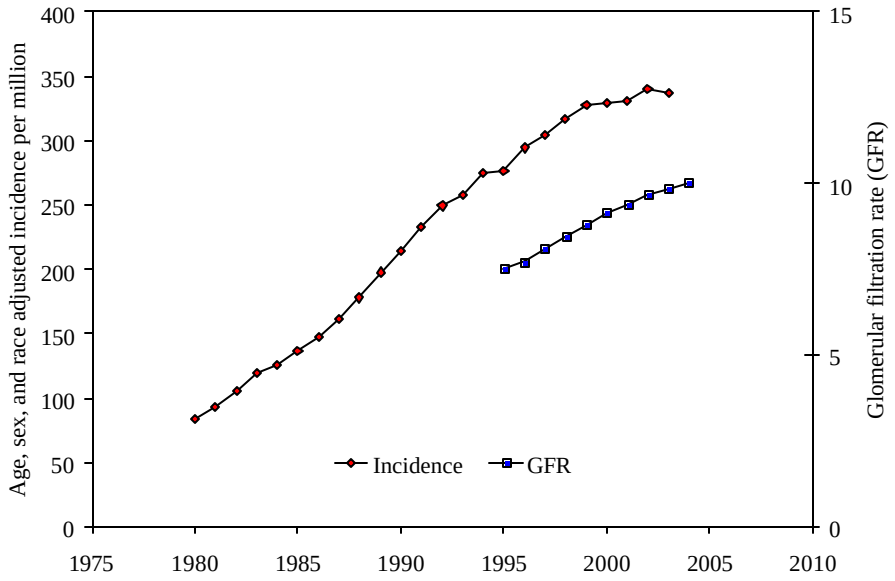
Figure 4: Trends in breast cancer incidence



Early stage tumors are tumors classified as "In situ" or "Localized" using the SEER historic stage A.

The USRDS registry records data on patients newly diagnosed with kidney failure prior to the initiation of dialysis. Kidney function at diagnosis can be measured using the glomerular filtration rate. Figure 5 shows trends in the overall incidence of kidney failure, on the primary y-axis, and the glomerular filtration rate at diagnosis, on the secondary y-axis. Between 1980 and 2004 the incidence rate increased from 100 per 1,000,000 persons to over 300 per 1,000,000 persons. The glomerular filtration rate at diagnosis increased by one third over the same period. While these data do not permit us to determine how much of the increase in the incidence rate is due to better detection versus clinical factors, they suggest that changes in detection patterns have played an important role.

Figure 5: Kidney failure indicence and kidney function at diagnosis



5. EXPLAINING CHANGING DETECTION PATTERNS

The evidence presented in the previous section suggests that treated prevalence is increasing partly because of early detection and treatment of pre-symptomatic and mildly symptomatic cases. In this section, we discuss four explanations for this shift in practice patterns: development of new treatment technology, development of new diagnostic technology, pharmaceutical company marketing practices, and "medicalization". These explanations are interrelated and are not mutually exclusive. For example, the development of new treatment technology will spur investment into diagnostic technology and vice versa.

5.1 New treatment technology

Most of the economic literature on rising medical costs assigns a central role to new treatment technology. Treatment technology leads to treatment expansion when the technology increases the benefits or reduces the costs of treating patients with mild

cases of disease. In terms of the model, the effectiveness of treatment is positively related to s. In the case of life-extending technology, effectiveness is positively related to both s and d .

In some instances new technology offers differential benefits to patients with less severe cases. An example would be breast conserving surgery for early stage breast cancer. In other cases new technology offers a uniform benefit for mild and severe cases alike, but the improvement is enough to induce patients with mild, previously untreated cases to seek care.

The development of selective serotonin re-uptake inhibitors (SSRIs) for depression is one of the most well-documented examples of technology-driven treatment expansion. Prior to the advent of selective serotonin re-uptake inhibitors, treatment options were limited, and the prescription drugs that were available had undesirable side effects. The most common treatment modality was psychotherapy, which entailed a significant time cost for patients. The introduction of an easy-to-use, effective drug greatly reduced both the monetary cost and time cost associated with treatment. The introduction of selective serotonin re-uptake inhibitors also solidified the idea that depression was at least in part biologically based, reducing the stigma associated with seeking care and increasing public support for parity mandates to equalize coverage of mental and physical illness.

SSRIs offered new opportunities for treating such cases because side effects were infrequent and mild and efficacy was high. The diffusion of SSRIs was facilitated by ease with which they could be prescribed by primary care physicians. The ability to treat depression in the primary care setting reduced barriers to treatment among patients with limited financial resources and patients who lacked the skill to navigate the health care system to access specialty care.

In the case of depression and some other conditions, such as gallstones, there is a direct link between the development of new technologies and recent increases in treated prevalence (Legoretta et al. 1993). For other conditions, however, treated prevalence has increased in the absence of major technological breakthroughs. Examples would include early stage prostate, breast, and colorectal tumors, asthma, diabetes, and kidney disease. It's not that treatment technology for these conditions is

static – treatment for early stage breast and prostate tumors is associated with fewer side effects today than in the past – but it is difficult to isolate major, discrete technological improvements in technology. It seems as if the forces driving treatment expansion in these cases are operating independently of changes in treatment technology.

5.2 Changes in diagnostic technology

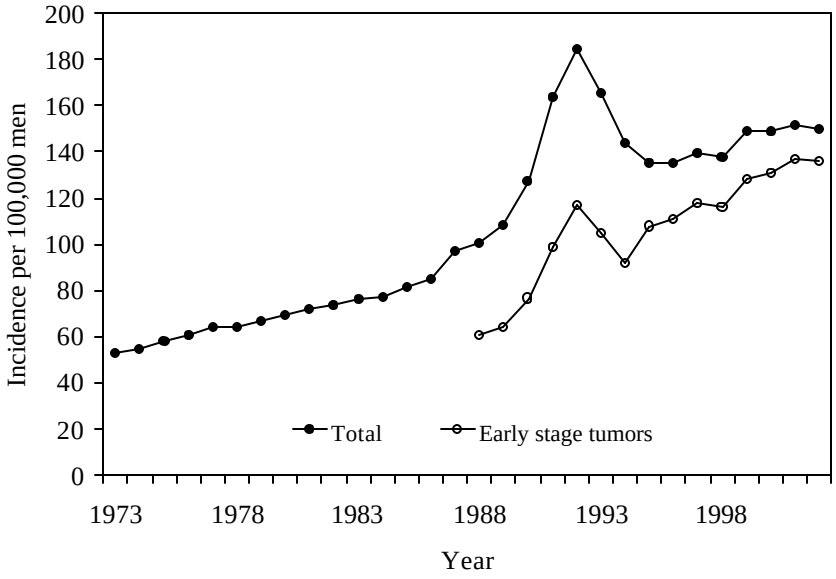
Of the ten conditions we examine, three (heart disease, cancer, and high blood pressure) are often diagnosed asymptomatically and three (COPD, diabetes, and kidney failure) are often diagnosed in mildly symptomatic forms. The treated prevalence of these conditions will be sensitive to changes in screening and diagnostic technology. In some cases new diagnostic technology is more accurate than existing technologies, in other cases new diagnostic technology is easier to use, less costly, or less invasive.

The case of the prostate specific antigen (PSA) test for prostate cancer illustrates how treated prevalence responds to new diagnostic technology. Prior to the late 1980s, men were screened for prostate cancer using only the digital rectal exam. Sometime around 1988, physicians began to use the PSA test, often in conjunction with the digital rectal exam, to detect early state prostate tumors. Because the PSA test is a blood test, it is associated with less patient discomfort than the digital rectal exam. It is also has a much higher true positive rate. Unlike the digital rectal exam, which enables physicians to detect only larger, "palpable" tumors via touch, the PSA permits detection of tumors that are only a few millimeters in diameter. Both features of the PSA test contributed to an increase in the treated prevalence of prostate cancer. Because of its ease of use, the PSA test was used on previously unscreened patients and, because of its high sensitivity, the PSA test detected tumors in patients who were previously classified as cancer-free using the digital rectal exam.

Figure 6 shows trends in total prostate cancer incidence rates and the incidence of early stage tumors. The introduction of PSA testing shows up as a sharp spike in the incidence rate from 1988 to 1993. After 1993, incidence rates stabilized at about 194% of the pre-PSA level. While treatment technology for early stage prostate cancer has

improved over this timeframe, the changes have been gradual. As in the case of breast cancer, early stage tumors account for all of the increase in total incidence rates (note that tumor staging data are not available pre-1988).

Figure 6: Trends in prostate cancer incidence



5.3 Pharmaceutical company marketing practices

A growing body of work, much of it highly polemic, blames pharmaceutical industry marketing practices for recent increases in treated prevalence (for example, Critser's (2005) "Generation Rx. How Prescription Drugs Are Altering American Lives", Moynihan and Cassel's (2005) "Selling Sickness: How the World's Biggest Pharmaceutical Companies Are Turning Us All into Patients"). Mild mental disorders such as attention deficit hyperactivity disorders and "lifestyle" conditions like erectile dysfunction are often cited as examples of diseases that have been "marketed". Much of the attention has focused on direct-to-consumer advertising, which urges consumers to "talk to your doctor" about signs and symptoms, but this is only one of several avenues by which drug companies can increase demand. Even before the rules governing direct-to-consumer advertising were relaxed in 1997, drug companies promoted aggressive

treatment of target conditions by encouraging physicians to screen for specific conditions, developing tools and technologies to facilitate diagnosis, and donating money to patient advocacy groups to promote "awareness".

The use of marketing techniques by drug companies to encourage detection and diagnosis is consistent with economic theories about the relationship between market power and firms' incentives to engage in market-expanding advertising (Grossman and Shaprio 1984; Iizuka 2004). Yet, it seems overly simplistic to attribute the rise in treated prevalence to marketing alone. To achieve the market power necessary to justify market-expanding advertising, a company must have a product that is truly innovative. (Having a patient is necessary but not sufficient to achieve market power, since on-patent drugs must compete with existing medications and non-pharmacological interventions.) Thus, the literature on the contribution of marketing to treatment expansion seems to be re-stating the hypothesis that technological innovation drives treatment expansion, albeit with strong claims about the welfare implications of treatment expansion. Of note, Iizuka (2004) finds that expenditures on direct-to-consumer advertising are positively related to product quality.

Some commentators carry the argument further and claim that recent increases in treatment expansion are the result of the 1997 decision by the Food and Drug Administration to relax the rules governing direct-to-consumer advertising. Rosenthal et al. (2003) studied the impact of direct-to-consumer advertising on prescription drug spending and found that though the relationship was positive, the post-1997 increase in direct-to-consumer advertising explained less than 20 percent of the observed increase in spending. This finding is consistent with the observation that treatment rates for many of the conditions we examine were on the rise prior to 1997 and treatment rates have increased for conditions like early stage cancer where treatment regimens consist mainly of off-patent drugs and non-pharmacological interventions. Based on this evidence, we conclude that pharmaceutical industry marketing practices are a mechanism by which new technology leads to treatment expansion, but ultimately pharmaceutical industry marketing fails as a stand-alone explanation for why treated prevalence is on the rise.

5.4 Medicalization

The term "medicalization" is used in medical sociology to describe the process by which the medical community defines new conditions and illnesses. Most of the literature focuses on the creation of entirely new categories of disease, as opposed to the expansion of existing conditions, but some authors have begun referring to the shift towards earlier diagnosis as the "medicalization of risk" (Klawiter 2005).

In seeking to explain medicalization, sociologists reject the "technological determinism" that characterizes much of the health economics literature on medical spending (Conrad 2005). Before scientists can invent a treatment for high blood pressure or "pre-diabetes", the medical community must classify these conditions as "diseases" and persons with the conditions must come to view themselves as "patients". Thus, sociologists focus on the social construction of illness by the medical profession, patient interest groups, and industry.

Much of the work on medicalization is descriptive, and the treatment of the dynamic aspects of medicalization often leaves much to be desired. Why are some conditions medicalized and not others? And why are some conditions only being medicalized today when it would have been advantageous to do so in the past? Nevertheless, the medicalization literature provides a unique and valuable perspective on the increase in treatment rates and shows the fallacy of viewing these trends solely in terms of technological innovation.

6. LOOKING AHEAD

Between 1946 and 1958 the Tulane University Medical School conducted examinations of 10,709 healthy adults in a test of "multiphasic screening" (Schenthal 1960). Participants from the community were subjected to a battery of examinations and laboratory tests. The study found that "…practically all (92%) of these subjects, who considered themselves well and declared themselves asymptomatic, had disease or pathological physiology which was amenable to diagnosis and treatment." The ability of even rudimentary (by today's standards) screening program like Tulane's to find

illness in the vast majority of the asymptomatic population should give pause to the notion that increases in chronic disease treatment rates are temporary and self-limiting. An anecdote reported in the New York Times (Kolata 2006) makes the point nicely:

> One day, as Dr. Meador tells it, a doctor-in-training was asked by his professor to define a well person. The resident thought for a moment. A well person, he said, is "someone who has not been completely worked up."

There is a strong push in medicine to take advantage of new diagnostic technologies to diagnosis disease in asymptomatic patients. Many universities are funding initiatives in "Predictive Health", the goal of which is to identify genetic markers of disease. According the website of the Institute for System's Biology (2006) in Seattle:

> The new approach to medicine, based on each individual's genetic makeup, will help us determine the probability of an individual contracting certain diseases, as well as reveal how an individual may respond to various treatments, thereby providing guidance for developing customized therapeutic drugs. Thus another use of the technologies and tools of systems biology will be to develop preventive treatments for individuals, based on their potential health problems, as indicated by their genetic makeup and current blood-protein markers.

Imaging technology is also getting more powerful. While it is still uncommon to use imaging technology for routing screening, there is growing interest in the use of CT scanners for colon cancer screening and GE Healthcare, a leading manufacturer of diagnostic equipment, is devoting resources towards increasing the use of it's scanning equipment in asymptomatic patients: "Our vision for the future is to enable a new 'early health' model of care focused on earlier diagnosis, pre-symptomatic disease detection and disease prevention." In summary, the focus of modern medicine on early detection suggests that, if anything, increases in treatment rates and treatment of asymptomatic cases are likely to accelerate in the near future.

7. CONCLUSION

We show that there have been increases in treated prevalence over the 1990s and that these increases are responsible for a large share of growth in total spending on major medical conditions. Understanding the factors behind the growth of treated disease prevalence is an important prerequisite for assessing the welfare implications of increasing medical expenditures. We identify two such factors. First, the obesity epidemic has led to increases in treatment rates for conditions closely linked to obesity, like diabetes. Second, a large portion of increases in treated prevalence appear to be driven by changes in practice patterns, with a focus on early detection and treatment of patients with mildly symptomatic or asymptomatic cases. The shift towards early detection defies easy explanation. Attributing all of the increase to technological innovation is probably too simplistic. While a complete discussion of the welfare implications of the shift towards early detection is beyond the scope of this article, we caution readers against the view, widely held by non-clinicians (Fuchs 1996; Schwartz et al. 2004), that early detection is always beneficial. Many screening procures are overused (Merenstein et al. 2006; Prochazka et al. 2005) and there is surprisingly little evidence to back many routine screening practices (Malm 1999).

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