CHAPTER THREE

The Economics of Risky Health Behaviors[1]

John Cawley* and Christopher J. Ruhm**

*Department of Policy Analysis and Management and Department of Economics, Cornell University, Ithaca, NY, USA
**Frank Batten School of Leadership and Public Policy, University of Virginia, Charlottesville, VA, USA

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Abstract

Risky health behaviors such as smoking, drinking alcohol, drug use, unprotected sex, and poor diets and sedentary lifestyles (leading to obesity) are a major source of preventable deaths. This chapter overviews the theoretical frameworks for, and empirical evidence on, the economics of risky health behaviors. It describes traditional economic approaches emphasizing utility maximization that, under certain assumptions, result in Pareto-optimal outcomes and a limited role for policy interventions. It also details non-traditional models (e.g. involving hyperbolic time discounting or bounded rationality) that even without market imperfections can result in suboptimal outcomes for which government intervention has greater potential to increase social welfare. The chapter summarizes the literature on the consequences of risky health behaviors for economic outcomes such as medical care costs, educational attainment, employment, wages, and crime. It also reviews the research on policies and strategies with the potential to modify risky health behaviors, such as taxes or subsidies, cash incentives, restrictions on purchase and use, providing information, and restricting advertising. The chapter concludes with suggestions for future research.

Keywords: health behaviors; alcohol; tobacco; smoking; drugs; obesity; diet; food; physical activity; public health; public policy; taxation; subsidies; addiction; externalities; advertising; information; behavioral economics; neuroeconomics; human capital; education; prices; sex; income; time preference; peers; bounded rationality; medical costs; employment; wages; crime; hyperbolic discounting

JEL Codes: I1; I18; I20; D01; D03; H2; D1; D6; D87

1. INTRODUCTION

Health has many determinants, including market goods and services such as medical care, investments of time, and environmental conditions such as air pollution, sanitation and water purity. However, in industrialized countries where morbidity and mortality are primarily related to chronic rather than infectious diseases, health behaviors are particularly important. Such health behaviors are the subject of this chapter and can be broadly construed as any action, or deliberate inaction, by an individual that affects his or her own health or the health of others. This chapter focuses on the specific behaviors—like smoking, drinking, diet, and physical activity—that have strong direct effects on own health. The empirical evidence cited in this chapter is primarily drawn from high-income countries, particularly the United States, so the analysis is particularly relevant for industrialized nations. Previous Handbook chapters have provided detailed discussions of individual health habits such as smoking (Chaloupka and Warner, 2000), alcohol consumption (Cook and Moore, 2000), and prevention (Kenkel, 2000). For the most part, we neither repeat nor update those discussions.[2] Instead, we provide a broad overview of theoretical frameworks and empirical evidence on the economics of health behaviors. In doing so, we examine traditional economics approaches emphasizing utility maximization that, under certain assumptions (e.g. perfect information and no externalities), result in Pareto optimal outcomes and at most a limited role for policy interventions. We also describe a variety of factors (e.g. market imperfections and hyperbolic time discounting) that can result in Pareto suboptimal outcomes in which government intervention has the potential to increase social welfare.

1.1. The Importance of Health Behaviors

An influential study by McGinnis and Foege (1993) estimated that approximately half of the 2.1 million deaths occurring in the United States in 1990 resulted from external modifiable risk factors. Their findings, summarized in the first column of Table 3.1, suggest that health behaviors play a major role. Tobacco use was responsible for almost a fifth of mortality in that year and the combined influence of smoking, diet, physical activity, and alcohol consumption accounted for 38 percent of deaths. Unsafe sexual behaviors, driving and illicit drug use accounted for another 3 percent of deaths. An update to this study (Mokdad et al., 2004, 2005), shown in the second column of Table 3.1, indicated that the situation was relatively similar in 2000, when 36 percent of deaths were related to smoking, diet, physical activity, and alcohol

[2] For more recent, but also generally more selective, reviews of the literature on alcohol consumption, and obesity, see: Cook and Moore (2002), and Cawley (2010).

Table 3.1 US Deaths Related to Modifiable Risk Factors, 1990 and 2000

Cause of Death	1990	2000
Tobacco	400,000 (19%)	435,000 (18%)
Poor diet/physical inactivity	300,000 (14%)	365,000 (15%)
Alcohol consumption	100,000 (5%)	85,000 (4%)
Microbial agents	90,000 (4%)	75,000 (3%)
Toxic agents	60,000 (3%)	55,000 (2%)
Motor vehicles	25,000 (1%)	43,000 (2%)
Fire arms	35,000 (2%)	29,000 (1%)
Sexual behavior	30,000 (1%)	20,000 (1%)
Illicit drug use	20,000 (1%)	17,000 (1%)
All modifiable risks	1,060,000 (50%)	1,159,000 (48%)

Note. Sources: 1990—McGinnis and Foege (1993); 2000—Mokdad et al. (2004, 2005). The estimate of deaths due to poor diet and physical inactivity was revised downward from 400,000 in Mokdad et al. (2004) to 365,000 in Mokdad et al. (2005). All other figures in the Year 2000 column are from Mokdad et al. (2004).

Table 3.2 US Deaths Related to Modifiable Risk Factors, 2005

Cause of Death	2005
Tobacco smoking	467,000
High blood pressure	395,000
Overweight—obesity (high BMI)	216,000
Physical inactivity	191,000
High blood glucose	190,000
High LDL cholesterol	113,000
High dietary salt (sodium)	102,000
Low dietary omega-3 fatty acids	84,000
High dietary trans fatty acids	82,000
Alcohol use	64,000
Low intake of fruits and vegetables	58,000
Low dietary polyunsaturated fatty acids	15,000

Note. Source: Danaei et al. (2009).

consumption and an additional 3 percent of deaths were attributable to unsafe sex, driving, or drug use.

The results of more recent research, examining a different set of risk factors for US mortality in 2005, are shown in Table 3.2 (Danaei et al., 2009). Whereas McGinnis and Foege (1993) and Mokdad et al. (2004, 2005) examine the composite risk factor of poor diet and physical inactivity, Danaei et al. (2009) separately break out the effects of: high BMI (to which they attribute 216,000 annual deaths), physical inactivity (191,000 deaths), high blood glucose (190,000 deaths), high LDL cholesterol (113,000 deaths), high dietary salt (102,000 deaths), low dietary omega-3 fatty acids (84,000 deaths), high dietary trans fatty acids (82,000 deaths), low intake of fruits and vegetables (58,000 deaths), and low dietary polyunsaturated fatty acids (15,000).

Table 3.3 Leading Causes of Death and Disability-Adjusted Life-Years (DALYs) in High Income Countries

Risk Factor	% of Deaths	% of DALYs
Tobacco use	17.9	10.7
High blood pressure	16.8	6.1
Overweight and obesity	8.4	6.5
Physical inactivity	7.7	4.1
High blood glucose	7.0	4.9
High cholesterol	5.8	3.4
Low fruit and vegetable intake	2.5	1.3
Urban outdoor air pollution	2.5	
Alcohol use	1.6	6.7
Occupational risks	1.1	1.5
Illicit drugs		2.1

Note. Source: World Health Organization (2009), Tables 1 and 2. Table shows top ten risk factors contributing to deaths or DALYs for countries with 2004 per capita incomes exceeding $10,066. A blank entry implies that the specified risk factor is not in the top ten. A given death or DALY may be attributed to multiple risk factors and the risk factors may interact with each other (e.g. obesity may cause high blood pressure).

All of these estimates should be interpreted with considerable caution because the sources of most deaths are multifactorial, making it quite difficult to ascertain the independent effect of specific determinants, and because of the difficulty of fully adjusting for potential confounding variables.[3] This uncertainty notwithstanding, modifiable behaviors represent an important determinant of premature death. In addition, mortality is only part of the negative consequences of poor health habits; morbidity must also be considered. For example, obesity is associated with high rates of arthritis, which is chronic and disabling but rarely deadly, and Type II diabetes, which can lead to medical complications such as blindness and amputation of toes or feet (Dixon, 2010). Smoking is similarly linked to a myriad of quality-of-life reducing health problems such as lung cancer, emphysema, and chronic obstructive pulmonary disease (US DHHS, 1990).

The World Health Organization (WHO) has recently examined how modifiable risk factors are related to both mortality and morbidity, as measured by disability-adjusted life years (DALYs) (World Health Organization, 2009). The results for high-income countries (those with 2004 per capita incomes in excess of $10,065), which are summarized in Table 3.3, differ from those presented in Tables 3.1 and 3.2 in that there is no attempt to identify a single (primary) cause of death or disability, nor to account for interactions between them (e.g. smoking may be a cause of hypertension). As a result, many of the risk factors may reflect a combination of health behaviors and medical treatments.

[3] For example, Flegal et al. (2005) calculate that the number of excess deaths associated with clinical weight classifications (relative to the normal weight category of $18.5 \leq BMI < 25$) were 112,000 for obesity ($BMI \geq 30$), negative 86,000 for overweight ($25 \leq BMI < 30$), and 34,000 for underweight ($BMI \leq 18$), which is difficult to reconcile with the Mokdad et al. (2005) estimate that 365,000 deaths are due to poor diet and physical inactivity.

The estimates from the WHO, shown in Table 3.3, rate smoking as the most damaging health behavior, responsible for 18 percent of deaths and 11 percent of DALYs. Excess body weight is third on the list, responsible for 8 percent of deaths and 7 percent of DALYs, and physical inactivity is fourth, responsible for 8 percent of deaths and 4 percent of DALYs. The second, fifth, and sixth ranked risk factors—high blood pressure, blood glucose, and cholesterol—are all affected by health behaviors such as smoking, physical inactivity, and diet. Indeed, only two of the risk factors listed—outdoor air pollution and occupational risks—are unrelated to individual health behaviors.

The WHO analysis underscores the importance of individual health behaviors in modern industrialized economies. This is in contrast to poorer nations, where infectious diseases and environmental risks play a greater role. For example, in countries with 2004 per capita incomes of $825 or less, the top ten risks of death included child underweight, unsafe water/sanitation/hygiene, and indoor smoke from solid fuels (ranked first, fourth, and sixth), all of which are a direct consequence of poverty (poverty could, in turn, affect behaviors). Low incomes are also important for suboptimal breastfeeding (ranked ninth) and limiting the availability of medical treatments that might offset the consequences of unsafe sex, which is the third ranked mortality risk. Tobacco use and physical inactivity play a smaller role in poorer countries—each is involved in around 4 percent of deaths and is ranked as the seventh and eighth risk factors.[4]

1.2. Trends in Health Behaviors

Figures 3.1 and 3.2 depict trends in a variety of health behaviors in the United States, based on information from a variety of sources (detailed in the figure notes).[5] Figure 3.1 illustrates alcohol consumption per capita, the prevalence of current smoking among adults, illicit drug use during the past year by high school seniors, daily calorie intake (separately for males and females), and obesity prevalence.[6] The results are mixed, with a trend toward healthier behaviors on some dimensions but not others. Most importantly, smoking prevalence fell by almost half among adults between 1974 and 2007 (from 37 to 20 percent), while obesity more than doubled (rising from

[4] The risks associated with DALYs appear to be even more directly related to poverty. Child underweight, unsafe water/sanitation/hygiene, unsafe sex, suboptimal breastfeeding, and indoor smoke from solid fuels are the top five risks, with vitamin A and zinc deficiencies ranking sixth and tenth. Rounding out the list are high blood pressure, alcohol use, and high blood glucose (ranked seventh through ninth). Smoking, overweight/obesity, physical inactivity, and illicit drug use are notably absent from this list.

[5] Some databases provide information on health behaviors for a broader set of countries (although completeness and comparability of the data sometimes present challenges. For instance, the *OECD Health Data* (www.oecd.org/health/healthdata) indicates food, alcohol, and tobacco consumption, and rates of overweight and obesity, for most OECD countries. Data on these behaviors, as well as on physical activity, oral health and health risks such as blood pressure, cholesterol, and diabetes, can be obtained from the World Health Organization's *WHO Global Infobase* (https://apps.who.int/infobase/).

[6] Data are unavailable in some years for many of these outcomes, with linear interpolation used to impute these missing values. Unless otherwise noted, obesity is defined throughout as a body mass index (BMI, calculated as weight in kilograms divided by height in meters squared) of 30 or higher.

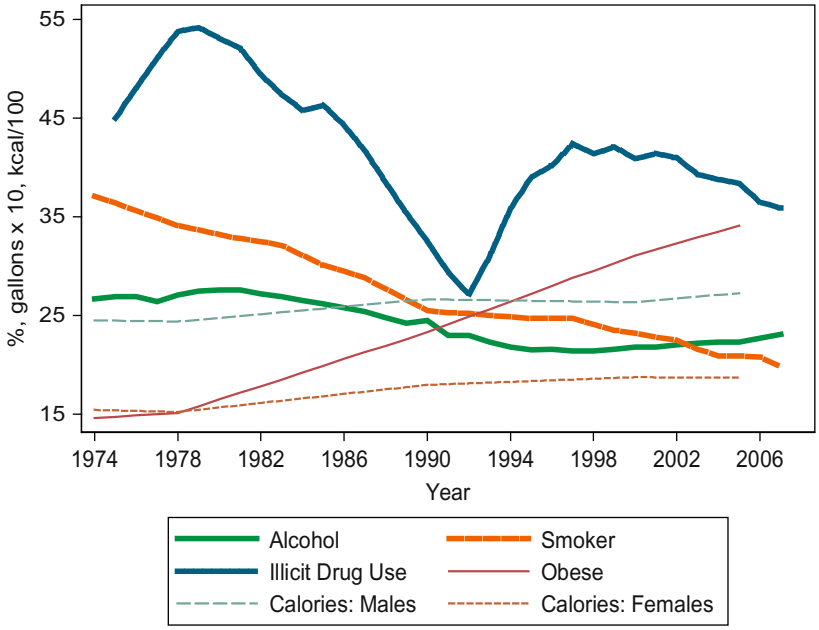

Figure 3.1 Trends in substance use, obesity and energy intake. *Note: Alcohol refers to apparent per capita ethanol consumption (tenths of gallons) for persons aged ≥14 years (source: www.niaaa.nih.gov/Resources/DatabaseResources/QuickFacts/AlcoholSales/consum01.htm). Smoker is the percent of adults who are current smokers (source: National Health Interview Survey, www.cdc.gov/tobacco/data_statistics/tables/trends/cig_smoking/index.htm). Illicit drug use indicates use in last year by 12th graders (source: Johnston et al., 2009). Obesity refers to persons aged ≥20 years with a body mass index ≥30 (source: NCHS, 2010). Calories are average daily energy intake (kcal/100) for persons aged ≥20 years (source: NCHS, 2010). Linear interpolation is used to fill in periods with missing data. The y-axis indicates the percent of the relevant population smoking or who are obese; number of gallons of alcohol consumption × 1/10, and kcal consumed × 1/100.*

15 percent in the early 1970s to 34 percent in 2003–2006). The increase in obesity was fueled by an increase in average daily calorie consumption (12 percent rise for males and 23 percent rise for females). Alcohol consumption per capita declined 20 percent between 1974 and 1997 but has increased modestly (by around 8 percent) since then. The effects of this change are ambiguous because light drinking may yield some health benefits (Gaziano et al., 1993; Thun et al., 1997). However, during 1997–2007 there was either no change or an increase in binge and heavy drinking (National Center for Health Statistics, 2010), which are likely to negatively affect health.[7] Finally, illicit drug use (among high school seniors) shows no clear time trend: it fell

[7] There is no evidence of reductions in heavy drinking (more than 14 drinks per week for males and 7 drinks per week for females) or binge drinking (5 or more drinks on a single occasion).

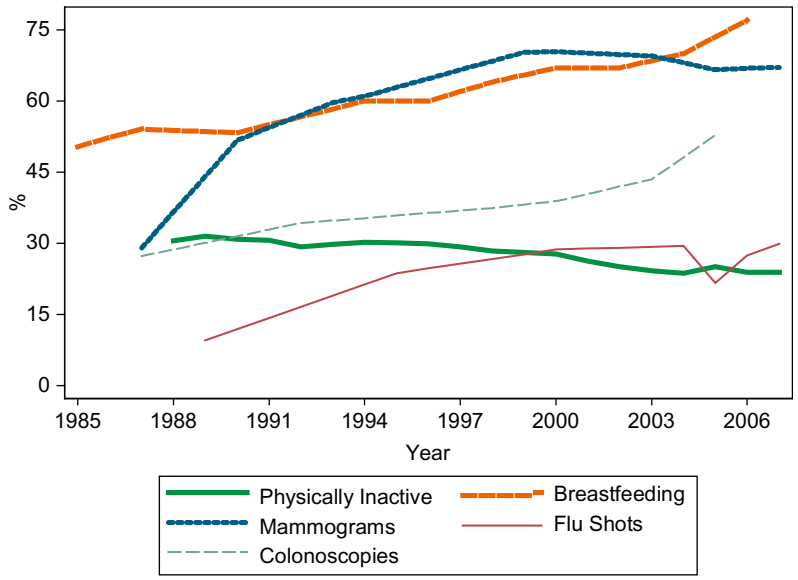

Figure 3.2 Trends in physical activity, breastfeeding and medical screening. *Note: Inactive adults are those reporting no leisure-time physical activity during the last month. Data are for 36 states (source: www.cdc.gov/nccdphp/dnpa/physical/stats/leisure_time.htm). Breastfeeding indicates infants ever breastfed (source: NCHS, 2010; McDowell et al., 2006). Mammograms refer to women aged ≥40 years receiving mammograms in last 2 years. Flu shots refers to percent of adults receiving influenza vaccination in the last 12 months (source: NCHS, 2010). Colonoscopy indicates the percentage of persons aged ≥50 years who have ever had a colonoscopy or sigmoidoscopy (source: http://progressreport.cancer.gov/doc_detail.asp?pid = 0&did = 0&chid = 72&coid = 718&mid = #trends). Linear interpolation is used to fill in periods with missing data.*

sharply from a peak of 54 percent in 1979 to a trough of 27 percent in 1992, before rising rapidly to 42 percent in 1997, after which it declined modestly.[8]

Figure 3.2 depicts trends in health behaviors such as physical inactivity (no leisure-time physical activity during the last month), medical screening tests (mammograms and colorectal endoscopy), vaccinations (flu shots), and breastfeeding. Obviously, these represent only a subset of possible health behaviors that could be considered and their inclusion is illustrative rather than exhaustive. Consistent data on these behaviors is available for a shorter period of time (with the exception of breastfeeding) than those presented in the previous figure; however, they tend to suggest that healthy behaviors are becoming increasingly common over time. Thus, mammography and colonoscopies have both become more widely used over time, as has influenza vaccination and the fraction of

[8] Illicit drugs include: marijuana, LSD, other hallucinogens, cocaine, heroin, other narcotics, amphetamines, barbiturates, or tranquilizers not under a doctor's orders.

infants that are breastfed.[9] There also appears to have been an increase in physical activity, although most adults do not meet recommended levels (Troiano et al., 2008).

Overall, changes in health behaviors since the 1970s (particularly the rapid decline in smoking) have mostly operated in the direction of improving overall health (Cutler et al., 2009); however, many of these beneficial trends ended or slowed by the early or mid-1990s. Moreover, the main exception to this pattern—the rapid and continuing growth in obesity—has important negative consequences. As always, it is difficult to extrapolate from the past to predict the future. Cutler et al. (2009) suggest that mortality risks will increase during the first two decades of the 21st century due to increases in obesity,[10] and Olshansky et al. (2005) raise the possibility that its rise may lead to reductions in life expectancy.[11]

1.3. Differences in Health Behaviors across Population Subgroups

Health-related behaviors differ, sometimes substantially, across population subgroups. Table 3.4 summarizes disparities in health behaviors like smoking, obesity, drinking, physical inactivity, two types of medical screening testing (mammograms and colorectal exams), sexually transmitted disease, and the use of sun protection. The estimates are based on 2008 data from the National Health Interview Survey (NHIS).[12] The subsamples examined are stratified by sex, race/ethnicity, age, education and annual family income; within those categories the averages and prevalences reported are unconditional. Because many of these factors may be correlated (i.e. better educated persons tend to be in families with higher incomes), the disparities observed should not be interpreted as causal. Table 3.5 displays probit estimates of the corresponding predicted subgroup differences after including controls for demographic characteristics.

The two tables reveal fairly similar patterns. Females are more likely than males to engage in certain healthy behaviors (they are less likely to smoke or binge drink, and are more likely to use sunscreen) but are less likely than males to engage in other healthy behaviors (they are less likely to engage in physical activity and are more likely to have sexually transmitted diseases (STDs), although the STD disparity may be due to biological differences in susceptibility rather than differences in behaviors).

Blacks and Hispanics are less likely than non-Hispanic whites to smoke or drink heavily, but are more likely to be obese and physically inactive. Minorities less commonly receive colorectal screening and Hispanic women are less likely to obtain mammograms

[9] The results for breastfeeding are part of a longer-term secular increase. For instance, 30 percent of infants were breastfed (for at least some period of time) in 1974 as compared to 53 percent in 1990 and 77 percent in 2006.

[10] Their estimates of mortality risk are not strictly limited to changes in health behaviors. In particular, they allow for direct effects of education and of blood pressure and cholesterol; the former influences health behaviors, while the latter are affected by them.

[11] However, Flegal et al. (2007) find that most types of mortality risk from obesity have been falling over time.

[12] See http://www.cdc.gov/nchs/nhis.htm for information on the NHIS.

Table 3.4 Percent of Group with Specified Health Behavior or Risk Factor, 2008

Group	Smoker	Obese	Heavy Drinker	Binge Drinker	Physically Inactive	Mammo-gram	Colorectal Screening	STD	Sun-screen
Full sample	20.6	27.4	5.5	22.7	38.2	57.8	42.3	3.0	16.9
Sex									
Male	23.1	27.0	6.2	31.9	36.0	—	43.1	2.0	9.7
Female	18.3	27.8	4.9	14.2	40.3	57.8	41.6	3.9	23.7
Race/ethnicity									
White (non-Hispanic)	22.0	26.2	6.5	25.1	35.2	59.8	45.8	2.6	19.6
Black (non-Hispanic)	21.3	36.1	3.4	14.3	47.4	59.0	37.6	4.9	5.9
Hispanic	15.8	31.3	3.9	21.7	46.9	47.1	25.2	3.4	13.0
Age (years)									
18–34	23.4	22.3	6.4	34.8	31.6	—	—	4.2	15.0
35–54	23.8	30.6	6.1	24.5	36.4	56.5	24.0	1.7	18.8
55–74	16.8	32.5	4.5	10.8	43.9	75.7	59.0	—	18.0
Education									
< High school graduate	27.5	33.3	4.9	15.7	61.9	47.4	31.9	2.3	8.3
High school graduate/GED	27.1	33.5	5.6	20.2	48.9	58.2	40.7	2.7	13.9
Some college	22.7	30.6	5.1	22.8	35.1	58.6	44.6	2.8	18.8
College graduate	8.9	21.4	5.0	22.2	22.0	62.4	48.2	2.7	25.8
Family income									
< $35,000	27.6	29.2	6.0	21.1	50.1	49.9	39.3	4.5	10.9
$35,000– $74,999	21.4	30.4	5.5	23.0	38.6	57.8	42.4	3.0	16.0
≥ $75,000	14.4	24.2	5.9	26.6	24.7	64.5	44.2	2.0	23.3

Note: Data refer to adults from the 2008 National Health Interview Survey and are weighted so as to be nationally representative. The results for education subgroups refer to individuals aged 25 and older. "Smoker" indicates current smoking and "obese" refers to having a body mass index of 30 or higher. "Heavy" drinkers refer to males (females) averaging > 14 (> 7) drinks per week during the last year and "binge" drinking refers to persons consuming 5 or more drinks during a single day at least once in the last year. Persons are considered "physically inactive" if they engaged in vigorous or moderate physical activity or strength training less than once per week. "Mammograms" indicates had a mammogram in the past two years for females aged 30 and higher. "Colorectal screening" indicates ever in lifetime had such screening for persons 40 and older. "STDs" indicate sexually transmitted diseases other than HIV/AIDS during the last five years for 18–49-year-olds. "Sunscreen" indicates always use sunscreen when outside on warm sunny days for more than one hour.

Table 3.5 Conditional correlations with Health Behaviors, 2008

Characteristic	Smoker	Obese	Heavy Drinker	Binge Drinker	Physically Inactive	Mammo-gram	Colorectal Screening	STD	Sun-screen
Female	−0.055	0.021	−0.017	−0.176	0.024	–	−0.011	0.017	0.134
	(0.004)	(0.006)	(0.002)	(0.004)	(0.006)		(0.007)	(0.004)	(0.006)
Black (non-Hispanic)	−0.054	0.121	−0.023	−0.085	0.075	0.066	−0.014	0.017	−0.113
	(0.006)	(0.009)	(0.003)	(0.005)	(0.008)	(0.010)	(0.010)	(0.006)	(0.004)
Hispanic	−0.125	0.041	−0.025	−0.051	0.062	0.033	−0.100	−0.001	−0.015
	(0.005)	(0.009)	(0.003)	(0.006)	(0.008)	(0.011)	(0.011)	(0.005)	(0.006)
Age: 25−34	0.362	0.128	0.069	0.457	−0.162	−0.519	–	0.024	0.039
	(0.014)	(0.013)	(0.012)	(0.017)	(0.010)	(0.009)		(0.003)	(0.010)
Age: 35−54	0.309	0.159	0.061	0.295	−0.110	−0.100	−0.382	–	0.042
	(0.013)	(0.012)	(0.010)	(0.017)	(0.010)	(0.012)	(0.009)		(0.009)
Age: 55−74	0.235	0.185	0.044	0.169	−0.050	0.098	−0.036	–	0.044
	(0.014)	(0.012)	(0.009)	(0.017)	(0.010)	(0.012)	(0.010)		(0.010)
High school graduate/GED	−0.017	−0.001	−0.001	0.021	−0.068	0.076	0.074	0.010	0.055
	(0.007)	(0.009)	(0.004)	(0.008)	(0.008)	(0.011)	(0.011)	(0.007)	(0.010)
Some college	−0.046	−0.012	0.001	0.030	−0.162	0.102	0.124	0.015	0.101
	(0.006)	(0.009)	(0.005)	(0.009)	(0.007)	(0.011)	(0.011)	(0.008)	(0.010)
College graduate	−0.139	−0.087	−0.009	−0.003	−0.223	0.126	0.153	0.020	0.160
	(0.004)	(0.008)	(0.004)	(0.008)	(0.007)	(0.012)	(0.012)	(0.009)	(0.012)
Income: $35,000−$74,999	−0.062	0.005	−0.002	0.009	−0.073	0.087	0.062	−0.011	0.040
	(0.005)	(0.007)	(0.003)	(0.006)	(0.006)	(0.009)	(0.009)	(0.003)	(0.006)
Income: ≥ $75,000	−0.105	−0.040	−0.002	0.028	−0.143	0.132	0.087	−0.019	0.076
	(0.004)	(0.007)	(0.004)	(0.007)	(0.007)	(0.010)	(0.010)	(0.003)	(0.008)
Baseline	0.207	0.296	0.051	0.197	0.405	0.564	0.424	0.032	0.169

Note: The table shows average predicted marginal effects from probit models that control for the specified covariates. Standard errors are in parentheses. Data refer to adults aged 25 and higher from the 2008 National Health Interview Survey. See Table 3.4 for definitions of the dependent variables. The reference group is non-Hispanic white male high school dropouts aged 75 or higher, with family incomes less than $35,000. "Baseline" estimates indicate average predicted values for the full sample, with covariates evaluated at their actual values.

but most of these differences are associated with correlated factors (like education and income) rather than race/ethnicity itself. Smoking and problem drinking tend to decrease with age, while obesity, physical inactivity, and medical screening tend to rise.

By far the strongest results are that higher socioeconomic status (SES), as proxied by educational attainment or family income, is generally correlated with healthier behaviors. For example, compared to high school dropouts, college graduates were 13.9 percentage points less likely to smoke, 8.7 percentage points less likely to be obese, 0.9 percentage points less likely to drink heavily, and 22.3 percentage points less likely to be physically inactive. In addition, they are 12.6 percentage points more likely to receive mammograms, 15.3 percentage points more likely to receive colorectal screening, and 16.0 percentage points more likely to use sunscreen when outside on warm sunny days. The only exception is that the highly educated were 2 percentage points more likely to have had an STD during the last five years. Income also appears to be associated with healthy behaviors, independent of education. Compared to persons with family incomes below $35,000, those with family incomes of at least $75,000 had relatively low rates of smoking, obesity, physical inactivity, and STDs; they also have a high prevalence of medical screening and sunscreen use. The exception to this pattern is that high family income is associated with modestly greater rates of binge drinking.

Previous evidence of healthier behaviors by more advantaged individuals was obtained in the two influential "Whitehall studies" of British civil servants (Marmot et al., 1978, 1991), which documented a strong positive relationship between occupational status, healthy behaviors, and life expectancy.[13] In these, and in many subsequent examinations, drinking is an exception, as it was in the NHIS data discussed above. For example, Adler et al. (1994) provide evidence of a negative association between SES (usually proxied by income or education) and smoking or physical inactivity but a positive correlation of SES with alcohol consumption. Cutler and Lleras-Muney (2010) show that education is positively associated with healthier behavior as regards to smoking, diet and obesity, health knowledge, household safety, medical testing, screening and vaccinations, and the control of high blood pressure and diabetes. Conversely, the highly educated were more likely to have ever used marijuana (but had smoked it less frequently within the last year) and had more often engaged in light (but not heavy) drinking.

Differences in health behaviors are one possible explanation for why socioeconomic status is positively related to health status and life expectancy. Interestingly, the original Whitehall studies, and much research that has followed (e.g. Lynch et al., 1996; Lantz et al., 2001), suggests that behaviors explain only a small fraction of the better health and longer life expectancy experienced by high individuals with high socioeconomic

[13] See Marmot and Wilkinson (2006) for an in-depth discussion of these issues.

status. However, using data from the British Health and Lifestyle Survey, Contoyannis and Jones (2004) and Balia and Jones (2008) potentially resolve this contradiction, showing that accounting for endogeneity in behavioral choices increases the estimated effect of behaviors on health outcomes and reduces the size of the residual SES effects. This occurs because persons with worse latent health tend to adopt healthier behaviors (e.g. someone who is diagnosed with cancer may quit smoking), introducing a negative bias into the predicted effects of health-enhancing behaviors.

Over time, SES-related gradients have widened for most, but not all, health behaviors. Probably most dramatic is the change for tobacco use, where a large gap has emerged over the last four decades because of larger reductions in smoking for more advantaged adults. Kanjilal et al. (2006) present evidence from the National Health and Nutrition Examination Surveys (NHANES) showing that the gap in current smoking between persons with more versus less than a high school education was 11.6 percentage points (33.5 vs. 45.1 percent) in 1971–1974 but had almost doubled to 21.5 points (17.1 vs. 38.6 percent) in 1999–2002; during the same period the smoking differential between the highest and lowest poverty-income-ratio (PIR) quartiles rose from 10.5 (33.5 vs. 44.0 percent) to 23.5 (13.9 vs. 37.4 percent) percentage points. Kenkel (2007) finds that the gap in smoking rates between those with and without a college degree grew from 2 percentage points in 1954 to 15 points in 1999. A substantial portion of the recent differential occurs because the highly educated tobacco users are much more likely than their less educated counterparts to quit smoking. For instance, in 2008, the quit ratio—defined as the percentage of persons who had ever smoked (more than 100 cigarettes) but who were *not* current tobacco users—was 45.7 percent for adults (aged 25 and over) without a high school diploma, compared to 80.7 percent of those with a graduate degree (Dube et al., 2009).

In contrast, most of the available evidence suggests that SES-related differentials in obesity have narrowed over time, as body weight has increased for all groups but at a somewhat faster rate for those with high education or income. Zhang and Wang (2004) find that between 1971–1974 and 1999–2000 the prevalence of obesity rose 16.2 percentage points (from 7.4 to 23.6 percent) for college educated men (aged 20 to 60) and 14.7 percentage points (from 12.0 to 26.7 percent) for those with less than a high school education. Over the same period the education gradient narrowed even more for women: the prevalence of obesity increased 22.6 percentage points (from 7.3 to 29.9 percent) for college educated women versus 12.9 percentage points (from 24.9 to 37.8 percent) for women with less than a high school education. The results of Chang and Lauderdale (2005) are also suggestive of a weakening in the negative relationship between income and obesity between 1971–1974 and 1999–2002, to the extent that income and obesity are now positively correlated for non-Hispanic black males. Among low SES individuals, the prevalence of obesity was already quite high in the early 1970s and so has grown relatively little over time, raising the possibility that differentials in

severe obesity might have continued to widen over time. However, Cutler et al. (2010) find that the combined prevalence of Class 2 obesity (BMI between 35.0 and 39.9) and Class 3 obesity (BMI of 40 or higher) rose between 1971–1974 and 1999–2004 by the same amount for adults with and without college educations: by 8 percentage points for males and 11 percentage points for females in both education groups.

There is weaker evidence on the behaviors that determine body weight: diet and physical activity. Popkin et al. (1996) find improvements in dietary quality between 1965 and 1989–1991, with larger gains for highly educated persons (who had worse diets at the beginning of the period but similar quality diets at the end of it). Casagrande et al. (2007) find that the consumption of fruits and vegetables changed little between 1988–1994 and 1999–2002, with possibly higher levels and slight increases observed for high SES individuals. Conversely, Kant and Graubard (2007) show that income and education-related differentials in the consumption of healthy foods declined between 1971–1975 and 1999–2002, largely due to a decrease among advantaged individuals. All of these findings should be considered preliminary and are not informative about net energy intake, which is of primary interest when one is concerned about obesity.

There is even less evidence on whether and how energy expenditure has changed over time across groups. Simpson et al. (2003) find that the prevalence of walking rose between 1987 and 2000 but without clear differences across education. On the other hand, Brownson et al. (2005) document modest increases in the probability of achieving recommended levels of physical activity for persons with 16 or more years of education but decreases for those with fewer than 12 years of schooling. However, they emphasize that this includes only leisure-time physical activities, and so does not provide information on other sources of energy expenditure (e.g. the strenuousness of work), which has declined over time, quite possibly differentially across groups.[14]

1.4. Health Economics Research on Health Behaviors

The introduction to Volume 1A of the *Handbook of Health Economics* (Culyer and Newhouse, 2000) includes an organizational chart of the field of health economics. Six of the eight categories of research relate to the health care sector and there is no explicit category for economics research on health behaviors; this is presumably included under the vague residual grouping of "what influences health, other than

[14] There have also been important changes in a variety of medical conditions—like hypertension, high blood pressure, and diabetes mellitus—that are determined by an interaction of health behaviors and medical care. Better control of blood pressure and cholesterol, particularly when combined with reductions in smoking, represent significant improvements in cardiovascular risk factors. These health risks have declined over the last three decades of the 20th century for virtually all groups but without clear differences across education or income categories (Kanjilal et al., 2006; Cutler et al., 2010). Conversely, diabetes diagnoses have increased dramatically over time, particularly for those with less education or low incomes (Kanjilal et al., 2006).

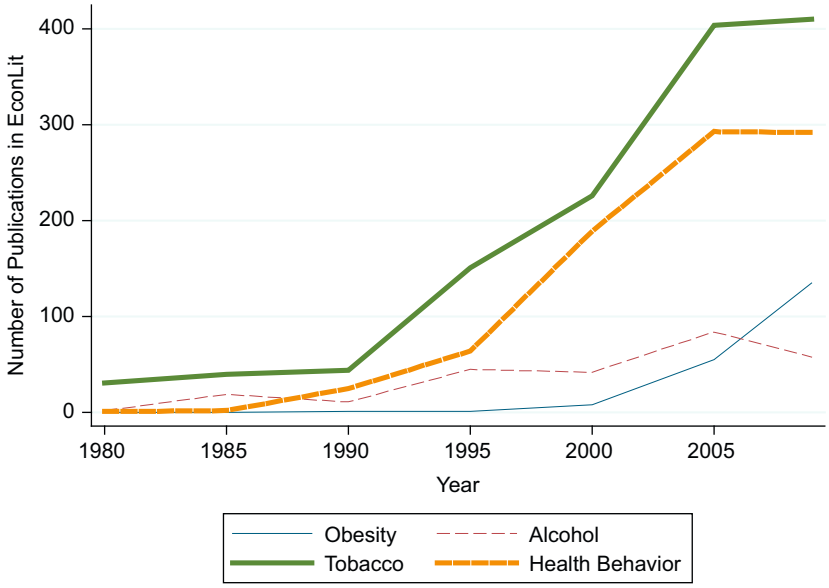

Figure 3.3 Number of economics publications examining health behaviors, 1980–2009. *Notes: Figure is based on year-specific searches of EconLit, a database of journal articles, dissertations, and working papers in economics. Obesity indicates the number of publications with the keyword "overweight" or "obesity." Alcohol indicates the number of publications with the keyword "alcohol" or "drinking." Tobacco indicates the number of publications with the keyword "tobacco," "cigarettes," or "smoking." Health behavior indicates the number of publications with the keyword "health behavior."*

health care?" (see Cawley and Kenkel, 2008). However, 50 percent of US health economists surveyed in 2005 reported studying "the behavior of individuals," which makes this as popular as any sub-specialty of health economics (Morrisey and Cawley, 2008), and economics research on health behaviors has increased considerably in the past three decades.[15] Figure 3.3 depicts the trend in the number of economics publications concerning various health behaviors identified in EconLit, the database of journal articles, dissertations, and working papers in economics. Between 1980 and 2009, the annual number of economics publications on obesity increased from 0 to 135, the number on alcohol rose from 2 to 99, those on tobacco or smoking grew from 31 to 410, and the number on health behaviors generally increased from 2 to 292.[16] These trends are likely due to many factors, including greater awareness of the

[15] While it seems likely that most of the health economists stating that they research "the behavior of individuals" study health behaviors, it is possible that some are referring to non-health behaviors.

[16] The annual number of publications in health care economics also rose substantially, but less than the increase in health behaviors research. For example, the annual number of publications in EconLit with the keyword "hospital" rose from 30 in 1980 to 235 in 2009, and the number with the keyword "medical care" rose from 5 in 1980 to 61 in 2009.

externalities associated with unhealthy behaviors (see, e.g., Manning et al.'s (1991) study on the external costs of smoking, heavy drinking, and sedentary lifestyles), growth in the availability of large secondary datasets allowing researchers to track and model unhealthy behaviors (e.g. the Behavioral Risk Factor Surveillance System (BRFSS) began in 1984 and the Youth Risk Behavior Surveillance System (YRBSS) started in 1991), and rising interest in government action to internalize the negative externalities associated with some health behaviors (e.g. the Federal tax increases on tobacco enacted during the 1990s).

The importance of health behaviors in explaining morbidity and mortality in economically developed countries, increasing richness of data available to study these behaviors, and the policy relevance of the related research questions, makes us optimistic about the future of research on health behaviors. We hope this chapter will be useful to the researchers contributing to that future literature and of broader interest to students and policy makers.

We have chosen not to divide this chapter into sections for smoking, alcohol consumption, drug use, and obesity; instead, the chapter is organized by the underlying economic concepts that relate to all behaviors. It is our hope that this organization will enable researchers to see common patterns and important differences across the various health behaviors, and bring researchers of specific health behaviors out of their separate silos to learn from the synergistic research on other health behaviors.

Interested readers are also referred to the previous Handbook chapters on smoking (Chaloupka and Warner, 2000), alcohol consumption (Cook and Moore, 2000), and prevention (Kenkel, 2000), the chapter on health behaviors among young people in the *Elgar Companion to Health Economics* (Kenkel, 2006), and that on health behaviors and addictions in the *Oxford Handbook of Health Economics* (Kenkel and Sindelar, 2011).

2. THE TRADITIONAL ECONOMIC APPROACH TO STUDYING HEALTH BEHAVIORS

2.1. Model of Health Capital

The foundation for much economics research on health behaviors is Michael Grossman's model of health capital (Grossman, 1972, 2000), which has been detailed in a previous volume in this series (Grossman, 2000).[17] Basic aspects of the model are that people receive an endowment of health capital at birth, which depreciates with

[17] Related empirical work is often inspired more by the intuition of this model than strict adherence to its theoretical features.

age but can be raised through investments; death occurs when the health stock falls below a minimum level. Health has both consumption and investment aspects, as it enters the utility function directly and determines the amount of healthy time available for market and non-market activities. People produce health by combining market goods and services with time, consistent with Becker's model of household production (Becker, 1976). For example, an individual might produce health by buying a treadmill and running shoes and spending time running on the treadmill.

Individuals allocate time and money to maximize the present discounted value of lifetime utility. Indirectly, length of life is a choice in the original model which contains no uncertainty. Specifically, the timing of death results from conscious decisions regarding health investments made with full knowledge of their implications for longevity.[18] Assuming that health has only investment aspects (i.e. it does not enter the utility function directly and is only valuable for producing healthy days), optimal health capital is characterized by an equality of the supply of health capital (i.e. the opportunity cost of health capital) and the demand for health capital (i.e. the marginal monetary return on health investments).

Application of the health capital model to health behaviors such as sleep and exercise is straightforward (see Kenkel, 2000): people invest in such behaviors until, at the margin, the return on investments in health equals the opportunity cost of health capital. However, the model also applies to unhealthy behaviors, which can be interpreted as negative investments in health. When the individual has solved the constrained maximization problem, the optimal participation in unhealthy behaviors will be characterized by an equality of the marginal costs of the unhealthy behavior (both the monetary cost of purchasing market goods like cigarettes and alcohol and the non-pecuniary cost of reduced health and shorter lifespan) and the marginal benefits (such as the instantaneous pleasure derived from consumption).

2.2. Education and Health Behaviors

A literature review by Grossman and Kaestner (1997) concluded that education is the most important correlate of good health for both individuals and groups; in particular, health is more strongly correlated with schooling than with occupation or income. In the model of health capital (Grossman, 1972), schooling may improve health by enhancing allocative efficiency (participation in healthier behaviors) or productive efficiency (obtaining more health from the same set of inputs). Cutler and Lleras-Muney (2010) provide an overview of the differences in health behavior by education, noting that in a parsimonious model estimated using the National Health Interview Survey data, an additional year of education is associated with a 3.0 percentage point lower

[18] However, at high ages, the depreciation rate of health capital may become so large that the individual is unable to afford sufficient investment flows to stay alive.

probability of being a current smoker, 1.4 percentage point lower probability of being obese, a 1.8 percentage point lower probability of being a heavy drinker, and a 0.1 percentage point lower probability of using marijuana in the past month.

Economists have used a variety of identification strategies to measure the causal effect of education on health behaviors. Focusing on the studies that used instruments that are both powerful and plausibly exogenous, the results are mixed. Three studies find evidence of a causal effect of education on smoking. Currie and Moretti (2003) instrument for a woman's education using college openings in her county of residence and find that education reduces the probability of smoking. De Walque (2007b) and Grimard and Parent (2007) exploit college attendance as a draft avoidance strategy during the Vietnam war and both find that college education reduces the probability of smoking by males.

Other studies are unable to reject the null hypothesis of no causal effect of education on health behaviors. Reinhold and Jurges (2009) examine the exogenous variation in education due to the abolition of school fees in Germany and conclude that there is no evidence that education causes reductions in smoking or obesity. Clark and Royer (2010) exploit two changes to British compulsory schooling laws that compelled a large percentage of the student population to stay in school longer. This exogenous variation in education had no detectable effect on smoking, drinking, diet, or exercise.

Other studies find mixed results. For example, Kenkel et al. (2006) exploit education policies (e.g. number of courses required to graduate from high school) as instruments and find that high school completion significantly reduces the probability of current smoking by adult men (but not women), but has no effect on overweight or obesity for either men or women.

2.3. Habit and Addiction

Marshall (1920) may have been the first work in which an economist addressed the phenomenon of habit or addiction; it contains the observation that "...the more good music a man hears, the stronger his taste for it is likely to become..." (Marshall, 1920, p. 94; Stigler and Becker, 1977). In this sense, the individual's utility function includes not just the current consumption of music C but also the stock of past consumption of music S.

In general, there are three characteristics of addiction. *Reinforcement* implies that the marginal utility of current consumption rises with the stock of past consumption ($U_{CS} > 0$). This is called adjacent complementarity because consumption of the good in adjacent time periods is complementary.[19] *Tolerance* implies that the stock of past

[19] Ryder and Heal (1973) also describe the possibility of distant complementarity, which they illustrate with the following example: "a person with distant complementarity who expects to receive a heavy supper would tend to eat a substantial breakfast and a light lunch. A person with adjacent complementarity would tend to eat a light breakfast and a substantial lunch in the same circumstances" (p. 5).

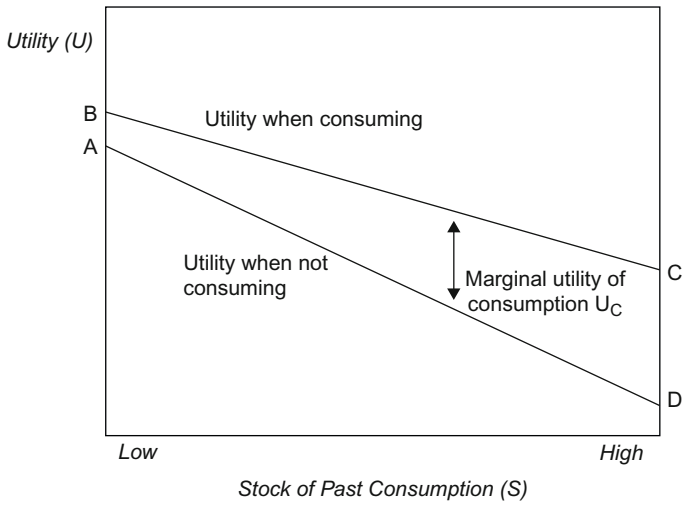

Figure 3.4 Graphical representation of characteristics of addiction. *Notes: Adapted from Rachlin (1997).*

consumption lowers utility ($U_S < 0$). This assumes that the addiction is harmful; one can also have a beneficial addiction (like exercise) in which the stock of past consumption raises utility ($U_S > 0$). Finally, *withdrawal* implies that there is a positive marginal utility of current consumption ($U_C > 0$).[20]

These characteristics of addiction are depicted in Figure 3.4, which is adapted from Rachlin (1997). The vertical axis is utility (U) and the horizontal axis is the stock of past consumption of the habitual or addictive good (S). The lower line AD shows the utility associated with each possible level of the stock of past consumption, conditional on not currently consuming the addictive good. The upper line BC shows the utility associated with each possible level of the stock of past consumption, conditional on currently consuming the addictive product. The graph illustrates withdrawal, as at every stock of past consumption, consuming the addictive good provides higher utility than abstaining: $U_C > 0$. The graph also illustrates tolerance. It depicts a harmful addiction because the stock of past consumption lowers utility: $U_S < 0$. For example, the first time an individual consumes the addictive substance, he has a stock of past consumption of zero, so he is at point B. After the individual has been a heavy user for sufficiently long to have the maximum stock of past consumption, when he consumes he is at point C, which is not only far below the utility he enjoyed during his first use of the addictive substance, it is also below the utility A that he enjoyed when he was still abstinent. Finally, the graph illustrates reinforcement, as the

[20] More complicated (but realistic) models of withdrawal include a kink in the marginal utility function at or near levels of recent previous use, as detailed below.

instantaneous marginal utility derived from consuming the addictive good is greater the higher the stock of past consumption: $U_{CS} > 0$. This illustrates that quitting a habit is harder the higher the stock of past consumption.

Houthakker and Taylor (1970) make the important empirical distinction between habitual (or addictive) goods, for which the stock of past consumption positively affects current use, and durable goods, for which prior consumption is negatively correlated with current use. They note that a good can be durable in the short term and habitual in the long term. For example, even someone who habitually overeats will, in the short term, experience satiation (resulting in a negative correlation in consumption in nearby time periods) but over the longer term, consumption will be positively correlated and thus food is habitual over longer periods of time.

2.3.1. Theory of Rational Addiction (TORA)

A landmark in the study of addiction is Becker and Murphy's (1988) Theory of Rational Addiction (TORA). In this model, addiction is optimal in the sense that it involves forward-looking utility maximization with stable preferences.[21] Previously, models of habit formation or addiction assumed that consumers were naïve: they realized that current consumption of the addictive good depended on its past consumption, but did not take into account the impact of current consumption on future consumption (Pollak, 1975).[22] An appealing aspect of the TORA is that it allows consumers to be sophisticated regarding the intertemporal dynamics of consumption and utility.

The TORA assumes that instantaneous utility depends on current consumption of the addictive good, the stock of past consumption of the addictive good, and current consumption of all other goods. Individuals allocate income to addictive goods and all other goods, taking into account the future consequences of their actions, including tolerance and reinforcement.[23] In the TORA, a person for whom a good is highly addictive (i.e. for whom the good has high adjacent complementarity) might knowingly consume enough to become an addict, because he calculates that by doing so he maximizes the present discounted value of utility. Becker and Murphy describe the model as applicable to a wide spectrum of behaviors, including harmful addictions such as smoking, drinking, gambling, using cocaine or heroin, and overeating, as well as beneficial addictions such as religiosity and jogging.

Formally, the consumer maximizes the present discounted value of lifetime utility by allocating her budget to consumption of an addictive good C and all other (non-addictive) goods Y. Current utility depends not just on instantaneous consumption of

[21] This does not mean that addicts will necessarily be glad to be addicted, a point to which we return below.

[22] For an example of a model of myopic habit formation, see Houthakker and Taylor (1970).

[23] A parsimonious rational addiction model expresses the adverse future consequences of substance use as a higher level of addictive stock, which causes disutility, but one could also incorporate into the model other adverse future consequences, such as probability of marital strife, criminal victimization, or arrest.

C and Y but also on the stock of past consumption of C, which is denoted S. (The stock of past consumption of all other (non-addictive) goods does not enter the utility function.) The lifetime utility function is:

$$U(0) = \int_0^T \mathrm{e}^{-\sigma t} U[Y(t), C(t), S(t)]\mathrm{d}t.$$

where σ is a constant rate of time preference. Reinforcement implies that $U_{CS} > 0$: a higher level of the addictive stock raises the marginal utility of consuming the addictive good. Tolerance implies that $U_S < 0$ for harmful addictions and that $U_S > 0$ for beneficial addictions.

The stock of past consumption S changes over time according to:

$$\dot{S}(t) = S(t) - S(t-1) = C(t) - \delta S(t) - h[D(t)],$$

where C is consumption of the addictive good in period t, δ is the exogenous depreciation rate in the addictive stock, and $D(t)$ represents expenditures on endogenous depreciation or appreciation of the stock. Consumers also face a lifetime budget constraint.

The TORA yields several important implications regarding the responsiveness of consumption to price. First, in almost any model of addiction, consumption at a point in time is related not only to current prices but also to past prices, because the latter determine the current addictive stock. Second, in models where agents exhibit foresight (like TORA but not myopic addiction), current consumption is also related to anticipated future prices because future prices will affect desired future consumption, which is a complement with the future level of the addictive stock; this in turn is affected by current consumption. Third, a future price change will have a greater impact on current demand the sooner it is anticipated, because individuals will then react to it earlier. Fourth, permanent price changes affect demand more than temporary ones, because forward-looking persons anticipate and make decisions based on future dynamics in prices. Fifth, the price elasticity of demand for the addictive good will be greater in the long run than in the short run, and that difference will rise with the level of addictiveness.

Finally, an arguably counter-intuitive implication is that the more addictive the good, the greater the long-run price elasticity of demand (see equation (18) and the related discussion in Becker and Murphy, 1988). The first reason that higher price leads to a reduction in quantity demanded is the usual reason that applies to all goods: the law of demand states that when price rises, the quantity demanded falls. However, for addictive goods there is a second reason that higher price leads to a reduction in quantity demanded: adjacent complementarity. Specifically, a rise in price that is expected to persist implies less consumption in the future, so it becomes optimal to hold a lower quantity of addictive stock, which is achieved by reducing consumption

today. The more addictive the good, the greater the adjacent complementarity, and the greater the extent to which current consumption falls in response to an expected reduction in future consumption. This prediction—that all else equals addiction implies a greater price elasticity—is in stark contrast to early models of addiction that hypothesized that addicts were irrational and therefore unresponsive to incentives (see Cawley, 2008).

2.3.2. Empirical Tests of Rational Addiction

Most papers testing the TORA use the empirical model developed by Chaloupka (1991) that represents a simplified version of the Becker and Murphy (1988) framework. The utility function is assumed to be quadratic, which yields linear first-order conditions and, with additional assumptions, the demand function:

$$C_t = \beta_0 + \beta_1 P_t + \beta_2 P_{t-1} + \beta_3 P_{t+1} + \beta_4 C_{t-1} + \beta_5 C_{t+1} + \varepsilon, \tag{3.1}$$

where P are prices and C are consumption levels of the addictive good in different time periods.[24] If omitted determinants of demand are autocorrelated then lags and leads of consumption will be correlated with the residual of current consumption, and OLS estimates of (3.1) will yield biased estimates of β_4 and β_5. A common strategy for dealing with this problem is to instrument for lagged and future consumption using further lags and leads of prices, under the assumption that any effect on current consumption of prices before $(t-1)$ or after $(t+1)$ must operate through their effects on consumption in $(t-1)$ or $(t+1)$ (Becker et al., 1994; Chaloupka, 1991).

The signs of the coefficients in (3.1) are used to test for addiction and forward-looking behavior. Table 3.6 lists predictions about the signs of the key coefficients implied by alternative hypotheses. Regardless of whether the good is addictive, consumption is always negatively correlated with contemporaneous price, because of the law of demand. The key test for addiction is whether past consumption raises current consumption; this is informative about adjacent complementarity. The key test for whether addiction is farsighted is whether current and future consumption are positively correlated—a rational (forward-looking) addict considers future events when choosing current consumption.

Table 3.6 also shows the possibly surprising prediction of the TORA that past and future prices are positively correlated with current consumption, after controlling for past and future consumption (see the discussion in Chaloupka (1991) concerning equations (3.2) and (3.3)).[25] Because the model already controls for past and future

[24] An interesting feature of this regression model is that the estimated coefficients on past and future prices, and past and future consumption, can be used to calculate the rate of time discount (σ) because $\beta_3 = \beta_2/(1+\sigma)$ and $\beta_5 = \beta_4/(1+\sigma)$.

[25] Gruber and Köszegi (2001) point out that, in many other applications, a positive correlation between future prices and current consumption is interpreted as a failed specification test of the model, not as evidence of forward-looking behavior.

Table 3.6 Testing Models of Addiction

Predicted Sign of Coefficient On:	Non-addictive	Myopic Addiction	Rational Addiction
$P(t)$	−	−	−
$P(t-1)$	0	+	+
$C(t-1)$	0	+	+
$P(t+1)$	0	0	+
$C(t+1)$	0	0	+

Note: Applies to coefficients from the Chaloupka (1991) empirical model of rational addiction.

consumption, for past (or future) prices to be higher holding constant past (or future) consumption, some unobserved correlate of demand must have changed. It is assumed that the change in unobservables persists today, implying higher demand today.[26]

Empirical tests of rational addiction have been conducted for: tobacco (Becker et al., 1991; Chaloupka, 1991), alcohol (Waters and Sloan, 1995), cocaine (Grossman and Chaloupka, 1998), and obesity (Cawley, 1999). Comprehensive reviews are available in Grossman (1993), Chaloupka (1996), Chaloupka and Warner (2000), and Cook and Moore (2000).

There are several challenges beyond those already mentioned to estimating empirical models of rational addiction. First, data on consumption of addictive goods may contain significant reporting error, (see, e.g., Brener et al., 2003). Individuals may fear prosecution if they report consumption of illegal substances, and stigma may lead to underreporting their use, even if legal.[27] Also, heavy consumers of some addictive products may be unable to accurately recall their consumption. Moreover, sales data provide a noisy measure of consumption because some purchases may be shared with others, stored until later, wasted, or transported across borders. In future research, biomarkers (e.g. levels of nicotine, alcohol, or drugs in the blood) could serve as more objective measures of consumption but, to provide statistical power, would need to be collected (ideally repeatedly) for large numbers of people.[28]

A second empirical challenge is that prices are generally measured with error. This is especially true for illegal drugs, but it can be remarkably challenging to determine prices faced by consumers even for cigarettes or alcohol because there are many varieties and brands (which may vary in quality), and because purchases may occur in a different state with a lower tax rate, on Native American reservations with no excise

[26] In the Chaloupka (1991) model, the long-run price elasticity of demand, defined as movement from one steady state of addictive consumption to another, is: $\varepsilon = (\partial C^{\star}/\partial P)\,(P/C^{\star}) = (\beta_1 + \beta_2 + \beta_3)/(1 - \beta_4 - \beta_5) \bullet (P/C^{\star})$.

[27] This may be especially true in certain subpopulations. For instance, pregnant women may be more likely to underreport smoking or drinking than the general population.

[28] In an early use of biomarkers to study health behaviors, Farrell and Fuchs (1982) used carbon monoxide in expired air samples and thiocyanate in blood samples to confirm the accuracy of self-reports of smoking in the Stanford Heart Disease Prevention Program data.

taxes, or over the internet. Even when prices are accurate, consumers act based on anticipated future prices that the econometrician does not observe.

Finally, some papers estimate variants of the rational addiction model using aggregate data, but these are unconvincing as there is no reason to believe that adjacent complementarity at the individual level should be detectable in aggregate data (Ferguson, 2000). Auld and Grootendorst's (2004) falsification test finds evidence of rational addiction, using conventional estimation techniques, for annual national aggregate quantities of milk, eggs, and oranges for Canada between 1961 and 2000. Moreover, their estimates suggest that milk is more addictive than cigarettes. They show that rational addiction can generally not be distinguished from serial correlation when one uses aggregate time-series data and that the discount rates implied in such situations are unreliable.[29]

2.3.3. Less-tested Implications of TORA

Under the TORA, greater adjacent complementarity implies a higher likelihood of unstable steady states. Becker and Murphy (1988) observe that, for many addictive goods, the distribution of consumption is bimodal. For example, few people consume small quantities of crystal meth or crack cocaine year after year; people tend to quickly converge to either a steady state with high consumption (addiction) or one with zero consumption (abstinence). Becker and Murphy suggest that exogenous shocks such as job loss or divorce could nudge a person out of the abstinence steady state, resulting in a rapid transition to the addicted steady state. Conversely, a shock such as a bad drug experience or "bottoming out" could lead an addict to quit "cold turkey" and eventually end up abstaining from use.[30]

In contrast to the large number of studies using empirical models of rational addiction to calculate price elasticities of demand, we know of no empirical work examining unstable steady states or the possibility that shocks drive a person from abstinence to addiction or vice versa. This may be due to a lack of large longitudinal datasets that include both accurate data on addictive consumption and credibly exogenous shocks for a substantial number of respondents. Moreover, the shocks leading to movement between steady states may be idiosyncratically person-specific and therefore difficult to identify in secondary data.

Consumption of addictive goods is sometimes characterized by cyclicality; for example, binging and purging with food, repeatedly quitting drinking but then falling

[29] As an alternative, Gruber and Köszegi (2001) examine responses to increases in future cigarette taxes that have been enacted but not yet implemented. They find that cigarette sales rise but consumption falls, suggesting that consumers are both stock-piling (to avoid the higher future prices) and reducing smoking (to reduce the future addictive stock), both consistent with forward-looking behavior.

[30] Becker and Murphy argue that, when addiction is strong, the only effective way to quit is to go "cold turkey" because any consumption is likely to lead the consumer back to the addicted steady state.

off the wagon, or cold turkey cessation of drugs followed by subsequent use. The original Becker and Murphy (1988) model, and the elaboration on it by Dockner and Feichtinger (1993), permit such cycles of addictive behavior by incorporating a second addictive stock into the utility function. Whereas the original addictive stock exhibits adjacent complementarity, the second stock exhibits adjacent substitutability (i.e. higher values of the stock lower the marginal utility of consumption). The first stock is assumed to have a high rate of depreciation (i.e. it is very sensitive to recent consumption) while the second stock depreciates more slowly (i.e. it is less sensitive to recent consumption). Suppose, for example, that an individual who has previously always abstained from the addictive good (so that both stocks are zero) begins to use the substance. The first addictive stock, which has a high depreciation rate (making it sensitive to recent events), increases rapidly, and the adjacent complementarity of the stock promotes greater future consumption. However, as the individual continues to consume in period after period, the second addictive stock (which has a low depreciation rate) increases. Eventually the effect of the second stock (characterized by adjacent substitutability) overwhelms the effect of the first stock (characterized by adjacent complementarity), and consumption begins to fall. The stock with adjacent complementarity depreciates quickly, and that with adjacent substitutability more slowly, so consumption remains relatively low even as both stocks depreciate. At some point, both stocks have fallen back to zero and the cycle may begin anew.

We are unaware of studies that have empirically tested the two-stock model of rational addiction. One reason may be that it is hard to measure or even define the two stocks (and little effort has been made in the theoretical literature to do so). It might be possible to define the stock with adjacent complementarity as "addiction" and that with adjacent substitutability as "bad health" but there is no clear justification for doing so and these concepts are difficult to measure. Other models that emphasize adjustment costs of deviating from recent levels of consumption (e.g. Dragone, 2009) may prove easier to estimate and test.

2.3.4. Rational Addiction with Learning and Uncertainty

A potential criticism of the TORA is that it predicts that addicts should be satisfied with their consumption patterns or, at least, view them as preferable to alternative states of the world in which they consume less of the addictive product and have a lower addictive stock. In an important extension, Orphanides and Zervos (1995) point out that this need not hold once the assumption of perfect foresight is relaxed. Specifically, inexperienced individuals are assumed to be uncertain about the harm of consuming the addictive good, with learning occurring over time. Persons with high addictive tendencies or excessive initial optimism (about the probability of avoiding addiction) will most frequently end up being addicts and will regret their addiction once it occurs.

Orphanides and Zervos (1995) assume there are two types of individual: "non-addicts," for whom $\theta = 0$, and "potential addicts," for whom $\theta = 1$. Individuals do not initially know to which group they belong but at time t assign a subjective probability, $P(t)$, to the likelihood that they are non-addicts. Utility at time t is:

$$U(Y(t), C(t)) + \theta\eta(t)\nu(C(t), S(t)),$$

where $U(.)$ represents the immediate reward from consuming the non-addictive good (Y) and the addictive product (C), ν is the detrimental addictive side-effect of past consumption, S is the stock of addictive capital, and η is a dichotomous variable indicating whether a harmful addiction has occurred, with $\Pr(\eta = 1)$ increasing in S. Individuals maximize discounted expected lifetime utility:

$$\max E\left\{\sum_{t=0}^{\infty} \delta^t[U(Y(t), C(t)) + \theta\eta(t)\nu(C(t), S(t))]\right\},$$

subject to a budget constraint and the equation of motion of the stock of addictive capital. δ is the discount factor.

The key distinction between this and the standard TORA model is the uncertainty in the last term: individuals do not initially know their type θ or the exact point at which addiction occurs. The subjective probability of being a non-addict ($\theta = 0$) is initially set at $P(0)$ and changes based on a Bayesian updating rule. If $\theta\eta(t) > 0$ then the consumer updates his beliefs so $P(t+1) = 0$. In other words, harm from consuming the potentially addictive good reveals to the individual that he cannot be a non-addict. Conversely, if $\theta\eta(t) = 0$, the consumer updates his beliefs so $P(t+1) > P(t)$, because consuming the addictive good without harm makes it more likely that he is a non-addict.

As in the standard model of rational addiction, the particularly interesting cases involve multiple steady states, and Orphanides and Zervos focus on the situation with two equilibria for potential addicts: one a low level of consumption capital, S_1, where harm does not occur, and the other a high and harmful level of addictive consumption, S_2.[31] The likelihood that potential addicts end up at the higher steady state primarily depends on the baseline probability $P(0)$, with individuals who are overoptimistic (i.e. have too large a value of $P(0)$) at greatest risk of becoming addicted. By construction, all addicts regret their decision, *ex post*.[32] The initial value of the crucial subjective probability, $P(0)$, is determined outside the model but the potential importance of peer

[31] Non-addicts consume an amount of the potentially addictive good that results in a stock intermediate between S_1 and S_2.

[32] Wang (2007) extends upon the framework of Orphanides and Zervos in three ways. His model is solved in continuous rather than discrete time. There is uncertainty about the ability to stop using the addictive product, as well as in the probability of becoming an addict—thus quitting behavior (including unsuccessful quit attempts) is the focus of this model. Finally, all individuals are potential addicts, but with initially unknown heterogeneity in the threshold for addiction.

influences, unwarranted optimism, and misinformation are emphasized. All of these determinants yield policy implications that are potentially quite different than those from the original TORA model (e.g. government policies might influence values of P (0)) and none have received detailed empirical examinations.

2.4. Price Elasticities of Health Behaviors

There are enormous research literatures estimating the price elasticity of demand for habitual or addictive substances. There are comprehensive reviews of this literature available for tobacco products (Chaloupka and Warner, 2000; Gallet and List, 2003), alcohol (Cook and Moore, 2000; Wagenaar et al., 2009), and food (Andreyeva et al., 2010). Grossman (2005) summarizes the empirical evidence on the importance of price as a determinant of the demand for cigarettes, alcohol, and illicit drugs. In this section we will cite the consensus estimates reported in the reviews and meta-analyses, and single out for special mention some studies that utilize exceptionally rich data or especially insightful methods. However, we caution that these studies vary in terms of data utilized and models estimated, so for specifics readers should refer to the original studies as well as the comprehensive literature reviews cited above.

Early studies estimated price elasticities of demand using aggregate data on sales and state taxes, and as a result were limited by multicollinearity, the discrepancy between sales and consumption, smuggling and cross-border shopping, and an inability to estimate elasticities separately for important subgroups such as youths or to measure the price elasticity of initiation or cessation. In response to these limitations, and thanks to the arrival of richer data, more recent studies have used longitudinal individual-level data on consumption. However, challenges persist in accurately measuring price, which may vary even within small geographic areas, and at any location vary by brand and quantity purchased. Common data sources for prices are: for tobacco the Tax Burden on Tobacco (Orzechowski and Walker, 2009), for alcohol and food the American Chamber of Commerce Researchers Association (or ACCRA) Cost of Living Index, and for illicit drugs the System to Retrieve Information from Drug Evidence (STRIDE).

Cigarettes are the addictive substance for which the most price elasticities of demand have been estimated. The *Handbook of Health Economics* chapter on smoking concludes that the price elasticity estimates for overall cigarette demand mostly fall within the range of −0.3 to −0.5 (Chaloupka and Warner, 2000). More recently, Gallet and List (2003) located 523 published estimates of the price elasticity of demand for cigarettes, the mean of which is −0.48, with a large standard deviation (0.43) and ranging from −3.12 to 1.41. They find that the median estimate of the price elasticity of demand for cigarettes is larger for the long run (−0.44, $N = 155$) than the short run (−0.40, $N = 368$), and larger for men (−0.50, $N = 24$) than women (−0.34,

$N = 15$). Aggregation of the data matters; the 87 studies using individual-level data found a smaller median price elasticity (−0.39) than the 101 studies using data at the level of the state or province (−0.60). See Table 3.7 for the median price elasticities of demand for cigarettes for different samples. The meta-analysis by Gallet and List (2003) finds that cigarette price elasticities are not affected by many aspects of the empirical model (e.g. whether accounting for rational addiction or estimating a double hurdle model, whether data are time series or cross-sectional, or whether data are from before or after the release of the 1964 Surgeon General's report on smoking); they conclude that estimation methods have little impact on estimates of the price elasticity of demand for cigarettes.

Some studies separately estimate the impact of price on smoking at the extensive margin. The consensus price elasticity of smoking participation is around −0.5 (Gilleskie and Strumpf, 2005; Grossman, 2005). Gilleskie and Strumpf (2005) show that higher cigarette prices lead to particularly large decreases in the probability of initiation by non-smokers. Conversely, in their preferred estimates, DeCicca et al. (2002) find no impact of cigarette taxes on smoking initiation by youths (both genders pooled) and, estimating models separately by gender, Cawley et al. (2004) estimate that smoking initiation by boys, but not girls, is sensitive to cigarette price.

The next largest relevant literature on price elasticities concerns food. Andreyeva et al. (2010) locate 160 studies that calculate the price elasticity of demand for major food categories. They find that the mean price elasticity of demand for food away from home is −0.81 ($N = 13$), for soft drinks is −0.79 ($N = 14$), for fats and oils is −0.48 ($N = 13$), and for sweets and sugars is −0.34 ($N = 13$). Consumption of high-nutrient, less energy dense foods is also sensitive to price; the mean price elasticity of demand for fruit is −0.70 ($N = 20$) and that for vegetables is −0.58 ($N = 20$). The few studies that calculated price elasticities of demand for food separately by income

Table 3.7 Estimates of Price Elasticity of Demand for Cigarettes

Category	Variable	Median Price Elasticity	Number of Estimates
Elasticity estimate	Short run	−0.40	368
	Long run	−0.44	155
Aggregation	Country	−0.40	335
	State/province	−0.60	101
	Individual	−0.39	87
Gender	Men	−0.50	24
	Women	−0.34	15
Age	Adult	−0.32	17
	Young adult	−0.76	22
	Teen	−1.43	8

Source: Gallet and List (2003), Table 2, column 1.

group found essentially no difference between the price sensitivity of low-income consumers and the population as a whole (Andreyeva et al., 2010). See Table 3.8 for mean price elasticities of demand for various food categories.

There are also a large number of studies estimating the price elasticity of demand for alcohol. Wagenaar et al. (2009) located 112 such studies containing a total of 1,003 estimates of price elasticity. The simple means of these price elasticities are −0.51 for alcohol as a whole ($N = 91$), −0.46 for beer ($N = 105$), −0.69 for wine ($N = 93$), and −0.80 for spirits ($N = 103$), and −0.28 for heavy drinking ($N = 10$). They conclude that there is "overwhelming" evidence that higher prices decrease consumption of alcohol (Wagenaar et al., 2009, p. 187). See Table 3.9A for price elasticities of demand for alcohol for different types of alcohol.

Gallet (2007) conducts a meta-analysis of 132 studies of the price elasticity of demand for alcohol. Across 1,172 published estimates, the median price elasticity is −0.535. The median price elasticity of demand for alcohol is larger in the long run (−0.816, $N = 148$) than in the short run (−0.518, $N = 1{,}024$). The one study located by Gallet (2007) that estimated the price elasticity of demand for alcohol separately by gender found that price elasticity is larger for women (−0.750) than men (−0.509). See Table 3.9B for price elasticities of demand for alcohol for different samples.

Table 3.8 Estimates of Price Elasticity of Demand for Food

Food Category	Mean Price Elasticity of Demand	Number of Estimates
Food away from home	−0.81	13
Soft drinks	−0.79	14
Fats/oils	−0.48	13
Sweets/sugars	−0.34	13
Fruit	−0.70	20
Vegetables	−0.58	20

Source: Andreyeva et al. (2010), Table 1.

Table 3.9A Estimates of Price Elasticity of Demand for Alcohol

Consumption	Mean Price Elasticity of Demand	Number of Estimates
All alcohol consumption	−0.51	91
Beer	−0.46	105
Wine	−0.69	93
Distilled spirits	−0.80	103
Heavy alcohol use	−0.28	10

Source: Wagenaar et al. (2009).

Table 3.9B Estimates of Price Elasticity of Demand for Alcohol

Category	Variable	Median Price Elasticity	Number of Estimates
Elasticity estimate	Short run	−0.518	1024
	Long run	−0.816	148
Aggregation	Country	−0.490	699
	State/province	−0.671	375
	Individual	−0.640	87
Gender	Men	−0.509	1
	Women	−0.750	1
Age	Adult	−0.556	22
	Young adult	−0.386	13
	Teen	1.167	1

Source: Gallet (2007), Table 2, column 1.

Table 3.10 Estimates of Price Elasticity of Demand for Various Illicit Drugs

Addictive Good	Estimate of Price Elasticity of Participation	Estimate of Price Elasticity of Demand Conditional on Use	Source
Marijuana	−0.3		Pacula et al. (2001)
Cocaine	−1.0	−0.3 to −0.4	Chaloupka et al. (1999)
Heroin	−0.89		Saffer and Chaloupka (1999)

Some studies estimate the price elasticity of demand for alcohol at the extensive margin. For example, Manning et al. (1995) calculate that a 10 percent increase in the price of alcohol decreases by 5.5 percent the probability that an individual is a current drinker.

A much smaller literature examines the price elasticity of demand for illicit drugs, generally focusing on the extensive margin of use; this literature confirms that even drug use is sensitive to price. Pacula et al. (2001) find that a 10 percent increase in the price of marijuana decreases its use at the extensive margin among high school seniors by 3 percent. Even the use of hard drugs is price sensitive. A permanent 10 percent increase in the price of cocaine is estimated to reduce the probability of its use by approximately 10 percent and to reduce by 3 to 4 percent the number of times cocaine users take the drug (Grossman and Chaloupka, 1998; Chaloupka et al., 1999). The price elasticity of heroin participation is −0.89, and is similar across race and gender groups (Saffer and Chaloupka, 1999). See Table 3.10 for price elasticities of demand for various illicit drugs.

Others have estimated price elasticities of demand for opium based on historic data from East Asia. Van Ours (1995) examines the opium market in the Dutch East Indies for 1923−1938 and estimates that the short-term and long-term price elasticity

of demand were about −0.7 and −1.0. Liu et al. (1999) use data from the opium market in Taiwan for 1914–1942 and calculate that the short- and long-run elasticities of demand were −0.48 and −1.38.

An important question is whether price sensitivity varies by intensity of use; i.e. when prices rise, is the reduction in consumption limited to casual users, or do heavy users decrease their consumption as well? The answer varies by substance. For alcohol, there is strong consistent evidence that the heaviest drinkers are the least sensitive to price. Manning et al. (1995) find that the price elasticity of demand for alcohol is U-shaped across drinking intensity; demand is relatively inelastic (−0.55) at the fifth percentile of drinkers, price elastic (−1.19) for the median drinker, and essentially zero at the 95th percentile. Likewise, Wagenaar et al. (2009), in their review of the literature, find a mean price elasticity of heavy drinking of −0.28 ($N = 10$), which is only a third of the overall price elasticity of alcohol consumption of −0.91 ($N = 91$). Similarly, Cook and Moore (2001) estimate that a one-dollar increase in the beer excise tax would reduce the prevalence of youth alcohol use by two percentage points, but would have no effect on binge consumption (Cook and Moore, 2001). In models estimated separately by gender, Markowitz and Grossman (2000) find that heavy drinking is elastic to the price of beer for women, but not men.

In contrast to alcohol, when it comes to food the heaviest consumers may be the most price sensitive. Auld and Powell (2009) estimate quantile regressions, which indicate that food prices have small effects on most of the population but have larger effects on youths above the 80th or so quantile of the distribution of BMI; e.g. the effect of fast food prices at the 90th or 95th quantile are three to five times higher than estimates for the entire population. Evidence is more mixed for smoking; price is a greater deterrent to heavy smoking (11 + cigarettes per day) than lighter smoking (6–10 cigarettes per day), but demand is relatively price elastic at both amounts (Gilleskie and Strumpf, 2005).

Another important question is whether youths are more or less price sensitive than adults, and the results are mixed. Gallet and List (2003), in their review of the literature, find that price elasticities of demand for cigarettes are larger for teens (−1.43, $N = 8$) and young adults (−0.76, $N = 22$) than for adults (−0.32, $N = 17$). However, more recent studies using richer data suggest that price has less impact on the smoking initiation of youths than that of adults (DeCicca et al., 2002, 2008a and b). When panel data are treated as repeated cross-sections, this research obtains estimates similar to those obtained in previous cross-sectional studies; e.g. the estimated price elasticity of teen smoking participation is around −0.7. However, when the longitudinal nature of the data is exploited by examining the smoking initiation decisions of non-smoking youths, cigarette taxes are found to have little impact on the probability of smoking initiation.

Table 3.11 Estimates of Income Elasticity of Demand for Cigarettes

Category	Variable	Median Income Elasticity	Number of Estimates
Elasticity estimate	Short run	0.28	295
	Long run	0.39	80
Aggregation	Country	0.33	341
	State/province	0.30	24
	Individual	0.06	10
Gender	Men	0.27	11
	Women	1.23	8
Age	Adult	0.06	6
	Young adult	0.05	1
	Teen	–	0

Source: Gallet and List (2003), Table 2, column 2.

For alcohol, the literature review by Gallet (2007) found a mean price elasticity of demand that was lower for young adults (−0.386, $N = 13$) than for adults (−0.556, $N = 22$). Saffer and Chaloupka (1999) find that the price elasticity of demand for cocaine and heroin is similar across age groups (Saffer and Chaloupka, 1999).

Another important question is whether the health behaviors of pregnant women are sensitive to price. Two studies (Colman, Grossman, and Joyce, 2003; Gruber and Köszegi, 2001) find that a 10 percent increase in cigarette prices is estimated to cause 10 percent of women to stop smoking during pregnancy. An important direction for future research is to better understand how price elasticities of demand vary across unhealthy behaviors, types of consumers (especially youths and pregnant women) and amounts of use.

2.5. Income and Health Behaviors

Income could either increase or decrease unhealthy behaviors. Income could lead to a rise in unhealthy behaviors if cigarettes, alcohol, drugs, and food are normal goods. However, good health and appearance may also be normal goods, leading one to invest more time and money in the production of health as income rises (Philipson and Posner, 1999).

Hundreds of published studies have calculated the income elasticity of smoking and drinking. Gallet and List (2003) located 375 published estimates of the income elasticity of cigarette smoking, the mean of which is 0.42, with a standard deviation equal to 0.49 and ranging from −0.80 to 3.03. They find that the median estimate of the income elasticity of demand for cigarettes is greater in the long run (0.39, $N = 80$) than in the short run (0.28, $N = 295$), and is greater for women (1.23, $N = 8$) than men (0.27, $N = 11$). Aggregation of the data matters; the 10 studies using individual data found a median income elasticity of 0.06, whereas the 24 studies using

Table 3.12 Estimates of Income Elasticity of Demand for Alcohol

Category	Variable	Median Income Elasticity	Number of Estimates
Elasticity estimate	Short run	0.676	901
	Long run	0.860	113
Aggregation	Country	0.768	581
	State/province	0.572	359
	Individual	0.213	74
Gender	Men	0.193	2
	Women	0.120	11
Age	Adult	0.267	30
	Young adult	0.328	4
	Teen	−0.001	2

Source: Gallet (2007), Table 2, column 2.

data at the level of state or province found a median income elasticity of 0.30. See Table 3.11 for estimates of the income elasticity of smoking for various samples.

Gallet (2007) documents 1,014 published estimates of the income elasticity of demand for alcohol, for which the median estimate is 0.69. As found for the price and income elasticities of demand for smoking, the income elasticity of demand for alcohol is sensitive to the aggregation of the data, with studies based on individual data finding a smaller median elasticity than those based on data at the state or province level. See Table 3.12 for estimates of the income elasticity of alcohol consumption for various samples.

In order to measure the causal effect of income on health behaviors, researchers have exploited a variety of natural experiments. A few papers have been able to use lottery winnings as an exogenous source of variation in income. Lindahl (2005) finds that higher lottery winnings reduce the probability of being overweight, but Apouey and Clark (2010) find that lottery winnings lead to an increase in smoking and social drinking. Other research uses variation in government income transfer policies as a source of exogenous variation. Cawley et al. (2010) find no detectable impact of income on weight or obesity, using as a natural experiment the Social Security notch that endowed certain cohorts of retirees with higher benefits. Schmeiser (2009) exploits variation across states in the generosity of the Earned Income Tax Credit (EITC) and is unable to reject the null hypothesis of no effect of income on weight for men. His results for women indicate that an additional $1,000 per year is associated with a gain of between 0.84 and 1.80 pounds. In a randomly assigned conditional cash transfer program in Mexico, a doubling of cash transfers to a household was associated with significantly higher BMI and prevalence of obesity among adults (Fernald et al., 2008a) but significantly lower BMI for age and prevalence of overweight among children (Fernald et al., 2008b).

2.6. The Role of Advertising

Economists have long debated how advertising affects consumer welfare. One possibility is that it provides valuable information about product attributes, quality, price, and lowers search costs. Alternatively, advertising may change consumer preferences or differentiate products in superficial ways, allowing higher prices to be charged. Joseph Stiglitz states flatly, "Most advertising is not informative. The typical Marlboro ad, with a cowboy smoking a cigarette, or a Virginia Slims ad, or a Budweiser Beer ad conveys no credible information concerning the nature of the product being sold, the price at which the product is sold, or where the product may be obtained" (Stiglitz, 1989, p. 842).

Advertising may allow oligopolists to differentiate their products, reduce cross-price elasticities of demand and thereby avoid price competition. This was the strategy of US cigarette manufacturers in the early 1920s, when producers tacitly colluded to keep prices high, and competed only on the basis of advertising, creating substantial barriers to the entry of potential new rivals (Adams, 1952).

The net effect of advertising may partly depend on the characteristics of a good. Advertisements for "search goods"—whose qualities are well known to consumers—may focus on price and availability, whereas those for "experience goods"—whose qualities can only be determined upon consumption—may include relatively little factual information on price or product characteristics (Carlton and Perloff, 2000).[33] This may be even truer for credence goods, whose qualities are difficult to evaluate, even after consumption. For example, a smoker may buy low-tar cigarettes under the assumption that they are less harmful, but whether that is true will not be clear even after the cigarettes are smoked. Deceptive advertising is particularly advantageous to firms selling experience or credence goods.[34]

Adding complexity is that advertising may be a complement to consumption of the advertised good, enhancing welfare even if it does not provide specific useful information about product characteristics or price (Becker and Murphy, 1993). However, Stiglitz (1989) expresses reservations about this possibility.

It is unclear to what extent unhealthy behaviors involve search, experience, or credence goods. Brands of cigarettes, alcohol, and food that one has not yet tried are experience goods. For many such goods, each unit of a specific brand is homogeneous, so after having tried the product once it becomes a search good. However, some product attributes may not be known even after consumption (e.g. the long-run

[33] For example, antidepressants are experience goods because they have idiosyncratic effects (regarding efficacy and side-effects) that are only revealed (to the patient and physician) after being used for an extended period of time.

[34] For example, all of the 58 advertisements (run during the first half of 1965) found to be deceptive by the Federal Trade Commission concerned experience, rather than search, qualities (Nelson, 1974).

health consequences of use), so there are certain ways in which these are credence goods.

Another ambiguity is whether advertising is cooperative (expanding the market by convincing new people to begin consuming the good) or competitive (increasing the advertised brand's share of a fixed market by stealing users from rival firms) or both. Limited empirical evidence suggests that advertising of soft drinks is competitive (Gasmi et al., 1992) while cigarette advertising is cooperative (Roberts and Samuelson, 1988).

Researchers examining how advertising influences unhealthy behaviors face several challenges, beyond those already mentioned. It is hard to measure an individual's exposure to advertising, or to find data that include both exposure to advertising and consumption of the advertised good. Perhaps most importantly, it is difficult to exploit exogenous variations in advertising in order to identify its impact on consumption. As pointed out by Avery et al. (2007, p. 449): "The relationship between advertising and consumption is literally a textbook example of simultaneous equations. . .Are consumers responding to the advertising or are advertisers responding to the consuming?"

Comprehensive literature reviews of the effect of advertising on tobacco consumption indicate that the evidence is mixed as to whether advertising increases use or has no detectable effect. Blecher (2008) identifies 18 studies that find no significant effect of advertising on smoking, and 17 analyses that uncover a significant positive impact. Saffer and Chaloupka (2000) classify studies of advertising and cigarette consumption according to whether the data are time series or cross-sectional. Among the time-series studies, nine find no effect of advertising and six find a small positive effect. All three cross-sectional studies examined indicate a positive effect of advertising.

Two meta-analyses of advertising elasticities of demand for cigarettes and alcohol find that use is less sensitive to advertising than to prices or income. Gallet and List (2003) located 137 published estimates of the advertising elasticity of cigarette smoking, the mean of which is 0.10, with a standard deviation equal to 0.13 and ranging from −0.10 to 069. Gallet (2007) located 132 studies containing 322 estimated advertising elasticities of alcohol consumption, of which the median estimate is 0.029.

Chou et al. (2008) merge data from the 1979 and 1997 National Longitudinal Surveys of Youth (NLSY79 and NLSY97) with information on weekly hours of television advertisements for fast food restaurants by designated market area (DMA) and year. They estimate that if youths were exposed to an additional half hour of fast food advertising per week, the probability of being overweight would rise by 2.2 percentage points (15 percent) for boys aged 3−11 years, 1.6 percentage points (or 12 percent) for girls aged 3−11 years, 2.5 percentage points (17 percent) for boys aged 12−18 years, and 0.6 percentage points (4 percent) for girls aged 12−18 years. This research is limited by a lack of information about fast food consumption (the outcome examined is BMI). Moreover, the child's exposure to advertising is estimated using

the number of hours that the child reports watching television and the ads aired in that DMA; the researchers do not know how many and which fast food commercials each child saw. Moreover, advertising in the DMA may be endogenous; fast food restaurants likely target advertising to areas whose residents are expected to have a high demand for fast food. Controlling for DMA fixed effects accounts for time-invariant differences in demand but there remains potentially endogenous variation in demand over time within DMAs.

Saffer and Dave (2006) pursue a similar strategy, merging individual-level data from the Monitoring the Future and NLSY97 with market-level data on alcohol advertising in television, newspapers, radio, and outdoor media. They examine the 75 largest DMAs in the US and find that advertising is positively, although modestly, correlated with the probability of alcohol use and binge drinking. They acknowledge that the results may be biased if advertising expenditures are a function of factors affecting demand for alcohol. This study also suffers the limitation of not being able to accurately estimate exposure to ads within DMAs.

Virtually all previous studies examining the impact of advertising on health behaviors are limited by the likelihood that advertising exposure is endogenous, e.g. through targeting of ads to consumers likely to demand the products. A recent review of the literature examining the impact of cigarette marketing on smoking criticizes this work for failing to address the endogeneity of marketing exposure and concludes that the findings "fall far short of those required to establish well-founded causal relationships" (Heckman et al., 2008, p. 43). The study that best estimates the effects of advertising exposure, while addressing the endogeneity of exposure, is Avery et al. (2007). Using the Simmons National Consumer Survey, the authors merge data on the number of advertisements for smoking cessation products in the specific magazine issues read by the respondent. To control for the selective targeting of ads, the authors control alternately for categories of magazines read (so variation in advertising exposure comes from, for example, reading *Time* instead of *Newsweek*) and for magazine fixed effects, thus exploiting variation over time in the number of ads in each magazine. The authors consistently find that exposure to magazine advertisements for smoking cessation products raises quit attempts, with weaker evidence of increases in successful quitting.[35]

2.7. Time Preference and Health Behaviors

The rate of time preference refers to an individual's willingness to exchange utility today for utility later: it is the marginal rate of substitution between current and future

[35] As a falsification test, they check whether future advertisements for smoking cessation products (in the same magazines the respondent currently reads) affect the current probability of quitting, and find that they do not.

utility (Becker and Mulligan, 1997). Suppose that an individual seeks to maximize the present discounted value of lifetime utility:

$$U = \sum_{t=1}^{T} \delta^t(U(C_t)) \quad \text{for } \delta = \frac{1}{1+\sigma},$$

where $U(C_t)$ is utility in period t. A higher rate of time preference σ indicates that the person is less patient (to a greater extent prefers utility today to utility tomorrow). The discount factor δ has the opposite correlation: a smaller discount factor implies less patience (future utility receives a weight closer to zero) and a higher discount factor implies greater patience (a higher weight for utility in later periods).

Victor Fuchs (1982) was one of the first economists to examine the relationship between rate of time preference and health behaviors, motivated by the large literature documenting a positive correlation between education and health. Fuchs (1982) argues that the correlation of education with good health could reflect differences in rate of time preference. Patient individuals are more likely to forego current utility in exchange for long-run benefits; this is likely to result in healthier behaviors (e.g. more exercising) and higher education, even if schooling has no causal effect on health. Fuchs finds that a more patient rate of time preference (elicited from questions about willingness to exchange a certain amount of money today for a larger amount in the future) is associated with greater schooling and usually also with healthier behaviors, although the point estimates are often small and not always statistically significant.

Time preference is notoriously difficult to measure. Most of the empirical literature attempting to do so uses one of two approaches. The first is to infer a discount factor from Euler equations of consumption (e.g. Lawrance, 1991) or wealth (e.g. Samwick, 1998) in different periods. A limitation of this approach is that identification of the rate of time preference is dependent on strong assumptions about functional form (Lawrance, 1991; Zhang and Rashad, 2008). A second common method is to survey respondents using hypothetical scenarios regarding willingness to exchange money today for (more) money in the future (e.g. Fuchs, 1982; Farrell and Fuchs, 1982). This approach is limited because people may not provide accurate answers to hypothetical scenarios and responses to such questions may instead measure expectations about interest rates, rates of return on investments, or attitudes toward risk, rather than discount factors.

A fundamental challenge for all methods of calculating the discount rate is that rates of time preference may vary across types of consumption. For example, an individual may be happy to save rather than spend money, but eager to defer physical pain rather than to experience it immediately. Moreover, the marginal rate of substitution for consumption in two particular periods may change over time or across the life-cycle.[36] These issues will be discussed in detail in section 3.

[36] See Frederick et al. (2002) for a general discussion of these issues.

Becker and Mulligan (1997) model time preference as endogenous. They point out that there is an incentive to reduce the rate of time discount because doing so raises the present discounted value of lifetime utility. They, along with Fuchs (1982), hypothesize that schooling may provide a method of decreasing one's rate of time discount and that this provides a possible mechanism through which education improves health. Becker and Mulligan (1997) further suggest that precommitment mechanisms (such as "Christmas clubs" that enforce saving) may be investments in learning patience. Parents may invest in reducing their children's discount rates, so that the youth will be more willing to make investments that involve short-term costs but long-term gain, which can yield benefits for health, human capital, and wealth. In the addiction model of Orphanides and Zervos (1998), discussed above, consumption of addictive substances raises the rate of time preference (reduces patience) but individuals are aware of this and account for it when making consumption decisions.

Farrell and Fuchs (1982) find that among white, non-Hispanic adults with between 12 and 18 years of schooling, in the Stanford Heart Disease Prevention Program, the negative correlation between eventual completed schooling and smoking is as strong at age 17 (when all were in the same grade, and differences in education had not yet arisen) as at age 24 (when they differed in attained education). Based on this, Farrell and Fuchs (1982) reject the hypothesis that years of schooling reduce smoking and conclude that omitted variables explain the observed correlation. They are unable to test which omitted variables are responsible, but hypothesize that one is rate of time discount.

Several recent papers demonstrate a correlation between body mass index (BMI) or obesity and proxies for rate of time preference such as savings rates (Komlos et al., 2004; Smith et al., 2005) or willingness to delay financial rewards or other gratification (Borghans and Golsteyn, 2006; Ikeda et al., 2010). Conversely, Chapman et al. (2001) find weak or no association between health behaviors (influenza vaccination, adherence to medication for high blood pressure, adherence to medication for high cholesterol) and time preference assessed using hypothetical scenarios. Khwaja et al. (2007) show that smokers and non-smokers have similar rates of time discount when the latter is proxied by willingness to undergo a colonoscopy and conclude that variation in discounting is not a major explanation for differences in smoking behavior.

Cutler and Glaeser (2005) argue that if discount rate heterogeneity explains variations in health behaviors, we should observe high within-person correlations across health behaviors—e.g. alcohol consumption should be higher among smokers than non-smokers. They test this using data for individuals 45 and older from the 1990 National Health Interview Survey on: current smoking, consumption of three or more alcoholic beverages per day, obesity, use of recommended hypertension medication, and (for women) receiving mammograms in the past three years. They find that the correlations across health behaviors are "surprisingly low" (p. 238), most below

10 percent, with the highest (alcohol and smoking) at 16 percent. They obtain similarly weak correlations using data from the Behavioral Risk Factor Surveillance System on smoking, drinking, obesity, seatbelt use, flu shots in the past year, and cancer screening. Changes over time in health behaviors (smoking, heavy drinking, being overweight, and physical inactivity) are also weakly correlated (in the Health and Retirement Study). While these results are consistent with the hypothesis that time preference is not a major determinant of health behaviors, other explanations are that these behaviors are primarily substitutes (rather than complements), and myopia about future consequences leads people to become addicted to certain unhealthy behaviors but not others.

3. ALTERNATIVE APPROACHES TO STUDYING HEALTH BEHAVIORS

3.1. Peer Effects

In the basic model of health capital, individuals make decisions in isolation from each other. Manski (2000) notes, however, that there are three channels through which individuals may affect each other. The first is through constraints on shared resources. For example, there may be only so many treadmills at the gym, or so many roster spots on the school sports team, and as a result one individual's decision to exercise or play sports can prevent another from doing the same. Second, individuals may influence each other's behavior through expectations. For example, teenagers may update beliefs about the marginal benefits and costs of risky behaviors by discussing their sexual experiences or observing each other's drug use. Third, individuals may directly affect each other's preferences.

This third mechanism is the focus of Leibenstein (1950). He emphasizes the role of "bandwagon" effects (deriving utility from consuming the same goods and services as peers), which makes the demand curve more elastic because, when price falls, demand increases both directly (because of lower prices) and indirectly (because others are more likely to be using the good). Conversely, "snob" effects work in the opposite direction. When something becomes common, people may not want to consume as much of it (perhaps because it no longer signals exclusivity). Snob effects make the demand curve less elastic because the increased consumption associated with a price decrease makes the good less desirable to consume. Bandwagon and snob effects imply that the market demand curve is not simply the horizontal summation of all individual demand curves in the market and that peer interactions matter in ways not captured by simple economic models.

For many addictive goods, consumers may strike a balance between bandwagon and snob effects. For example, teenagers may wish to rebel against the majority (consistent with a snob effect), but probably not in an utterly unique way that leaves them isolated from everyone else, so they choose to emulate a small subset of peers, consistent with a limited bandwagon effect.

A rapidly growing empirical literature has investigated peer effects in health behaviors. Manski (2000) notes three possible explanations for the correlation of behaviors within groups: (1) *endogenous interactions*, where behavior of the group affects behavior of the individual; (2) *contextual interactions*, where exogenous characteristics of group members (such as age or family background) affect behavior of the individual; and (3) *correlated effects*, a non-social effect in which the group behaves similarly because they have similar characteristics or environment. For example, smoking may be correlated within youth peer groups for any or all of these reasons. There may be endogenous interactions such as peer smoking increasing own tobacco consumption because smoking is a bandwagon activity. There may be contextual interactions if teens who hang out with older kids tend to have smoking peers (because the peers are older), and having older friends (whether smokers or not) is associated with more frequent initiation of smoking. Finally, there may be correlated effects in the sense that low-income youth tend to hang out together, and low income is associated with smoking initiation.

The source of correlated group behaviors has important implications for public policy. For example, smoking cessation programs targeted to individual teens may have spillover effects to peers if there are endogenous interactions, but not if there are only contextual interactions or correlated effects.

Manski (1993, 2000) emphasizes the difficulty in empirically distinguishing between these effects because of the "reflection problem": the observed correlation between an individual and his peers is a composite of both the impact of peers on the individual and of the individual on the peers. Researchers have sought to overcome the reflection problem by: assuming a specific length of lag between mean group behaviors and those of the individual, modeling individual behavior as a specific non-linear function of group behavior (i.e. assuming that individuals are responsive to some feature of the group distribution of behavior other than the mean), or using instrumental variables approaches that exploit exogenous variation in either the behavior of group members or the membership of the peer group. Even if endogenous interactions can be proven, Manski (2000) argues that such a finding is only useful if one can demonstrate that the mechanism is preferences (e.g. the stigma of drug consumption falls when its use rises), expectations (e.g. youths learn how pleasurable drugs can be by seeing others enjoy them), or constraints (e.g. search costs for drugs are reduced when friends buy and use them).

The nature and variety of empirical investigations on peer effects can usefully be illustrated by focusing on studies of obesity, which have received considerable

attention, particularly in response to a study of data on adults from the Framingham Heart Study that concluded obesity spreads within social networks (Christakis and Fowler, 2007). That study does not exploit any exogenous variation in either peer group membership or in the behavior of peers; instead it attempts to control for correlated effects by controlling for lags of both respondent obesity and peer obesity in a model that regresses respondent contemporaneous obesity on peer contemporaneous obesity status. Key findings are that the chances of becoming obese rose by 57, 40 and 37 percent, respectively, if the respondent had a friend, sibling, or spouse who became obese. These findings could reflect selection (friends and spouses choosing each other based on future weight or weight trajectories), correlated effects due to shared environments (such as local food prices and availability, or availability of exercise opportunities), or true endogenous interactions (i.e. causal peer effects). Christakis and Fowler (2007) and Fowler and Christakis (2008) argue that there is likely to be a true peer effect because the estimated effects are stronger for pairs who both list each other as friends than pairs in which one but not both individuals claim friendship. They also find that geographic proximity of peers does not matter—the authors interpret this second result as ruling out a common environment effect. However, even for true endogenous interactions, one might expect proximity to be important if the size of the peer effect depends on the frequency of interactions with the peer.

Cohen-Cole and Fletcher (2008a) investigate the sensitivity of the findings in Christakis and Fowler (2007) using a different dataset (the National Longitudinal Survey of Adolescent Health or Add Health) and age group.[37] When using the same regression model, they largely replicate the findings of Christakis and Fowler; however, when controls for school-specific time trends (as a proxy for the environment) were included, the peer correlations fall more than 30 percent, suggesting the importance of omitted group-level characteristics.[38] In addition, they conduct falsification tests, using the Add Health data, showing that Christakis and Fowler's (2007) regression model generates apparent network effects for outcomes for which true peer effects are unlikely (e.g. acne, height, and headaches) and that these disappear after controlling for environmental confounders.[39] They conclude that the method used by Christakis and Fowler (2007) is not sufficiently specific to separate true network effects from spurious correlations due to insufficient controls for the local environment.

[37] Add Health is a nationally representative sample of 7−12th graders first interviewed in 1994−95.

[38] However, Fowler and Christakis (2008) interpret this work as supporting their own, pointing out that their original estimates are within the 95 percent confidence intervals of Cohen-Cole and Fletcher (2008a).

[39] Falsification tests apply the empirical methods to outcomes for which the hypothesized relationships should *not* exist. Large or statiscally significant associations suggest problems with the empirical strategy. In a classic falsification test, Dranove and Wehner (1994) used a standard method of testing for demand inducement for medical care in a situation where it should not hold: pregnancies. Their finding that obstetricians/gynecologists appear to increase the number of pregnant women suggests shortcomings of the standard test.

Identifying the correct peer group is a challenge. "However severe the reflection problem may be when group composition is known, the problem becomes insurmountable when group composition is unknown" (Manski, 2000, p. 129). Moreover, peer groups may vary across outcomes; for example, a teenage boy might experience bandwagon effects for physical fitness from his sports teammates, for risky sexual activity from his classmates, and for alcohol from his older brother. In practice, researchers examining health behaviors have examined a variety of peer groups, almost always driven by their opportunistic availability in secondary data, including: classmates (Lundborg, 2006; Argys and Rees, 2008), friends (Christakis and Fowler, 2007; Cohen-Cole and Fletcher, 2008b), siblings (Christakis and Fowler, 2007), spouses (Christakis and Fowler, 2007), neighbors (Christakis and Fowler, 2007; Case and Katz, 1991), and college roommates (Yakusheva, 2010; Duncan et al., 2005).[40]

The method of instrumental variables can be applied to estimate peer effects by utilizing exogenous variations in peer behavior. For example, Trogdon et al. (2008) and Renna et al. (2008) instrument for the weight of a peer using the obesity status of the peer's parents. The validity of these instruments is questionable, however, because friendships could also be selected on the basis of obesity status, with obese youths relatively likely to have obese parents. This strategy may also suffer from a second-order case of the reflection problem—friend's parents' weight may be affected by friend's weight which in turn may be affected by the respondent's weight.

A second strategy for identifying causal peer effects is to instrument for peer behavior using exogenous variation in peer group membership; this has most frequently concerned classmates or roommates. For instance, Argys and Rees (2008) use birth date relative to the cutoff for starting kindergarten to generate exogenous variation in the age of the youth relative to classmates; they find that females with older peers are more likely to use substances (marijuana, alcohol, tobacco) but find few peer effects for boys. Lundborg (2006) assumes that while schools may be chosen by parents, the specific classroom within a grade is randomly assigned; utilizing across-classroom differences in peer behavior by controlling for school and grade fixed effects, he finds effects of classmates on binge drinking, smoking, and use of illicit drugs. Duncan et al. (2005) use randomized roommate assignment and find that boys who binge drank in high school consume more alcohol in college if their roommate binge drank in high school. No such peer effects were found for boys who did not binge drink in high school, or for

[40] Other relevant peer groups may include an online community or adolescents seen on television. In Christakis and Fowler (2007), "friends" were those that respondents listed as being able to get in touch with them in case they had moved and the surveyors were unable to find their new address or phone number. These might be friends or merely those most likely to know their whereabouts. Add Health asked respondents to list their five closest male and five closest female friends. This survey also contains information on a large number of students in the same schools, allowing researchers to use classmates as another peer group. In both the Framingham and Add Health data, one is able to explore the importance of symmetry: do two respondents list each other as friends, or does one person list the other as a friend but it is not reciprocated?

girls, or for marijuana use or sexual behavior for either boys or girls. Yakusheva et al. (2009) also exploit random roommate assignment and find that female college students gained less weight during freshman year when their roommate was heavier. Carrell et al. (2010) exploit random assignment of United States Air Force Academy cadets to squadrons (of approximately 30) with whom cadets live, eat, study, and compete in intramural sports. They find substantial peer effects; e.g. that the effect on the respondent's current fitness of friends' high school fitness is nearly 40 percent as strong as the effect of the respondent's own high school fitness. These peer effects are caused primarily by friends who are the least fit; this may be due to the sample consisting of unusually fit individuals who may not have much room for improvement.

In the Moving to Opportunity experiment, adults receiving a waiver to move to a higher-income neighborhood were 5 percentage points less likely to be obese than similar persons not receiving a waiver (Kling et al., 2007); this intent-to-treat estimate is consistent with those moving to more advantaged neighborhoods adopting a new set of peers with healthier habits. An alternative explanation is that the design of, and amenities in, the new neighborhoods better facilitate healthy eating and physical activity.

Another general empirical challenge in the peer effects literature is correctly modeling which aspects of the distribution of behavior in the peer group are relevant. Most analyses model individual behavior as a function of the central (especially average) tendencies of the peer group, but the presence or absence of extreme actions could be more important. High outliers ("bad apples") and low outliers ("straight arrows") might be especially influential on behavior. For example, the probability that a female college student develops an eating disorder might be influenced by the number of sorority sisters who are underweight, rather than average body weight of sorority members.

3.2. Information Constraints

Individuals may lack the information needed to accurately assess the costs and benefits associated with various health behaviors. One important question is whether individuals understand how health behaviors alter the risks of morbidity and mortality. If individuals underestimate the risks associated with unhealthy behaviors, government intervention to either directly provide the missing information or require disclosure of information by producers could be warranted. Kenkel (1991) provides evidence that health knowledge is related to smoking, drinking, and exercise in the expected directions, but that even some highly knowledgeable persons have poor health habits.

A large economics literature has examined consumer awareness of, and sensitivity to, information on the health consequences of smoking. Smoking is especially informative because its risks are well understood and have been widely publicized since the

1964 Surgeon General's report (US Department of Health, Education and Human Welfare, 1964).

A landmark study by Viscusi (1990) found that adults were much more likely to *overestimate* than to *underestimate* the extent to which smoking raises the risk of lung cancer. Specifically, he estimated that the true lifetime risk of lung cancer for smokers was between 5 and 10 percent but respondents to a national telephone survey estimated that the risk was 43 percent. Assuming the true risk was 10 percent, approximately 90 percent of the sample overestimated the risk, with 51 percent thinking it exceeded 50 percent. Smokers had lower risk estimates than non-smokers but still overstated the true risk fairly dramatically—the average stated subjective risk was 37 percent; 86 percent estimated greater than a 10 percent lifetime risk for smokers and 42 percent put the odds above 50 percent. These risks are overestimated even if respondents' answers were based on all of the risks of smoking (e.g. including those from heart disease, strokes, and emphysema).

Analyses of data from the Health and Retirement Survey (HRS) reach a starkly different conclusion. Schoenbaum (1997) finds that 50–62-year-old heavy smokers had expectations of living to age 75 that were almost twice as high as actuarial predictions. Khwaja et al. (2007), using the same data, find that, for the sample as a whole, subjective beliefs about survival were similar overall to the objective data but that current smokers were overly optimistic about survival while those who had never smoked were relatively pessimistic.[41] Smith et al. (2001) estimate that heavy smokers are more optimistic about their self-assessed longevity than their smoking behavior would warrant.[42]

One explanation for the difference in the aforementioned findings is that individuals may suffer from optimism bias; i.e. they may have accurate knowledge of population risks but still underestimate their personal risk. Viscusi's (1990) question "Among 100 cigarette smokers, how many do you think will get lung cancer because they smoke?" may measure knowledge of risks to the general population, whereas the HRS data analyzed by Schoenbaum (1997) and Khwaja et al. (2007) contained respondents beliefs that they personally would live to age 75.

Supporting this possibility, Smith et al. (2001) show that smokers and non-smokers in the HRS respond to health information differently: smokers dramatically reduce their subjective life expectancies when confronted with smoking-related health shocks but decrease their subjective life expectancies less than non-smokers in response to

[41] Objective survival probabilities were based on actual mortality within the HRS sample, rather than on life tables. Khwaja et al. (2009) find that 50–70-year-olds relatively accurately predict their probability of survival to age 75 regardless of their smoking status.

[42] Similarly, in an interesting analysis of secondary life insurance markets (viatical settlements) for individuals with HIV/AIDS, Bhattacharya et al. (2009) provide evidence that relatively healthy individuals understate their remaining life expectancy while those who are relatively unhealthy overstate it.

non-smoking-related health shocks. The authors conclude that smokers may not personalize the risks of tobacco use unless there is clear evidence that it is negatively affecting their own health. If true, this implies that general information (such as that required by labeling laws) may have only a limited effect on behavior until individuals incur a health shock related to the unhealthy behavior.

3.3. Time-inconsistent Preferences and Hyperbolic Discounting

Models of intertemporal choice have typically been based on the exponential discounting model of Samuelson (1937); e.g. the Samuelson framework is the basis for the optimization problem in the rational addiction model discussed above. A key feature is that the discount rate between any two periods t and $t+1$ is a constant (d), implying that the discount rate between periods t and $t+n$ is d^n, for all n. Such behavior is often referred to as "time consistent" because the marginal rate of substitution for consumption in any two periods remains constant over time.

Exponential discounting quickly became standard in models of intertemporal decisions because it provides a straightforward method of extending single-period utility maximization into a multi-period context, not because it accurately depicts the way that such decisions are actually made. To the contrary, as discussed below, individuals often have "time-inconsistent" preferences, e.g. discount rates are higher for intertemporal trade-offs that occur in the near future than for longer time horizons. This is called "hyperbolic" discounting (Ainslie, 1991).[43]

A key implication of hyperbolic discounting is "present-biased" preferences; the trade-off between utility in the current versus the next period is greater than that for any two adjacent periods in the future. Hyperbolic discounting results in time-inconsistent behavior. For example, assume a person is willing to trade one "util" of happiness in period n in the future for two utils received in period $n+1$. With exponential discounting and stable preferences, a person who is willing to make that trade when periods n and $n+1$ are in the distant future will also be willing to make that trade when it actually is period n. However, with hyperbolic discounting, there may be a preference reversal; the person might be willing to trade one util in period n for two utils in period $n+1$ while those periods are in the distant future, but when it actually is period n the individual might suddenly decide that more than two utils would be needed in the next period to compensate them for giving up a util immediately. Consider an individual who is choosing on Monday how much ice cream to eat on Friday. An exponential discounter will carry out on Friday the plans made on Monday (assuming no changes in income, prices, or other relevant variables) because preferences are time consistent. However, a hyperbolic discounter may plan on Monday to skip ice cream on Friday, but when Friday comes she may experience a preference reversal, suddenly being unwilling to

[43] This idea was first formalized by Strotz (1955–1956).

deny herself the current utility from consuming the ice cream. Thus, time-inconsistent preferences lead to self-control problems and future plans to engage in healthy behavior are consistently undone by (abnormally) high present discount rates.

Time-inconsistent preferences can affect many health behaviors. Plans made the night before to exercise in the morning will not be realized when the alarm clock goes off, intentions to consume alcohol in moderation will be undone as the immediate pleasures of having "just one more drink" are repeatedly acted upon, and so forth. Individuals who are naïve about their time-inconsistent preferences may be endlessly optimistic about their ability to improve future health behaviors. Those who are more sophisticated may seek precommitments that compel their future self to adhere to healthy behaviors. For instance, they may avoid bringing home ice cream, knowing that doing so will result in overeating. Or they may plan to run with a friend, aware that this will make it harder for them to skip the workout. Each of these tactics is designed to ensure that binding decisions are made when the marginal rate of substitution between two future periods is relatively low, rather than later, when the marginal rate of substitution will change in such a way that incentivizes immediate gratification.[44]

By far the most common way that economists have modeled hyperbolic discounting is using the quasi-hyperbolic (or $\beta-\delta$) framework developed by Laibson (1997), based on the functional form first used by Phelps and Pollak (1968) in their study of optimal intergenerational savings. Specifically, the utility function is characterized by:

$$U_t = u(C_t) + \beta \sum_{n=1}^{T-n} \delta^t \, u(C_{t+n}), \tag{3.3}$$

where $u(\cdot)$ is utility in the specified period, C is a composite consumption good, T is the time horizon over which utility is measured (known with certainty), with $\beta \leq 1$ and $\delta \leq 1$. The key implication is that the discount factor for consumption between period t and $t+1$ is $\beta\delta$, whereas that between any two future periods, $t+j$ and $t+j+1$ (for $j > 0$) is δ. If $\beta = 1$ this reduces to exponential discounting. However, if $\beta < 1$, the discount factor is lower (and the discount rate higher) for immediate than future consumption trade-offs. This results in time-inconsistent preferences and self-control problems similar (but not identical) to those in more general forms of hyperbolic discounting and that can be relatively easily incorporated into standard economic models of intertemporal choice.

Substantial research suggests that hyperbolic discounting explains many real-world decisions more accurately than standard exponential discounting. For example, Thaler

[44] Sophistication is a double-edged sword. O'Donoghue and Rabin (1999) point out that sophisticated hyperbolic discounters are more likely than naïve hyperbolic discounters to develop precommitment strategies but may also more frequently "preoperate"—realizing that they are unlikely to be able to stick to their plan, they abandon it earlier. For example, a sophisticated hyperbolic discounter may realize that she is unlikely to be able to stick to her diet and may cease even trying, whereas a naïve hyperbolic discounter may naïvely start diet after diet.

(1981) found that the future payments required to make individuals indifferent between receiving a prize now versus later yielded implied (per period) discount rates that declined dramatically as the length of time increased, consistent with hyperbolic discounting.[45] Angeletos et al. (2001), and the references contained therein, indicate that a wide variety of time preference experiments show that decision makers are more impatient in the short run than the long run. Frederick et al. (2002) examine a large number of empirical studies and find that discount factors increase (discount rates decrease) as the time horizon of the study increases but that this relationship disappears when studies covering less than one year are eliminated. Such results are consistent with hyperbolic discounting.[46]

Gruber and Köszegi (2001) incorporate time-inconsistent preferences in a model of smoking and distinguish between sophisticated and naïve agents, where the former understand that their preferences are time inconsistent but the latter do not. A key prediction is that future price increases reduce current consumption (in models that do not also control for future consumption). Because this is generally the key test for rational addiction, standard econometric tests (focusing on responses to future changes) cannot distinguish between the TORA and similar frameworks with time-inconsistent preferences.[47] Cutler et al. (2003) also use an informal model with hyperbolic discounting to consider (but not formally test) how time-inconsistent preferences affect eating decisions and obesity, and the resulting consequences for social welfare.[48]

3.4. Cognitive Limitations and Bounded Rationality

Hyperbolic discounting does not change the utility maximization assumptions standard in economic models, only the method of discounting. However, it is possible that individuals cannot (or for whatever reason, do not) maximize utility when faced with highly complex problems. Such limitations are the basis of the models of bounded rationality and "satisficing" developed by Herbert Simon (1984) and applied by economists to many decision processes (e.g. see Thaler and Sunstein, 2009), including those related to health behaviors.

For instance, in Suranovic et al.'s (1999) modified rational addiction framework, an indi

where B is the current benefit of smoking, L is the fully discounted future loss from smoking, and C is the adjustment cost to changing tobacco use from recent levels. The key assumptions are that most utility losses occur at or near the end of life (and so are discounted heavily at young ages) and that adjustment costs are zero for smoking at or above recent levels but positive for lower amounts of use.[49] The model is forward looking, in that future health costs are accounted for, but myopic in that the consequences of current smoking on future adjustment costs are not considered. It allows for several realistic consequences that are either difficult to explain or require strong assumptions using models with fully rational addiction. For instance, gradual rather than "cold turkey" withdrawal may occur if addiction is "weak" (i.e. if adjustment costs for deviating from past history rise at an increasing rate), and quitting may become more likely late in life, because the number of periods until losses are incurred decreases so that these are discounted less heavily. Importantly, by failing to fully account for the consequences of current smoking on future quitting costs, individuals may enter a consumption "trap," strengthening the potential case for policy intervention.

Akerlof (1991) provides a sophisticated analysis in which bounded rationality leads to choices that separately are close to utility maximizing but, in combination, can result in large errors. Key aspects of his formulation are that decision makers are slightly biased toward present rather than future utility (as described above) in ways that they are either unaware of or do not fully account for. The result is that they avoid large mistakes at any single point in time but may make small errors that accumulate across periods.

Procrastination is an example of such behaviors. Individuals may intend to start an exercise program or stop smoking at a date in the near future but time inconsistency repeatedly prevents these intentions from being realized. Akerlof's analysis also emphasizes the role of peer influences in encouraging these decision errors, particularly in situations, as with gangs or cults, where the social milieu may be constructed to encourage initially small but cumulatively large changes in behaviors.

In Rubinstein's (2003) model of bounded rationality, agents simplify choices by applying "similarity relations." Specifically, when considering uncertain and multidimensional outcomes, individuals do not fully account for dimensions of the choices that are "similar" but instead focus on those characteristics that are dissimilar. Consider lotteries of the form (X, p), in which a payout X is won with probability p and a payout of zero is won with probability $1 - p$. Rubinstein argues that, faced with the choice between (\$3,000, 0.25) and (\$4,000, 0.2), the difference in the size of the prize will be the decisive factor because the probabilities (0.20 and 0.25) will be interpreted as "similar" (and thus the difference will be ignored) whereas the prize amounts (\$3,000 versus

[49] By contrast, Dragone (2009) has developed a fully rational model of eating in which there are costs to any changes (either positive or negative) in food consumption. Under specified assumptions, overshooting will cause individuals to oscillate between gaining and losing weight, before eventually converging on the steady state.

$4,000) will be interpreted as dissimilar.[50] He argues that this model explains some observed time-inconsistent behavior better than hyperbolic discounting. Specifically, he argues that individuals may perceive "10 years from now" and "11 years from now" to be similar, but perceive "today" and "a year from now" as dissimilar.

Education is likely to be correlated with cognitive ability (which is measured with error, if at all), possibly explaining some of the strong positive relationship between schooling and healthy behaviors noted in section 1 of this chapter. Education might also influence behaviors in other ways (e.g. by being correlated with or causally affecting discount rates, health knowledge, or access to high-quality medical care) but an emerging body of research suggests that differences in cognition partly explain variations in health behaviors.

Cutler and Lleras-Muney (2010) conclude that about 30 percent of the average education gradient in a wide variety of health behaviors in the United States and Great Britain is related to disparities in cognitive ability, with high-level processing being more important than measures of memory. Interestingly, cognitive ability is more important at later ages than earlier in life, suggesting that cognitive skills are learned (and helping to rule out explanations based on factors that are confounded with education and cognitive skill levels at a point in time). Similarly, using Australian data, Antsey et al. (2009) find that cognitive skill (proxied by verbal ability and processing speed) is correlated with vitamin and mineral supplement use, high rates of physical activity and light/moderate alcohol use, and reduced smoking.[51]

Chen and Lange (2008) and Lange (2010) examine how education is related to breast, colorectal, and cervical cancer screening, with particular attention paid to differences between objective versus subjective cancer risk. A key result is that subjective risks of highly educated individuals more accurately reflect objective risks than do those of counterparts with less schooling, and that differences in subjective risks are more closely linked to the relevant screening decisions. These education differences do not appear to occur because of a positive correlation between schooling and income or the quality of medical care but could reflect the more scientific world-view of those with more schooling. One overall conclusion is that the highly educated are better at processing information related to medical risks and the behaviors required to ameliorate them (although it is not clear that schooling causes these differences).

De Walque (2007a) demonstrates that a series of HIV/AIDS prevention campaigns in Uganda during the late 1980s and early 1990s resulted in larger reductions in the incidence of HIV infection for highly educated young men and (particularly) women than for their counterparts with less schooling, with increased condom use playing a key role. Although alternative explanations cannot be completely ruled out, the

[50] Conversely, when choosing between ($3,000, 1) and ($4,000, 0.8) both the prize amounts and probabilities are dissimilar so that other criteria (like maximizing expected payouts) will be used.

[51] This study does not identify the direction of causation.

greater ability of highly educated persons to process the information provided in these campaigns may represent a significant source of these schooling-related differences.[52]

Rosenzweig and Schultz (1989) show that education increases the ability of couples to successfully use complicated contraceptive methods (rhythm or withdrawal) but with no corresponding differences for simpler methods (e.g. the pill or IUD). Contraceptive effectiveness also increased with schooling following unplanned pregnancies. Both results suggest that increased cognitive ability favorably influences behaviors, particularly when information is limited or idiosyncratic.

Goldman and Smith (2002) show that education increases the probability that individuals with HIV or diabetes adhere to the complicated medical regimes developed to treat these diseases and that this superior health management is linked to better outcomes.[53] Although this evidence refers to disease management, the same mechanisms seem likely to operate for health behaviors; for example, many of the measures of adherence (particularly for diabetes) are behavioral in nature.[54] They further show that the education differential in adherence to diabetes treatment largely disappears after controlling for scores on the Wechsler Adult Intelligence test (measuring high-level abstract reasoning), further indicating the key role of cognitive skills.[55]

3.5. Non-traditional Models

The models described above deviate from standard economic models of constrained maximization by adding constraints related to information or cognitive processing or by incorporating time-inconsistent preferences. However, attention has increasingly been paid, particularly in the areas of behavioral economics and neuroeconomics, to decision processes that differ more fundamentally from those traditionally used in economics. Kahneman (1994) distinguishes between "decision utility" and "experience utility." The key distinction is that individuals, when attempting to optimize based on decision utility, may incorrectly forecast the hedonic experiences (realized well-being) of different decisions (experience utility) and these errors may be systematic.

[52] Similarly, De Walque (2007b) and Grimard and Parent (2007) find that education has a negative causal effect on smoking—using IV procedures exploiting college attendance as a strategy to avoid the Vietnam war—but they are not able to say whether the effect of schooling reflects increased cognitive processing abilities or other factors, such as education-related decreases in discount rates, improvements in access to information, or wage increases.

[53] Maitra (2010) confirms that the highly educated have better diabetes treatment adherence but raises questions about the extent to which this explains better self-reported health.

[54] For instance, Goldman and Smith (2002) show that highly educated diabetics self-monitored their blood glucose and self-tested their blood or urine more frequently than their counterparts with less schooling.

[55] Less educated persons also have higher rates of undiagnosed diabetes, controlling for health insurance coverage (Smith, 2007), which is suggestive of a behavioral response although it could also reflect differences in access to care. Lleras-Muney and Lichtenberg (2005) show that educated individuals tend to use more recently developed drugs, particularly in cases where learning is required (e.g. when drugs are repeatedly purchased to treat a medical condition), and that these effects are unlikely to result from differences in insurance or access to medical care.

Kahneman and Thaler (2006) offer the example of someone grocery shopping while hungry: she may buy overly large quantities of food because her current hunger leads her to overestimate the experience utility of eating in the future.

A possible reason for systematic errors is that decisions are influenced by immediate emotional experiences, called "visceral factors" by Loewenstein (2000), such as anger, fear, thirst, hunger, or sexual desire. Loewenstein argues that such factors have been traditionally discounted by economists because they fluctuate rapidly (although often in highly predictable ways) and because their impact is underestimated during "cool states" when individuals are not under their influence. In "hot" states, where visceral factors are operative, individuals "who otherwise display 'normal' decision-making behavior. . .behave in ways that give the appearance of extreme discounting of the future" (p. 430).[56] In Laibson's (2001) cue theory of consumption, repeated pairing of a cue and a consumption good eventually creates complementarity between the cue and consumption of the good (i.e. the presence of the cue raises the marginal utility of consumption). He gives examples such as the smell of baking cookies and the sound of ice falling into a whiskey tumbler. Thus, cues can generate cravings in addicts and can be used in marketing to increase the consumption of food or alcohol. In contrast to the TORA, this model predicts high-frequency variations in craving and marginal utility, which can lead to seemingly random patterns of consumption. (Although Laibson notes that "cue effects can be captured using minor variants" of the TORA; see p. 82.) An important implication of the cue theory is that sophisticated consumers will actively engage in strategic cue management, which is consistent with the philosophy of, for example, Alcoholics Anonymous that its members must avoid people and locations (and, of course, the sight or smell of alcohol) that could be cues that would lead to cravings and falling off the wagon. This theory also implies that there are negative externalities associated with cues, with consequent implications for policy to ensure that the production of cues does not exceed the level that would maximize social welfare.

A number of economists have used models of "multiple selves" to characterize decision-making processes. Thaler and Shefrin (1981) postulate that consumer behavior represents an internal battle between a farsighted "planner," who values utility received in the distant future, and a myopic "doer," who prefers immediate gratification. The doer controls decisions but can be constrained by the farsighted planner through expenditures of (costly) willpower, (costly) precommitment devices restricting the choices available to or the trade-offs faced by the doer, or other techniques for achieving self-control (e.g. rules, mental accounting, and framing). Similarly,

[56] For example, Lerner and Keltner (2001) provide evidence that anger (but interestingly not fear) is associated with more risk-taking choices. Ariely and Loewenstein (2006) find that the willingness to engage in unsafe sex or in morally questionable behavior to obtain sexual gratification is higher for sexually aroused than non-aroused persons and that individuals poorly predict how sexual arousal will influence their behavior.

Fudenberg and Levine (2006) view decision problems as a game between a long-run patient self and a series of short-run impulsive selves. The long-run actor may again choose self-control actions influencing the utility function of the myopic self, even though short-run costs must be incurred to reduce the future cost of self-control. Brocas and Carrillo (2008) use a principal–agent approach in which the individual is split into a myopic but informed system (the agent) and a forward-looking but uninformed system (the principal) who maximizes the expected utility of all of the agents. Because the principal lacks complete information, she will optimally delegate certain choices to the agents but not others. For instance, the principal will offer the agent pairs of positively correlated labor supply-consumption choices that limit the consumption of "tempting" goods" but otherwise leave the agent free to make consumption decisions. This, for example, may take the form of strict prohibition on the consumption of addictive products, while allowing complete freedom in the use of those that are non-addictive.[57]

3.5.1. Brain Structure and Decision Making

Many of the models just discussed link decision making to the structure of the brain. In Thaler and Shefrin (1981), decisions of the "planner" primarily reside in the prefrontal cortex, while those of the "doer" occur in the more primitive limbic system (described below). Fudenberg and Levine (2006) similarly appeal to evidence that short-term impulsive behavior and long-term planned behavior occur in different parts of the brain. Brocas and Carrillo (2008) explicitly appeal to neuroscience evidence of multiple brain systems that split individual decision making into two processes.

The following elements of brain anatomy provide insight into these characterizations.[58]

- The human brain evolved by *adding* new capabilities rather than *replacing* those previously existing. The brain stem and cerebellum, which developed first, control autonomic functions such as heartbeat and breathing. Surrounding this is the limbic system (the amygdala, thalamus, hypothalamus, and hippocampus), which coordinates sensory inputs to generate subjective feelings and drives states like anger, pleasure, and aggression. The neocortex, which came last, consists of the occipital, parietal, temporal and frontal lobes (that deal with sensory processing), and the prefrontal cortex which is the locus of abstract thinking, conceptualization, and planning.
- The limbic system responds to cues and stimuli without accounting for the long-term consequences of current actions, whereas the deliberative system, located in the neocortex, involves higher cognitive processes that do consider long-term consequences. The limbic and deliberative systems operate in parallel to yield

[57] In a somewhat related model of "temptation utility," Gul and Pesendorfer (2001) show that individuals may choose to limit the available choice set so as to avoid needing to exercise costly self-control.

[58] This discussion draws heavily on Ruhm (2010), which in turn is based on material in MacLean (1990), Massey (2002), Bernheim and Rangel (2004), Loewenstein and O'Donoghue (2004), and Camerer et al. (2005).

differences in perception and memory, so that emotional feelings exist independently of rational assessments. We argue that many decisions about health behaviors involve an interaction of rational calculations with processes based on emotions, chemical responses, and feelings.

- The limbic system often acts upon external stimuli *before* deliberative processes take place in the neocortex. The number of neural connections running from the limbic system to the cortex also far exceeds those in the reverse direction, suggesting that emotional impulses frequently overwhelm cognitive processes.

Bernheim and Rangel (2004) draw on this neuroscience evidence in developing their model of cue-triggered decision processes. Specifically, they assume the brain contains a hedonic forecasting mechanism that learns from experience. However, the consumption of addictive substances interferes with this normal learning process by acting directly on the limbic system. Over time this system will be activated upon the presentation of the cues and the individual will enter a "hot" mode in which rational utility-maximization processes are bypassed. When the individual is in a "cold" mode, he makes decisions rationally with recognition of future consequences but in hot modes these cognitive processes do not operate.[59] The sophisticated decision maker is aware of this and rationally chooses a lifestyle that reduces the possibility of being cued into hot modes.

This model differs from the TORA in a number of ways. Most importantly, consumption of the addictive good is frequently viewed to be a mistake because stochastic shocks (i.e. encountering cues that trigger hot modes) cause decisions to diverge from rationality.[60] Cue-triggered addiction can also account for behaviors such as intentional use followed by half-hearted and later concerted attempts at abstention, or intentional recidivism.

One could alternatively model behavior as the result of multiple simultaneously operating brain systems, without an extreme division into hot and cold modes. For example, in Loewenstein and O'Donoghue (2004), decisions reflect the interaction between the cognitively sophisticated deliberative system (located in the prefrontal cortex) and a rapidly responding affective system (occurring in more primitive brain structures).[61] The affective system primarily controls behavior but the deliberative system exerts influence through the use of costly cognitive effort or willpower.[62] Exposure to (potentially learned) cues and stimuli can trigger affective system

[59] Metcalfe and Mischel (1999) provide an earlier and less formal treatment of the hot and cool states of decision-making.

[60] Addiction also does not necessarily require adjacent complementarity in this model.

[61] Similar decision processes are modeled in the psychological literature without explicit linkages to brain function. For example, in cognitive-experiential self-theory, information is processed by "experiential" and "rational" systems that operate in parallel and are interactive: the experiential system is automatic, preconscious, rapid, and non-verbal; the rational system is analytic, deliberative, slow and affect-free (Epstein, 2003).

[62] Thaler and Sunstein (2009) refer to these as the "automatic" and "reflective" systems.

responses but, in contrast to "hot/cold" models, the deliberative system almost always exerts at least some influence. The standard model of rational choice corresponds to the special case in which the deliberative system is in full control (i.e. exerting cognitive effort is costless) and hot states occur when the affective system is in full control (i.e. exerting cognitive effort has infinite cost).

This model provides insight into a variety of behaviors. First, individuals may simultaneously do one thing while wishing they were actually doing another (e.g. "I should not be eating this donut"). Second, actions may frequently be influenced by transitory emotional states that result from affective system stimuli. Third, hyperbolic discounting and preference reversals occur naturally because future planning reflects deliberative processes but immediate decisions are strongly influenced by the affective system. Fourth, discount rates may vary across types of consumption (because affective system responses will be more important for some types than others) and situations (depending on how strongly the affective system has been triggered and how much willpower has been depleted). Fifth, the model can explain phenomena such as loss aversion (if the affective system weights losses more heavily than gains) and non-linear probability weighting (individuals may consistently overestimate the probability of unlikely events and underestimate the probability of likely events) that are difficult to reconcile with standard utility maximization.

Ruhm (2010) has developed and applied a variant of Loewenstein and O'Donoghue's model in his examination of overeating and obesity. In his model, food consumption is influenced by the affective and deliberative systems and overeating has become more common over time partly because of lower food prices (as in traditional economic models) and partially because of the greater sophistication of "food engineering" whereby food products are increasingly designed to appeal to the affective system.

3.5.2. Behaviors Difficult to Explain using Traditional Models

Many aspects of generally observed behavior are difficult to reconcile with the traditional economic models in which decision makers are fully rational.[63] For example, Bernheim and Rangel (2004) point out that addicts typically describe their substance use to be a mistake even, in some instances, while they are in the act of taking the drug.

The data also suggest that the use of a single discount rate—whether exponential or hyperbolic—is unlikely to adequately describe many aspects of decision making. For instance, Frederick et al. (2002) summarize a large body of evidence indicating that: gains are discounted more than losses (loss aversion); small amounts are discounted more than large amounts; improving sequences are preferred to worsening sequences; and discount rates differ dramatically across situations and types of

[63] Although this section focuses on non-rational behavior, the consequences will, in practice, often be difficult to distinguish from non-exponential (e.g. hyperbolic) discounting. Either can cause, for example, self-control problems.

consumption.[64] Many discounting "anomalies" can be explained if decision making is based on multiple brain systems. Evidence that such brain modularity is important has been obtained from patients with brain damage. For example, persons with deficient limbic system function are less able to engage in gradual learning or to acquire conditioned responses to emotional stimuli, while individuals with damage to their prefrontal cortex exhibit impaired decision making, with a particular inability to act on long-term goals (see Loewenstein and O'Donoghue, 2004; Camerer et al., 2005; or Brocas and Carrillo, 2008 for useful discussions of this literature). Some neuroscience evidence obtained from magnetic resonance imaging (MRI) supports the possibility that multiple-system models of decision making arise directly from the structure of the brain (e.g. McClure et al., 2004); however, there remains ambiguity about these MRI results (e.g. see Glimcher et al., 2007).

The empirical data also suggest that the deliberative system exerts less power when cognitive processing resources are limited (when self-control is depleted) than when it is not. For example, Shiv and Fedorikhin (1999) describe an experiment in which subjects are asked to memorize either a two-digit or a seven-digit number and were then asked to choose a snack: either chocolate cake or fruit salad. Those who were asked to memorize a seven-digit number were more likely than those asked to memorize a two-digit number to request the chocolate cake (this was particularly true for those with high self-rated measures of consumer impulsivity), which the authors interpret as evidence that greater cognitive processing demands increase the likelihood that choices are driven by lower-order affective reactions rather than by higher-order processes like thinking or reasoning. More recently, Vohs et al. (2008) have shown that the cognitive effort involved in making choices reduces self-control among a variety of dimensions.[65]

The frequent use of precommitment or other self-control devices is cited as evidence of time-inconsistent preferences of which agents have at least some awareness. For example, Gruber and Köszegi (2001) highlight the use of socially managed incentives to reduce smoking, such as announcing a New Year's resolution to quit smoking to create embarrassment for oneself if one resumes smoking. Ruhm (2010) emphasizes the increasing frequency of bariatric surgery, which can be viewed as an extremely strong precommitment strategy. The drug Antabuse is another market-generated precommitment device: by taking it in the morning an alcoholic can ensure that if he consumes alcohol later in the day he will be made ill by the interaction of the drug

[64] Consistent with this, Cutler and Glaeser (2005) show that within-person correlations across health behaviors are quite low both at a point in time and when looking at behavioral changes across time. This variation results in part from genetic factors but behavior-specific situational influences are also important.

[65] They examine how making choices among consumer goods or college course options were related to physical stamina, persistence, procrastination, and the ability to perform cognitive calculations. In each case, the group required to make choices experienced greater reductions in self-control or cognitive processing ability.

with the alcohol. A key distinction is that rational addicts may pay for technologies that help them quit or weaken their addiction but they should not be willing to pay to limit their future choices. Conversely, under alternative models, agents frequently make choices they will subsequently regret and so, under some circumstances, may choose to voluntarily constrain their future options.

Experimental and non-experimental evidence suggests that individuals recognize their time-inconsistent preferences and act strategically to at least partially overcome them. Using data from three health clubs, Della Vigna and Malmendier (2006) compare the behavior of members to non-members, where the latter are allowed to use the clubs by paying for each visit. They find that members have higher per visit average costs and overestimate their future attendance. Those with monthly memberships are more likely, than annual members, to stay enrolled beyond one year (despite paying a higher fee for the flexibility to quit each month) and their attendance declines over time, in contrast to the increases observed for annual members. The researchers believe that overconfidence about future self-control provides the most likely explanation for these results.

Considerable ingenuity is required to distinguish between rational and non-rational use of addictive products. Most previous "tests" of the TORA identify forward-looking behavior without distinguishing between complete or partial foresight, or between time-consistent preferences and self-control problems. Gruber and Köszegi (2001) emphasize that both the TORA and models of hyperbolic discounting imply responsiveness of current consumption to anticipated future price changes and the authors show that forward-looking behavior by smokers need not indicate rational behavior.[66]

Because such direct tests are difficult to obtain, researchers have begun to use indirect evidence to distinguish between the two classes of models. Using data from the United States and Canada, Gruber and Mullainathan (2005) find that the happiness of smokers is increased when taxes are raised, consistent with sophisticated awareness of time-inconsistent preferences but probably not with rational addiction (because that model implies that higher prices reduce utility). Similarly, Kan's (2007) IV estimates suggest that Taiwanese smokers who intend to quit are relatively supportive of smoking bans or cigarette tax increases. This is interpreted as evidence of a demand for self-control devices that are predicted with time-inconsistent preferences and forward-looking behavior.[67] Ruhm (2010) tests a variety of predictions of a two-system model

[66] Specifically, they find that announced future tobacco taxes have a positive effect on current cigarette *sales*, a result that suggests forward-looking behavior as consumers stock up prior to price hikes in order to save money. However, current *consumption* (not sales) appears to decline in response to future tax increases, as predicted by both TORA and less than fully rational models in which consumers have some foresight.

[67] However, the appropriateness of the instruments (awareness of the health risks of smoking and weight loss attempts) is questionable as quit attempts may increase the disutility of second-hand smoke, prompting support for these measures.

of overeating and obesity, with food engineering, against a standard model with full rationality. Among the most important predictions are that the frequency of eating mistakes—as evidenced by weight loss attempts—and of the consumption of engineered foods (that are high in fat and salt) will have increased over time, particularly for heavy individuals, and that actual weight will have increased over time without an accompanying rise in desired weight. All of these predictions are borne out using data for adults in the US.

3.6. Short-term Effects

The discussion on health behaviors to this point has emphasized long-term factors and influences. (For example, education is anticipated to have lasting effects on decisions.) However, health behaviors are also influenced by short-term factors, including some that are poorly explained by many standard models. We discuss two examples below.

3.6.1. Full Wallets Hypothesis

According to the permanent income hypothesis (PIH), short-term changes in income should have little influence on consumption decisions because spending is based on "permanent" rather than current income (Hall, 1978). However, liquidity constrained individuals may not be able to smooth consumption as the PIH model predicts; moreover, decision makers with time-inconsistent preferences, or operating using the non-traditional models described above, may not even try to smooth consumption. The empirical evidence indicates that violations of PIH are common and that even very short-term changes in income affect a variety of types of consumption, including many health behaviors. This is sometimes referred to as the "full wallets" hypothesis.

Stephens (2003) showed that for persons receiving a major portion (at least 70 percent) of their income from Social Security, expenditures on "instantaneous consumption goods" (expenditures on food away from home and entertainment) increase by 33 percent on the day Social Security checks are received and by 35 percent on the next day, relative to average spending. The interpretation is that consumers have "full wallets" immediately after receiving their checks, which results in higher spending because consumers are liquidity constrained at other times of the month.[68] A similar responsiveness to the receipt of pay checks has been observed in the United Kingdom (Stephens, 2006) and to the receipt of food stamp benefits in the United States (Wilde and Ranney, 2000).[69]

[68] Such behavior could occur without liquidity constraints if retailers discount prices at the beginning of the month (when Social Security checks are received). Instead, Hastings and Washington (2010) show that food prices decline slightly over the month (by about 3 percent between the first and fourth weeks of the month) and that almost all of the change in food expenditures is due to variation in the quantities of food purchased.

[69] Energy intake also falls at the end of the "food stamp month" for those who shop infrequently.

Shapiro (2005) expands on this last result, showing that caloric intake declines 10 to 15 percent over the food stamp month. This is interpreted as evidence for quasi-hyperbolic discounting because, under exponential discounting, an annual discount rate of 146 percent would be needed to explain these results.[70] Mastrobuoni and Weinberg (2009) find even greater evidence of declining within-month food consumption for persons receiving 80 percent or more of their income from Social Security and who have little or no savings: annual discount factors are 0.08 for these persons, compared to flat consumption profiles for recipients with savings. The probability of consuming less than the recommended daily calories also increases dramatically toward the end of the benefit month for the former group but not for the latter.

The potential food insufficiency described above could have deleterious health consequences for at least some individuals. However, most detrimental consequences of short-term changes in income appear to work in the opposite direction (i.e. "full" wallets are more harmful than "empty" wallets). Riddell and Riddell (2006) show that intravenous drug users are much more likely to be admitted to Vancouver hospitals with overdoses in the two days after welfare checks are received than at other times of the month.[71] Dobkin and Puller (2007) obtain similar results for drug-related admissions, particularly for cocaine overdoses, among California recipients of Supplemental Security Income (SSI) or Disability Income (DI). Interestingly, they do not find a corresponding "pay check" effect, suggesting that the consequences across population segments may be heterogeneous.[72]

Such spikes in drug use due to full wallets have severe negative health consequences. Riddell and Riddell (2006) show that the death rates of SSI beneficiaries increase 22 percent on the day of benefit receipt—generally the first of the month. Mortality also increases at the beginning of the month for other reasons. Phillips et al. (1999) find that the overall US death rate rises by 0.9 percent in the first week of the month due to large increases in mortality from substance abuse (13.8 percent) but also because of increases in mortality due to homicide, suicide, other external causes, motor vehicle accidents, and liver disease with mention of alcohol (6.5, 5.3, 4.6, 2.8 and 2.6 percent, respectively).[73] They also uncover evidence of smaller (less than 1.0 percent) but still significant first-of-the month effects for deaths due to respiratory or circulatory disorders, neoplasms, and liver disease without mention of alcohol.

[70] Evidence is also provided that the implied discount rates increase dramatically (0.24 percentage points per day) over the food stamp month.

[71] These effects may be reinforced by cue-driven behavior (as in the models of Laibson, 2001 or Bernheim and Rangel, 2004 discussed above) because the majority of drug users in Vancouver live in close proximity of each other.

[72] Also, the SSI/DI impact on alcohol-related hospital admissions is smaller than that for other drugs—possibly because acute health problems are less common for alcohol.

[73] They point out that alcohol or substance abuse seems likely to play an indirect role in many deaths from some of the other sources (e.g. homicide and suicide).

Using data from the 1973–2005 Multiple Cause of Death Files, Evans and Moore (forthcoming) confirm that mortality increases by 0.9 percent in the first week of the month, relative to the preceding week. This is due in part to a 3.0 percent rise in deaths due to substance abuse (versus a 0.8 percent increase in other fatalities) but because substance abuse deaths are relatively rare, the absolute within-month fluctuations in mortality are much greater for deaths from non-substance-related causes.[74] They also show that first-of-the-month increases in various types of consumption (e.g. food and non-food items, lottery tickets, movie box office receipts, as well as foot traffic at malls, retail and apparel establishments) are greatest for the groups most likely to be liquidity constrained (the less educated, government transfer payment recipients, low-income households), and that within-month variation in mortality declines with education. Their overall conclusion is that deaths rise at the beginning of the month because many inherently risky activities increase when liquidity constrained consumers have full wallets.

3.6.2. Macroeconomic Fluctuations and Health Behavior

Many aspects of health-related behaviors exhibit a counter-cyclical variation. Evidence that mortality is procyclical dates back more than 80 years (Ogburn and Thomas, 1922; Thomas, 1927) but it is only in the last 15 years that these patterns, and the mechanisms for them, have begun to be understood. A major empirical innovation has been the use of data containing multiple geographic locations observed at several points in time, allowing the use of panel data methods, in particular the inclusion of location-specific fixed effects and general time effects.[75]

In a series of papers, Ruhm has provided evidence that, when economic conditions (typically proxied by unemployment rates) weaken, heavy drinking and drunk-driving, smoking, obesity, and physical inactivity decrease and diets improve (Ruhm, 1995, 2000, 2005a; Ruhm and Black, 2002). Two potential mechanisms for these effects have been highlighted. First, income reductions during bad economic times appear to reduce some types of unhealthy consumption (e.g. drinking), as in the discussion of full wallet effects above. Second, some healthy behaviors (e.g. exercise) are time intensive and work hours are generally procyclical.

Other research examining these health behaviors, using similar techniques, generally also finds that behaviors become healthier in bad times. Evidence that alcohol sales and driving problems decline during a weak macroeconomy has been provided by Evans and Graham (1988), Ettner (1997), and Freeman (1999), among others; Dee (2001) uncovered a drop in alcohol use and heavy consumption, but also an increase

[74] They estimate that there are 647 extra deaths from substance abuse during the first week of the month, compared with 3,636 additional fatalities from other sources.

[75] Ruhm (2008) provides a detailed discussion of these issues, and a review of the related empirical literature.

in binge drinking.[76] Gruber and Frakes (2006) verify the decline in smoking and Courtemanche (2009) shows that shorter work hours decrease obesity because they are associated with increases in exercise and reduced consumption of fast food and prepared processed foods. Xu and Kaestner (2010) use an instrumental variables approach to show that the lower work hours occurring during economic downturns cut smoking and increase exercise while Edwards (2008) indicates that having more non-work time increases sleeping, socializing, and time spent caring for the elderly. On the other hand, Charles and DeCicca (2008) find that obesity and BMI rise for men with low *ex ante* probabilities of employment.[77]

Interestingly, better health during economic downturns occurs even though screening tests (mammograms, pap smears, and colorectal exams) are less often received (Ruhm, 2000) and doctor visits and hospital episodes decrease (Ruhm, 2003; Xu and Kaestner, 2010). However, there are exceptions. Dehejia and Lleras-Muney (2004) find that pregnant women obtain earlier and more extensive prenatal care when the economy is weak and Ruhm (2007) shows a similar increase in sophisticated treatments for heart disease (e.g. coronary bypass and angioplasty) among senior citizens.

The macroeconomic variations in health behaviors provide one reason why mortality is procyclical. Research using the empirical methods just discussed generally predicts that a 1 percentage point increase in unemployment reduces total mortality by 0.3 to 0.5 percent, with a similar decline in deaths from coronary heart disease and much larger decreases in deaths from external causes (particularly traffic accidents); conversely, cancer mortality changes little because such fatalities are unlikely to respond to short-term changes in behaviors.[78]

It is not obvious whether these behavioral changes represent rational or non-rational responses to changing incentives. On the one hand, individuals will optimally substitute labor supply from periods when wages are low to those when they are high, implying that it may be optimal to devote less time to health-enhancing behaviors when the economy is robust, even if doing so increases the risk of death. On the other hand, Evans and Moore (2009) show that the causes of death with high macroeconomic fluctuations are the same ones that exhibit large within-month variations, presumably due to full wallets effects that are inconsistent with intertemporal optimization.

[76] However, Arkes (2007) finds that drinking and drug use by teenagers is counter-cyclical.

[77] More complete discussions of this literature, including evidence for countries outside the United States, are provided by Ruhm (2006) or Ruhm (2008).

[78] For more detailed reviews of previous research examining how macroeconomic conditions influence mortality, see Ruhm (2005b, 2006, 2008).

4. ECONOMIC CONSEQUENCES OF HEALTH BEHAVIORS

We next discuss the challenges to identifying the causal impact of health behaviors and summarize the substantial body of research examining such consequences for a variety of outcomes including: medical care costs, education, employment, wages, and crime.

4.1. Reasons to (and not to) Conduct Cost of Behavior Studies

Cost of Illness (COI) studies calculate the difference in medical costs between those with and without a specific medical condition, controlling for observable characteristics. Such analyses can be conducted not just for diseases like cancer and diabetes but also for health behaviors such as alcoholism, smoking, drug abuse, and a sedentary lifestyle; we refer to these as Cost of Behavior (COB) studies.

Costs can be divided into direct payments for medical care and indirect costs for which no payments are made—such as productivity losses due to job absenteeism or premature death. Public health advocates use COB studies to lobby for greater expenditures to improve health behaviors. However, such arguments are often circular. For example, some health behaviors have substantial medical resources devoted to them, and thus a high COB, but that does not necessarily justify still greater spending (see, e.g., Shiell et al., 1987). Because medical expenses arise from the decision to treat the outcomes of unhealthy behaviors, a simplistic (but not necessarily desirable) way to reduce these costs is simply to refuse to treat such consequences. Another limitation is that COB studies tend not to consider marginal effects (of expanding or reducing the scale of interventions) but instead assume that unhealthy behaviors can be completely eradicated (Shiell et al., 1987).[79] Given these shortcomings, it is sometimes argued that health economists should devote their efforts not to COI or COB studies but instead to studying the cost effectiveness of specific interventions designed to change health behaviors (e.g. Roux and Donaldson, 2004).

There are, nevertheless, several situations where COB studies may be of interest. First, they indicate the amount of medical resources currently devoted to treatment—e.g. how much does the US spend treating alcoholism? Second, they enhance our understanding of disparities across gender, race, or income. Third, they provide help in calculating external costs, which may justify government intervention (Zohrabian and Philipson, 2010; Baumol, 1972). Fourth, they represent one input into cost-effectiveness analyses of candidate interventions, by providing estimates of the value of avoided unhealthy behaviors.

[79] Still another potential difficulty is in distinguishing between annual and lifetime costs. For example, Fang and Gavazza (2007) provide evidence that greater investments in medical care prior to age 65 are associated with reduced medical expenditures after that age, along with commensurate reductions in total lifetime spending.

4.2. Challenges to Identifying the Consequences of Health Behaviors

It is critically important to accurately estimate the causal effects of health behaviors on outcomes (e.g. medical care costs and wages). However, it is frequently the case that only correlations are estimated, which provide limited information because they reflect three factors: (1) the causal impact of unhealthy behaviors on outcomes; (2) the impact of poor outcomes on unhealthy behaviors (*reverse causation*); and (3) the influence of omitted variables that affect both unhealthy behaviors and poor outcomes (*confounding*).

In order to estimate the causal effect of unhealthy behavior on an outcome of interest, the most convincing research design would randomly assign large numbers of otherwise similar individuals into treatment and control groups, with the treatment group then compelled to engage in unhealthy behaviors. Comparing differences in outcomes between the treatment and control groups would then generate a consistent estimate of the impact of unhealthy behaviors. Fortunately for subjects, and unfortunately for researchers, such randomized experiments are neither ethical nor feasible. As an alternative, economists frequently seek out "natural experiments" that exploit exogenous variation in health behaviors; i.e. not the result of reverse causation (e.g. poor labor market outcomes causing unhealthy behaviors) or confounding (e.g. differences in risk aversion or rate of time preference).

Such natural experiments are often exploited using the method of instrumental variables (IV). However, the instruments (i.e. the natural experiments) must be both powerful and valid.[80] In terms of power, a rule of thumb is that the F statistic for the null hypothesis that the coefficients on the instruments are jointly equal to zero, in the first stage of two-stage least squares, should be 10 or higher (Stock et al., 2002). There is no simple convincing test of instrument validity (see, e.g., French and Popovici, 2011). With multiple instruments, over-identification tests can be conducted.[81] However, such over-identification tests are only reliable when the instruments are both powerful and valid (French and Popovici, 2011; Wooldridge, 2002).[82] As a result, McCloskey (1998) argues that instruments are ultimately accepted (or rejected) based on rhetoric— the author must make a convincing logical argument for their validity. French and Popovici (2011) observe that IV studies of the consequences of health behaviors conducted during the 1990s often used intuitive or

[80] Angrist and Krueger (2001) and Angrist and Pischke (2009) summarize theoretical and practical difficulties associated with using instrumental variables to identify causal effects; Auld (2006) and Auld and Grootendorst (2011) examine these challenges in the context of health behaviors.

[81] For instance, the two-stage least squares residuals can be regressed on all exogenous variables (the instruments and other regressors); the F statistic testing the hypothesis that the coefficients on the instruments are jointly equal to zero is then computed.

[82] More generally, the requirements of power and validity interact in that the bias resulting from an invalid instrument is greater the weaker it is. With sufficiently weak instruments, bias may be larger in IV than OLS estimates and the "cure can be worse than the disease" (Bound et al., 1995).

theoretic arguments in favor of instrument validity, whereas those published in the 2000s papers relied more heavily on statistical evidence.

French and Popovici (2011) summarize 60 studies, published between 1990 and 2009, that use IV methods to measure the impact of using alcohol, illicit drugs, and tobacco on a variety of economic outcomes. They note that economists have used the following variables as instruments for risky behaviors: family history of risky behaviors, religiosity, and state policies affecting access and taxes. However, they note that these instruments are now more widely regarded as potentially invalid because of unobserved heterogeneity and policy endogeneity.

Prices of, or taxes on, addictive substances may be valid instruments for their consumption, but power may be lacking. Other, more recently used instruments for risky behaviors are described in subsequent sections. As in many fields of economics, the search for powerful and valid instruments is ongoing in this literature.

In considering the impacts of health behaviors, it is important to note that some behaviors (like smoking) are never health enhancing. In contrast, increased calorie consumption can reduce the risk of mortality if one is underweight (Flegal et al., 2005, 2007) and moderate consumption of alcohol may improve cardiovascular health (Cook, 2007). Such non-linear effects of consumption represent an additional challenge for empirical work measuring the consequences of healthy behaviors.

4.3. Impacts on Medical Care Costs

Relatively few studies have used econometric techniques to measure the *causal* effect of health behaviors on medical care utilization or costs.[83] In contrast, many investigations have, in the tradition of COI studies, estimated the correlation between behaviors and medical costs. For instance, Dorothy P. Rice, one of the pioneers of COI studies, quickly moved from estimating the direct costs of conditions such as cancer and AIDS to estimating the direct costs of health behaviors such as smoking, alcohol abuse, and drug abuse.

Many studies have examined the medical care costs of smoking; Sloan et al. (2004) identifies at least 165 published between the 1960s and 2002. For example, Rice et al. (1986) calculated that $14.4 billion was spent in the US in 1980 treating smoking-related illness. In an unusually detailed and careful analysis, Sloan et al. (2004) calculate that smoking at age 24 is associated with $3,757 higher lifetime medical expenditures for women and $2,617 greater expenditures for men (in year 2000 dollars).

Rice et al. (1991) estimated that the direct medical care costs of alcohol abuse were $6.8 billion and those related to drug abuse were $2.1 billion in 1985. Cook (2007) reviews COB studies of alcohol abuse, the most recent of which (Harwood,

[83] Other methods, such as propensity score matching, could be used to estimate the causal effect of risky health behaviors on medical care costs, but to our knowledge have not yet been used for this purpose.

2000) calculates that the medical consequences of alcohol abuse (including fetal alcohol syndrome) totaled $19 billion in 1998. The Office of National Drug Control Policy calculates that $15.8 billion was spent on medical treatments and prevention of drug abuse in 2002 (Office of National Drug Control Policy, 2004). French et al. (2000) compared self-reported health service utilization among drug users and non-users and calculate that chronic drug users and injecting drug users generated $1,000 per year in excess health services utilization relative to non-drug users. Finkelstein et al. (2009) analyze data from the Medical Expenditure Panel Survey (MEPS) and calculate that, in 2006, obese people (i.e. those with a body mass index of 30 or higher) had medical spending that was $1,429 (in 2008 dollars) or 41.5 percent higher than that for healthy-weight people (those with a body mass index of 18.5 to 25). They calculate, across all payers, $85.7 billion (in 2008 dollars) was spent treating obesity in 2006, which represents 9.1 percent of all medical spending that year. To reiterate, each of these studies estimates the correlation of health behaviors with medical care costs, not the causal effect.

A smaller number of studies use IV methods to estimate the causal effect of health behaviors on medical care utilization and costs. McGeary and French (2000) use access to drug markets, neighborhood sightings of intoxicated individuals, and drug sales as instruments for chronic drug use.[84] They estimate that chronic drug use raises the probability of an emergency room visit by 30 percent for females and 36 percent for males. Balsa et al. (2008) examines how alcohol consumption affects health care utilization, instrumenting for alcohol consumption using state alcohol and drug policies and other state characteristics (including, curiously, average precipitation). They are unable to reject the exogeneity of alcohol consumption and thus prefer their non-IV estimates which show that moderate drinking decreases the likelihood of emergency room visits for both sexes and hospitalizations for women but not men. Cawley and Meyerhoefer (2010) estimate the impact of obesity on medical care costs, using obesity status of a biological child to instrument for weight of the parents. Obesity is found to raise annual medical care costs by $2,741 (in 2005 dollars), which is more than four times as large as the corresponding OLS estimate ($676). They hypothesize that OLS results suffer attenuation bias due to measurement error in self-reported weight.

An important direction for future research is to obtain more comprehensive and reliable estimates of the causal effects on medical costs of health behaviors.

4.4. Impacts on Education

The effects of alcohol consumption on educational outcomes have been frequently studied (see the meta-analysis by Lye and Hirschberg, 2010). For instance, the IV

[84] The validity of these instruments is questionable if they are correlated with unobserved socioeconomic status.

estimates of Renna (2007) suggest that binge drinking decreases the probability of graduating high school (by age 19) by as much as 5.2 percent for women and 14.5 percent for men.[85] Using state policies as instruments for drinking, Chatterji (2006a) finds that high school alcohol use has little effect on educational attainment. Cook and Moore (1993) instrument for alcohol consumption using cross-state variation in minimum legal drinking age and conclude that high school seniors who are frequent drinkers (or frequently drunk) eventually complete 2.2 fewer years of college. Dee and Evans (2003) disagree with using across-state variation in such laws out of concern for unobserved heterogeneity across states. Instead, they estimate two-sample IV models in which teen drinking is instrumented using within-state variation in minimum legal drinking ages, and they conclude that teen drinking has no detectable effect on high school completion, college entrance, or college persistence. Koch and Ribar (2001) instrument for age of first alcohol use with sibling age of alcohol initiation, and conclude that delaying the start of drinking has small effects on educational attainment—a one-year delay in onset is predicted to raise completed education by roughly one-tenth of a year. On the whole, these findings suggest that heavy alcohol consumption by youths decreases educational attainment, but that moderate use may not have a detectable effect.

IV methods have been less commonly used to estimate the impact of drug use on educational outcomes. Chatterji (2006b) uses state drug policies and middle school characteristics as instruments for drug use during high school but uncovers no evidence of significant effects on educational attainment. This may be due in part to weak instruments.

Two recent investigations—Fletcher and Lehrer (2009) and Ding et al. (2009)—use genetic markers associated with brain chemistry to instrument for obesity and other health conditions when examining how the former is related to educational outcomes. However, this requires the unattractive identifying assumption that genes that affect brain chemistry do not affect educational attainment, except through obesity and other regressors; in fact, the specific genes used as instruments in these papers have been linked to many other conditions that could also affect education, such as alcoholism, schizophrenia, aggression, and violence (see Cawley et al., 2011). Notwithstanding this caveat, Fletcher and Lehrer uncover little evidence that being overweight during adolescence influences completed years of schooling; however, Ding et al. conclude that obesity lowers grade point average (GPA) by 0.45 (roughly one standard deviation).

Sabia (2007), when using parental obesity status to instrument the weight of the respondent, finds that a 50 to 60 pound (approximately two standard deviation) weight increase reduces the GPA of white females by 8 to 10 percentile points (e.g.

[85] For women, binge drinking is instrumented using state alcohol taxes and state minimum legal drinking ages. Those variables do not strongly predict binge drinking by men, so religiosity and whether a parent was a problem drinker are instead used for men. However, these are of questionable validity as they may directly affect education.

from the median to around the 40th percentile) with little evidence of effects on the GPA of white males or non-whites.

4.5. Impacts on Employment

The most frequently studied consequences of health behaviors are labor market impacts such as a lower probability of employment or lower wages. For example, a large literature examines how alcohol consumption affects employment. Mullahy and Sindelar (1996) instrument for problem drinking using state beer and cigarette taxes, and per capita ethanol sales. Their estimates suggest that problem drinking is associated with statistically insignificant reductions in the probability of employment. Using the same data and similar specifications but allowing for non-linear effects, Terza (2002) finds that the reduction in employment associated with problem drinking is statistically significant. In contrast, instrumenting for problem drinking using indicator variables for whether county of residence is wet or dry, Feng et al. (2001) uncover a positive and significant association between problem drinking and male employment, and an insignificant association for females. Using Finnish data and a large number of instruments, Johansson et al. (2007) conclude that alcohol dependency lowers the probability of employment by 50 percent for men and 40 percent for women.[86]

Negative effects of drug use on employment have also been fairly widely documented. For instance, DeSimone (2002) instruments drug use with the regional price of cocaine and state decriminalization of marijuana, and concludes that marijuana and cocaine use lower employment probabilities by 15−17 and 23−32 percent, respectively. Similarly, French et al. (2001) find that chronic drug use lowers employment probabilities by 9 percentage points for both sexes but that light or casual use has no detectable effect. MacDonald and Pudney (2000) estimate a joint model of occupational attainment, unemployment, and drug use using data of British young adults; they find that past hard drug use and current drug use are both associated with a higher probability of current unemployment.

Studies of the impact of obesity on employment obtain mixed results. Cawley (2000) uses weight of a biological child as an instrument for the weight of the mother and finds no significant impact of weight on female employment. Morris (2007) uses the local area prevalence of obesity as an instrument and concludes that obesity reduces the employment of both males and females. Norton and Han (2008) estimate the effect of obesity on employment, instrumenting for obesity using a similar set of genetic markers related to brain chemistry as were used by Fletcher and Lehrer (2009) to study the impact of obesity on educational outcomes. Using this IV method, Norton and Han (2008) find no effect of lagged obesity on the employment of either men or women. Rooth (2009) conducted an audit study in which fictitious job

[86] The instruments include parental alcohol or mental health problems, religiosity, and respondent diabetes or asthma.

applications were submitted for real job openings. Applications sent with a photo of an obese male (female) were 6 (8) percentage points less likely to receive a job interview than non-obese counterparts.

4.6. Impacts on Income, Earnings, and Wages

Cook (2007) notes that alcohol consumption is generally positively correlated with earnings; he refers to this as the "drinker's bonus" but suspects it is the result of reverse causality or confounding. However, a number of high-quality studies suggest that the effect may be causal. Auld (2005) examines how drinking and smoking affect income, assuming that religiosity and alcohol/tobacco prices affect smoking/drinking but not income directly. His estimates suggest that moderate alcohol consumption raises income by 10 percent and heavy drinking raises it by 2 percent (relative to abstaining), while smoking reduces income by 24 percent. Van Ours (2004) instruments for drinking and smoking using dichotomous indicators of initiation before age 16 and concludes that alcohol use raises men's wages by 10 percent while smoking reduces them by the same amount; neither drinking nor smoking affects female earnings.

Early studies of the NLSY that addressed the endogeneity of drug use found mixed results, including finding positive effects of drug use on wages for at least some groups and/or specifications (e.g. Kaestner, 1991; Register and Williams, 1992; Kaestner 1994). Using data of male workers residing in Amsterdam, and with an identification strategy based on the discrete factor method, van Ours (2007) estimates that marijuana use lowers wages by around 10 percent but that cocaine use has no wage effect.[87]

The large recent literature estimating how weight affects wages generally finds an obesity earnings penalty for women (especially white females), with less consistent results for men, including sometimes a wage premium associated with being overweight (e.g. Cawley, 2004; McLean and Moon, 1980).[88] Using weight of a biological sibling as an instrument, Cawley (2004) estimates that a two standard deviation (roughly 65 pound) increase in weight is associated with 18 percent lower wages for white females; however, the OLS estimates, which Hausman tests imply are preferable, are only half as large. Using parental BMI as an instrument in a non-parametric model, Kline and Tobias (2008) uncover obesity wage penalties for British men and women. Conversely, using as instruments for obesity the genes related to brain chemistry mentioned above, Norton and Han (2008) fail to detect an obesity wage penalty for young adults of either sex. Gregory and Ruhm (2011) employ flexible functional forms to show that the wages of women begin to decline after a BMI of around 23. Since this is well within the "healthy" weight range, they speculate that this may

[87] Parental cannabis use and the presence of children are used as instruments. The absence of an effect of cocaine use on wages may be due to lack of statistical power.

[88] McLean and Moon hypothesize that among mature men, large size may signal power and accomplishment; they label this the "portly banker" effect.

reflect returns to "beauty" (Hamermesh and Biddle, 1994) rather than obesity *per se*. They also find that male wages decline at higher levels of BMI. European studies using weight of relatives as instruments also find obesity wage penalties for women but less so for males (e.g. Lundborg et al., 2007; Brunello and d'Hombres, 2007; Atella et al., 2008; Greve, 2008), although the effects of obesity vary across nations.[89]

4.7. Impacts on Crime

Substance use may lead to criminal activity, for instance by increasing aggression or lowering inhibitions; see the discussion of "hot" states in section 3. Related research spans the literatures in health economics and the economics of crime, and can generally be divided into studies directly relating substance use to crime and those evaluating how substance abuse policies or sin taxes (rather than substance use itself) are associated with criminal behavior.

French and Maclean (2006) instrument underage drinking by young adults with parental drinking problems and state beer taxes. They find that, among males (females), consuming 12 or more alcoholic drinks in the past year is associated with a 30.9 (2.8) percentage point increase in the probability of vandalizing property, a 28.9 (19.8) percentage point increase in the probability of stealing, and a 50.1 (57.2) percentage point increase in the probability of committing any illegal act.

Other studies use indirect measures of substance use. Fryer et al. (2005) find that an index of cocaine prevalence (based on the factors of arrests, emergency room visits, drug busts, and newspaper stories involving cocaine) predicts higher homicide rates among black youths. Grogger and Willis (2000) estimate that the introduction of crack cocaine into a city (based on FBI crime reports) raised urban crime rates by roughly 10 percent.

Taxes that raise the monetary cost of alcohol or drugs, and policies that increase the time cost of acquiring alcohol or drugs, are generally associated with decreases in crime. Carpenter and Dobkin's (2010) comprehensive review of research on alcohol regulation concludes that higher alcohol taxes and age-based restrictions on alcohol availability reduce crime. Markowitz (2005) finds little evidence that increases in drug or alcohol prices reduce violent victimizations but higher beer taxes do appear to reduce alcohol-related assaults.[90] Carpenter (2005, 2007) shows that zero tolerance drunk-driving laws for minors reduced male nuisance crimes such as vandalism, public drunkenness, and disorderly conduct by 1–2 percent but have no effect on violent crime. Biderman et al.'s (2009) difference-in-differences estimates indicate that homicides were reduced by 10 percent when Sao Paulo Metropolitan Area municipalities adopted laws mandating closing hours for bars and restaurants to restrict alcohol

[89] Brunello and d'Hombres find that BMI lowers earnings more in Southern than Northern Europe; Lundborg et al. (2007) find particularly large obesity wage penalties in Central Europe.

[90] The data set utilized, the National Crime Victimization Survey, provides information about whether alcohol and drugs were involved in an incident.

consumption. Dobkin and Nicosia (2009) find that after the US shut down two plants producing more than half of the precursors to production of methamphetamines, felony arrests for methamphetamines fell by half, but there were no significant reductions in property or violent crime. Weatherburn et al. (2002) find that the heroin "drought" in Australia was accompanied by a sharp increase in robberies and breaking and entering offenses, but these quickly declined.

A general methodological concern is that local crime rates are sometimes used as an instrument for drug and alcohol use (e.g. see the review in French and Popovici, 2011), whereas some of the research described here concludes that crime is a consequence of such use.

5. STRATEGIES FOR MODIFYING HEALTH BEHAVIORS

The first fundamental theorem of welfare economics states that perfectly functioning free markets are Pareto efficient and that government intervention cannot increase social welfare (see, e.g., Mas-Colell et al., 1995). However, there frequently exist failures in markets for addictive substances or other goods involving unhealthy behaviors. The traditional economic approach is for government intervention to fix these market failures. For example, in the case of a negative externality in consumption A. C. Pigou advocates a tax on consumption equal to the amount of the externality, thus internalizing costs of the externality to the decision maker and resulting in socially optimal levels of consumption (see, e.g., Baumol, 1972). Another example is that, if consumers lack relevant information, the government can require that manufacturers provide the missing information, leading to socially optimal decisions.

The economic approach differs from the public health perspective. For example, the economic perspective is that the socially optimal levels of unhealthy behaviors are characterized by marginal social benefits equaling marginal social costs, whereas the public health perspective seems to be that the socially optimal prevalence of smoking and obesity is zero. The public health perspective is generally supportive of government action to reduce unhealthy behaviors, whether or not there are market failures. Interestingly, recent work emphasizing time-inconsistent or other non-optimizing economic behavior may partially reconcile differences between these two approaches, suggesting situations where government involvement may raise social welfare even in the absence of market failures.

5.1. Taxes and Subsidies

5.1.1. Economic Rationale for Taxation

Unhealthy behaviors such as smoking, excessive drinking, and obesity impose substantial external costs. As discussed, Pigovian taxes—which are set equal to the external

cost—can correct these distortions and result in socially optimal levels of consumption by agents who equalize marginal social benefit with marginal social cost.

Measuring the external costs of unhealthy behaviors will always be difficult because randomized controlled trials are unethical and researchers must rely on observational data, with the result that confounding factors (e.g. rate of time preference, cognitive ability, or mental health) may bias estimates and lead to an overstatement of external costs. On the other hand, causal effects may be understated if the economically disadvantaged are both more likely to engage in unhealthy behaviors and have less access to medical care. One promising direction for future research is to find and exploit natural experiments in which behaviors vary but other determinants of medical costs do not.

Manning et al.'s (1991) comprehensive study of participants in the RAND Health Insurance Experiment examined both the costs (e.g. medical costs, sick leave, early retirement, disability benefits, and lost wage tax revenue) and the "benefits" (e.g. lower payments from retirement pensions and long-term care insurance) associated with smoking, drinking alcohol, and a sedentary lifestyle. They conclude that lifetime external costs (in 1986 dollars) amount to $1,000 per smoker, $19,000 per heavy drinker, and $1,650 per sedentary person. Put another way, the external costs amount to 15 cents per pack of cigarettes smoked, 54 cents per excess drink, and 24 cents for each mile not walked.

Sloan et al. (2004) provide an even more comprehensive investigation of the external costs associated with smoking that considers: disability life-years, lifecycle earnings, Social Security and Medicare benefits, as well as spouse morbidity, disability, and mortality. They estimate that the lifetime external cost of smoking (net of cigarette taxes paid) is $3,829 (in 2000 dollars) for a female 24-year-old smoker and $8,001 for a corresponding 24-year-old male; the latter amounts to $1.44 per pack of cigarettes.

Obesity imposes external costs through both public and private health insurance. In 2008, obesity-related illness cost taxpayers $19.7 billion through Medicare and $8 billion through Medicaid, while private health insurance plans paid $49 billion in 2008 to treat obesity-related illnesses (Finkelstein et al., 2009). Using the method of instrumental variables in which respondent weight is instrumented using the weight of a biological child, Cawley and Meyerhoefer (forthcoming) estimate that the causal impact of obesity on medical care costs for Medicaid recipients is $3,674 (2005 dollars), of which $3,521 is paid by Medicaid; however, given the relatively small sample, these estimates are not statistically significant.

If consumers have time-inconsistent preferences, the optimal tax should include not only external costs but also the internal costs that consumers impose on themselves. Using such reasoning, Gruber and Köszegi (2001) estimate that cigarette taxes should be raised by an additional dollar per pack, while Sloan et al. (2004) obtain dramatically higher ($32.78/pack) estimates of the internal costs of smoking. Consistent

with this, Gruber and Mullainathan (2005) report that smokers in the US and Canada are happier in jurisdictions (states or provinces) with higher cigarettes taxes, as might occur with time-inconsistent preferences—because the government is helping them to smoke less, which they would prefer but otherwise could not do on their own.

To internalize the externalities associated with smoking it is clear that cigarettes should be taxed. However, for other unhealthy behaviors the ideal tax policy is less obvious. For instance, moderate drinking may yield medical benefits, as discussed above, implying that higher alcohol taxes could have negative effects on health for moderate consumers. The situation is even more complex for obesity.[91] The most direct application of the Pigovian logic would be to tax body fat; for example, charging the obese a higher premium for their public and private health insurance. For example, section 2705 of the 2010 Patient Protection and Affordable Care Act (PPACA) allows employers to provide premium discounts, rebates, or rewards of up to 30 percent of employee-only insurance premiums (up to 50 percent with approval from the Secretary of Health and Human Services) if they participate in qualifying wellness programs, such as those to promote healthy weight. A decision instead to tax the behaviors, goods, or services that contribute to obesity leads to the different problem of determining the ideal scope and structure of such taxes. For example, tax policy is hindered by the impossibility of dividing foods into those that are "bad" (promote obesity) or "good" (prevent obesity). Recently, some public health researchers have called for taxes on full-calorie soft drinks (e.g. Brownell et al., 2009; Brownell and Frieden, 2009). However, the approach of taxing or subsidizing specific foods raises many questions. For example: should fruit juices that are high in calories also be taxed? Does the answer depend on the vitamin content of the juice? Should diet soft drinks that are low in calories be subsidized? Or should diet soft drinks be taxed because they may promote the habit of consuming sweets or may be complements with other energy-dense foods (Brownell et al., 2009)? Obesity is the result of energy imbalance; specifically, more calories consumed than burned or excreted. Thus, obesity can equally well be attributed to insufficient physical activity as to excessive caloric intake. Does this imply that taxes should be increased on complements to sedentary behaviors, such as televisions, video games, or internet access?

Optimal taxation becomes still more complicated when allowing for cognitive limitations, bounded rationality or other sources of decision errors. For instance, Bernheim and Rangel (2004) point out that many unhealthy behaviors represent mistakes (which have a stochastic component), implying that higher taxes may unfairly penalize people whose genetic endowment predisposed them to drug addiction or

[91] For a discussion of the issues surrounding taxes on energy-dense foods to promote healthy eating, see Cawley (forthcoming).

other undesirable outcomes. On the other hand, such individuals may most desire government policies as precommitment devices (Gruber and Mullainathan, 2005).

In contrast to the complexities of taxing unhealthy behaviors, the economic argument for eliminating *subsidies* that promote unhealthy behaviors seems quite strong. For example, current US agricultural policies expand the availability and reduce the cost of "program" crops—like soybeans and corn—that have become major inputs into processed energy-dense food products (Wallinga et al., 2009; Cawley and Kirwan, 2011). Removing such subsidies would raise the relative prices of such foods, with the desirable effect of reducing body weight, although, depending on the elasticity of consumption to the price of agricultural commodities, this effect may be small (Cawley and Kirwan, 2011).

5.1.2. Concerns about Regressivity of Taxes

Vertical equity suggests that those with greater ability to pay should be taxed more heavily than those with less ability to pay (Rosen, 2002). Because those of low socioeconomic status tend to be more likely to engage in many unhealthy behaviors including tobacco use, physical inactivity, and poor diet (see, e.g., Pampel et al., 2010, and Cutler and Lleras-Muney, 2010), Pigovian taxes on products like cigarettes and energy-dense foods will tend to be regressive. Thus there may be a trade-off between the two socially desirable goals of vertical equity and population health. In principle, this trade-off can be avoided by pairing the excise tax with a means-tested income transfer, with the combined effect of allowing the substitution effect of the price change to affect behavior while eliminating the income effect of the price change that would lower utility (see, e.g., Perloff, 2008). In practice this may be difficult to implement. Moreover, it is important to note that taxes imposed on behaviors disproportionately engaged in by the poor will not automatically be regressive, because elasticity of demand for the taxed product may decline with income. However, this also raises the question of whether an attempt should be made to design taxes to focus on those whose (unhealthy) behaviors are most price elastic.

5.1.3. Extent to which Tax is Passed through to Retail Price

In perfectly competitive markets, the pass-through of a tax to retail prices is $\frac{S}{S-D}$, where S and D are the price elasticities of supply and demand (see, e.g., Perloff, 2008). Thus, the pass-through of a tax is bounded by zero and one. However, with imperfectly competitive markets, taxes may be "overshifted" such that prices can rise by more than the amount of the tax (Besley and Rosen, 1999). The logic is that manufacturers may use the tax hike as an opportunity to raise prices, although this begs the question of why the apparently collusive oligopolists were not previously maximizing profits. One possibility is that concerns about fairness constrain profit seeking (Kahneman et al., 1986). For instance, price increases with no apparent justification

may cause an outcry among consumers that cuts sales and profits; tax increases then provide manufacturers with an excuse to raise prices.

Studies confirm that tax pass-through rates sometimes exceed 100 percent. For sales tax rates ranging from 0 to 8.25 percent, the tax pass-through rate has been estimated to be 100 percent for fast food hamburgers and over 100 percent for Coca-Cola (Besley and Rosen, 1999). Chaloupka and Warner (2000) review evidence that the pass-through exceeds 100 percent for cigarettes, while Young and Bielinska-Kwapisz (2002) and Kenkel (2005) found pass-through of greater than 100 percent when alcohol taxes were increased.

5.1.4. Cross-border Shopping, Smuggling, and Excise Tax Evasion

When excise taxes are higher in one jurisdiction than another, there is an incentive for organized or casual cross-border shopping (more pejoratively called "smuggling"). Beatty et al. (2009) present evidence suggesting that differentials across international borders in rates of alcohol and tobacco taxation result in economically important amounts of cross-border shopping and tax avoidance behavior. Chaloupka and Warner (2000) conclude that a large proportion of cigarette sales in states with low cigarette taxes represent smuggling to higher-tax states and describe how cross-border shopping between the US and Canada has varied with differences in their tobacco taxes. Tosun and Skidmore (2007) show that when West Virginia raised its food sales tax from 0 to 6 percent in 1990, food sales in border counties fell by 8 percent, as consumers increasingly made purchases in neighboring states that taxed food at a lower rate or exempted it from sales taxation altogether.

The extent of these effects will generally vary depending on how close customers are to jurisdictional borders. For example, most Texas residents will find cross-border shopping to be uneconomical because of the time and travel costs involved. Conversely, the District of Columbia is so small that it is relatively easy and cheap for residents to shop across the border. Thus, it is estimated that food demand in DC is highly elastic to local taxes, with each 1 percentage point increase in the food sales in the District of Columbia, relative to neighboring states, reducing food purchases in the District by 7 percent (Fisher, 1980).

The implication of this literature is that individual cities and states whose citizens live close to state borders may find that excise taxes reduce domestic sales but have little impact on consumption. If states do not take into account the impact of their lower excise taxes on the health behaviors of residents of neighboring states, there may be a "race to the bottom" in which states jockey to have excise taxes slightly lower than their neighbors in order to increase tax revenue. Excise taxes set at the national level would prevent the race to the bottom, but at the cost of preventing states from tailoring such tax policy to their circumstances.

5.1.5. Effect of Taxes on Unhealthy Behaviors

Section 2 provides detailed information on price elasticities of demand for unhealthy behaviors. The estimates presented there confirm that consumption of even addictive goods is responsive to price, which implies that tax policy can be used to reduce the prevalence of unhealthy behaviors.

Other research explicitly examines the effects of taxes in particular (rather than variation in prices from other sources) on health behaviors. For instance, an analysis of almost 30 years of state-level data found that a 1 percent increase in beer taxes is associated with a 1.0 percent decrease in youth drinking (Carpenter et al., 2007). Others estimate that a one-dollar increase in the beer excise tax would reduce the prevalence of youth alcohol use by two percentage points, but with no effect on binge consumption (Cook and Moore, 2001). Forster and Jones (2001) use duration analysis to study the decisions to initiate and quit smoking; they estimate that a 5 percent increase in the cigarette tax would reduce by 2 to 3.5 percent the total number of years spent smoking. The 1998 Master Settlement Agreement between cigarette manufacturers and the State Attorneys general immediately increased cigarette prices by 43.5 cents per pack (nearly 20 percent) and raised prices further during the next two years. This reduced smoking rates by 13 percent for youths and 5 percent among adults (Sloan et al., 2004) but smoking by pregnant women fell by less than 3 percent (Levy and Meara, 2006).

Recent research suggests that food taxes may not have much impact on caloric intake or obesity. For example, existing soft drink taxes have no detectable effect on child weight (Fletcher, Frisvold and Tefft, 2010). This may be due to the fact that existing taxes on soft drinks are quite small—they average 2.7 percent of soft drink prices (Fletcher et al., 2010). Larger taxes have been proposed and might have bigger impacts on consumer behavior, but convincing evidence of this has not yet been obtained. For instance, Chouinard et al. (2007) estimate that even a 10 percent *ad valorem* tax on fat in dairy products (milk, cream, cheese, butter, ice cream, and yogurt) would reduce average fat consumption by less than a percentage point. Based on their review of the literature, Powell and Chaloupka (2009) conclude that small taxes and subsidies are not likely to significantly reduce obesity or BMI, but non-trivial price hikes might have a measurable effect.

Consumers may respond strategically to tax increases. For example, smokers respond to higher cigarette prices by switching to cigarettes that are higher in tar and nicotine per cigarette (Farrelly et al., 2004). In addition, smokers respond to increases in cigarette taxes by extracting more nicotine per cigarette (Adda and Cornaglia, 2006). For most age groups, this compensating behavior is so large that the average daily tar intake is unaffected by cigarette taxes, with one study documenting *increases* in tar and nicotine consumption for 18−24-year-olds (Evans and Farrelly, 1998).

More generally, consumer responsiveness may depend on the salience of the tax (Chetty et al., 2009). For instance, taxes that are added at the register may have less effect on purchases than those that are included in product list prices and thus are seen when consumers make decisions of what to buy. These issues are particularly relevant for efforts to impose Pigovian taxes designed to produce socially optimal levels of consumption.

Another empirical challenge is that variation across jurisdictions in taxes may be correlated with variation across jurisdictions in voter sentiments regarding unhealthy behaviors (DeCicca et al., 2002). For example, cigarette taxes are low in Kentucky but high in California. This may partly occur because Kentucky is a major tobacco-growing state, and California is not; it may also reflect differences across states in preferences about health.

Failure to control for policy endogeneity may bias coefficient estimates of the impact of taxes on health behaviors. For example, DeCicca et al. (2008b) measure state anti-smoking sentiment using data from the Tobacco Use Supplements of the Current Population Survey, which ask respondents their opinions on anti-smoking policies such as clean indoor air laws and restrictions on tobacco promotion and advertising. They then show that controlling for state anti-smoking sentiment leads to a reduction in the estimated price elasticity of demand for cigarettes and that the failure to do so leads to overestimates of the price responsiveness of youth smoking.

5.1.6. Effect of Taxes on Outcomes Subsequent to Consumption

A large literature estimates the effect of alcohol taxes on outcomes subsequent to consumption. (Parallel literatures are less complete for smoking or eating because those behaviors are not contributors to crime, and a related literature does not exist for drugs because illicit drugs are not taxed.) Many of the studies on the effects of alcohol taxes estimate reduced-form models using state-level data, so the results need to be interpreted with caution, given concerns about policy endogeneity, modest within-state variation in taxes, and omitted variables.

Alcohol taxes (most commonly beer taxes) are negatively correlated with physical child abuse committed by women but not men (Markowitz and Grossman, 2000), child homicides (Sen, 2006), teen abortions (Sen, 2003), gonorrhea and syphilis (Chesson et al., 2000), work days lost due to industrial injuries (Ohsfeldt and Morrisey, 1997) and male but not female suicides (Markowitz et al., 2003). Pacula (1998) shows that beer and marijuana are complements, with the result that higher beer taxes reduce marijuana use.

The effects of alcohol taxes on motor vehicle fatalities have been widely studied with most research suggesting a strong negative association (e.g. Cook, 1981; Chaloupka et al., 1993; Ruhm, 1996; Young and Bielinska-Kwapsiz, 2006). Some

(but likely not all) of this inverse relationship is probably spurious or reflects policy endogeneity (Dee, 1999; Mast et al., 1999; Young and Likens, 2000). Cook and Moore (1993) show that higher alcohol taxes are associated with increased educational attainment but Dave and Kaestner (2002) find little association between alcohol taxes and labor market outcomes. Cook et al. (2005) estimate that a permanent reduction of 1 percent in alcohol consumption (whether through taxes or another policy) would have a negligible effect on the death rate of those aged 35–69 years but Cook and Tauchen (1982) show that higher alcohol taxes reduce cirrhosis mortality, presumably due to decreases in heavy drinking.

5.2. Cash Incentives for Healthy Behaviors

The TORA predicts that individuals engage in unhealthy behaviors when the discounted lifetime benefits exceed the discounted lifetime costs. However, other models emphasize the likelihood of mistakes. In these cases (and even with rational addiction if there is *ex ante* uncertainty about outcomes) agents will often attempt to change their unhealthy behavior but fail to do so. Indeed, this is why Orphanides and Zervos (1995) and Bernheim and Rangel (2004), among others, suggest that harm reduction policies such as subsidized rehabilitation may be desirable.

One possible way to "help people help themselves" is to provide financial incentives for reductions in drinking, drug use, weight, or food consumption, or for increases in physical activity.[92] Such incentives are potentially useful for several reasons. First, the benefits of behavior change may otherwise not be salient, because their magnitude is not known with any degree of certainty. Second, the benefits of behavior change may not be immediate. (In contrast, the *costs* of behavior change, such as withdrawal, usually are immediate.) For this reason, the effectiveness of rewards generally declines the further in the future they occur (Ainslie, 1975). Third, time-inconsistent preferences may result in preference reversals and an inability to adhere to plans for more healthful behavior.

Offering immediate cash rewards for behavior change may help to solve these problems. Interestingly, even small incentives may be effective if they are salient and provided in exchange for clearly defined short-term objectives. Even small rewards may be effective because people tend not to compare payoffs to their income or wealth but instead "bracket" them—i.e. consider them in isolation (Read et al., 1999; Kahneman and Tversky, 1979). Financial rewards can also be structured to create precommitment devices, helping to reduce problems created by time-inconsistent

[92] Financial incentives can also be used to encourage patients to receive screening tests, show up for appointments or adhere to recommended regimes for taking prescription medications. In the review by Giuffrida and Torgerson (1997), 10 of 11 studies found that financial incentives improve patient compliance on these outcomes.

preferences. For example, recovering addicts might post a bond that is automatically forfeited if they relapse.

Contingency management offers incentives for addicts to remain abstinent by providing them with vouchers that can be exchanged for market goods in exchange for negative results on drug tests (Higgins et al., 2002).[93] This program was originally devised for cocaine addicts but has since been applied to the treatment of addiction to alcohol, marijuana, nicotine, and opiates. A meta-analysis of voucher-based reinforcement therapy found overwhelming evidence of increased abstinence; the vouchers raised compliance by an average of 30 percent, with larger effect sizes for rewards that were more valuable or were delivered immediately (Lussier et al., 2006). However, contingency management appears to be more effective at treating use of opiates and cocaine than tobacco (Prendergast et al., 2006). A striking feature of these programs is the relatively high success rates obtained for small vouchers—as little as $2.50 for a single negative test for cocaine (Higgins et al., 2002) or a $137 average payment over a three-month period (Petty and Martin, 2002).

The results for weight loss efforts are more mixed. Cawley and Price (2011) find that worksite programs offering modest cash rewards for specific reductions in weight (e.g. $30 per quarter for a 10 percent weight reduction) were not successful—the treatment group lost slightly less weight over a 12-month period than the control group—although modest improvements were obtained when the treatment group posted substantial ($110) bonds that were only refunded upon successful achievement of year-end weight loss goals.[94] Similarly, Finkelstein et al. (2007) present evidence of modest weight loss at three months but no difference at six months for financial rewards ranging from $7 to $14 per percentage point of weight reduction (after six months), and Butsch et al. (2007) fail to detect significant effects at 12 weeks for a treatment group offered a $150 refund of their enrollment fee if they lost 6 percent of their initial weight.

Burger and Lynham (2010) examine data from the British bookmaker William Hill of 51 people who placed bets that they would be able to lose a specific number of pounds (verified by a physician) over a period of time. Weight loss averaged 78 pounds (from a starting mean of 263 pounds) over an average of 243 days. However, despite payoffs averaging $2,332, roughly 80 percent of bettors failed to meet their weight loss goals. This is an interesting example of the private market offering precommitment mechanisms for time-inconsistent consumers and it is possible that many individuals considered themselves better off for having participated because they lost weight, even if they "lost" their bet.

[93] Vouchers are awarded instead of cash because recovering addicts might be tempted to spend cash on drugs.

[94] However, attrition was extremely high in this study, 51.2 percent after one quarter and 76.4 percent after one year.

Giné et al. (2010) implemented a voluntary precommitment program for smoking cessation whereby smokers deposited funds for six months and had these deposits returned to them if they tested negative for nicotine use, with the funds forfeited to charity for positive urine tests. Those who participated were three percentage points more likely than a control group to have quit smoking at six months, and this effect persisted at 12 months.

The success of such interventions may often be highly dependent on the precise structure of incentives. For example, Volpp et al. (2008) uncovered substantial short-run weight loss (at 16 weeks) for a program in which participants successfully meeting weight loss goals were entered into a lottery with a one-in-five chance of receiving a small ($10) reward and a one-in-one hundred probability of obtaining a large ($100) reward. The short-term weight loss was dramatic (weight reductions averaging 16 pounds), but the longer-term effects (at seven months) were less so. Similarly, Volpp et al. (2006) found that modest financial incentives combined with enrollment in a smoking cessation program substantially reduced tobacco use in the short term (at 30 and 75 days), but not in the longer run (at six months). On the other hand, in a follow-up intervention, Volpp et al. (2009) found evidence of decreased smoking even at 18 months, possibly because the incentives were of relatively large size and dependent on longer-term behavioral changes.[95]

5.3. Restrictions on Purchase or Use

A variety of policies have been implemented with the goal of directly restricting availability or raising the time costs for using unhealthy products. A large literature has examined the impacts of state minimum legal drinking ages (MLDA), which make it more difficult for teens to acquire alcohol. Wagenaar and Toomey (2002) conducted an in-depth review of research conducted between 1960 and 2000, covering periods of both falling (during the 1970s) and then rising (during the 1980s) drinking ages. They concluded that the evidence persuasively indicates an inverse relationship between the MLDA and youth alcohol consumption, traffic crashes, and other social problems (like suicides, homicides, and vandalism). However, they also find that the evidence is insufficient to say whether the effects vary across subpopulations, such as for college students. More recent analyses confirm these effects. For instance, Cook and Moore (2001) find that youths who are younger than the minimum purchase age for alcohol in their state are 5.5 percentage points less likely to drink in the past 30 days and are 2.5 percentage points less likely to binge drink. A recent analysis of almost 30 years of state-level data concluded that increases in the MLDA during the

[95] Individuals were paid $250 for not smoking three or six months after program completion and an additional $400 if still abstinent at nine or 12 months. However, reflecting the difficulty of making permanent behavioral changes, only 9.4 percent of the treatment group abstained from smoking at nine or 12 months (compared to 3.6 percent of the controls).

1970s and 1980s reduced drinking participation and heavy drinking by 4 percent among high school seniors (Carpenter et al., 2007). However, minimum purchase ages may have the unintended consequence of leading youths to switch from alcohol to drugs: DiNardo and Lemieux (2001) estimate that raising the state MLDA from 18 to 21 increases the prevalence of youth marijuana consumption by 2.4 percentage points.

Laws barring youth possession, use, and/or purchase of tobacco also deter smoking participation by teens but with little evidence of changes at the intensive margin for adolescent and young adult smokers (Tauras, Markowitz, and Cawley, 2005).

A large body of research suggests that restrictions on smoking in public places and private workplaces (e.g. clean indoor air laws) reduce the prevalence of tobacco use (see Chaloupka and Warner, 2000, for an extensive review). Some evidence (see Picone, Sloan, and Trogdon, 2004) suggests that bans on smoking in public places also reduce alcohol consumption by women (but not men), which for women is consistent with complementarity between smoking and drinking. Bitler et al. (2010) show that the impact of clean indoor air laws varies by industry, with larger reductions in smoking among bartenders than for those employed in other industries (e.g. schools, restaurants, and government).

Most related studies are unable to examine the extent to which smoking was displaced from public places to private places such as homes, or the related issue of whether such laws affect the exposure of non-smokers to environmental tobacco smoke (ETS) in public places or private homes. Two investigations of these issues yield somewhat conflicting results. Adda and Cornaglia (2010) suggest that smoking bans in the US displaced smoking from public to private places, with the net result of increased exposure to ETS by non-smokers, particularly those sharing a household with smokers. However, Carpenter et al. (2011) find that Canadian bans on public smoking led to large reductions in ETS exposure in public places for both smokers and non-smokers, and that these laws did not significantly affect ETS exposure in homes. However, they do estimate that non-smokers' exposure to ETS increased at building entrances. Neither Adda and Cornaglia (2010) nor Carpenter et al. (2011) find a significant impact of smoking bans on the probability of smoking.

A potential limitation of all of this research is that estimates may be biased by policy endogeneity. Gallet et al. (2006) find that the adoption of clean indoor air laws is correlated with state characteristics such as political affiliation, urban population, per capita income, and tobacco production.

A dramatic policy enacted to restrict the consumption of an addictive substance was Prohibition, which outlawed the sale and purchase (but not use) of alcohol in the United States from 1919 to 1933. Individual-level data on alcohol consumption do not exist for this era, but the impact of Prohibition on heavy alcohol consumption has been estimated using deaths from cirrhosis of the liver or alcoholism (Miron and

Zwiebel, 1991) and police records regarding arrests for drunkenness (Dills et al., 2005). Both proxy measures suggest that alcohol consumption initially fell sharply, to around 30 percent of the previous level, immediately after the enactment of Prohibition, before rebounding over the next several years to between 60 and 70 percent of its prior level (Miron and Zwiebel, 1991). These proxy measures also suggest that, after the repeal of Prohibition, heavy drinking initially declined but returned to the pre-Prohibition level after a decade.

Drug legalization may be analogous to the repeal of Prohibition. Miron (2003) estimates that the black market prices of cocaine are 2 to 4 times higher than they would be if the drug was legal and that heroin prices are 6 to 19 times as high. Legalization would therefore decrease prices substantially, resulting in higher consumption (see section 2 of this chapter for estimates of the price elasticity of demand for illicit drugs).

Consumption of illicit drugs is deterred not only by higher prices but also by the legal penalties for purchase and possession. For example, fines for possession and the probability of arrest decrease marijuana use among young adults (Farrelly et al., 2001). However, doubling the fines for marijuana possession would reduce the probability of use by youths by less than 1 percent, while decriminalization would increase it by 4 to 5 percent (Chaloupka et al., 1999; Saffer and Chaloupka, 1999). On the other hand, doubling of the fines for cocaine possession would reduce corresponding use by roughly 4 percent (Chaloupka et al., 1999).

Some studies have examined the impact of increased drug law enforcement on drug price, purity, and consumption. For example, Weatherburn et al. (2002) investigate the Australian heroin "drought," in 2000, that partly resulted from increased law enforcement. Using a survey of 165 heroin users in that country's largest heroin market, they found that the drought raised prices and lowered purity of heroin, reduced consumption and rates of overdose, but that the associated health benefits were partially offset by increased use of other drugs, most commonly cocaine. Dobkin and Nicosia (2009) examine the impact of the US government's decision in 1995 to shut down two suppliers providing more than half of the precursors used to produce methamphetamine. Focusing on California, the authors find that the supply of methamphetamine was halved, purity declined from 90 to 20 percent, and the price tripled. Use of the drug among arrestees declined 55 percent, and related hospital admissions fell 50 percent. However, the impact was largely temporary, with the price restored to its original level within four months and other outcomes returned to their original levels within 18 months (suggesting that meth producers were able to find substitute ingredients). In contrast to Weatherburn et al. (2002), Dobkin and Nicosia (2009) find little evidence of substitution away from the newly expensive drug and towards other drugs.

An empirical challenge to measuring the causal effect of policies on health behaviors is that the policies are endogenous, and are more likely to be adopted in states

where voter sentiment is against such unhealthy behaviors. For example, Cawley and Liu (2008) find that state laws to prevent or reduce childhood obesity (such as mandatory physical education for school-aged children) are more likely to be enacted in states with large gaps between the desired and actual weight of adults. Carpenter et al. (2007) find that alcohol consumption fell just as much 1−2 years before as 1−2 years after a rise in the minimum legal drinking age; they interpret this as evidence that increases in the minimum legal drinking age are endogenous responses to high levels of teen drinking (they find no evidence that zero tolerance underage drunk-driving laws are endogenously adopted).

For this reason, simple estimates of the impact of policies on health behaviors may suffer from omitted variables bias. Ruhm (1996) uses state fixed effects to control for differences across states in (e.g.) unobserved social attitudes against drinking and finds that omitted variables strongly affect parameter estimates for policies designed to deter drunk driving (but do not much affect the estimates for alcohol taxes).

5.4. Providing Information

Information is generally a public good, and as a result is underprovided by private markets (see, e.g., Perloff, 2008). When consumers have incomplete information, free markets may fail to maximize social welfare (Mas-Colell et al., 1995), providing an efficiency rationale for the government either to deliver the missing information or to require suppliers to do so. Orphanides and Zervos (1995) discuss how information, education, or counter-advertisement efforts may be desirable, even with perfect rationality, to reduce *ex ante* errors in subjective probabilities—particularly because individuals tend to underestimate their own probabilities of becoming addicts, in part because they overstate rates of substance use by peers. Information that reduces the divergence between subjective and objective risk assessments may therefore improve *ex post* utility.

Consumers sometimes respond strongly to the provision of new information. A dramatic example is the release of the first Surgeon General's report on smoking and health in 1964, which was followed by an immediate 5 percent decrease in smoking; other research indicates that both warning labels on cigarette packs and paid anti-smoking advertisements significantly cut tobacco use (Chaloupka and Warner, 2000). However, the reductions were larger for more highly educated individuals (Grossman, 2000), perhaps reflecting differences in cognitive ability. Information about adverse health consequences can also decrease the use of other addictive goods. For instance, perceived risk of harm from regular use is negatively correlated with the probability of smoking marijuana in the past year (Pacula et al., 2001).

These findings are not limited to addictive products. In an experiment in Kenya, teenagers in randomly selected schools were provided information that the HIV

infection was more common among adult males than teenage boys. This information led to a 61 percent reduction in the impregnation of teenage girls by adult males, as girls substituted away from unprotected sex with older men toward condom-utilizing sex with teenage boys (Dupas, 2011). Information campaigns to prevent HIV/AIDS in Africa have the largest impact for better educated persons (De Walque, 2007a).

Consumers also respond to nutritional information. The Nutrition Labeling and Education Act (NLEA) required manufacturers of packaged foods to provide information about their products in the form of the Nutrition Facts panel. One study concludes that this increased the consumption of iron and fiber, without affecting consumption of total or saturated fat or cholesterol (Variyam, 2008). However, other research suggests that the Nutrition Facts panel led more consumers to choose low-fat options (Mathios, 2000) and that the NLEA lowered obesity among white females by 2.4 percentage points (Variyam and Cawley, 2006). Notably, competition between food manufacturers did not result in this information being voluntarily provided prior to the government mandate (Mathios, 2000). Between 1975 and 1985, government campaigns to encourage lower consumption of fats successfully reduced the fat intake of US women. Even larger decreases were observed after 1985, when food companies received permission to make health claims about their products (Ippolito and Mathios, 1995). Most discussion of policies to counter externalities involves an increased role for government, but this is an example of how in certain instances decreasing regulation can reduce market failures, improve efficiency, and enhance social welfare.

One recent policy innovation, implemented in New York City in 2008 and as part of the US health care reform bill in 2010, requires calorie labeling on menus and menu boards in restaurant chains. Elbel et al. (2009) found that the New York City labeling law raised the percentage of customers who reported seeing calorie labels at four major fast-food chains (relative to controls in Newark, NJ, which does not have a labeling law); however, calories, saturated fat, sodium, or sugar in the food actually purchased did not change. On the other hand, Bollinger et al. (2011) found that the New York City law reduced calories per transaction at Starbucks by 6 percent (15 calories), almost entirely due to decreases in calories from food (rather than beverages).[96] Wisdom et al. (2010) summarize the experimental data showing that customers provided calorie information at a fast-food restaurant ordered meals with around 60 fewer calories than those not receiving the information.

[96] They utilize rich data on every transaction at Starbucks stores in New York City and the control cities Boston and Philadelphia (with no calorie posting), from January 1, 2008 until February 28, 2009—before and after the law's April 1, 2008 implementation. Most of the reduction in calories was due to consumers buying fewer food items, rather than substituting to lower calorie foods.

5.5. Advertising Restrictions

A common public health response to a high prevalence of unhealthy behaviors involving legal substances such as tobacco, alcohol, or energy-dense foods is to ban or regulate advertisements for these products, or to call for voluntary limits on advertising by manufacturers. (For a description of the history of US regulation of cigarette advertising, see Nelson, 2006.)

Saffer and Chaloupka (2000) examine the impact of various bans on cigarette advertising in 22 OECD countries during the period 1970–1992. They conclude that comprehensive bans (i.e. bans on such ads on television, radio, print, outdoors, movies, sponsorship and at point of purchase) can reduce tobacco consumption but that more limited restrictions have little or no effect. However, the results of later research are more ambiguous. Blecher (2008) extends the approach of Saffer and Chaloupka (2000) to 30 developing countries over the period 1990–2005 and finds that both comprehensive and limited policies to restrict tobacco advertising reduce smoking. Nelson (2003) examines data for 20 OECD countries over the period 1970–1995 and concludes that Saffer and Chaloupka's (2000) results are not robust to the use of stationary data in the form of consumption growth rates, or to controlling for other policies (such as warning labels), or analyzing different time periods; Nelson (2003) concludes that advertising bans, whether comprehensive or limited, do not affect cigarette consumption, which is also the conclusion of meta-analysis of nine studies examining the US government's 1971 ban on television broadcast advertising of cigarettes (Nelson, 2006).

While television advertisements of cigarettes have been illegal in the US since 1971, television advertisements of liquor were kept off the airways by a voluntary agreement among manufacturers until November 1996, when the liquor industry's national trade organization agreed to lift the self-imposed ban (see Frank, 2008).

Nelson (2010) examines the relationship between advertising bans and alcohol consumptions in 17 OECD countries between 1975 and 2000. He criticizes earlier studies for failing to control for the stringency of other alcohol policies, speculating that this may have led to omitted variables bias in estimates of the impact of the advertising bans. In his preferred models, Nelson (2010) detects no impact of alcohol advertising bans on the demand for alcohol.

Recently, researchers have begun to estimate the possible impact of regulation of advertisements for energy-dense foods. Using data from the NLSY79 and NLSY97, Chou et al. (2008) estimate that a ban on fast-food television advertisements would reduce the prevalence of overweight among 3–11-year-olds by 18 percent and the fraction of overweight adolescents (12–18-year-olds) by 14 percent. Eliminating the tax deductibility of TV advertising for fast-food companies (which the authors state would raise the price of advertising by 54 percent) is estimated to decrease the share of

overweight children by 7 percent and of overweight adolescents by 5 percent. However, these estimates do not account for targeting of advertising towards heavier youths.

5.6. Defaults and Choice Architecture

Behavioral economists emphasize that individuals frequently make systematic mistakes, raising the possibility that social welfare can be improved by changing the default options to account for factors such as procrastination, lack of self-control, and status quo bias. A key component of these interventions, sometimes referred to as "libertarian" or "asymmetric" paternalism (Loewenstein et al., 2007; Thaler and Sunstein, 2009), is that few limitations are placed on the available choice set but small cognitive costs are charged for individuals to select options that are perceived by planners to represent *ex post* mistakes. A risk of this strategy is that planners may underestimate the knowledge and sophistication of consumers, and may needlessly distort decision making, lowering social welfare.

There are many potential applications of behavioral economics to unhealthy behaviors; to date, it has been applied most to food consumption. Examples of proposed policy changes include moving energy-dense items to less convenient locations in school cafeterias, making water rather than soft drinks the default beverage option for fast-food meals; and making food choices several hours before the meal will be eaten (Just, 2006; Loewenstein et al., 2007).

While such policies hold considerable promise, empirical analyses of them have just begun to be conducted and it will be some time before we have high-quality evaluations of these interventions. To provide one example, Wisdom et al. (2010) find that making lower-calorie sandwiches more salient, by listing them first on the menu, had no effect on total calories consumed because, although these sandwiches were more often ordered, the calorie savings were compensated for by increased consumption of other products. However, a "stronger" intervention, in which individuals had to unseal an envelope with additional menu choices, to purchase more caloric items, did reduce total energy intake.

6. FUTURE DIRECTIONS

Economic research on health behaviors has reached the stage of "early adolescence." At the beginning of this chapter, we documented the remarkable increase over the past two decades in economics journal articles devoted to risky health behaviors. During the "infancy" period, economists demonstrated to skeptical health professionals and policy makers that economic factors really do play a role in determining health behaviors like drinking and smoking. Much of this research was documented at

length in dedicated chapters in volume 1B of this Handbook (Chaloupka and Warner, 2000; Cook and Moore, 2000; Kenkel, 2000). The early effort was highly successful in convincing public health researchers and practitioners that health behaviors are responsive to prices and other incentives. As a result, taxes are now routinely used by state and federal governments in their attempts to reduce smoking and alcohol consumption and are receiving considerable attention in current efforts to reduce obesity.

The "toddler" years were dominated by theoretical development and empirical testing of the model of rational addiction. This work has also been extremely influential, particularly among economists, for most of whom the TORA is often the default model for examining health behaviors. This framework has many attractive features, including emphasizing the role of prices and forward-looking behavior, an appreciation of the distinction between long-run and short-run elasticities, and for demonstrating that even seemingly undesirable outcomes can be consistent with fully rational decision making. With additional assumptions it can explain many interesting phenomena like quitting "cold turkey," cycles of binging and purging, and entry into addiction following adverse life events.

That said, the assumptions required for the TORA—including perfect foresight, complete optimization, and time-consistent preferences—may be violated for many

substance use policies to be exogenous, but the median voter theorem implies that states with strong voter sentiment against unhealthy behaviors will enact policies designed to discourage them (e.g. DeCicca et al., 2008; Cawley and Liu, 2008) and policies may be enacted in response to high levels of substance use (see, e.g., Carpenter et al., 2007). While there is growing appreciation of the problem of policy endogeneity, there is mixed evidence of the extent to which it biases estimates (see, e.g., Carpenter et al., 2007 and Ruhm, 1996), making this an important area for additional research.

Future empirical investigations will certainly benefit from new data collection, but also from better use of existing data. For instance, there is increased awareness in obesity research that body mass index is a noisy measure of fatness. Recently, economics studies have begun to use alternative measures of fatness, such as percent body fat and waist circumference (see, e.g. Burkhauser and Cawley, 2008; Johansson et al., 2009; Wada and Tekin, 2010), but economists are limited by the available data; for instance, many secondary data sets include self-reported weight and height but not more accurate measures of fatness. Biomarkers are also becoming more available in data used by economists, suggesting that such research will become more common in the future.[97] For example, Adda and Cornaglia (2006) examine the concentration of cotinine, a metabolite of nicotine, in bodily fluids and find that smokers compensate for tax hikes by extracting more nicotine from each cigarette smoked. In subsequent work, Adda and Cornaglia (2010) use data on cotinine concentration to determine whether taxes and public smoking bans affect exposure to environmental tobacco smoke. For an in-depth discussion of the uses and limitations of biomarkers in social science data, see volume 55 (2009), issue 2 of *Biodemography and Social Biology*.

More in-depth analysis will be facilitated by richer data. For example, recent research on smoking (Loomis et al., 2006), drinking (Bray et al., 2009), and food purchases (Zhang et al., 2008) documents purchases by individual consumers using scanner data from retail establishments, rather than relying on aggregate sales data or recall in consumer surveys. Similarly, heart rate monitors, pedometers, and accelerometers are being used to measure physical activity (e.g. see Berlin et al., 2006), although each has limitations when applied to general populations (Sirard and Pate, 2001).

However, data limitations continue to pose obstacles for many potentially interesting analyses. For example, it is hard to survey the severely drug addicted because they may not have a permanent residence or phone; as a result, even large social science data sets may not provide statistical power for an analysis of heavy drug users. Another example is that data on food consumption tends to be collected over brief periods (e.g. using 24-hour dietary recalls) in repeated cross-sections; longitudinal data would better

[97] For information on biomarkers in US population-based data see: http://biomarkers.uchicago.edu/studiescollectingbiomarkers.htm.

allow for more in-depth study of the dynamics of eating and weight changes. Information on mental health, and its determinants, is generally not as good as that for physical health and large secondary data sets rarely provide reliable information on job and non-job sources of stress. The field would also benefit from better theoretical definitions of peer and reference groups, and data corresponding to these categorizations.

Perhaps most importantly, most of the empirical evidence summarized in this chapter focuses on a limited set of health behaviors—particularly smoking, drinking, substance use, and obesity. This reflects a relative paucity of research on other outcomes, although some research has been conducted on behaviors like risky sexual activity and prostitution (Oettinger, 1999; Levine, 2001; Gertler et al., 2005), immunizations (Philipson, 1996; Mullahy, 1999), and seatbelt or motorcycle helmet use (Carpenter and Stehr, 2008; Dee, 2009).[98] Future analyses of a wider array of health behaviors are likely to be highly informative.

6.2. Future Research using Non-traditional Models

This chapter highlights the promise of emerging non-traditional models that marry strengths of the standard rational economic framework with an understanding of biological considerations, and which incorporate insights from behavioral economics and neuroeconomics. From a modeling perspective, the most important development to date has been the use of quasi-hyperbolic discounting, which provides a straightforward method of incorporating time-inconsistent preferences into otherwise conventional frameworks. However, this is just one way of capturing such behavior, and it does not account for other aspects of observed decision making, such as the apparent heterogeneity of discount rates across types of purchases or alternative mental states. We anticipate that there will be active research over the coming decades aimed at more realistic modeling decision making related to health behaviors. Several areas of study seem particularly promising.

Increased attention is being paid to the role of genetic determinants of health, motivated in part by the mapping of the human genome and the inclusion of genetic markers on datasets that are frequently used by economists (e.g. the National Longitudinal Survey of Adolescent Health). To date, most economics research in this area has used genetic markers as instrumental variables when examining how specific behaviors or health conditions affect outcomes such as educational attainment and school performance (Ding et al., 2009; Fletcher and Lehrer, 2009) or labor market outcomes like employment or wages (Norton and Han, 2008).[99] Thoughtful use of genetic markers in health economics will require awareness of the following issues

[98] Chapters on prevention (Kenkel, 2000) and infectious diseases (Philipson, 2000) in volumes 1A and 1B of this Handbook cover some of these issues in greater detail.

[99] Goldman et al. (2005) provide a detailed discussion of how genetics influence addictive behaviors but do not integrate this into an economic model.

(see, e.g., Conley, 2009 and Cawley et al., 2011). First, behaviors are often influenced by multiple genes (they are polygenic) in ways that are difficult to quantify. Second, behavior is often the result of complicated interactions between genes and environment. Finally, genes tend to affect multiple health behaviors and conditions, implying that genes may be invalid instruments in many contexts.

The emerging field of neuroeconomics also offers promise, some of which may have begun to be realized through the use of brain scans undertaken while individuals engage in the decision making related to health and other behavioral outcomes (e.g. McClure et al., 2004; Glimcher et al., 2007; Hare et al., 2009). However, it is not yet clear to what extent brain structure is a dominant determinant of economic behavior nor, even if it is, whether current methods of examination provide useful information. Thus, it is difficult to refute Rubinstein's (2008, p. 493) conclusion that brain studies, while "fascinating," have not yet yielded fundamental insights that change economics.

Additional interdisciplinary work with biomedical researchers is almost certainly desirable, in part because health behaviors are influenced by the system in which medical care is provided. For example, evidence suggests that physician counseling raises the likelihood that sedentary individuals increase physical activity (Calfas et al., 1996) and that tobacco users stop smoking (Stead et al., 2008) but many patients, particularly ethnic minorities, do not receive such advice from their doctors (US Department of Health and Human Services, 2009). A second reason is that while economists are especially well trained in addressing potential design problems in randomized experiments—such as attrition or substitution bias, heterogeneous treatment effects, or treatment contamination—they generally lack a corresponding understanding of biological or medical aspects of the interventions.

Finally, insights from other social sciences—particularly psychology and sociology—are likely to provide rich additions to traditional economic models. Indeed, some of the most exciting recent economic research on health behaviors has incorporated factors such as peer groups, social capital and relative status, all of which originally arose from other social science disciplines. In addition, many stylized facts that are central to behavioral economics were first identified by psychologists. One of the great strengths of economics is its ability to incorporate useful theories and findings from other disciplines while retaining a central role for incentives, trade-offs and constrained optimization. Continuation of this process is likely to allow exciting progress to be made in understanding the determinants of health behaviors and in developing public policies and interventions that can enhance social welfare.

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