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THE EFFECT OF PHYSICIAN ADVICE ON ALCOHOL CONSUMPTION: COUNT REGRESSION WITH AN ENDOGENOUS TREATMENT EFFECT

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SUMMARY

Although there are encouraging trends, alcohol abuse continues to be a significant public health problem. Econometric studies of alcohol demand have yielded a great deal of information for alcohol abuse prevention policy. These studies suggest that higher alcohol taxes and stricter drunk-driving policies can reduce heavy drinking and drunk driving. In this paper we explore the role physician advice plays in the campaign to prevent alcohol-related problems. Compared to alcohol taxation, physician advice is a more precisely targeted intervention that does not impose extra costs on responsible drinkers. Compared to the resource costs of arresting, processing, and punishing drunk drivers, physician advice may be a lower-cost intervention. To provide a basis for alcohol policy analysis, we use an alcohol demand framework to test whether physician-provided information about the adverse consequences of alcohol abuse shifts demand to more moderate levels. There are three aspects of our alcohol demand model that complicate the estimation: (1) the dependent variable is non-negative (it is a count variable—number of drinks consumed); (2) a non-trivial number of sample observations have zero values for the dependent variable; and (3) because the data we use is non-experimental, the treatment variable indicating receipt of advice from a physician may be endogenous. We implement an estimation method that is specifically designed to deal with these three complicating factors. Our results show that advice has a substantial and significant impact on alcohol consumption by males with hypertension, and that failing to account for the endogeneity of advice masks this result.

1. INTRODUCTION

Although there are encouraging trends, alcohol abuse continues to be a significant public health problem. In 1996 the proportion of traffic fatalities that were alcohol-related reached a 20-year low of 32.4%; from 1970 to 1995 the age-adjusted death rates from liver cirrhosis dropped by 47.3%; and per capita consumption of ethanol in 1996 was second only to 1995 in being the lowest level in 34 years.[1] But there is still a long way to go. Data from the 1993 National Longitudinal Alcohol Epidemiology Survey indicate that the 1-year prevalence of combined alcohol abuse and dependence was 7.41%, representing almost 14 million Americans (Grant *et al.*, 1994). It has been estimated that over 100,000 deaths a year are attributable to alcohol (US Department of Health and Human Services, 1993).

In this paper we explore the role physician advice plays in the campaign to prevent alcohol-related problems. By providing advice and counseling physicians have the opportunity to influence

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[1] These are the most recent estimates available from surveillance reports of the National Institute on Alcohol Abuse and Alcoholism (Yi *et al.*, 1998; Saadatmand *et al.*, 1998; Williams *et al.*, 1998).

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their patients' drinking practices before serious alcohol-related problems and alcohol dependence develop. Controlled clinical trials have demonstrated the efficacy of physician advice in the context of specific interventions. In these trials patients have been randomly assigned to an advice treatment group or a control group that receives no special treatment. Observed differences in drinking outcomes for members of the advice group versus the control are used to estimate the advice treatment effect. Bien, Miller, and Tonigan's (1993, p. 319) review of the clinical trial literature 'places brief counseling among the most strongly supported intervention modalities for alcohol problems...'[2] In a multinational trial the World Health Organization (WHO) Brief Intervention Study Group (1996) found that at-risk drinkers who received advice reported drinking approximately 17% less on average than those in the control group. Fleming *et al.* (1997) found that 12 months after a baseline survey, problem drinkers given brief physician advice consumed about 40% less alcohol, had 46% fewer binge drinking episodes, and engaged in 63% less excessive drinking. These reductions were statistically significantly greater than those seen in the control group (where consumption fell by 18%, binge drinking fell by 21%, and excessive drinking fell by 32%.

Econometric studies of alcohol demand have yielded a great deal of information for alcohol abuse prevention policy, but have not yet considered the role of physician advice. A series of studies find that higher alcohol taxes and stricter drunk driving policies can reduce heavy drinking and drunk driving (Cook and Tauchen, 1982; Chaloupka, Grossman and Saffer, 1993; Kenkel, 1993; Mullahy and Sindelar, 1994; Ruhm, 1996). However, Manning, Blumberg and Moulton (1995) and Kenkel (1996) find a subset of very heavy drinkers whose demand seems unresponsive to price, putting some limits on alcohol taxation as a prevention policy. Compared to alcohol taxation, physician advice is also a more precisely targeted intervention that does not impose extra costs on responsible drinkers. Compared to the resource costs of arresting, processing, and punishing drunk drivers, physician advice may be a lower cost intervention. To begin to provide a basis for alcohol policy analysis, we use an alcohol demand framework to test whether physician-provided information about the adverse consequences of alcohol abuse shifts demand to more moderate levels. Because of data limitations, we are only able to explore the effect of physician advice about drinking for patients with hypertension. Despite the limited applicability of our findings, this study is an important exploration of the usefulness of physician advice as a possibly under-rated and under-studied tool in the arsenal of approaches to reduce alcohol abuse.

Our study of physician advice extends several lines of research in empirical health economics. A number of studies estimate consumer demand functions for health-related goods. Several studies find that health information is an important determinant of consumer decisions about smoking, diet, drinking, and exercise (Lewit, Coate, and Grossman, 1981; Viscusi, 1990; Ippolito and Mathios, 1990, 1995; Kenkel, 1991). The theoretical and empirical literature in health economics has also long recognized that the physician provides information and advice, along with medical care. However, most previous studies have focused on the physician's advice about the appropriate level of medical care (Dranove, 1988; Kenkel, 1990). We broaden the focus, and study the information-providing role of physician's advice about a health-related but non-medical consumer good, alcohol.[3]

[2] Similar conclusions are drawn by the US Preventive Services Task Force Report (1995, p. 572) and the Eighth Special Report to Congress on Alcohol and Health (US Department of Health and Human Services, 1993).

[3] Similar to our approach, Jones (1994) and Jones and Yen (1994) include advice from either a physician or a family member as a determinant of cigarette demand. Both studies find some counter-intuitive results, where advice is associated with more smoking, but estimating the effect of advice is not the main focus of these studies.

There are three aspects of our alcohol demand model that complicate the estimation: (1) the dependent variable is non-negative (it is a count variable—number of drinks consumed); (2) a non-trivial number of sample observations have zero values for the dependent variable; and (3) because the data we use is non-experimental, the treatment variable indicating receipt of advice from a physician may be endogenous. We implement an estimation method that is specifically designed to deal with these three complicating factors, which are in fact present in many contexts in empirical health economics.

We also attempt to bridge a gap left by medical studies of data from clinical trials. Controlled clinical trials are considered the gold standard for evaluating the efficacy of an intervention, i.e. evaluating whether the intervention can work under a set of tightly controlled conditions. But demonstrated efficacy in a trial does not necessarily translate into effectiveness in everyday practice (Teutsch and Harris, 1996, p. 5). Two aspects of the protocol for the WHO (1996) trial and other trials limit their general applicability. First, the samples include only at-risk drinkers; at baseline the typical daily consumption in the WHO trial averaged over 4 drinks (over 2 ounces of absolute ethanol). Second, the content of the physician advice provided is carefully defined. Econometric analysis of non-experimental data provides a look at the effectiveness of physician advice as it is given in everyday practice.

In the next section we develop an empirical model to show that in non-experimental data the receipt of physician advice is potentially endogenous. Section 3 describes the econometric methodology that yields a consistent estimate of the treatment effect of advice in the presence of such endogeneity. Section 4 details the data from the 1990 Health Interview Survey, and provides an overview of the extent of consumer information and the prevalence of physician advice. Our econometric results, reported in Section 5, show that advice has a substantial and significant impact on alcohol consumption, and that failing to account for the endogeneity of advice masks this result. The final section summarizes and concludes.

2. AN EMPIRICAL MODEL OF ALCOHOL DEMAND AND PHYSICIAN ADVICE

The empirical model begins with the standard assumption that the consumer makes choices about health-related goods to maximize utility subject to the appropriate budget constraints and household production technology (Grossman, 1972). Alcohol use, like other health-related goods, is assumed to provide utility directly, and to enter as an input into the household production of good health. In a typical case, the consumer must decide if the direct utility effect of drinking exceeds the utility effect of the predicted health consequences. The consumer's maximizing choice implicitly defines alcohol demand to be a function of income, prices, and utility and health production function parameters (Rosenzweig and Schultz, 1983). Physician advice enters the model because the consumer's demand for alcohol depends on his or her prediction of the health consequence—that is, on the consumer's understanding of the parameters of the health production function. Advice from a physician may change this understanding and shift the demand curve to the left.

The empirical demand for alcohol can be generically written as

$$D = f(A, x, \xi) \tag{1}$$

where A is a binary variable indicating physician advice. Other observable alcohol demand determinants, denoted by the vector x, include socioeconomic characteristics such as age, schooling, and income. Unobservable demand influences are captured by ξ, a random error term.

The next step is to model consumer demand for advice. In non-experimental data the observed receipt of advice is generated by the optimizing behaviour of consumers and physicians. Physician advice is potentially valuable to the consumer because it provides information about the parameters of the health production function that show the health consequences of lifestyle choices. The *ex ante* benefits of information depend on the consumer's expectations about whether the information will change actions that determine utility. This is based on Hirshleifer and Riley's (1979) discussion of the expected value of information. The cost of advice-seeking is influenced by factors such as health insurance coverage for physician visits. Optimizing consumer behavior implies a demand function for advice as information given by

$$a = g(z, \nu) \tag{2}$$

where z is a row vector of observable determinants of the benefits and costs of advice to the consumer, ν is a random error denoting unobservable advice determinants and a is a latent index of the net value of physician advice—the higher the value of a, the more likely it is that advice will be sought from a physician. The latent index a is not observable; instead, we observe the binary variable A indicating the receipt of physician advice. The binary variable A takes a value of 1 if the net value of physician advice is positive ($a > 0$), and 0 otherwise.

The empirical problem is that important determinants of advice receipt may be unobserved, and that these unobservable effects may be correlated with the random component of the demand equation (1). For example, health-minded individuals may have a higher than average propensity to seek advice, and a simultaneously higher than average propensity to avoid unhealthy behaviors like heavy drinking. On the other hand, it may be that unobservable influences on drinking are positively related to the error term in the advice equation (2). For example, in the data used in this study, we observe the weekly consumption of alcohol, but we do not observe whether or not the individual has been diagnosed as an alcoholic. Diagnosed alcoholics may drink more, *ceteris paribus*, and may be more likely to receive advice from the physician. These arguments suggest correlation between ξ and ν (indeterminate in sign), leading to correlation between A and ξ in equation (1).

Physician behaviour may reinforce the tendency for the most health-minded consumers to receive the most advice. To the extent consumers value advice, the physician has an incentive to supply it during office visits. The physician may be able to charge higher fees, or improve patient satisfaction with payoffs for long-run physician/patient relationships. Since the physician's incentives to supply advice depend in part on the consumers' valuations of it, the physician may attempt to provide advice to the most health-minded consumers.

Alternatively, professional norms and ethical concerns may prompt physicians to screen and provide advice to the consumer who needs it the most, whether or not such a consumer will place a high (or even positive) value on the advice. For example, and related to the specific context of our empirical study, the US Preventive Services Task Force (USPSTF) (1995), p. 46) recommends that all patients with confirmed hypertension should receive appropriate counseling about alcohol consumption.[4] The possibility that a physician might provide advice, even when doing so hurts financially, contradicts a simplistic model of profit-maximization, but is consistent with the broader notion of utility-maximization. An altruistic physician is willing to sacrifice some

[4] The USPSTF recommendation is not very clear on what constitutes appropriate counseling; for example, it does not discuss possible differences between light versus heavy drinking in hypertension.

profits for the psychic return of providing advice where it is most needed. This is similar to the model of physician behaviour suggested by Evans (1974) in the context of supplier-induced demand, where medical professionalization leads to attitudes that counteract financial incentives to provide excess care. Becker (1974, 1976) discusses more generally how economic models can incorporate altruism. In either case just discussed, to the extent the physician observes consumer characteristics unobservable to the econometrician, this again enhances the potential for correlation between A and ξ. The sign of the correlation is an empirical question.

Correlation between A and ξ for any of the above-mentioned reasons would cause classical endogeneity bias in the conventional (ordinary least squares) estimation of equation (1). As discussed earlier, dealing with the potential endogeneity of A is complicated by the fact that the dependent variable is by nature non-negative (count) valued and is zero-valued for a large proportion of the sample. For this reason we apply an extension of the method suggested by McGeary and Terza (1998), which is a more flexible version of the technique suggested by Terza (1998).[5]

We treat equation (1) as a semi-reduced-form equation, and incorporate the potential endogeneity of A without identifying the complete structure that generates observed receipt of advice and alcohol demand. Several alternative structures could be behind equation (1). In the alcoholism example discussed above, past drinking is a determinant of the receipt of advice, and both past drinking and advice receipt help determine current drinking. But with past drinking hard to observe, physicians might follow various rules of thumb in pursuit of various objectives (e.g. profits versus ethical concerns), in which case past drinking may or may not be a determinant of advice receipt. To sort out these alternative structures would require richer longitudinal data. Our approach focuses on the problem of estimating the treatment effect of advice on alcohol demand without specifying the full structure.

3. THE ECONOMETRIC MODEL AND ESTIMATOR

We chose the following simple probit-type specification for the physician advice model characterized by equation (2):

$$A = \begin{matrix} 1 \text{ iff } a > 0 \\ 0 \text{ otherwise} \end{matrix} \tag{3}$$

where $a = z\alpha + \nu$; A, z, and ν are defined as in equation (2); α is a conformable row vector of parameters to be estimated, and ν is standard normally distributed (conditional on z and x). In specifying the alcohol demand equation (1) we take account of the fact that the dependent variable (D) is non-negative (number of drinks consumed in the past two weeks) and, assume that

$$E[D|x, z, A, \nu] = h(x\beta + \gamma A + \theta\nu, \omega) \tag{4}$$

[5] There are a number of econometric techniques that have been used to handle count data and/or observed zeros for the dependent variable in contexts that do not involve an endogenous treatment affect: (1) tobit analysis (Tobin, 1958); (2) the sample selection approach of Heckman (1976, 1978, 1979); (3) the exponential conditional mean regression model of Wooldridge (1992) and Mullahy (1997, 1998); (4) hurdle models as described in Mullahy (1986); (5) the Poisson regression model; (6) the negative binomial regression model; (7) the two-part model of Duan *et al.* (1983); and (8) the zero-altered Poisson model of Greene (1994). The latter four approaches have been compared by Grootendorst (1995). In models with neither count data nor zero values for the dependent variable, the popular two-stage technique originated by Heckman (1978) is most often used to deal with an endogenous treatment effect.

where D and x are defined as in equation (1); β is the column vector of parameters conformable with x; γ denotes the advice treatment effect, θ and ω are scalar parameters; and

$$h(q,\omega) = \begin{array}{lll} (1+\omega(q))^{1/\omega} & \text{for} & \omega \in (-\infty,0)\cup(0,\infty) \\ \exp\{q\} & \text{for} & \omega = 0 \end{array}$$

The form of the demand equation (4) is based on the inverse Box-Cox (1964) (IBC) formulation originally proposed by Wooldridge (1992) and implemented in an endogenous switching context by McGeary and Terza (1998). This specification is quite flexible and nests two common functional forms, i.e. the linear model ($\omega = 1$) and the exponential model ($\omega = 0$). Note also that equation (4) accommodates the potential endogeneity of A through direct inclusion of ν in the specification of the conditional mean of D—if $\theta = 0$ then A is exogenous and a conventional least squares estimator will be unbiased (consistent). The parameter θ therefore embodies the endogeneity of the regressor A in the drinking equation (4). For example, if unobservable aspects of the individual's personality simultaneously increase the likelihood that the individual will receive advice from a physician *and* his consumption of alcohol, we would expect θ to be positive. In this case, otherwise unbiased methods of estimating β will produce biased results because positive (and negative) influences on alcohol consumption that are *actually* due to unobservable influences will be spuriously attributed to the effect of physician advice. Estimation methods for the exponential version of this model ($\omega = 0$) are proposed by Terza (1998).[6]

We adopt the regression specification in equation (4) for two reasons. First, the conventional model with an additive normally distributed error term would violate the restriction on the range of the dependent variable. In the conventional model proposed by Heckman (1978), the additive error term is assumed to be bivariate normally distributed with ν (conditional on x and z). The endogeneity of A would then be accounted for in estimation by including a correction term (inverse mills ratio) in the regression specification, and then applying a conventional least-squares estimation technique. The problem with this approach in the present context is that the assumed normality of the regression error term holds open the possibility that the value of the dependent variable D could be negative. As can be seen in expressions (3) and (4) this is overcome in our formulation through direct inclusion of ν as an unobserved (or omitted) regressor.[7] Our model thus allows for the potential endogeneity of A while maintaining the integrity of the restriction on the range of the dependent variable. Moreover, our model is less parametric than the conventional Heckman-type model because neither the full distributional specification of D (conditional on x and z) nor the joint normality of D and a are required.

The second reason for our specifying the model as in equation (4) is that it appropriately accommodates observed zero values of D. The conventional semi-log version of Heckman's method in which $D = \exp\{x\beta + \gamma A + \zeta\}$ and ζ is joint normally distributed with ν is thwarted by the fact that, in the presence of zeros, the model cannot be linearized via the log transformation.

The model is estimated using the two-stage procedure developed by McGeary and Terza (1998). This technique, which is an extension of the approach taken by Terza (1998), is similar to the method developed by Heckman (1978) but takes into account the fact that the dependent variable in the drinking equation is limited in range. In the first stage, maximum likelihood probit analysis is

[6] Alternative methods for dealing with endogenous switching (sample selection and endogenous treatment effects) in count data models have been suggested by Greene (1994), Lee (1997), and Weiss (1995).

[7] A similar approach is taken by Mullahy (1997) in dealing with more general forms of endogeneity in exponential regression models.

used to estimate α, the vector of parameters of the advice equation. In the second stage, non-linear least squares is applied to

$$D = h^*(x, z, A, \beta, \theta, \hat{\alpha}) + e \tag{5}$$

where

$$h^*(x, z, A, \beta, \theta, \hat{\alpha}) = E[D|x, z, A]$$
$$= A\frac{\int_{z\hat{\alpha}}^{\infty} h(x\beta + \gamma A + \theta\nu, \omega)\phi(\nu)\mathrm{d}\nu}{\Phi(z\hat{\alpha})} + (1 - A)\frac{\int_{-\infty}^{z\hat{\alpha}} h(x\beta + \gamma A + \theta\nu, \omega)\phi(\nu)\mathrm{d}\nu}{1 - \Phi(z\hat{\alpha})}$$

$\hat{\alpha}$ is the first-stage probit estimate of α, e is the random error term, and $\phi(\cdot)$ and $\Phi(\cdot)$ denote the standard normal pdf and cdf, respectively.[8]

The model is technically identified through functional form. However, our empirical model also implies a set of exclusion restrictions, i.e. variables to be included in z as determinants of advice receipt that can be excluded from x, the set of alcohol demand determinants. Because physician advice is a type of medical care, we assume that factors including health insurance status, physician contacts, and health problems influence the probability of advice receipt by influencing both the frequency and content of patient–physician contacts, but are not direct determinants of the demand for alcohol. Standard models of the demand for alcohol as a consumer good provide a theoretical basis for our exclusion restrictions. Moreover, our specification of the alcohol demand model is consistent with previous empirical research (cited in the introduction above) that omit these variables as direct determinants of alcohol demand. However, because alcohol is a health-related good, the validity of excluding physician contacts and health problems from the alcohol demand equation could be questioned. Accordingly, we test this restriction (described later).

One last practical advantage of our approach for estimating the determinants of alcohol demand deserves comment. As in a number of recent studies, we rely on self-reported measures of drinking behaviour. Although Anda *et al.* (1987, 1988) find that self-reported measures similar to the one used below are well correlated with objective measures of alcohol-related crashes and injuries, measurement error is an obvious concern. In Appendix A we show that under certain reasonable assumptions our estimator will be consistent in the presence of measurement error in the drinking variable (D) and/or the binary advice variable (A). This follows from the fact that such measurement errors serve only to introduce additional sources of correlation between the unobservables in the drinking equation and the unobservables in the advice equation, a problem which our estimation strategy is designed to correct.

4. THE DATA

The data are from responses to the 1990 National Health Interview Survey core questionnaire and special supplements. Table I provides the definitions and means of the variables used in the analysis. The dependent variable for the analysis is the number of alcoholic beverages consumed in the last two weeks. This is calculated as the product of self-reported drinking frequency (the number of days in the past two weeks with any drinking) and drinking intensity (the average

[8] GAUSS© software for this estimator is available upon request from Joseph Terza.

number of drinks on a day with any drinking). For the analysis, lifetime abstainers and former drinkers with no drinking in the past year are excluded from the sample. Observations on the dependent variable are non-negative and often zero-valued (21%), reflecting current year drinkers with no consumption in the past two weeks. Thus the above-mentioned 'zeros problem' cannot be ignored in our econometric analysis.

Physician advice about drinking is based on respondents' answers to the following question: 'Have you ever been told by a physician to drink less?' This measure is used as the key explanatory variable to test whether information provided by the physician shifts demand towards less drinking.[9] The question about advice was asked in context as a way to reduce high blood

Table I. Variable means and definitions

Variable label	Definition	Mean
Dependent variable		
D	Total drinks	14.697
Advice		
A	Drinking advice	.279
Socioeconomic variables (x and z)		
EDITINC	Monthly income ($1000)	2.575
AGE30	$30 < \text{age} \leq 40$	.180
AGE40	$40 < \text{age} \leq 50$	.195
AGE50	$50 < \text{age} \leq 60$	.182
AGE60	$60 < \text{age} \leq 70$	.199
AGEGT70	$70 < \text{age}$	.122
EDUC	Years of schooling	12.925
BLACK	Black d.v.	.133
OTHER	Non-white, non-black	.018
MARRIED	Married	.645
WIDOW	Widowed	.052
DIVSEP	Divorced or separated	.160
EMPLOYED	Employed	.666
UNEMPLOY	Unemployed	.029
NORTHE	Northeast	.217
MIDWEST	Midwest	.275
SOUTH	South	.295
MEDICARE	Insurance through Medicare	.252
MEDICAID	Insurance through Medicaid	.031
CHAMPUS	Military insurance	.059
HLTHINS	Health insurance	.814
REGMED	Reg. source of care	.821
DRI	See same doctor	.721
MAIORLIM	Limits on major daily activ.	.086
SOMELIM	Limits on some daily activ.	.077
HVDIAB	Have diabetes	.061
HHRTCOND	Have heart condition	.146
HADSTROKE	Had stroke	.036

$n = 2467$; # of observations for which $D = 0$ is 527.

[9] As reviewed by Dufour (1996), evidence is accumulating that low and moderate levels of alcohol consumption can be associated with health benefits (e.g. protection against coronary heart disease). It is unlikely that individuals in our sample received advice about the health benefits of drinking because (1) the evidence was more speculative in 1990, the survey year; and (2) the sample is restricted to those with high blood pressure, and drinking was already a well-established risk factor for that chronic condition.

pressure. Accordingly, for our analysis we use a sub-sample of 2467 males who are current drinkers and report having been told at some time that they had high blood pressure.[10]

The remaining variables in Table I are measures of socioeconomic characteristics used as additional explanatory variables in the alcohol demand equation, and a set of variables measuring health insurance, physician contacts, and health problems which are specified as determinants of the probability of advice receipt.[11] In this sub-sample of males with hypertension the average age is 51, and about 30% are not in the labour force, mainly due to retirement. The measure of income is the sum of personal income from all sources including wages and salaries, self-employment, social security and other pensions, public assistance, and interest and dividends.

As can be seen in Table I, about 28% of drinkers with hypertension report having been advised to drink less. Physicians could be providing advice more often than individuals report receiving it. However, findings from surveys of physicians tend to support the patterns found in surveys of individuals. For example, a survey of primary care physicians in Massachusetts found that less than half (47%) routinely ask their patients about exercise and diet habits, although more asked about smoking (90%) and drinking (85%) (Wechsler *et al*., 1983). Physicians may ask about their patients' drinking and still fail to provide advice. Wells *et al*. (1984, 1986) find that, at most, only about half of the physicians routinely counsel a high percentage of all patients with poor health habits. Based on their findings, Wells *et al*. (1984) suggest that 'doctors may operate on a self-referential principle such as "As long as your patient drinks less than you do, he's okay" (p. 2848). Others have noted that physicians may not provide advice because they do not believe it is effective (Wechsler *et al*., 1983). Whatever the reasons, the evidence from surveys of either individuals or physicians confirm that physicians often fail to provide advice.

Before we turn to the econometric estimation of the determinants of alcohol demand, Table II presents cross-tabulations of alcohol outcomes and health information levels by advice status, to provide a first look at some of the hypothesized relationships in the data. On average alcohol consumption is higher for those who had received advice to drink less. This is true for total consumption over the past two weeks (the dependent variable used below), as well as for both the frequency and intensity of drinking in the past two weeks. In addition, people receiving advice self-report more occasions of drunk driving in the past year. These patterns suggest that physicians may target advice to their heavier drinking patients, as in the alcoholism example discussed above. In non-experimental data this targeting may mask any treatment effect where advice lowers consumption.

[10] Restricting the sample to males makes sense because both high blood pressure and problem drinking are much more prevalent among males than females. The sample restrictions could raise an issue of bias if health problems determine drinking status and hence sample selection, but evidence suggests that health problems are not the main reason most non-drinkers do not drink. The National Health Interview Survey asked people who were not drinkers in the current year their main reason for not drinking. Of males who were not current drinkers, almost 75% responded that their main reason for not drinking was one of the following: 'no need/not necessary', 'don't care for/dislike it', 'religious/moral reasons', or 'brought up not to drink'. That is, most non-drinkers simply are not in the market for alcoholic beverages for exogenous reasons. About 12% of non-drinkers gave as their reason 'medical/health reasons', and a fraction of a per cent identify themselves as recovering alcoholics. Looking at the receipt of advice by non-drinkers and their reasons for not drinking provides some evidence that there may be a small selection effect where some people are not in the sample because of the health effects of drinking.

[11] Measures of alcohol prices and availability cannot be merged with the National Health Interview Survey because the public use version removes geographic identifiers. As long as such measures are uncorrelated with the individual characteristics included as explanatory variables in our model, the omission of prices and availability measures should not cause important bias problems.

Table II. Alcohol outcomes and health information by advice status

	Advice not received	Advice received
Alcohol outcomes		
Total drinks in past two weeks	12.64	20.43
Drinking frequency in past two weeks	4.38	5.57
Drinking intensity in past two weeks	2.12	2.85
Drunk driving in past year	0.77	2.30
Health information		
Per cent agreeing that high blood pressure definitely increases the chance of heart disease	66.98	71.44
Per cent agreeing that high blood pressure definitely increases the chance of a stroke	75.76	76.33
Per cent agreeing that alcohol is strongly associated with high blood pressure	39.76	56.05
Per cent agreeing that alcohol definitely increases the chance of throat cancer	17.29	25.16
Per cent agreeing that alcohol definitely increases the chance of liver cirrhosis	81.91	85.14
Per cent agreeing that alcohol definitely increases the chance of mouth cancer	14.10	21.34

At the same time, the patterns in Table II suggest that people who have received advice to drink less are better informed about the health consequences of drinking. Recalling that the advice was given in context as a way to reduce high blood pressure, it is notable that 56% of those receiving advice agreed that alcohol consumption is strongly associated with high blood pressure, compared to only 40% of those who did not receive advice. Smaller differences suggest that alcohol advice receipt is associated with somewhat better information about the other health consequences of drinking and about the consequences of high blood pressure. It is intriguing that compared to those who did not receive advice, people who received advice are better informed about the health consequences of drinking but still drink more. This is additional suggestive evidence that there may be a treatment effect of advice which is masked in the non-experimental data by the targeting of advice to the heavier drinkers.

The observed extent of health information in the cross-section is consistent with individuals acquiring and incorporating new findings into their information sets at different rates and sometimes slowly. This suggests that there is at least the potential for physician advice on drinking to play an important information-providing role. In particular, the physician may be able to fill in the gaps in the individual's information set and tailor the advice to specific circumstances. It should be pointed out that it is not clear whether individuals' information sets tend in the long run towards accuracy. For example, Viscusi (1990) provides evidence that the extensive anti-smoking campaign has led many people to overestimate the risk of lung cancer due to smoking. Whether the resulting information set is more accurate, and thus the choices 'better' in the sense of being closer to the privately optimal decisions, cannot be addressed.

5. RESULTS

Table III presents results from alternative specifications of the alcohol demand functions. In the development of the econometric model in Section 3, the potential endogeneity of the advice variable and the non-negativity of the dependent variable (number of drinks) were of particular concern. For this reason six sets of results are presented in the columns of Table III from left to right: (1) OLS results that account for neither non-negativity nor endogeneity (Uncorrected OLS); (2) results from a model that assumes a fixed exponential functional form and thus accounts

Table III. Comparison of estimators for the alcohol demand equation

Variable	Uncorrected OLS		Uncorrected exponential		Uncorrected IBC		Corrected OLS		Corrected exponential		Corrected IBC	
	Coeff.	*t*-stat.	Coeff.	*t*-stat.	Coeff.	*t*-stat.	Coeff.	*t*-stat.	Coeff.	*t*-stat.	Coeff.	*t*-stat.
$A(\gamma)$	7.671	7.542*	0.455	6.304*	0.328	6.223*	−18.564	−1.821	−1.400	−1.614	−1.131	−2.110*
θ							15.790	2.615*	1.143	2.208*	0.846	2.685*
ω											−0.112	−3.667*
CONSTANT	20.825	7.100	3.167	14.445	2.672	16.558	30.345	6.166	4.256	5.853	2.816	12.022
EDITINC	0.063	0.668	0.003	1.095	0.002	1.105	0.041	0.759	0.001	0.218	−0.001	−0.352
AGE30	0.147	0.085	0.013	0.104	0.011	0.119	2.483	1.256	0.209	1.167	0.139	1.246
AGE40	−0.865	−0.493	−0.088	−0.663	−0.071	−0.713	0.826	0.438	0.053	0.316	−0.136	−1.161
AGE50	−2.107	−1.178	−0.121	−0.836	−0.086	−0.790	−0.517	−0.262	0.017	0.095	−0.044	−0.412
AGE60	−1.604	−0.842	−0.123	−0.805	−0.094	−0.833	−0.141	−0.064	0.035	0.182	0.003	0.027
AGEGT70	−5.189	−2.313*	−0.516	−2.781*	−0.394	−2.836*	−4.584	−2.016*	−0.444	−2.066*	−0.338	−2.394*
EDUC	−0.388	−2.461*	−0.033	−2.795*	−0.025	−2.821*	−0.713	−3.374*	−0.061	−3.162*	−0.040	−3.089*
BLACK	−5.809	−4.174*	−0.451	−3.923*	−0.333	−3.867*	−2.929	−1.808	−0.258	−1.696	−0.158	−1.672
OTHER	−6.536	−1.910	−0.506	−1.780	−0.375	−1.723	−4.242	−1.654	−0.310	−0.950	−0.194	−0.828
MARRIED	−2.157	−1.506	−0.160	−1.454	−0.115	−1.421	−1.301	−0.860	−0.097	−0.727	0.018	0.203
WIDOW	1.123	0.448	0.203	0.939	0.162	1.021	3.552	1.192	0.389	1.397	0.398	2.312*
DIVSEP	3.093	1.771	0.197	1.432	0.145	1.446	5.169	2.380*	0.364	2.037*	0.379	3.150*
EMPLOYED	0.467	0.355	0.032	0.305	0.020	0.258	−0.801	−0.529	−0.065	−0.506	−0.041	−0.465
UNEMPLOY	9.331	3.232*	0.462	1.969*	0.321	1.929	10.201	2.132*	0.549	2.073*	0.373	2.580*
NORTHE	−0.854	−0.616	−0.008	−0.071	0.001	0.011	−0.115	−0.085	0.065	0.510	0.123	1.498
MIDWEST	−0.991	−0.755	−0.016	−0.172	−0.007	−0.096	−1.360	−1.060	−0.048	−0.430	−0.010	−0.133
SOUTH	−1.105	−0.848	−0.066	−0.695	−0.044	−0.628	−1.268	−1.024	−0.084	−0.758	0.034	0.443
SSR	1215105.4		1209883.6		1209362.2		1210928.6		1205159.7		1173015.5	

* Statistically significant at the 5% level.

for non-negativity but does not allow for the potential endogeneity of advice (Uncorrected exponential); (3) Wooldridge-type IBC estimates that account for non-negativity but not for endogeneity (Uncorrected IBC); (4) OLS results with a Heckman-type correction for endogenous advice (see Heckman, 1978) but no provision for non-negativity (Corrected OLS); (5) non-linear least squares results that account for both endogeneity and non-negativity, but assume a fixed exponential functional form (see Terza, 1998) (Corrected exponential); and (6) estimates via the technique described in Section 3, that is based on Wooldridge's IBC model, account for endogeneity and non-negativity, and allow flexibility in the functional form of the regression (Corrected IBC).

As can be seen from columns one to three, estimates that ignore the endogeneity of advice lead to the counterintuitive conclusion that physician advice has a *positive* and *statistically significant* effect on drinking. The results in columns four to six were all obtained via methods that correct for endogeneity. In all these cases the advice treatment effect is *negative* as expected though it is statistically significant at the 5% level only for the corrected IBC estimates in column six. Note that the parameter θ captures the potential correlation between alcohol demand and the unobservables that determine receipt of advice. In all the models corrected for endogeneity (columns four to six) θ is positive and statistically significant at the 5% level—a result that would be consistent with the alcoholism example discussed earlier. These results reveal the importance of correcting for the endogeneity of advice in estimation.

Correcting for endogeneity raises several specification issues that can be explored. Recall that in addition to non-linearities, the model is identified through exclusion restrictions involving a set of eleven variables related to health insurance status, physician contacts, and health problems. A likelihood ratio test supports the joint significance of this set of variables in the first stage model of advice receipt, suggesting we have identified important determinants of advice receipt.[12] The validity of excluding physician contacts and health problems as direct determinants of alcohol demand could be questioned. In order to test this exclusion we estimated the model with those variables included and then conducted a Wald test of the null hypothesis that the coefficients of these variables are zero and were not able to reject the exclusion restriction, providing further support for the specifications reported in Table III.[13]

It is also clear from Table III that accounting for the non-negativity of the dependent variable and allowing for flexibility in the functional form of the regression are important. The former is evidenced by the smaller sum of squared residuals (SSR) for the Corrected exponential and Corrected IBC models versus the Corrected OLS specification; the latter follows from the fact that ω is significantly different from zero. Moreover, the estimated advice treatment effect is statistically significant at any reasonable level for the Corrected IBC model, which accounts for non-negativity in the most flexible way. In the other models that correct for the endogeneity of the advice variable the treatment effect is negative but not statistically significant at the 5% level. The importance of allowing for flexibility of functional form is demonstrated by the statistical significance of the IBC parameter (ω). Rejection of the null ($H_0 : \omega = 0$) is tantamount to rejection of the exponential

[12] Results from the first-stage probit model of advice receipt are presented in Appendix B. The test statistic (distributed χ^2 with 11 degrees of freedom) for the restriction that the set can be omitted from the probit model is 65.57, which is significant at better than the 0.001 level.

[13] The value of the Wald statistic ($\chi_{(7)}$) for testing the joint significance of *REGMED, DR1, MAJORLIM, SOMELIM, HVDIAB, HHRTCOND*, and *HADSTROK* is 0.864.

formulation. Moreover, because the estimated value of ω is negative, statistical significance in this context implies rejection of the linear model ($\omega = 1$).

Because the Corrected IBC model appears to yield the best fit for the data and leads to rejection of the Corrected exponential model, we focus on the results obtained in the sixth column of Table III. The results support the prediction that physician advice shifts alcohol demand down to more moderate levels. Because of the non-linear specification the magnitude of the effect is not readily interpretable from the estimated coefficient. We compute the marginal effect as the change in the conditional mean function attributable to physician advice as if it were exogenously given, *ceteris paribus*. To compute this effect we must choose a starting point on the alcohol consumption curve and assume that $\theta = 0$ (this imposes the exogeneity condition). At the median of the dependent variable, the point estimate of the effect of physician advice is a reduction in two-week drinking by almost $4\frac{1}{2}$ drinks, or by about 72%. The 95% confidence interval around this point estimate implies the reduction in two-week drinking could be as small as about $\frac{1}{2}$ drink (a 9% reduction) or as large as almost $5\frac{1}{2}$ drinks (a 90% reduction). The predicted change in drinking varies depending upon the starting point (results available upon request), but is fairly close to these results in percentage terms.

Is the estimated effect of physician advice on drinking too large to be credible? The evidence from controlled clinical trials, where credibility comes from the experimental design, helps put our econometric results into perspective. In the WHO trial, at-risk drinkers who received advice decreased their average 2-week consumption by 14 drinks (WHO, 1996), compared to our point estimate that advice causes a reduction of $4\frac{1}{2}$ drinks. Of course, because the subjects in the WHO trial at baseline were heavy drinkers their consumption fell by a smaller amount in percentage terms: an average 17% reduction compared to our point estimate of a 72% reduction. However, the confidence interval around our point estimate includes the possibility that even in percentage terms the effect of advice in our sample was as small as or smaller than the estimated effect in the samples of at-risk drinkers in the WHO trial.

Our point estimate, that physician advice has a relatively larger effect in our sample than that found in clinical trials, seems sensible for several reasons. First, the average baseline drinking in the WHO sample was over four times the average in our sample. Thus the clinical trials provide evidence for a narrow range and unusually high level of drinking where the effect of advice could be low if at-risk drinkers are more resistant to changing their consumption. Second, due to data availability we focused on the effectiveness of advice about drinking in the context of hypertension control. This context is again much different than the context of the clinical trials. On balance, it seems plausible that moderate drinkers are more willing to reduce consumption to achieve the specific health goal of a lower blood pressure, compared to the samples in the clinical trials. Finally, it should be noted that the evidence from clinical trials suggest that 5 minutes of advice has as much impact as more involved interventions (Bien, Miller and Tonigan, 1993; WHO, 1996). This helps alleviate the suspicion that the effectiveness of advice might be more limited than its efficacy, if in everyday practice advice is less intensive than under the trial protocols.

The other explanatory variables that emerge as statistically significant determinants of the demand for alcohol are: age, schooling, marital status, and employment status. In keeping with the focus of this paper these results will not be discussed in detail, but it should be noted that the broad patterns are consistent with previous studies.

6. CONCLUSIONS

In this paper we use a microeconometric model and non-experimental data to estimate the effectiveness of physician advice on alcohol consumption. The results support our contention that the receipt of advice should be considered a potentially endogenous explanatory variable. When we correct for endogeneity, we find evidence that physician advice can lead to a reduction in alcohol consumption. This evidence suggests that the efficacy of physician advice as demonstrated in clinical trials may translate into effectiveness in everyday practice.

With evidence that physician advice encourages healthier lifestyles, the prevalence of advice becomes a matter of policy concern. There are several factors that limit the applicability and usefulness of our results for alcohol policy analysis. First, the effect of physician advice on drinking is estimated for a sample of male hypertensives. With almost one quarter of adults having hypertension, this is a limited but not trivial subsample of the population. The effect of advice might be smaller (or larger) in other subsamples and in the general population. Second, the estimated effect of advice is imprecise, so strong policy conclusions based on the point estimate of the effect are unwarranted.

Nevertheless, illustrative calculations suggest that policies to encourage physician advice about drinking to patients with hypertension are likely to yield substantially more benefits than costs. Our estimates imply that 5 minutes of advice from a physician could reduce the median drinker's 2-week alcohol consumption by almost $4\frac{1}{2}$ drinks, or about 117 drinks on an annual basis. Manning *et al.* (1991) estimate the external costs heavy drinkers impose on the rest of society, which allows us to place a dollar value on the social benefits of reduced alcohol consumption. Adjusted for inflation, their best estimate of the external cost per drink is \$0.33, implying that the physician's intervention yields about \$39 (117 times \$0.33) of social benefits. To estimate the social cost of the intervention, we can value the patient's time at the average hourly earnings (\$11.44/hour) and the physician's time at the average net income per hour of patient care (\$72.80/hour).[14] Calculated this way the cost of the 5-minute intervention is about \$7.00, so the net social benefits are about \$32 per drinker given advice.

The above calculations are illustrative rather than definitive. By focusing on external costs these calculations ignore the benefits to the drinker himself. A more complete cost–benefit analysis would add the benefits of improving the drinker's information, which could be substantial. On the other side of the ledger, a more complete cost–benefit analysis should also include the resource costs of training programmes needed to encourage physicians to provide more advice. If every hour of physician training costs \$100 (physician time plus instruction costs) and yields only 10 new drinking patients advised, the social costs per drinker advised rise to \$17. The policy of encouraging physician advice is still estimated to yield net social benefits of \$22 per drinker. For comparison, Fleming *et al.* (1998) use results from a controlled clinical trial to estimate the benefits and costs of brief physician advice for problem drinking. They find a similarly high ratio of benefits to costs, but because they are considering a more intensive intervention for a group of problem drinkers the absolute magnitude of net benefits per patient is much larger (\$920.93).[15]

[14] These figures come from the *Statistical Abstract* (US Bureau of the Census, 1996), Table 659, Average Hourly and Weekly Earnings, and Table 184, Medical Practice Characteristics.

[15] On the cost side, Fleming *et al.* (1998) estimate the costs of two 30-minute visits, compared to the 5-minute session in our illustrative calculation. The shorter intervention seems a more plausible description of advice in everyday practice and other studies (Bien, Miller and Tonigan, 1993; WHO, 1996) indicate that interventions of this length can significantly reduce drinking. On the benefits side, participants in their intervention group reduced alcohol consumption by an average

The limited evidence available tends to suggest that a more complete cost–benefit analysis would conclude that encouraging physician advice in everyday practice is a desirable policy. Given the importance of physician advice, failing to give advice could be viewed as physician incompetence or medical error, a topic of recent policy concern (see Kohn, Corrigan, and Donaldson, 2000). So put differently, the available evidence suggests that policies to reduce this type of medical error are likely to generate more benefits than costs.

It is also interesting to compare the effectiveness of physician advice to taxation, a prominent alternative alcohol control policy. Several recent estimates suggest that the price elasticity of heavy drinking may be about −1.0.[16] If so, to achieve the same 72% reduction in drinking that we estimate would result from advice would require substantial tax hikes to increase the average price of alcohol by about 72%. Even if they could be enacted, large alcohol tax hikes have the disadvantage of imposing large costs on responsible moderate drinkers. Physician advice has the potential to be a policy tool that is both effective and can be precisely targeted. The effectiveness of physician advice on drinking in other population subgroups and in the general population deserves greater attention.

An important subject for future work is the extent to which lack of access to medical care is an important barrier to the receipt of physician advice on lifestyle-related topics. Our preliminary results suggest that lack of access does not appear to be the main barrier. For example, the results of the first-stage selection equations (reported in Appendix B) do not show a consistent pattern that people with health insurance are more likely to receive lifestyle advice. These results are relevant to the question of whether health insurance reforms are likely to have much of an impact on the prevalence of advice. However, much more analysis of these patterns is required before any policy implications should be drawn.

APPENDIX A THE EFFECT OF MEASUREMENT ERROR

Under certain reasonable assumptions our estimator will be consistent in the presence of measurement error in the drinking variable (D) and/or the binary advice variable (A). To simplify the argument consider the case in which the drinking equation is linear. We have

$$E[D^*|x, z, A^*, \varepsilon] = x\beta + \gamma A^* + \varepsilon \tag{A1}$$

$$a^* = z\alpha + \zeta \tag{A2}$$

$$A^* = 1 \text{ iff } a^* > 0$$

where the '*' denotes the true (unreported) value of the variable, with

$$D^* = D + \eta$$

$$a^* = a + \psi$$

of 419 drinks per patient per year, compared to our estimate of 117 drinks per patient per year. This difference is to be expected because their study focuses on a population of very heavy drinkers. Fleming *et al*. (1998) do not base their estimate of benefits on the reduction in alcohol consumption. Instead, they collected data on health care utilization, legal events and motor vehicle accidents to build up an estimate of the benefits of the intervention. As a result their estimate is more complete than ours, reflecting both external benefits and benefits to the drinkers.

[16] Manning, Blumberg and Moulton (1995) estimate that at the 90th percentile of drinkers the price elasticity of demand is −1.19. Kenkel (1996) estimates that the price elasticity of the frequency of drinking is −0.83 (on average, for males). Both studies find evidence that a subset of heavy drinkers may be entirely unresponsive to price increases, another reason to prefer alternative policy interventions to combat alcohol abuse.

D = the reported amount of drinking

a = the latent index underlying the reported physician advice variable

ε = a random error term

and η and ψ are the respective measurement errors. The general endogeneity problem arises because the unobservables that influence drinking (ε) may be correlated with the unobservables that influence physician advice (ζ). This is can be stated formally as

$$\text{cov}(\varepsilon, A^*|x, z) = \text{cov}(\varepsilon, \zeta|x, z) \neq 0$$

Possible bias due to measurement error arises because η may be correlated with A^* and/or ψ may be correlated with D^*. The former would hold, for instance, if people who have been told to cut back their drinking actually tend to report that they have (even if they haven't); the latter would be true if people who have not reduced their drinking tend to report that they received no advice to do so (even if they have). These two possibilities can be stated formally as

$$\text{cov}(\eta, A^*|x, z) = \text{cov}(\eta, \zeta|x, z) \neq 0$$

and

$$\text{cov}(\psi, D^*|x, z) = \text{cov}(\psi, \varepsilon|x, z) \neq 0$$

Rewriting equations (A1) and (A2) in their observable forms yields

$$E[D|x, z, A, \varepsilon, \eta] = x\beta + \gamma A + \varepsilon - \eta \tag{A3}$$

$$a = z\alpha + \zeta - \psi \tag{A4}$$

$$A = 1 \text{ iff } a > 0$$

Now let $\nu = \zeta - \psi$. It is clear that under the above assumptions neither $\text{cov}(\nu, \varepsilon|x, z)$ nor $\text{cov}(\nu, \eta|x, z)$ is equal to zero. Suppose, however, that these correlations can be manifested as

$$E[\varepsilon|\nu, x, z] = \theta_1 \nu$$

and

$$E[\eta|\nu, x, z] = \theta_2 \nu$$

as would be the case under bivariate normality of each of $(\nu, \varepsilon|x, z)$ and $(\nu, \eta|x, z)$. We could then rewrite equation (A3) as

$$E[D|x, z, A, \nu] = x\beta + \gamma A + \theta_1 \nu + \theta_2 \nu = x\beta + \gamma A + \theta \nu \tag{A5}$$

where $\theta = \theta_1 + \theta_2$. With equation (A5) and the assumed normality of $(\nu|x, z)$ we could then consistently estimate β, γ, and θ via Heckman's two-stage technique. These estimates would be consistent despite measurement error in *both* D and A.

Note that equation (A5) is similar to equation (4) in the paper. The only difference is that instead of assuming linearity of the right-hand-side, we assume the non-linear inverse Box–Cox (IBC) form—$h(x\beta + \gamma A + \theta\nu, \omega)$. In allowing for the IBC formulation, there are two required adjustments to the above argument; neither of which affects the substance of the argument. First, in writing the drinking equation (A1), and its observable form (A3), the measurement error term in D^* (i.e. η) will have to be involved in D^* in a non-linear way. For example,

$$D^* = Dm(\eta)$$

where $m(\eta)$ is defined such that

$$E[D|x, z, A, \varepsilon, \eta] = \frac{h(x\beta + \gamma A + \varepsilon, \omega)}{m(\eta)} = h(x\beta + \gamma A + \varepsilon - \eta, \omega)$$

In the case in which $\omega = 0$ [so that $h(q, 0) = \exp\{q\}$] we would write

$$m(\eta) = \exp\{\eta\}$$

and

$$E[D|x, z, A, \varepsilon, \eta] = \frac{\exp\{x\beta + \gamma A + \varepsilon\}}{\exp\{\eta\}} = \exp\{x\beta + \gamma A + \varepsilon - \eta\}$$

The second adjustment to the argument that non-linearity requires is that the covariances between ν and ε, and ν and η be such that they can be manifested as in the following IBC analog to equation (A5):

$$E[D|x, z, A, \nu] = h(x\beta + \gamma A + \theta\nu, \omega)$$

which is equation (4) in the paper.

In summary, our estimator remains consistent in the presence of measurement error in the drinking variable, the advice variable, or both. This follows from the fact that such measurement errors serve only to introduce additional sources of correlation between the unobservables in the drinking equation and the unobservables in the advice equation.

APPENDIX B FIRST-STAGE RESULTS FROM PROBIT MODELS OF DRINKING ADVICE

Dependent variable = A	Coefficient	t-statistic
CONSTANT	−0.519	−2.680
EDITINC	−0.002	−0.268
AGE30	0.292	2.657*
AGE40	0.190	1.682
AGE50	0.165	1.428
AGE60	0.193	1.455
AGEGT70	0.186	1.095
EDUC	−0.032	−3.247*
BLACK	0.279	3.397*
OTHER	0.194	0.944
MARRIED	0.143	1.574
WIDOW	0.280	1.867
DIVSFP	0.252	2.351*

(*continued overleaf*)

Dependent variable = *A*	Coefficient	*t*-statistic
EMPLOYED	−0.047	−0.521
UNEMPLOY	0.115	0.655
NORTHE	0.097	1.143
MIDWEST	−0.013	−0.156
SOUTH	−0.013	−0.164
MEDICARE	−0.064	−0.586
MEDICAID	0.135	0.829
CHAMPUS	0.166	1.431
HLTHINS	−0.217	−2.651*
REGMED	0.167	1.520
DR1	−0.029	−0.306
MAJORLIM	−0.002	−0.013
SOMELIM	0.081	0.787
HVDIAB	0.101	0.880
HHRTCOND	0.125	1.504
HADSTROK	0.138	0.950

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