Nicotine addiction and cigarette consumption: a psycho-economic model

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Abstract

The ‘rational addiction’ model of cigarette smoking downplays the influence of social factors and biological dependency. This paper develops a simple model of these factors and presents some causality tests that indicate that nicotine content ‘causes’ smoking with the direction being negative. ©2000 Elsevier Science B.V. All rights reserved.

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Keywords: Nicotine; Addiction; Smoking

1. Introduction

On 20th June 1997 the US tobacco industry reached a historic 368 billion dollar compensation deal with state and federal governments. The deal proposed severe restrictions on the sale, advertising and distribution of tobacco products and smoking in public places. Perhaps the most important feature was that the product would be regulated as a dangerous drug and nicotine steadily withdrawn from cigarettes. The deal was finally dropped because it failed to get through congress. However, its passage was indicative of the tremendous extent to which the tide has turned in the public perception of smoking. The inspiration for the bill was the performance of seven CEO’s from cigarette manufacturing corporations who proclaimed in congress that nicotine was not, in any way, addictive. Combined with the emerging evidence that they have been purposefully manipulating nicotine levels, to

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hook young smokers, this has severely damaged their public image to the point where the proposed deal was an attempt to stave off punitive lawsuits (Goldberg, 1998). The proposal of such draconian measures is in contrast to the vogue for Becker’s rational addiction approach amongst economists which might be seen as endorsing a laissez-faire attitude (see e.g. Winston, 1980). The rational addiction approach claims to draw support from empirical studies of smoking behavior. These studies all use the number of cigarettes per capita as the explanatory variable without taking account of the nicotine content of the cigarettes. This neglects the fact, clearly recognized in the new deal, that the major cause of addiction to cigarette smoking is the presence of nicotine in the cigarette.

In this paper, I discuss the various ways in which the psychopharmacological addictiveness of smoking influences demand. I then develop a model which incorporates this and some of the factors highlighted in the psychological literature on smoking. Using the data of Simonich (1991), I present causality tests on US quarterly series which indicate that nicotine ‘causes’ cigarette smoking and not vice versa. The relationship is negative. This raises problems with the effectiveness of a policy of producing health gains by lowering nicotine content to reduce the damage from cigarette smoking.

2. Background

The societal flow of messages; ‘smoking may seriously damage your health’ etc. has been gathering pace since the Surgeon General’s Report of January 1964. Surveys indicate that many smokers are highly aware of such messages. However, for the individual, it is possible to believe such messages yet act differently. Partly this may be because nicotine is addictive on a basic chemical level and also through secondary reinforcement via ‘scripts’ (Van Raaij, 1990, pp. 168/9). A script is a scenario of interlocking consumption elements which have a reinforcing quality. For example, some people consume cigarettes, alcohol and the other elements of an exciting night life jointly; each is an element of a script such that if the others are present an individual feels a need for the missing element. In addition, there is the cognitive dissonance factor whereby the smoker blocks out the personal relevance of the risk by representing to themselves such beliefs as ‘I am not really a heavy smoker’ ‘If I did not smoke I would have so much stress that I would be more likely to fall ill’ ‘My father smoked like a train and he lived until he was 89’ etc. Indeed, the first promulgations of the cognitive dissonance theory (e.g. Festinger, 1957) used smoking as a prime example.

Jarvik (1977) reports that studies on individuals show that the puff rate goes up, along with the number of cigarettes consumed, when manufacturers lower the intrinsic nicotine content. The rise in cigarette consumption is statistically significant but that of the puff rate is not. The history of the lettuce cigarette is of interest with respect to these characterizations of smoker behavior. In the 1970’s, lettuce cigarettes failed to sell indicating that the ‘hit’ is essential i.e. script elements are not enough by themselves to satisfy the smoker. The relative failure of the nicotine patch shows that the hit by itself is not enough. One is lead to the conclusion that both script and hit elements figure prominently in smoking.

Economists have, since 1933, produced a plethora of studies of the demand for cigarettes (see Cameron, 1998). The commodity has been recognized as addictive in various ways. Work using a simple static demand function has usually expected a ‘low’ elasticity on the
grounds that it will be hard to cut consumption when prices rise. The asymmetric response of addicts to price changes has been considered only by Young (1983); Godfrey (1986) and Conniffe (1995). Houthakker and Taylor (1970) formulate a dynamic demand model in which consumers have a ‘psychological stock’ of habits which is a function of past consumption. This is purely a ‘black box’ approach; no one has ever ventured a conceptual discourse on what exactly the psychological stock of habits is. The current vogue, in cigarette demand estimates, is the ‘rational addiction’ model of Becker et al. (1994) which is again really a stock of habits approach with the catch all black box being ‘human capital’ ‘addiction capital’ or ‘consumption capital’ depending on whether you are Becker and Stigler (1977); Leonard (1989) or Chaloupka (1991). Mochrie (1996) echoes the feelings of these writers: ‘There is no need to examine the nature of this consumption capital, and it seems quite acceptable to treat it as a convenient fiction called into existence to obviate anomalies in the existing theory of the consumer’.

Addiction by its very nature is a pharmacological process. This suggests that maybe we can work with an underlying demand function for ‘kicks’ ‘highs’ or ‘buzzes’ and incorporate the chemical delivery mechanism of these into the model. All consumers can be assumed to have some latent demand for ‘highs’; after all the pursuit of these has been found at all times and in all cultures. It is not clear, in much of the literature, exactly why people do demand addictive goods. In Becker and Murphy (1988) a good is a good despite the identity of the authors. In Leonard (1989) goods are combined with time to make the ‘commodities’ of Becker’s time allocation model. In the model of Chaloupka (1991) smoking and the stock of habits are inputs in a production function for ‘relaxation’. This broadens the picture somewhat, and has a basis in chemistry. Chaloupka further highlights the pharmacological addiction aspect by stating that cigarette demand is a good vehicle for the application of the rational addiction model precisely because of nicotine’s pharmacological addictiveness. However, he makes no attempt to control, in his empirical work, for nicotine delivery in the cigarettes smoked.1

Only four studies, in the vast literature, of the economics of smoking have controlled for temporal heterogeneity in product characteristics. Schneider et al. (1981) and Porter (1986) model cigarette consumption as a function of the market share of filter cigarettes and low tar cigarettes. Their argument is not however one about the content of the cigarette smoked. Rather, the filter and low tar variables are proxies for changes in attitude attendant upon the 1953 and 1964 health scares. The coefficients on these variables are significant and negative but there are severe specification problems; a static demand equation with a Durbin-Watson of 0.98 and the presence of very strong trends in the filter and tar variables suggests the possibility of a ‘spurious’ regression. Simonich (1991) introduces the average nicotine yield per cigarette into the demand equation. He hypothesizes that lower nicotine cigarettes will result in increased cigarette demand. He uses a static log-linear demand equation in which all the variables have been detrended. This is judged to be correctly specified on the basis of a first-order Durbin-Watson test. No account is taken of fourth-order serial correlation. The nicotine variable has a negative coefficient but is insignificant. The equation is part of a three equation model but consumption does not feature in the equation for average nicotine

1 Admittedly the data set used by Chaloupka does not have any nicotine measurement in it. Nevertheless, it is surely time that economists constructed suitable data rather than constantly mining second-hand sources.
yield. A new paper by Evans and Farrelly (1998) concludes, using US time series data, that the tax induced price rises have resulted in a switch to greater tar and nicotine content cigarettes thereby negating the intended health gains. They conclude that taxes should be based on tar and nicotine content rather than the cigarette itself.

3. Model

In this section, I develop a simple model of cigarette consumption which attempts to draw together the threads from economics, psychology and biology discussed above. Let there be two goods; one \((X)\) which is ‘normal’ in the sense that it does not deliver any chemical alterations to the brain state nor is it imbued with any learned components and another \((Z)\) which delivers chemical ‘hits’ to the brain and also provides utility from being a constituent of ‘scripts’ \((M)\) (Van Raaij, 1990, pp. 168/9). As discussed above, a script is a scenario of interlocking consumption elements which have a reinforcing quality. We can distinguish between private and social scripts although this distinction is not incorporated in the formal model. In the private script, one might be smoking as an aid to completing a solo work task or relaxing after a meal. In the social script, the tension and excitement of the presence of other people are factors in generating the desired level of nicotine consumption. The basic idea of the script is that smoking the cigarette becomes intertwined with the experiences which are temporally joint with the nicotine consumption. Over a process of time these associations may be a non-separable element of the utility from the smoking act. This specific aspect is not explicitly incorporated in the formal model for the purposes of simplification. The mental process of association may engender brand loyalty as the style of packet, name of the cigarette etc. can potentially become part of the overall pleasure. The script appears to be an independent element in cigarette consumption. Attempts to curb smoking by supplying nicotine patches etc. have not been particularly successful because the ritual of physically consuming the cigarette is missing and thus the secondary reinforcement with other activities is absent. The utility function is:

\[
U = U(H, M, X, W)
\]  

(1)

where the variables are defined as follows; \(H\) = the ‘hit’ delivered to the brain by the process of inhalation, \(M\) is the standardized volume of ‘scripts’ in which smoking \((Z)\) occurs and \(W\) is the perceived stock of health capital arbitrarily treated here as some single valued positive index of physical wellbeing. \(X\) is the composite good to represent consumption of goods which are not cigarettes and not pharmacologically activating (this is a ‘no other drugs’ version of the model). The available menu of scripts is treated here as being exogenously given. The delivery of hits, scripts and health capital is through the production functions:

\[
H = H(M, Z)
\]  

(2)

\[
W = W(Z, X, k)
\]  

(3)

where \(M\) enters into \(H\) because it modifies the rate and incidence of puffing i.e. smoking in a night club when meeting people would differ, even for the same person, from when in a situation of stress or isolation.
where \( k \) is a filter variable to represent cognitive dissonance (Gilad et al., 1987). There is a threshold level \( k^* \) which can be exceeded by the flow of messages indicating that smoking is a serious health risk. If this happens then there is a switch-over in (1) similar to the ‘flip-flop’ utility function of Winston (1980) who emphasizes the problem of self control, but does so without incorporating ‘hits’ or scripts.

The \( k^* \) filter will be exceeded by events which make the risk more ‘salient’ to the individual such as witnessing at first hand the details of a smoking related death from cancer. For example, the director of the National Society for Non-Smokers, in England, ‘gave up after a surgeon friend forced him to watch the removal of cancerous lung’ (Greenfield and Rosenberg, 1986, p. 10). If there is a switch in perceptions, the smoker could simply reduce nicotine intake by smoking less or puffing less on their current brand. However, the market has an incentive to provide devices to assist self control (up to a point) such as low tar and filter tip cigarettes. Yet the problem of backsliding remains as the apprehensive smoker could slip back to an old higher danger brand or use the supposedly safer brand in a more dangerous way.

For the purposes of illustration, I now provide a version of the model with some specific functional forms. Prices and Income \((Y)\) are assumed to be fixed. Scripts are exogenous to the level of income and, less restrictively, all other variables. A multiplicative form is assumed for the utility and stock of health capital functions. There is a constant puff or hit rate per cigarette modified by the exposure through script activity. The model is thus:

\[ U = A \cdot X^\beta \cdot H^\gamma \cdot M^\nu \cdot W^\xi \quad (4) \]
\[ H = \gamma \cdot Z \cdot M \quad (5) \]
\[ W = \zeta \cdot B \cdot X^\theta \cdot Z^\eta + (1 - \zeta) \cdot \iota \cdot X^\kappa \quad (6) \]
\[ Y = P_z \cdot Z + P_x \cdot X \quad (7) \]

where \( \zeta = 0 \) if \( k < k^* \) and \( \zeta = 1 \) if \( K > k^* \), and the expected signs are:

\[ \alpha, \beta, \gamma, \xi, \eta, \kappa > 0 \quad \eta < 0 \quad \kappa > 0 \text{ or } \kappa < 0 \]

there is ambiguity about the \( \kappa \) parameter as it may be the case that, at the margin, more of other goods may be seen as bad for health e.g. overeating.

After substituting (5) and (6) into (4) and maximizing subject to (7) we obtain the demand function which reflects two states of mind. The aggregate data will be a weighted average of these two states of mind demand functions. The separate functions are:

\[ Z = Y \cdot \frac{(\alpha + \eta \cdot \xi)}{(P_z \cdot (\beta + \theta \cdot \xi + \alpha + \eta \cdot \xi))} \quad \text{when } \zeta = 1 \quad (8) \]
\[ Z = P_z \cdot \alpha \cdot \frac{Y}{(P_x^2 \cdot \beta + P_x^2 \cdot \kappa + P_z^2 \cdot \alpha)} \quad \text{when } \zeta = 0 \quad (9) \]

My interest in this paper is in the impact of nicotine content on demand. This will be shown in the response to two of the parameters in the model that is a higher nicotine content will raise the absolute values of \( \alpha \) and \( \eta \) due to fear and there will be an offsetting increase in \( \alpha \) through the pharmacological effects of smoking on the brain.
Potentially there is also a possible route through modification of $\gamma$ but I am treating this as constant for the purposes of simplification and also because this seems plausible in the light of the clinical studies discussed earlier. There are three relevant derivatives as the $\eta$ parameter does not feature when $\zeta = 0$. These are:

$$f_{z\alpha} = Y \cdot \frac{(\beta + \theta \cdot \xi)}{Pz \cdot (\beta + \theta \cdot \xi + \alpha + \eta \cdot \xi)^2} \text{ for } \zeta = 1$$ (10)

$$f_{z\alpha} = Pz \cdot Y \cdot Pz^2 \frac{(\beta + \kappa)}{(Pz^2 \cdot \beta + Pz^2 \cdot \kappa + Pz^2 \cdot \alpha)^2} \text{ for } \zeta = 0$$ (11)

$$f_{z\eta} = Y \cdot \xi \cdot \frac{(\beta + \theta \cdot \xi)}{Pz \cdot (\beta + \theta \cdot \xi + \alpha + \eta \cdot \xi)^2} \text{ for } \zeta = 1$$ (12)

Given the expected signs of the parameters (10) and (11) will be positive (unless there is a large enough negative value of $\kappa$) and thus nicotine will be positively related to demand although the size of the effect will depend on the value of $k$ the exogenous variable which dictates the distribution over $\zeta$.

The sign of (12) will also be positive by implication but as $\eta < 0$ this predicts a negative relationship between nicotine content and the demand for cigarettes. Consequently, the relationship is indeterminate leading us to an empirical approach in the next section.

4. Empirical results

The data used here cover 1959Q3–1983Q4, in the United States, and are taken from Simonich (1991). The variables are CIGPC = Number of cigarettes sold in the domestic economy divided by the total population of those aged over 14 years old and NIC = sales-weighted average nicotine yield per cigarette. The latter series was computed by Phillip Morris staff using nicotine yields from laboratory tests. It was also found necessary to introduce the dummy SG64 equal to one for the 1964Q1 observation to overcome the appearance, in diagnostic tests, of extreme non-normality in the regression residuals. This captures a sudden, but immediately reversed, decline in smoking in response to the health fears aroused by the report of the Surgeon General in January 1964.

The estimating equations are thus:

$$\text{CIGPC} = a0 + a1 \cdot \text{CIGPC} + a2 \cdot \text{NIC} + a3 \cdot \text{SG64} + u$$ (13)

$$\text{NIC} = b0 + b1 \cdot \text{CIGPC} + b2 \cdot \text{NIC} + b3 \cdot \text{SG64} + v$$ (14)

where $u$ and $v$ are classical disturbance terms.

The CIGPC variable is a measure of the $Z$ variable in the above discussion.

With not measured directly here, a feature common to the literature, but the possibility of an abrupt switch in it through the $k$ factor is approximated by the SG64 variable. The causation from NIC to CIGPC has been extensively discussed above in terms of the opposing forces.

2 I have interpolated the missing values in the earlier part of the series.
Table 1
Cigarette and nicotine equations

<table>
<thead>
<tr>
<th></th>
<th>CIGPC</th>
<th>NIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>INT</td>
<td>170.05</td>
<td>−0.037</td>
</tr>
<tr>
<td></td>
<td>(1.55)</td>
<td>(0.48)</td>
</tr>
<tr>
<td>CIGPC(−1)</td>
<td>0.367</td>
<td>0.00012</td>
</tr>
<tr>
<td></td>
<td>(3.83)</td>
<td>(1.75)</td>
</tr>
<tr>
<td>CIGPC(−2)</td>
<td>−0.099</td>
<td>−0.00012</td>
</tr>
<tr>
<td></td>
<td>(0.96)</td>
<td>(1.61)</td>
</tr>
<tr>
<td>CIGPC(−3)</td>
<td>0.167</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>(1.62)</td>
<td>(1.39)</td>
</tr>
<tr>
<td>CIGPC(−4)</td>
<td>0.294</td>
<td>0.000041</td>
</tr>
<tr>
<td></td>
<td>(3.024)</td>
<td>(0.6)</td>
</tr>
<tr>
<td>NIC(−1)</td>
<td>−297.241</td>
<td>1.145</td>
</tr>
<tr>
<td></td>
<td>(1.91)</td>
<td>(10.45)</td>
</tr>
<tr>
<td>NIC(−2)</td>
<td>263.376</td>
<td>−0.006</td>
</tr>
<tr>
<td></td>
<td>(1.12)</td>
<td>(0.36)</td>
</tr>
<tr>
<td>NIC(−3)</td>
<td>−34.777</td>
<td>−0.22</td>
</tr>
<tr>
<td></td>
<td>(0.145)</td>
<td>(1.35)</td>
</tr>
<tr>
<td>NIC(−4)</td>
<td>122.217</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>(0.79)</td>
<td>(0.62)</td>
</tr>
<tr>
<td>SG64</td>
<td>−142.78</td>
<td>−0.04</td>
</tr>
<tr>
<td></td>
<td>(3.95)</td>
<td>(1.58)</td>
</tr>
<tr>
<td>R Squared</td>
<td>0.577</td>
<td>0.987</td>
</tr>
<tr>
<td>F(9, 84)</td>
<td>12.7398</td>
<td>688.14</td>
</tr>
<tr>
<td>DW-statistic</td>
<td>1.7842</td>
<td>1.92</td>
</tr>
<tr>
<td>CHI-SQ(4) AR(4)</td>
<td>7.8095</td>
<td>2.09</td>
</tr>
<tr>
<td>CHI-SQ(1) Reset</td>
<td>0.0171</td>
<td>0.005</td>
</tr>
<tr>
<td>CHI-SQ(2) Normality</td>
<td>1.1446</td>
<td>4.08</td>
</tr>
<tr>
<td>CHI-SQ(1) Heteroscedast</td>
<td>1.2737</td>
<td>0.05</td>
</tr>
</tbody>
</table>

of the ‘hit’ and ‘health fear’ parameters for those for whom \( k > k^* \). The reverse causation has also been hinted at; viz. falling consumption due to health scares, if the fear effect dominates the hit effect, may induce product innovations in the form of allegedly ‘safer’ cigarette.

As is usual in causality testing it is not possible to make any a priori stipulation about appropriate lag length. Given the periodicity of the data and the limits of degrees of freedom, four periods are chosen here although the results are not sensitive to changes in this.

The results of estimating (1) and (2) by OLS are shown in Table 1.3 The diagnostic tests indicate that this is a generally satisfactory specification although there is some suggestion of fourth order serial correlation in (1). The F(4, 84) test is 2.72 for the ‘nicotine causes smoking hypothesis’ which is significant at the 3.5 percent level whilst that for the ‘smoking causes nicotine’ hypothesis is 1.196 which is significant at the 32 percent level. It seems from the above that there is unidirectional causation running from nicotine to cigarettes. Only the first of the four NIC terms is individually significant. Given that it is negative there is support for the idea that the term shown in (12) above may be dominating the combined

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3 As this is a traditional bivariate causality test we do not take account of income and price explicitly. In the literature (Cameron, 1998) income elasticities have tended to zero and the price elasticity has been about −0.3. Any price effects in this study will be compounded in the quantity lags.
effects of the terms shown in (10) and (11), in aggregate cigarette demand, during this particular period of American history.

Taking into account only the NIC term which is significant at the 5 percent level, and evaluating at the means, the elasticity of demand with respect to nicotine is $-0.43$. The notable feature of this is that it is less than one with this being statistically significant if we work back to the relevant point estimate. Thus, although there is a compensation effect of weaker cigarettes leading to more smoking it is less than 100 percent suggesting that the current ‘phase out nicotine’ approach to cigarette regulation will work to some extent. However, its effect would, on this evidence, be much more muted than seems to be envisaged in the debate in the popular media.

5. Discussion

Surprisingly, only four studies in the econometric literature on smoking have included variables to measure changes in the chemical nature of cigarettes. This empirical neglect is mirrored in the theoretical literature which, having now reached the stage of domination by rational addiction models, has completely overlooked social and pharmacological aspects to the smoking decision. The key to this view is the emphasis on the long run as the planning horizon with individuals having extravagant capacities for foresight. My approach focuses on smoking as behavior which shows a short-run orientation precisely because it is habituated in nature with this habituation being due, to some extent, to chemical dependency. This approach fits in with the bounded rationality espoused by (Akerlof, 1991, p. 5). ‘...I do not agree that the model of forward-looking, rational behavior accurately describes the way in which individuals decide on drug or alcohol intake. Most drug abusers, like most chronically overweight individuals, fully intend to cut down their intake, since they recognize that the long-run cost of their addiction exceeds its benefits. They intend to stop-tomorrow. Individuals following the procrastination model are both maximizing and knowledgeable and yet their decisions are not fully rational.’

In the explicit form of the model I allowed for two opposing effects of nicotine dependence. The first of these works through the high or thrill of a ‘hit’ on a cigarette, whilst the second works through the fear of health damage which is only acted upon when an information filter is triggered. In such a context, the volume of smoking may rise or fall attendant upon a change in nicotine content. This potential offset is overlooked in the current stringent policy measures proposed in the USA, and about to spread in Europe, to control nicotine as a fully listed addictive drug.

Using data from the latter of the studies, which took account of the nicotine in a cigarette, I have found indication of Granger causality from nicotine content to cigarette smoking but not vice versa. The relationship is negative indicating that smokers respond to lower nicotine delivery by increasing consumption. This usage of cigarettes seems more consistent with the self control or cognitive dissonance models dismissed by Becker and Murphy (1988) than the rational addiction model which they put forward. Obviously, I would not be so

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4 There are occasional health economics scholars, such as Hsieh (1998), who adopt a Bayesian learning model to the absorption of health information.
bold as to claim that the empirical work here presented constitutes some overwhelming counter-proof to a proof they have hitherto established but there is least a case to be made for the opposition and hopefully this paper goes some way towards making it.

References


5 The rational addiction model has been plagued by estimated discount rates which seem a long way from ‘rational’ as normally understood. Throughout Becker et al. (1994) this problem is effaced by invoking non-removable biases in the data. Whilst such arguments may have some validity in the context of unusually high discount rates, they do not help in the case of the NEGATIVE discount rates sometimes found.