

An Assessment of Causal Inference in Smoking Initiation Research and a Framework for Future Research

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ABSTRACT

Reliably identifying the causal factors underlying youth smoking initiation is an important part of developing effective smoking prevention programs and shaping other types of smoking-related policies. The establishment of reliable scientific evidence in support of a causal link between cigarette advertising and youth smoking initiation depends both on rich longitudinal data as well as careful empirical applications. We examine basic principles of empirical scientific investigation of potential causal relationships, discuss findings of recent research on causal factors of youth smoking, and evaluate evidence from the public health literature regarding the effects of cigarette advertising on youth smoking.

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I. Introduction

The causal factors underlying youth smoking initiation are central to the challenge of developing effective smoking prevention programs and the shaping of many other types of smoking-related policies. These causal factors, specifically the ones that potentially may be influenced by the conduct of cigarette manufacturers, also have been a central issue in much of the recent smoking-related litigation. These high-profile legal cases have focused on the claim that tobacco company conduct significantly influenced youth-smoking initiation rates. In particular, cigarette advertising and tobacco company public reluctance to acknowledge the health risks of smoking have been purported to be key factors influencing youth smoking decisions.¹

In this paper, we examine the basic principles of empirical scientific investigation that have been adopted to establish causal relationships, and apply these principles to evaluate the evidence underlying the link between cigarette advertising and youth smoking initiation. The causal link between tobacco-company marketing and youth smoking generally has been accepted as empirically established outside the economics literature, despite the fact that the underlying evidence fails to meet commonly-applied econometric standards. We review some of these papers in the public health literature that have been cited as evidence supporting the causal link between cigarette advertising and youth smoking, and explain why these papers fail to support this causal link. Further, we also review the economics literature that provides evidence for a multi-causal model for youth risky behaviors, including smoking. We identify studies in which

¹ When we use the term advertising, we are referring to “non-price” related marketing, such as magazine advertisements, t-shirts and sport sponsorships, which have an image component. Price-related marketing, such as coupons, price discounts or package give-aways, are not referred to as advertising. The qualitative effects of price-related promotions are not in dispute, as well-established evidence in the literature supports a causal relationship between price and cigarette consumption, as is true with most goods and services. See Chaloupka and Warner (2001) for further discussion of studies on cigarette demand.

researchers have found that many characteristics affecting choices to participate in risky behaviors are formed at very early ages.

Empirical demonstration of causation is fundamental to guiding effective development of interventions in many areas of social life. Scientific principles are especially important for developing smoking-related policies, as enormous resources currently are being devoted to develop programs that attempt to reduce youth smoking propensities. However, many of these policies (and suggested policies) focus on causal factors that have not been scientifically established, but rather merely assumed to affect smoking initiation. More effective policies potentially could be developed by distinguishing causal factors through more reliable empirical methodologies. Without identifying factors that lead to smoking initiation through sound estimation techniques, current prevention efforts may turn out to have little or muted benefits.²

II. Causal Factors of Smoking Initiation and The Scientific Model

An important goal of policy analysis is to identify and measure causal relationships, as many confounding correlations exist which do not reflect structural relations underlying actual outcomes. Understanding these underlying structural relations allows empirical versions of estimated models to more accurately forecast the effects of interventions. As is well recognized in the econometrics literature, precise identification and measurement of causal effects is a difficult task in the area of human choice, since input parameters of investigated relationships may reflect unobserved individual characteristics and choices. Thus, correlations between

² Youth smoking prevention programs are not immune to the shortcomings that have afflicted other types of social interventions, where well-intended programs, implemented by well-meaning agencies or institutions, turned out to have negligible or even unanticipated harmful effects. For example, Martin and Grubb (2001) found that some labor market programs appear to yield negative rates of return when the effects are compared to program costs. In a similar vein, subsequent studies have concluded that “displacement effects”—newly trained workers simply displace non-trained workers—may be sizeable, so that total employment is only marginally affected by training programs, again, contrary to expectations, as discussed in Davidson and Woodbury (1993) and Heckman et al.(1998).

endogenous input parameters and observed outcomes may simply represent individual sorting on latent characteristics, rather than structural relations between input and output variables.

Many times in other disciplines, biases associated with endogeneity do not hinder estimation of structural models because observed variables are not affected by individual choice. For example, to evaluate the effect of a particular drug on a particular disease, researchers may be able to perfectly control factors such as the presence of other diseases, dosage regimens, alternative drug usage, and diet. Further, they may be able to precisely observe other parameters that potentially influence disease outcomes, such as age, weight, and other individual characteristics. After identifying statistical relationships between drug usage (and other input parameters) and disease remission, researchers can tentatively draw conclusions regarding the drug's effect.

The task of estimating intervention effects becomes far more difficult when the goal is to evaluate a public policy. Measuring the effect of a social policy, such as welfare reform or restrictions on cigarette marketing, significantly increases the number of factors (both observable and unobservable), and the interactions of factors, that need to be accounted for in the analysis. The complexity of modeling human choice requires far more elaborate theoretical and empirical modeling than would be required to draw causal inferences regarding drug efficacy in a controlled experiment environment.

The explicit recognition of this complexity in the econometric literature has promoted the development of sophisticated empirical (and theoretical) modeling to allow causal inferences regarding human behavior. Unlike causal models in epidemiology and statistics, which typically focus solely on outcomes of treatments, econometric models not only model treatment outcomes, but also the role of choice in the treatment employed. Thoughtfully constructed structural models

in economics consider both the relationship between the input factors determining outcomes and the choices leading to the observation of input factors.

Well-specified empirical models are based on careful reasoning about the potential underlying causal relations and will distinguish among alternative explanations for the same phenomenon. A model of human behavior must not only model the outcomes, but the choices that give rise to the outcomes, and the set of factors that drive these choices (and that may have led to these outcomes). This process is important because researchers observe the outcomes of choices made, and therefore need to distinguish between the effects of the factors that led to the choice from the effects of the choice itself.

Similar problems face most analyses of public policy. The goal, as with most scientific study, is to establish or reject theorized causal relationships. Researchers construct hypotheses, design empirical models or experiments to isolate these factors, gather appropriate data, and implement statistical tests (for model selection and causal inferences). For informative studies on individual choice, rich data is not only desirable but almost always essential. Ideally, researchers would like data that accurately measures the factor being tested (or a good proxy for it), and precise measures of other causal factors, to isolate the effect of the factor being studied. The use of appropriate empirical methodology, including robustness (or sensitivity) tests, provides a basis for interpreting results as scientific evidence of a causal relation. Replication of results by other researchers provides additional support for a sound analysis from which one may draw causal inferences.³

In addition to designing social policies based on empirically identified casual relationships, policymakers face a very complex program evaluation task. At its base is the simple fact that one does not observe counterfactuals, or particularly, how individuals who

experience intervention programs would have done absent that experience. During policy evaluation periods, program designs that base participation on exogenous criteria greatly minimize this evaluation difficulty, however these types of designs may be difficult to implement.⁴

III. Empirically Identified Casual Factors of Youth Smoking

In economics, the test of whether an input is a “causal” factor considers whether outcomes would be changed if that input, and that input alone, were varied. This distinction exists in other scientific disciplines as well. In sociology and epidemiology, which rely upon statistical models to obtain estimates of risk, causal interpretations might indicate differential risks associated with variation of just the variable of interest (all else held constant)

The available evidence in the developing literature on adolescent risky behavior, including smoking, supports a multi-causal model for youth smoking, as many factors have been empirically linked to youth smoking in this literature. These factors include price, parental influences, risk preferences, peer influences, and access.⁵ This literature provides a strong empirical basis in support of a causal relationship between many of these factors and youth smoking, such as price and risk preferences.

For example, a key finding of the economic research on smoking is that increases in the health or direct money costs of smoking lead to declines in smoking propensities. A number of studies have estimated the effect of the direct money price of cigarettes on smoking demand. One of these studies, by Lewit and Coate (1982), found that smoking by young adults (ages 20

³ See Heckman (2005) “The Scientific Model of Causality” for further detailed discussion.

⁴ Otherwise, people who are selected for particular programs could be different in latent characteristics, and hence differences in group outcomes may not be accurately captured by available statistical measures. The importance of designing youth smoking interventions on sound empirical investigations and follow-up evaluations is recognized in disciplines outside of economics. For example, see Biglan et. al. (2004).

⁵ See Becker, et al., (1994) and Chaloupka, (1991) for further elaboration.

through 24) is much more responsive to price than smoking by older adults. Similarly, Lewit, Coate and Grossman (1981) found that propensities for adolescents (ages 12 through 17) declined with increases in cigarette prices, and that adolescent cigarette consumption was more sensitive to price than aggregate demand for other age groups.⁶

Alternatively, many studies have focused on the responsiveness of consumer demand to changes in other components of smoking costs, such as restrictions on access, restrictions on use, and changes in information regarding the long-term health effects of smoking. For example, restrictions on youth access to tobacco, when rigorously enforced, have been found to reduce youth smoking.⁷ Smoking participation also has been found to respond to the release of new information regarding associated health effects, indicating that consumers take into account the health costs of smoking in deciding whether or not to smoke. Similarly, the probability of quitting has been shown to increase with the length of time an individual has smoked, which implies that smokers give greater weight to potential health costs as they become more imminent.⁸

A further finding that is emerging from a variety of recent studies is that characteristics of many sorts are shaped at early ages and these characteristics greatly affect overall development and choices to participate in risky behaviors. Heckman, Stixrud, and Urzua (2006) examine the effects of cognitive and non-cognitive skills, formed at early ages, on behavioral outcomes, including smoking. They find that higher cognitive and non-cognitive skills decrease the

⁶ A consensus across the various literatures supports a multi-causal view, not only of youth smoking, but also of other risky behaviors often initiated in youth, such as drinking alcohol, smoking marijuana, engaging in unprotected sex and experimenting with hard drugs. A synthesis of studies on adolescent behavior, Kipke, *Risks and Opportunities* (1999) concludes: "Indeed, there is mounting evidence that most biological changes interact with a wide range of contextual, psychological, social, and environmental factors that affect behavior."

⁷ See Chaloupka and Pacula (1998).

⁸ Douglas (1998).

probability of smoking by age 18. In Cunha, et al. (2005), childhood development stages are explicitly examined, and the returns to early life cycle investments in skills are found to be high. Further, the authors find that factors observed early in childhood can explain many “risky” behaviors, and that early remediation efforts can be effective in improving adolescent outcomes.⁹

This finding that common factors, related to ability, self-regulation and self-perception, are powerful explanatory variables for adolescent behavioral problems has strong implications regarding future intervention programs. The potential focus of these interventions go well beyond programs targeted solely to adolescents, as many identified factors are determined at very early ages of the child (long before adolescence). For example, one of the major determinants is family --family environments, as well as genetic factors. Family environments include the utero environment created by the mother’s own behavior, such as her own smoking, drinking or stress factors operating on the fetus. The important lesson that is emerging from this literature is that abilities of many sorts are shaped at early ages and they greatly affect child development and child choices, consequently early childhood interventions are important.

Developing and accurately estimating a robust structural model of youth smoking initiation is a complex task, both because it involves many different factors and because these factors likely occur early in childhood development. Hence, identifying advertising as a causal factor (and measuring the magnitude of the related effect) requires rich data sources that include not only information on parental inputs, peer characteristics, exposure to cigarette marketing and other potential influences, but also requires that these variables be observed for extended periods of time (back to early periods of childhood development).¹⁰

⁹ Other examples of such studies include: Bowles et al. (2001), Heckman and Rubenstein (2001), Knudsen (2004), and Turkheimer et al. (2003).

¹⁰ Research on adolescent risk-taking behavior identifies many risk factors associated with drug and tobacco use. These factors include individual factors, such as depression, attention deficit disorder, early

Finally, self-selection may present a very difficult obstacle in measuring causal factor effects for smoking initiation, as researchers have found that many risk-taking behaviors are highly correlated with one another. For example, a study by Morral, McCaffrey and Paddock (2005) investigated whether a common-factor cause model of “drug use propensity” could explain both marijuana and cocaine use, in comparison to a “gateway” theory that using one drug (marijuana) acts as a gateway to using the other (cocaine). Other researchers also have investigated gateway theories and presented evidence that frequent use of one drug leads to increased use of other drugs.¹¹ Distinguishing between these gateway theories versus individual-specific latent characteristics that lead to a variety of risk-taking behavior requires both rich data and a careful econometric methodology.

IV. Youth Smoking Initiation and Evaluation of Advertising as a Causal Factor

As discussed in the previous section, developing and estimating a robust model of youth smoking initiation is a complex task. It is in this context that other hypothesized causal factors, such as advertising, must be evaluated. In this section, we outline the several studies in the public health literature examining the causal link between cigarette advertising and youth smoking initiation. Specifically, we focus on the central studies presented by the Department of Justice in the recent federal tobacco litigation and discuss the reliability of causal claims based on this body of research.

A central claim, in the recent tobacco litigation case, was that the risk of adolescent smoking initiation would be reduced if tobacco-company advertising was reduced, all else equal.

and persistent antisocial, aggressive or rebellious behavior, and impulsiveness; and social influences, such as low levels of parental supervision, parental/sibling attitudes toward and use of drugs, legal penalties and sanctions, prices, and peer use of drugs. Maternal smoking is another predictor of adolescent smoking.

¹¹See Pacula (1998).

The economic interpretation of the government’s claim was that cigarette advertising was a “causal factor” of youth smoking decisions. The establishment of reliable evidentiary support of this claim would include the following elements: (1) valid measurements of both smoking outcomes and alleged causal factors, as well as other probable causes, (2) a clear argument demonstrating that other potential causes of smoking initiation, other than the one in question, have been accounted for, and (3) replicability to allow other researchers to follow and duplicate the analysis. The actual analyses provided as evidence for this claim fell far short of this threshold, as we describe below.

A. Overview of Evidence Provided in Support of the Proposition That Youth Smoking is Caused by Cigarette Advertising

The evidentiary support provided by the government primarily drew upon the public health literature, and specifically the group of papers discussed in the Cochrane Review.¹² The stated objective of the authors of the Cochrane Review was to “assess the effects of tobacco advertising and promotion on nonsmoking adolescents’ future smoking.” (Lovato 2003, 1) Their methodology was to select a set of studies from a search of published studies meeting certain criteria, including a longitudinal design with follow-up from baseline advertising exposure, receptivity or attitudes. Based on their review of nine longitudinal studies, the authors state:

Longitudinal studies consistently suggest that exposure to tobacco advertising and promotion is associated with the likelihood that adolescents will start to smoke. Based on the strength of this association, the consistency of findings across numerous observational studies, temporality of exposure and smoking behaviors observed, as well as the theoretical plausibility regarding the impact of advertising, we conclude that tobacco advertising and promotion increases the likelihood that adolescents will start to smoke.¹³

As their conclusion suggests, the authors attempt to satisfy “causal criteria” to support

¹² Lovato, C., G. Linn, L.F. Stead, and A. Best, “Impact of Tobacco Advertising and Promotion on Increasing Adolescent Smoking Behaviours,” *The Cochrane Database of Systematic Reviews*, Issue 3 (2003)

¹³ (Lovato 2003, 1)

their findings. Causal criteria include: consistency, strength of association, specificity, temporality, coherence/plausibility and dose/response relationship.¹⁴ However, the longitudinal studies relied upon by the authors are flawed, and therefore application of causal criteria to flawed studies does not support a finding of causality.

While the Cochrane Review relies on nine studies, we focus on two of the studies included, Pierce et. al. (1998) and Biener and Siegal (2000), as these articles themselves claim to provide support for a causal relationship between tobacco company advertising and youth smoking uptake.¹⁵ Further, both of these articles were promoted as the strongest evidentiary support for a causal link between cigarette advertising and youth smoking. Both Pierce (1998) and Biener and Siegal (2000) use measures of “receptivity” to tobacco advertising to predict later smoking behavior. Receptivity measures were based on factors such as owning or using a promotional item, being able to name a cigarette brand, or having a favorite cigarette ad. Baseline smoking behavior and attitudes were elicited with various questions about current and intended smoking behavior.

Pierce et. al. (1998) found that their measure of receptivity to advertising in the baseline survey positively correlated with future progress towards smoking uptake. The participants’ receptivity to tobacco company advertising and promotions was based on the response to questions: “Have you ever bought or received a promotional item?” or “Would you ever use a promotional item?”¹⁶ Those who responded affirmatively to both questions were deemed

¹⁴ Such criteria have been discussed in the public health, and other literature, as necessary to establish causality, and explicitly have been referenced in publications related to smoking, including the 1994 and 2004 Surgeon General Reports, Henneken (1987) and Susser (1991). The Cochrane Review authors cite Susser (1991) for criteria to evaluate causality.

¹⁵ Other Cochrane Review articles include Alexander (1983), Armstrong (1990), Charlton (1989), Diaz (1998), Pucci (1999), Sargent (2000), and While (1996). Pierce (1998) and Biener (2000) were the only studies with follow-up three or more years after baseline.

¹⁶ The data were from a random-digit dial telephone baseline survey of adolescents aged 12 to 17 in

“highly receptive” and had greater progression towards smoking in follow-up.

Biener and Siegal (2000) looked at the relationship between receptivity to tobacco advertising and promotions to becoming an established smoker (smoking more than 100 cigarettes by follow up) and concluded that the “progression to established smoking over a 3-year period was about 3 times higher for those who owned or were willing to use a tobacco promotional item at baseline.”¹⁷ The authors report that “adolescents who were highly receptive to marketing in 1993 were more than twice as likely to become an established smoker by 1997 compared with those who had low receptivity.”

B. The Evidence Does Not Scientifically Support A Causal Link Between Cigarette Advertising And Youth Smoking

Despite the representations, the Cochrane Review studies do not reliably investigate the causal link between cigarette advertising and youth smoking initiation, as these studies are flawed in terms of both the methodology and the data employed. These flaws are fundamental, and therefore these studies potentially lead to very misleading interpretations regarding the relationship between cigarette marketing and youth smoking initiation.¹⁸

There are three central reasons why the studies cited in the Cochrane Review are flawed. The first problem is that these studies do not develop empirical causal models that are theoretically and statistically robust. The estimating equations implemented do not entertain or test against plausible alternative specifications of the data and model. The methods for including

California in 1993 with a follow-up in 1996. The authors also classify participants “susceptibility” to smoking based on responses to questions on anticipated future behavior. Pierce et. al. (1998,512).

¹⁷ The methodology implemented was telephone survey to adolescents between the ages of 12 and 15 in Massachusetts, with a follow-up interview between November 1997 and February 1998. Receptivity was defined as “high” for those participants who owned a cigarette promotional item and could name a cigarette brand ad; “moderate” for those who either owned a promotional item or could name a cigarette brand; and “low” for those who did neither. Biener and Siegal (2000,410).

¹⁸ The uncertainty of the effects of advertising on smoking behavior is explicitly recognized in the economics literature. See Duffy (1996) and Chaloupka and Warner (1999) for further discussion.

(and excluding) explanatory variables are purely ad hoc, as is the choice of the statistical model itself. The researchers present no reliable empirical or theoretical analyses to justify any aspect of model selection, and therefore do not address whether important variables are omitted, whether included variables may be endogenous, or whether underlying assumptions for statistical inferences are consistent with measurable empirical distributions.

The second problem is that these studies ignore well-established principles of statistical analyses of human behavior. Specifically, these studies ignore the consequences of human choice for the validity of their statistical analyses. Their procedures do not capture, and indeed, do not entertain the possibility of deliberate human responses to complex real world stimuli, such as advertising. Thus, the studies ignore commonly-accepted techniques and methods for conducting statistical analyses of human behavior, such as the method of control functions, the method of instrumental variables and other modern methods of statistical inference in causal models.

The explicit disregard for endogenously-determined right-hand-side variables in the empirical implementations is most acute for the employed advertising measures. Specifically, the authors treat ownership of cigarette promotional items by a teenager, such as hats and t-shirts with cigarette brand emblems, as being exogenously determined. For example, both Biener and Siegal (2000) and Pierce et. al. (1998), use ownership of cigarette promotional items as a measure of advertising exposure. Teenagers' recollection of cigarette brands is also employed as an additional measure of teen marketing exposure. The problem is that these proxies for participants' advertising and marketing exposure are plausibly related to already existing unobserved preferences to smoke among the adolescents studied. In other words, participants who have greater preferences for smoking might seek out and obtain more tobacco-related items, even if the items themselves have no independent causal effect on their desire to smoke.

The marketing exposure metrics are then correlated with smoking initiation measures to draw inferences on the causal relationship. However, the simple correlation between the possession of such smoking related items and future smoking behavior does not indicate that the items caused smoking, as this inference does not account for the effects of self-selection. Participants who already are more likely to smoke would be more likely to be classified as having high exposure to cigarette advertising, all else equal. Therefore, observing a correlation between the exposure measures and smoking uptake is not reliable evidence of any causal effect.

A third flaw is that, even if the marketing exposure were exogenously determined, these metrics have not been shown to be related to any actual advertising exposure experienced by the participants of these studies. That is, the advertising exposure variables have not been demonstrated to vary across sample participants according to variations in advertising and non-price marketing campaigns. Therefore, these exposure measures in the public health literature may not provide any information regarding advertising experienced by survey participants.

The strong inferences made from simple observed statistical associations in the public health literature on youth smoking initiation represents a key point of differentiation between carefully applied econometric analyses and other types of statistical inquiries. The acceptance of causal relationships, without estimation of structural specifications based on sound theoretical frameworks, has been recognized as important an limitation in other scientific disciplines as well. For example, Freedman (1999) provides numerous examples of faulty causal inference in statistical studies and discusses the “limits of current statistical techniques for making causal inferences from patterns of association.”¹⁹

¹⁹ See p. 243. On p. 248, Freedman also states that “...many empirical papers published today, even in the leading journals, lack a sharply focused research question; or the study design connects the hypotheses to the data collection only in a very loose way. Investigators often try to use statistical models not only to control for confounding, but also to correct basic deficiencies in the design or the data.” Further,

V. Conclusion

The findings in the public health literature linking tobacco-company (non-price) marketing campaigns emerge from empirical implementations that fall far short of those required to establish well-founded causal relationships. These studies do not accurately model human behavior, as these studies ignore how human choice affects the measurement for both “treatment” and outcome. The biases that emerge from flawed modeling, measurement and interpretation have been fundamental topics in the econometrics field, and this research provides helpful lessons for empirical studies of human behavior in other disciplines as well. Not addressing the potential role individual choices have in shaping the choice of or acceptance of a tobacco item, or other receptivity measures, which are taken to be surrogates for advertising in many public health studies, makes cited results unreliable.

In sum, the establishment of reliable scientific evidence in support of a causal link between cigarette advertising and youth smoking initiation depends both on rich longitudinal data as well as careful empirical applications. These data would need to measure a variety of actual and potential causal factors throughout childhood development and the associated studies would need to explicitly recognize the seriousness of potential biases. Finally, replicability of measured statistical relations would also be an essential part of developing a reliable scientific basis from which to draw conclusions on the effects of tobacco-company advertising.

Freedman (2004, 267) states: “[C]ausal relationships cannot be inferred from a data set by running regressions unless there is substantial prior knowledge about the mechanisms that generated the data.

Bibliography

- Becker, Grossman and Murphy, "An Empirical Analysis of Cigarette Addiction," 84 *Amer. Econ. Rev.* 3 (1994), 396-418
- Biener and Siegel, "Tobacco Marketing and Adolescent Smoking: More Support for a Causal Inference," *American Journal of Public Health*, March 2000, Vol. 90, No.3
- Biglan, Anthony, Patricia A. Brennan, Shannon L. Foster, K. Stenius , and Harold D. Holder, *Helping Adolescents at Risk: Prevention of Multiple Problem Behaviors*, New York: Guilford Press (2004).
- Bowles et al., "The Determinants of Earnings: A Behavioral Approach," *Journal of Economic Literature*, 39(4): 1137-1176, 2001
- Chaloupka, "Rational Addictive Behavior and Cigarette Smoking," 99 *J. Pol. Econ.* 4 (1991), 722-42
- Chaloupka FJ, Pacula RL "An Examination of Gender and Race Differences in Youth Smoking Responsiveness to Price and Tobacco Control Policies," NBER Working Paper 6541, April 1998
- Chaloupka and Warner, "The Economics of Smoking," National Bureau of Economic Research, NBER Working Paper 7047, March 1999
- Cunha, Heckman, et al., "Interpreting the Evidence on Life Cycle Formation," (revised March 28, 2005)
- Davidson, C. and S. Woodbury, "The Displacement Effect of Reemployment Bonus Programs," *Journal of Labor Economics*, 11: 575-605 (1993)
- Douglas, "Duration of the Smoking Habit," 36 *Economic Inquiry* 1 (1998), 49-64
- Martyn Duffy , "Econometric Studies Of Advertising, Advertising Restrictions and Cigarette Demand: A Survey," *International Journal of Advertising*, 1996, 15: 1-23
- Duffy, "An Econometric Study of Advertising and Cigarette Demand in the United Kingdom," *Int'l Journal of Advertising*, Vol. 15, pp. 262-284 (1996). 17
- Freedman, "From Association to Causation: Some Remarks on the History of Statistics," *Statistical Science*, Vol. 14, No. 3, p. 243-258, 1999.)
- Freedman, "Graphical Models For Causation, And The Identification Problem," *Evaluation Review* 28: 267-293, 2004.
- Heckman, James J., "The Scientific Model of Causality," revised, April 28, 2005.

Heckman, James J., L. Lochner, and C. Taber, "General Equilibrium Treatment Effects: A study of Tuition Policy," *American Economic Review*, 88: 381-386 (1998).

James J. Heckman and Yona Rubinstein, "The Importance of Noncognitive Skills: Lessons from the GED Testing Program," *American Economic Review*, 91(2):145:149, 2001

Heckman, James J. , Stixrud, J. and Sergio Urzua, 2006. "The Effects of Cognitive and Noncognitive Abilities on Labor Market Outcomes and Social Behavior," NBER Working Papers 12006, National Bureau of Economic Research, Inc.

Knudsen, "Sensitive Periods in the Development of the Brain and Behavior," *Journal of Cognitive Neuroscience*, 16(8): 1412-1425, 2004

Lewit and Coate, "The Potential for Using Excise Taxes to Reduce Smoking," 1 *J. Health Econ.* 2 (1982), 121-145.

Lewit, Coate & Grossman, "The Effects of Government Regulation on Teenage Smoking, 24 *J. Law & Econ.*, (1981)

Lovato, C., G. Linn, L.F. Stead, and A. Best, "Impact of Tobacco Advertising and Promotion on Increasing Adolescent Smoking Behaviours," *The Cochrane Database of Systematic Reviews*, Issue 3 (2003)

Martin, John P. and David Grubb, "What Works and for Whom: A review of OECD Countries' Experiences with Active Labor Market Policies," *Swedish Economic Policy Review*, 8:9-56 (2001)

Morrall, McCaffrey, & Paddock, "Reassessing the Marijuana Gateway Effect" Drug Policy Research Center: October 2001.

Pacula, "Adolescent Alcohol and Marijuana Consumption: Is There Really a Gateway Effect?" National Bureau of Economic Research: January 1998.

Pierce, Choi, Gilpin, Farkas and Berry, "Tobacco Industry Promotion of Cigarettes and Adolescent Smoking," *The Journal of the American Medical Association*: 1998.

Saffer and Chaloupka, "The Effect of Tobacco Advertising Bans on Tobacco Consumption", *Journal of Health Economics*, 2000(19):1117-1137

Saffer and Chaloupka, "Tobacco Advertising: Economic Theory and International Evidence," National Bureau of Economic Research, working paper 6958, February 1999.

Turkheimer et al., "Socioeconomic Status Modifies Heritability of IQ In Young Children," *Psychological Science*, 14(6): 623-628, 2003.