10
Treatment effects: *IV*

In this chapter we continue the discussion of treatment effects but replace ignorable treatment strategies in favor of instrumental variables and exclusion restrictions. Intuitively, instrumental variables are a standard econometric response to omitted, correlated variables so why not employ them to identify and estimate treatment effects. That is, we look for instruments that are highly related to the selection or treatment choice but unrelated to outcome. This is a bit more subtle than standard linear *IV* because of the counterfactual issue. The key is that exclusion restrictions allow identification of the counterfactuals as an individual's probability of receiving treatment can be manipulated without affecting potential outcomes.

We emphasize we're looking for good instruments. Recall that dropping variables from the outcome equations that should properly be included creates an omitted, correlated variable problem. There doesn't seem much advantage of swapping one malignant inference problem for another — the selection problem can also be thought of as an omitted, correlated variable problem.

10.1 Setup

The setup is the same as the previous chapter. We repeat it for convenience then relate it to common average treatment effects and the Roy model to facilitate interpretation. Suppose the *DGP* is

outcomes:[1]

$$Y_j = \mu_j(X) + V_j, j = 0, 1$$

selection mechanism:[2]

$$D^* = \mu_D(Z) - V_D$$

and observable response:

$$\begin{aligned} Y &= DY_1 + (1-D)Y_0 \\ &= \mu_0(X) + (\mu_1(X) - \mu_0(X))D + V_0 + (V_1 - V_0)D \end{aligned}$$

where

$$D = \begin{array}{ll} 1 & D^* > 0 \\ 0 & otherwise \end{array}$$

and Y_1 is (potential) outcome with treatment and Y_0 is (potential) outcome without treatment. The outcomes model is the Neyman-Fisher-Cox-Rubin model of potential outcomes (Neyman [1923], Fisher [1966], Cox]1958], and Rubin [1974]). It is also Quandt's [1972] switching regression model or Roy's income distribution model (Roy [1951] or Heckman and Honore [1990]).

10.2 Treatment effects

We address the same treatment effects but add a couple of additional effects to highlight issues related to unobservable heterogeneity. Heckman and Vytlacil [2005] describe the recent focus of the treatment effect literature as the heterogeneous response to treatment amongst otherwise observationally equivalent individuals. Unobservable heterogeneity is a serious concern whose analysis is challenging if not down right elusive.

In the binary case, the treatment effect is the effect on outcome of treatment compared with no treatment, $\Delta = Y_1 - Y_0$. Some typical treatment effects include: *ATE*, *ATT*, *ATUT, LATE*, and *MTE*. *ATE* refers to the average treatment effect, by iterated expectations, we can recover the unconditional average treatment effect from the conditional average treatment effect

$$\begin{aligned} ATE &= E_X[ATE(X)] \\ &= E_X[E[\Delta \mid X = x]] = E[Y_1 - Y_0] \end{aligned}$$

---

[1] Separating outcome into a constant and stochastic parts, yields

$$Y_j = \mu_j + U_j$$

Sometimes it will be instructive to write the stochastic part as a linear function of X

$$U_j = X\beta_j + V_j$$

[2] To facilitate discussion, we stick with binary choice for most of the discussion. We extend the discussion to multilevel discrete and continuous treatment later in chapter 11.

In other words, the average effect of treatment on outcome compared with no treatment for a random draw from the population.

ATT refers to the average treatment effect on the treated,

$$\begin{aligned} ATT &= E_X\left[ATT\left(X\right)\right] \\ &= E_X\left[E\left[\Delta \mid X=x, D=1\right]\right] = E\left[Y_1 - Y_0 \mid D=1\right] \end{aligned}$$

In other words, the average effect of treatment on outcome compared with no treatment for a random draw from the subpopulation selecting (or assigned) treatment.

ATUT refers to the average treatment effect on the untreated,

$$\begin{aligned} ATUT &= E_X\left[ATUT\left(X\right)\right] \\ &= E_X\left[E\left[\Delta \mid X=x, D=0\right]\right] = E\left[Y_1 - Y_0 \mid D=0\right] \end{aligned}$$

In other words, the average effect of treatment on outcome compared with no treatment for a random draw from the subpopulation selecting (or assigned) no treatment.

For a binary instrument (to keep things simple), the local average treatment effect or *LATE* is

$$\begin{aligned} LATE &= E_X\left[LATE\left(X\right)\right] \\ &= E_X\left[E\left[\Delta \mid X=x, D_1 - D_0 = 1\right]\right] = E\left[Y_1 - Y_0 \mid D_1 - D_0 = 1\right] \end{aligned}$$

where D_j refers to the observed treatment conditional on the value j of the binary instrument. *LATE* refers to the local average or marginal effect of treatment on outcome compared with no treatment for a random draw from the subpopulation of "compliers" (Imbens and Angrist [1994]). That is, *LATE* is the (discrete) marginal effect on outcome for those individuals who would not choose treatment if the instrument takes a value of zero but would choose treatment if the instrument takes a value of one.

MTE (the marginal treatment effect) is a generalization of *LATE* as it represents the treatment effect for those individuals who are indifferent between treatment and no treatment.

$$MTE = E\left[Y_1 - Y_0 \mid X=x, V_D = v_D\right]$$

or following transformation $U_D = F_{V|X}\left(V\right)$, where $F_{V|X}\left(V\right)$ is the (cumulative) distribution function, we can work with $U_D \sim Uniform\left[0,1\right]$

$$MTE = E\left[Y_1 - Y_0 \mid X=x, U_D = u_D\right]$$

Treatment effect implications can be illustrated in terms of the generalized Roy model. The Roy model interpretation is discussed next.

10.3 Generalized Roy model

Roy [1951] introduced an equilibrium labor model where workers select between hunting and fishing. An individual's selection into hunting or fishing depends on his aptitude as well as supply of and demand for labor.[3] A modest generalization of the Roy model is a common framing of selection that frequently forms the basis for assessing treatment effects (Heckman and Robb [1986]).

Based on the *DGP* above, we identify the constituent pieces of the selection model.

Net benefit (or utility) from treatment is

$$\begin{aligned} D^* &= \mu_D(Z) - V_D \\ &= Y_1 - Y_0 - c(W) - V_c \\ &= \mu_1(X) - \mu_0(X) - c(W) + V_1 - V_0 - V_c \end{aligned}$$

Gross benefit of treatment is[4]

$$\mu_1(X) - \mu_0(X)$$

Cost associated with treatment is

$$c(W) + V_c$$

Observable cost associated with treatment is

$$c(W)$$

Observable net benefit of treatment is

$$\mu_1(X) - \mu_0(X) - c(W)$$

Unobservable net benefit of treatment is

$$-V_D = V_1 - V_0 - V_c$$

where the observables are $\begin{bmatrix} X & Z & W \end{bmatrix}$, typically Z contains variables not in X or W, and W is the subset of observables that speaks to cost of treatment.

Given a rich data generating process like above, the challenge is to develop identification strategies for the treatment effects of interest. The simplest *IV* approaches follow from the strongest conditions for the data and typically imply homogeneous response. Accommodating heterogeneous response holds economic appeal but also constitutes a considerable hurdle.

---

[3] Roy argues that self-selection leads to lesser earnings inequality than does random assignment. See Heckman and Honore [1990] for an extended discussion of the original Roy model including identification under various probability distribution assignments on worker skill (log skill).

[4] For linear outcomes, we have $\mu_1(X) - \mu_0(X) = (\mu_1 + X\beta_1) - (\mu_0 + X\beta_0)$.

10.4 Homogeneous response

Homogeneous response is attractive when pooling restrictions across individuals (or firms) are plausible. Homogeneous response implies the stochastic portion, U_j, is the same for individuals receiving treatment and not receiving treatment, $U_1 = U_0$. This negates the interaction term, $(U_1 - U_0) D$, in observed outcome and consequently rules out individual-specific gains. Accordingly, $ATE = ATT = ATUT = MTE$. Next, we review treatment effect identification conditions for a variety of homogeneous response models with endogenous treatment.

10.4.1 Endogenous dummy variable IV model

Endogenous dummy variable *IV* regression is a standard approach but not as robust in the treatment effect setting as we're accustomed in other settings. Let L be a linear projection of the leading argument into the column space of the conditioning variables where X includes the unity vector ι, that is,

$$\begin{aligned} L(Y \mid X) &= X\left(X^T X\right)^{-1} X^T Y \\ &= P_X Y \end{aligned}$$

and Z_i be a vector of instruments. Identification conditions are

Condition 10.1 $U_1 = U_0$ *where* $U_j = X\beta_j + V_j$, $j = 0, 1$,

Condition 10.2 $L(U_0 \mid X, Z) = L(U_0 \mid X)$, *and*

Condition 10.3 $L(D \mid X, Z) \neq L(D \mid X)$.

Condition 10.1 is homogeneous response while conditions 10.2 and 10.3 are exclusion restrictions. Conditions 10.1 and 10.2 imply observed outcome is

$$Y = \mu_0 + (\mu_1 - \mu_0) D + X\beta_0 + V_0$$

which can be written

$$Y = \delta + \alpha D + X\beta_0 + V_0$$

where $\alpha = ATE$ and $V_0 = U_0 - L(U_0 \mid X, Z)$. As D and V_0 are typically correlated (think of the Roy model interpretation), we effectively have an omitted, correlated variable problem and *OLS* is inconsistent.

However, condition 10.2 means that Z is properly excluded from the outcome equation. Unfortunately, this cannot be directly tested.[5] Under the above conditions, standard two stage least squares instrumental variable (*2SLS-IV*) estimation (see chapter 3) with $\{\iota, X, Z\}$ as instruments provides a consistent and asymptotically normal estimate for *ATE*. That is, the first stage discrete choice (say, logit

[5]Though we might be able to employ over-identifying tests of restrictions if we have multiple instruments. Of course, these tests assume that at least one is a legitimate instrument.

or probit) regression is

$$D = \gamma_0 + X\gamma_1 + Z\gamma_2 - V_D$$

and the second stage regression is

$$Y = \delta + \alpha\widehat{D} + X\beta_0 + V_0$$

where $\widehat{D} = \hat{\gamma}_0 + X\hat{\gamma}_1 + Z\hat{\gamma}_2$, predicted values from the first stage discrete choice regression.

10.4.2 Propensity score IV

Stronger conditions allow for a more efficient *IV* estimator. For instance, suppose the data satisfies the following conditions.

Condition 10.4 $U_1 = U_0$,

Condition 10.5 $E[U_0 \mid X, Z] = E(U_0 \mid X)$,

Condition 10.6 $\Pr(D = 1 \mid X, Z) \neq \Pr(D = 1 \mid X)$ *plus*
$\Pr(D = 1 \mid X, Z) = G(X, Z, \gamma)$ *is a known parametric form (usually probit or logit), and*

Condition 10.7 $Var[U_0 \mid X, Z] = \sigma_0^2$.

The outcome equation is

$$Y = \delta + \alpha D + X\beta_0 + V_0$$

If we utilize $\{\iota, G(X, Z, \gamma), X\}$ as instruments, *2SLS-IV* is consistent asymptotically normal (*CAN*). Not only is this propensity score approach more efficient given the assumptions, but it is also more robust. Specifically, the link function doesn't have to be equal to G for *2SLS-IV* consistency but it does for *OLS* (see Wooldridge [2002], ch. 18).

10.5 Heterogeneous response and treatment effects

Frequently, homogeneity is implausible, $U_1 \neq U_0$. Idiosyncrasies emerge in both what is observed, say $X\beta_0 \neq X\beta_1$, (relatively straightforward to address) and what the analyst cannot observe, $V_0 \neq V_1$, (more challenging to address). Then observed outcome contains an individual-specific gain $(U_1 - U_0) D$ and, usually, $ATE \neq ATT \neq ATUT \neq MTE$. In general, the linear *IV* estimator (using Z or G as instruments) does not consistently estimate *ATE* (or *ATT*) when response is heterogeneous, $U_1 \neq U_0$. Next, we explore some *IV* estimators which may consistently estimate *ATE* even though response is heterogeneous.

10.5.1 Propensity score IV and heterogeneous response

First, we return to the propensity score and relax the conditions to accommodate heterogeneity. Let $U_j = X\beta_j + V_j$ where $E[V_j \mid X, Z] = 0$. Identification conditions are

Condition 10.8 *conditional mean redundancy,* $E[U_0 \mid X, Z] = E[U_0 \mid X]$ *and* $E[U_1 \mid X, Z] = E[U_1 \mid X]$,

Condition 10.9 $X\beta_1 - X\beta_0 = (X - E[X])\gamma$,

Condition 10.10 $V_1 = V_0$, *and*

Condition 10.11 $\Pr(D = 1 \mid X, Z) \neq \Pr(D = 1 \mid X)$ *and* $\Pr(D = 1 \mid X, Z) = G(X, Z, \gamma)$ *where again* G *is a known parametric form (usually probit or logit).*

If we utilize $\left\{\iota, G(X, Z, \gamma), X - \overline{X}\right\}$ as instruments in the regression

$$Y = \mu_0 + X\beta_0 + \alpha D + \left(X - \overline{X}\right) D\gamma + V_0$$

2SLS-IV is consistent asymptotically normal (*CAN*).

We can relax the above a bit if we replace condition 10.10, $V_1 = V_0$, by conditional mean independence

$$E[D(V_1 - V_0) \mid X, Z] = E[D(V_1 - V_0)]$$

While probably not efficient, α consistently identifies *ATE* for this two-stage propensity score *IV* strategy utilizing $\{\iota, G, X, G(X - E[X])\}$ as instruments.

10.5.2 Ordinate control function IV and heterogeneous response

Employing control functions to address the omitted, correlated variable problem created by endogenous selection is popular. We'll review two identification strategies: ordinate and inverse Mills *IV* control functions. The second one pioneered by Heckman [1979] is much more frequently employed. Although the first approach may be more robust.

Identification conditions are

Condition 10.12 *conditional mean redundancy,* $E[U_0 \mid X, Z] = E[U_0 \mid X]$ *and* $E[U_1 \mid X, Z] = E[U_1 \mid X]$,

Condition 10.13 $g_1(X) - g_0(X) = X\beta_1 - X\beta_0 = (X - E[X])\gamma$,

Condition 10.14 $V_1 - V_0$ *is independent of* $\{X, Z\}$ *and* $E[D \mid X, Z, V_1 - V_0] = h(X, Z) + k(V_1 - V_0)$ *for some functions* h *and* k,

Condition 10.15 $\Pr(D = 1 \mid X, Z, V_1 - V_0) = \Phi(\theta_0 + X\theta_1 + Z\theta_2 + \varrho(V_1 - V_0))$, $\theta_2 \neq 0$, *and*

Condition 10.16 $V_1 - V_0 \sim N\left(0, \tau^2\right)$.

The model of observed outcome

$$Y = \mu_0 + \alpha D + X\beta_0 + D\left(X - E\left[X\right]\right)\gamma + \xi\phi + error$$

can be estimated by two-stage *IV* using instruments

$$\left\{\iota, \Phi, X, \Phi\left(X - E\left[X\right]\right), \phi\right\}$$

where Φ is the cumulative standard normal distribution function and ϕ is the ordinate from a standard normal each evaluated at $[X_i, Z_i]\,\widehat{\theta}$ from probit. With full common X support, *ATE* is consistently estimated by α since ϕ is a control function obtained via *IV* assumptions (hence the label ordinate control function).

10.5.3 Inverse Mills control function IV and heterogeneous response

Heckman's inverse Mills control function is closely related to the ordinate control function. Identification conditions are

Condition 10.17 *conditional mean redundancy,* $E\left[U_0 \mid X, Z\right] = E\left[U_0 \mid X\right]$ *and* $E\left[U_1 \mid X, Z\right] = E\left[U_1 \mid X\right]$,

Condition 10.18 $g_1\left(X\right) - g_0\left(X\right) = \left(X - E\left[X\right]\right)\delta$,

Condition 10.19 $\left(V_D, V_1, V_0\right)$ *is independent of* $\left\{X, Z\right\}$ *with joint normal distribution, especially* $V \sim N\left(0, 1\right)$, *and*

Condition 10.20 $D = I\left[\theta_0 + X\theta_1 + Z\theta_2 - V_D > 0\right]$ *where* I *is an indicator function equal to one when true and zero otherwise*.

While this can be estimated via *MLE*, Heckman's two-stage procedure is more common. First, estimate θ via a probit regression of D on $W = \left\{\iota, X, Z\right\}$ and identify observations with common support (that is, observations for which the regressors, X, for the treated overlap with regressors for the untreated). Second, regress Y onto

$$\left\{\iota, D, X, D\left(X - E\left[X\right]\right), D\left(\frac{\phi}{\Phi}\right), \left(1 - D\right)\frac{-\phi}{1 - \Phi}\right\}$$

for the overlapping subsample. With full support, the coefficient on D is a consistent estimator of *ATE*; with less than full common support, we have a local average treatment effect.[6]

[6]We should point out here that this second stage *OLS* does not provide valid estimates of standard errors. As Heckman [1979] points out there are two additional concerns: the errors are heteroskedastic (so an adjustment such as White suggested is needed) and θ has to be estimated (so we must account for this added variation). Heckman [1979] identifies a valid variance estimator for this two-stage procedure.

The key ideas behind treatment effect identification via control functions can be illustrated by reference to this case.

$$E\left[Y_j \mid X, D=j\right] = \mu_j + X\beta_j + E\left[V_j \mid D=j\right]$$

Given the conditions, $E\left[V_j \mid D=j\right] \neq 0$ unless $Corr\left(V_j, V_D\right) = \rho_{jV_D} = 0$. For $\rho_{jV_D} \neq 0$,

$$E\left[V_1 \mid D=1\right] = \rho_{1V_D}\sigma_1 E\left[V_D \mid V_D > -W\theta\right]$$
$$E\left[V_0 \mid D=1\right] = \rho_{0V_D}\sigma_0 E\left[V_D \mid V_D > -W\theta\right]$$
$$E\left[V_1 \mid D=0\right] = \rho_{1V_D}\sigma_1 E\left[V_D \mid V_D \leq -W\theta\right]$$

and

$$E\left[V_0 \mid D=0\right] = \rho_{0V_D}\sigma_0 E\left[V_D \mid V_D \leq -W\theta\right]$$

The final term in each expression is the expected value of a truncated standard normal random variate where

$$E\left[V_D \mid V_D > -W\theta\right] = \frac{\phi\left(-W\theta\right)}{1-\Phi\left(-W\theta\right)} = \frac{\phi\left(W\theta\right)}{\Phi\left(W\theta\right)}$$

and

$$E\left[V_D \mid V_D \leq -W\theta\right] = -\frac{\phi\left(-W\theta\right)}{\Phi\left(-W\theta\right)} = -\frac{\phi\left(W\theta\right)}{1-\Phi\left(W\theta\right)}$$

Putting this together, we have

$$E\left[Y_1 \mid X, D=1\right] = \mu_1 + X\beta_1 + \rho_{1V_D}\sigma_1\frac{\phi\left(W\theta\right)}{\Phi\left(W\theta\right)}$$

$$E\left[Y_0 \mid X, D=0\right] = \mu_0 + X\beta_0 - \rho_{0V_D}\sigma_0\frac{\phi\left(W\theta\right)}{1-\Phi\left(W\theta\right)}$$

and counterfactuals

$$E\left[Y_0 \mid X, D=1\right] = \mu_0 + X\beta_0 + \rho_{0V_D}\sigma_0\frac{\phi\left(W\theta\right)}{\Phi\left(W\theta\right)}$$

and

$$E\left[Y_1 \mid X, D=0\right] = \mu_1 + X\beta_1 - \rho_{1V_D}\sigma_1\frac{\phi\left(W\theta\right)}{1-\Phi\left(W\theta\right)}$$

The affinity for Heckman's inverse Mills ratio approach can be seen in its estimation simplicity and the ease with which treatment effects are then identified. Of course, this doesn't justify the identification conditions — only our understanding of the data can do that.

$$ATT\left(X, Z\right) = \mu_1 - \mu_0 + X\left(\beta_1 - \beta_0\right) + \left(\rho_{1V_D}\sigma_1 - \rho_{0V_D}\sigma_0\right)\frac{\phi\left(W\theta\right)}{\Phi\left(W\theta\right)}$$

by iterated expectations (with full support), we have

$$ATT = \mu_1 - \mu_0 + E[X](\beta_1 - \beta_0) + \left(\rho_{1V_D}\sigma_1 - \rho_{0V_D}\sigma_0\right) E\left[\frac{\phi(W\theta)}{\Phi(W\theta)}\right]$$

Also,

$$ATUT(X,Z) = \mu_1 - \mu_0 + X(\beta_1 - \beta_0) - \left(\rho_{1V_D}\sigma_1 - \rho_{0V_D}\sigma_0\right)\frac{\phi(W\theta)}{1-\Phi(W\theta)}$$

by iterated expectations, we have

$$ATUT = \mu_1 - \mu_0 + E[X](\beta_1 - \beta_0) - \left(\rho_{1V_D}\sigma_1 - \rho_{0V_D}\sigma_0\right) E\left[\frac{\phi(W\theta)}{1-\Phi(W\theta)}\right]$$

Since

$$\begin{aligned} ATE(X,Z) &= \Pr(D=1 \mid X,Z)\, ATT(X,Z) \\ &\quad + \Pr(D=0 \mid X,Z)\, ATUT(X,Z) \\ &= \Phi(W\theta)\, ATT(X,Z) + (1-\Phi(W\theta))\, ATUT(X,Z) \end{aligned}$$

we have

$$\begin{aligned} ATE(X,Z) &= \mu_1 - \mu_0 + X(\beta_1 - \beta_0) \\ &\quad + \left(\rho_{1V}\sigma_1 - \rho_{0V_D}\sigma_0\right)\phi(W\theta) - \left(\rho_{1V}\sigma_1 - \rho_{0V_D}\sigma_0\right)\phi(W\theta) \\ &= \mu_1 - \mu_0 + X(\beta_1 - \beta_0) \end{aligned}$$

by iterated expectations (with full common support), we have

$$ATE = \mu_1 - \mu_0 + E[X](\beta_1 - \beta_0)$$

Wooldridge [2002, p. 631] suggests identification of

$$ATE = \mu_1 - \mu_0 + E[X](\beta_1 - \beta_0)$$

via α in the following regression

$$\begin{aligned} E[Y \mid X,Z] &= \mu_0 + \alpha D + X\beta_0 + D(X - E[X])(\beta_1 - \beta_0) \\ &\quad + D\rho_{1V_D}\sigma_1\frac{\phi(W\theta)}{\Phi(W\theta)} - (1-D)\rho_{0V_D}\sigma_0\frac{\phi(W\theta)}{1-\Phi(W\theta)} \end{aligned}$$

This follows from the observable response

$$\begin{aligned} Y &= D(Y_1 \mid D=1) + (1-D)(Y_0 \mid D=0) \\ &= (Y_0 \mid D=0) + D[(Y_1 \mid D=1) - (Y_0 \mid D=0)] \end{aligned}$$

and applying conditional expectations

$$\begin{aligned} E[Y_1 \mid X, D=1] &= \mu_1 + X\beta_1 + \rho_{1V_D}\sigma_1\frac{\phi(W\theta)}{\Phi(W\theta)} \\ E[Y_0 \mid X, D=0] &= \mu_0 + X\beta_0 - \rho_{0V_D}\sigma_0\frac{\phi(W\theta)}{1-\Phi(W\theta)} \end{aligned}$$

Simplification produces Wooldridge's result.

10.5.4 Heterogeneity and estimating ATT by IV

Now we discuss a general approach for estimating *ATT* by *IV* in the face of unobservable heterogeneity.

$$\begin{aligned} ATT(X) &= E[Y_1 - Y_0 \mid X, D = 1] \\ &= \mu_1 - \mu_0 + E[U_1 - U_0 \mid X, D = 1] \end{aligned}$$

Identification (data) conditions are

Condition 10.21 $E[U_0 \mid X, Z] = E[U_0 \mid X]$,

Condition 10.22 $E[U_1 - U_0 \mid X, Z, D = 1] = E[U_1 - U_0 \mid X, D = 1]$, *and*

Condition 10.23 $\Pr(D = 1 \mid X, Z) \neq \Pr(D = 1 \mid X)$ *and* $\Pr(D = 1 \mid X, Z) = G(X, Z; \gamma)$ *is a known parametric form (usually probit or logit).*

Let

$$\begin{aligned} Y_j &= \mu_j + U_j \\ &= \mu_j + g_j(X) + V_j \end{aligned}$$

and write

$$\begin{aligned} Y &= \mu_0 + g_0(X) + D\{(\mu_1 - \mu_0) + E[U_1 - U_0 \mid X, D = 1]\} \\ &\quad + D\{(U_1 - U_0) - E[U_1 - U_0 \mid X, D = 1]\} + V_0 \\ &= \mu_0 + g_0(X) + ATT(X) D + a + V_0 \end{aligned}$$

where $a = D\{(U_1 - U_0) - E[U_1 - U_0 \mid X, D = 1]\}$. Let $r = a + V_0$, the data conditions imply $E[r \mid X, Z] = 0$. Now, suppose $\mu_0(X) = \eta_0 + h(X)\beta_0$ and $ATT(X) = \tau + f(X)\delta$ for some functions $h(X)$ and $f(X)$. Then, we can write

$$Y = \gamma_0 + h(X)\beta_0 + \tau D + Df(X)\delta + r$$

where $\gamma_0 = \mu_0 + \eta_0$. The above equation can be estimated by *IV* using any functions of $\{X, Z\}$ as instruments. Averaging $\tau + f(X)\delta$ over observations with $D = 1$ yields a consistent estimate for ATT, $\frac{\sum D_i(\tau_i + f(X_i)\delta)}{\sum D_i}$. By similar reasoning, $ATUT$ can be estimated by averaging over the $D = 0$ observations, $-\frac{\sum D_i(\tau_i + f(X_i)\delta)}{\sum(1 - D_i)}$.

10.5.5 LATE and linear IV

Concerns regarding lack of robustness (logical inconsistency) of ignorable treatment, or, for instance, the sometimes logical inconsistency of normal probability assignment to unobservable expected utility (say, with Heckman's inverse Mills *IV* control function strategy) have generated interest in alternative *IV* approaches.

One that has received considerable attention is linear *IV* estimation of local average treatment effects (*LATE*; Imbens and Angrist [1994]). We will focus on the binary instrument case to highlight identification issues and aid intuition. First, we provide a brief description then follow with a more extensive treatment. As this is a discrete version of the marginal treatment effect, it helps provide intuition for how instruments, more generally, can help identify treatment effects.

For binary instrument Z,

$$LATE = E\left[Y_1 - Y_0 \mid D_1 - D_0 = 1\right]$$

where $D_1 = (D \mid Z = 1)$ and $D_0 = (D \mid Z = 0)$. That is, *LATE* is the expected gain from treatment of those individuals who switch from no treatment to treatment when the instrument Z changes from 0 to 1. Angrist, Imbens, and Rubin [1996] refer to this subpopulation as the "compliers". This treatment effect is only identified for this subpopulation and because it involves counterfactuals the subpopulation cannot be identified from the data. Nonetheless, the approach has considerable appeal as it is reasonably robust even in the face of unobservable heterogeneity.

Setup

The usual exclusion restriction (existence of instrument) applies. Identification conditions are

Condition 10.24 $\{Y_1, Y_0\}$ *independent of* Z*,*

Condition 10.25 $D_1 \geq D_0$ *for each individual, and*

Condition 10.26 $\Pr(D = 1 \mid Z = 1) \neq \Pr(D = 1 \mid Z = 0)$.

Conditions 10.24 and 10.26 are usual instrumental variables conditions. Conditional 10.25 is a uniformity condition. For the subpopulation of "compliers" the instrument induces a change to treatment when Z takes a value of 1 but not when $Z = 0$.

Identification

LATE provides a straightforward opportunity to explore *IV* identification of treatment effects. Identification is a thought experiment regarding whether an estimand, the population parameter associated with an estimator, can be uniquely identified from the data. *IV* approaches rely on exclusion restrictions to identify population characteristics of counterfactuals. Because of the counterfactual problem, it is crucial to our *IV* identification thought experiment that we be able to manipulate treatment choice without impacting outcomes. Hence, the exclusion restriction or existence of an instrument (or instruments) is fundamental. Once identification is secured we can focus on matters of estimation (such as consistency and efficiency). Next, we discuss *IV* identification of *LATE*. This is followed

by discussion of the implication of exclusion restriction failure for treatment effect identification.

For simplicity there are no covariates and two points of support $Z_i = 1$ and $Z_i = 0$ where

$$\Pr(D_i = 1 \mid Z_i = 1) > \Pr(D_i = 1 \mid Z_i = 0)$$

Compare the outcome expectations

$$\begin{aligned} & E[Y_i \mid Z_i = 1] - E[Y_i \mid Z_i = 0] \\ = \; & E[D_i Y_{1i} + (1 - D_i) Y_{0i} \mid Z_i = 1] \\ & - E[D_i Y_{1i} + (1 - D_i) Y_{0i} \mid Z_i = 0] \end{aligned}$$

$\{Y_1, Y_0\}$ independent of Z implies

$$\begin{aligned} & E[Y_i \mid Z_i = 1] - E[Y_i \mid Z_i = 0] \\ = \; & E[D_{1i} Y_{1i} + (1 - D_{1i}) Y_{0i}] - E[D_{0i} Y_{1i} + (1 - D_{0i}) Y_{0i}] \end{aligned}$$

rearranging yields

$$E[(D_{1i} - D_{0i}) Y_{1i} - (D_{1i} - D_{0i}) Y_{0i}]$$

combining terms produces

$$E[(D_{1i} - D_{0i})(Y_{1i} - Y_{0i})]$$

utilizing the sum and product rules of Bayes' theorem gives

$$\begin{aligned} & \Pr(D_{1i} - D_{0i} = 1) E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = 1] \\ & - \Pr(D_{1i} - D_{0i} = -1) E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = -1] \end{aligned}$$

How do we interpret this last expression? Even for a strictly positive causal effect of D on Y for all individuals, the average treatment effect is ambiguous as it can be positive, zero, or negative. That is, the treatment effect of those who switch from nonparticipation to participation when Z changes from 0 to 1 can be offset by those who switch from participation to nonparticipation. Therefore, identification of average treatment effects requires additional data conditions. *LATE* invokes uniformity in response to the instrument for all individuals. Uniformity eliminates the second term above as $\Pr(D_{1i} - D_{0i} = -1) = 0$. Then, we can replace $\Pr(D_{1i} - D_{0i} = 1)$ with $E[D_i \mid Z_i = 1] - E[D_i \mid Z_i = 0]$ and

$$\begin{aligned} & (E[Y_i \mid Z_i = 1] - E[Y_i \mid Z_i = 0]) \\ = \; & \Pr(D_{1i} - D_{0i} = 1) E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = 1] \\ = \; & (E[D_i \mid Z_i = 1] - E[D_i \mid Z_i = 0]) E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = 1] \end{aligned}$$

From the above we can write

$$\begin{aligned}
& \frac{E[Y_i \mid Z_i = 1] - E[Y_i \mid Z_i = 0]}{E[D_i \mid Z_i = 1] - E[D_i \mid Z_i = 0]} \\
= & \frac{\Pr(D_{1i} - D_{0i} = 1) E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = 1]}{E[D_i \mid Z_i = 1] - E[D_i \mid Z_i = 0]} \\
= & \frac{(E[D_i \mid Z_i = 1] - E[D_i \mid Z_i = 0]) E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = 1]}{E[D_i \mid Z_i = 1] - E[D_i \mid Z_i = 0]} \\
= & E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = 1]
\end{aligned}$$

and since

$$LATE = E[Y_{1i} - Y_{0i} \mid D_{1i} - D_{0i} = 1]$$

we can identify *LATE* by extracting

$$\frac{E[Y_i \mid Z_i = 1] - E[Y_i \mid Z_i = 0]}{E[D_i \mid Z_i = 1] - E[D_i \mid Z_i = 0]}$$

from observables. This is precisely what standard *2SLS-IV* estimates with a binary instrument (developed more fully below).

As *IV* identification of treatment effects differs from standard applications of linear *IV*,[7] this seems an appropriate juncture to explore *IV* identification. The foregoing discussion of *LATE* identification provides an attractive vehicle to illustrate the nuance of identification with an exclusion restriction. Return to the above approach, now suppose condition 10.24 fails, $\{Y_1, Y_0\}$ not independent of Z. Then,

$$\begin{aligned}
& E[Y_i \mid Z_i = 1] - E[Y_i \mid Z_i = 0] \\
= & E[D_{1i}Y_{1i} + (1 - D_{1i}) Y_{0i} \mid Z_i = 1] \\
& - E[D_{0i}Y_{1i} + (1 - D_{0i}) Y_{0i} \mid Z_i = 0]
\end{aligned}$$

but $\{Y_1, Y_0\}$ not independent of Z implies

$$\begin{aligned}
& E[Y_i \mid Z_i = 1] - E[Y_i \mid Z_i = 0] \\
= & E[D_{1i}Y_{1i} + (1 - D_{1i}) Y_{0i} \mid Z_i = 1] \\
& - E[D_{0i}Y_{1i} + (1 - D_{0i}) Y_{0i} \mid Z_i = 0] \\
= & \{E[D_{1i}Y_{1i} \mid Z_i = 1] - E[D_{0i}Y_{1i} \mid Z_i = 0]\} \\
& - \{E[D_{1i}Y_{0i} \mid Z_i = 1] - E[D_{0i}Y_{0i} \mid Z_i = 0]\} \\
& + \{E[Y_{0i} \mid Z_i = 1] - E[Y_{0i} \mid Z_i = 0]\}
\end{aligned}$$

Apparently, the first two terms cannot be rearranged and simplified to identify any treatment effect and the last term does not vanish (recall from above when $\{Y_1, Y_0\}$ independent of Z, this term equals zero). Hence, when the exclusion

[7] Heckman and Vytlacil [2005, 2007a, 2007b] emphasize this point.

restriction fails we apparently cannot identify any treatment effects without appealing to other strong conditions.

Sometimes *LATE* can be directly connected to other treatment effects. For example, if $\Pr(D_0 = 1) = 0$, then $LATE = ATT$. Intuitively, the only variation in participation and therefore the only source of overlaps from which to extrapolate from factuals to counterfactuals occurs when $Z_i = 1$. When treatment is accepted, we're dealing with compliers and the group of compliers participate when $Z_i = 1$. Hence, $LATE = ATT$.

Also, if $\Pr(D_1 = 1) = 1$, then $LATE = ATUT$. Similarly, the only variation in participation and therefore the only source of overlaps from which to extrapolate from factuals to counterfactuals occurs when $Z_i = 0$. When treatment is declined, we're dealing with compliers and the group of compliers don't participate when $Z_i = 0$. Hence, $LATE = ATUT$.

Linear *IV* estimation

As indicated above, *LATE* can be estimated via standard *2SLS-IV*. Here, we develop the idea more completely. For Z binary, the estimand for the regression of Y on Z is

$$\frac{E[Y \mid Z = 1] - E[Y \mid Z = 0]}{1 - 0} = E[Y \mid Z = 1] - E[Y \mid Z = 0]$$

and the estimand for the regression of D on Z is

$$\frac{E[D \mid Z = 1] - E[D \mid Z = 0]}{1 - 0} = E[D \mid Z = 1] - E[D \mid Z = 0]$$

Since Z is a scalar the estimand for *IV* estimation is their ratio

$$\frac{E[Y \mid Z = 1] - E[Y \mid Z = 0]}{E[D \mid Z = 1] - E[D \mid Z = 0]}$$

which is the result utilized above to identify *LATE*, the marginal treatment effect for the subpopulation of compliers. Next, we explore some examples illustrating *IV* estimation of *LATE* with a binary instrument.

Tuebingen-style examples

We return to the Tuebingen-style examples introduced in chapter 8 by supplementing them with a binary instrument Z. Likelihood assignment to treatment choice maintains the state-by-state probability structure. Uniformity dictates that we assign zero likelihood that an individual is a defier,[8]

$$p_D \equiv \Pr(s, D_0 = 1, D_1 = 0) = 0.0$$

[8]This assumption preserves the identification link between *LATE* and *IV* estimation. Uniformity is a natural consequence of an index-structured propensity score, say $\Pr(D_i \mid W_i) = G\left(W_i^T \gamma\right)$. Case 1b below illustrates how the presence of defiers in the sample confounds *IV* identification of *LATE*.

Then, we assign the likelihoods that an individual is a complier,

$$p_C \equiv \Pr(s, D_0 = 0, D_1 = 1)$$

an individual never selects treatment,

$$p_N \equiv \Pr(s, D_0 = 0, D_1 = 0)$$

and an individual always selects treatment,

$$p_A \equiv \Pr(s, D_0 = 1, D_1 = 1)$$

such that state-by-state

$$\begin{aligned} p_1 &\equiv \Pr(s, D_1 = 1) = p_C + p_A \\ p_0 &\equiv \Pr(s, D_0 = 1) = p_D + p_A \\ q_1 &\equiv \Pr(s, D_1 = 0) = p_D + p_N \\ q_0 &\equiv \Pr(s, D_0 = 0) = p_C + p_N \end{aligned}$$

Since $(Y_j \mid D = 1, s) = (Y_j \mid D = 0, s)$ for $j = 0$ or 1, the exclusion restriction is satisfied if

$$\Pr(s \mid Z = 1) = \Pr(s \mid Z = 0)$$

and

$$\begin{aligned} \Pr(s \mid Z = 1) &= p_1 + q_1 \\ &= p_C + p_A + p_D + p_N \end{aligned}$$

equals

$$\begin{aligned} \Pr(s \mid Z = 0) &= p_0 + q_0 \\ &= p_D + p_A + p_C + p_N \end{aligned}$$

probability assignment for compliance determines the remaining likelihood structure given $\Pr(s, D)$, $\Pr(Z)$, and $p_D = 0$. For instance,

$$\Pr(s, D = 0, Z = 0) = (p_C + p_N) \Pr(Z = 0)$$

and

$$\Pr(s, D = 0, Z = 1) = (p_D + p_N) \Pr(Z = 1)$$

since

$$\Pr(s, D = 0) = (p_C + p_N) \Pr(Z = 0) + (p_D + p_N) \Pr(Z = 1)$$

implies

$$p_N = \Pr(s, D = 0) - p_C \Pr(Z = 0) - p_D \Pr(Z = 1)$$

By similar reasoning,

$$p_A = \Pr(s, D = 1) - p_C \Pr(Z = 1) - p_D \Pr(Z = 0)$$

Now we're prepared to explore some specific examples.

Table 10.1: Tuebingen IV example treatment likelihoods for case 1: ignorable treatment

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D=1 \mid s)$	0.32	0.0	0.08
compliers: $\Pr(s, D_0=0, D_1=1)$	0.0128	0.0	0.0512
never treated: $\Pr(s, D_0=0, D_1=0)$	0.01824	0.32	0.55296
always treated: $\Pr(s, D_0=1, D_1=1)$	0.00896	0.0	0.03584
defiers: $\Pr(s, D_0=1, D_1=0)$	0.0	0.0	0.0
$\Pr(Z=1)=0.3$			

Table 10.2: Tuebingen IV example outcome likelihoods for case 1: ignorable treatment

state (s)	*one*		*two*		*three*	
$\Pr\begin{pmatrix} Y,D,s, \\ Z=0 \end{pmatrix}$	0.021728	0.006272	0.224	0.0	0.422912	0.025088
$\Pr\begin{pmatrix} Y,D,s, \\ Z=1 \end{pmatrix}$	0.005472	0.006528	0.096	0.0	0.165888	0.026112
D	0	1	0	1	0	1
Y	0	1	1	1	2	1
Y_0	0	0	1	1	2	2
Y_1	1	1	1	1	1	1

Case 1

Given $\Pr(Z=1) = 0.3$, treatment likelihood assignments for case 1 are described in table 10.1. Then, from

$$\begin{aligned}\Pr(s, D=1) &= (p_C + p_A)\Pr(Z=1) + (p_D + p_A)\Pr(Z=0) \\ &= \Pr(D=1, Z=1) + \Pr(D=1, Z=0)\end{aligned}$$

and

$$\begin{aligned}\Pr(s, D=0) &= (p_D + p_N)\Pr(Z=1) + (p_C + p_N)\Pr(Z=0) \\ &= \Pr(D=0, Z=1) + \Pr(D=0, Z=0)\end{aligned}$$

the *DGP* for case 1, ignorable treatment, is identified in table 10.2. Various treatment effects including *LATE* and the *IV*-estimand for case 1 are reported in table 10.3. Case 1 illustrates homogeneous response — all treatment effects, including *LATE*, are the same. Further, endogeneity of treatment is ignorable as Y_1 and Y_0 are conditionally mean independent of D; hence, *OLS* identifies the treatment effects.

Case 1b

Suppose everything remains the same as above except treatment likelihood includes a nonzero defier likelihood as defined in table 10.4. This case highlights

Table 10.3: Tuebingen IV example results for case 1: ignorable treatment

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1]$ $= -0.6$	$p = \Pr(D = 1) = 0.064$
$IV - estimand = \frac{E[Y\|Z=1]-E[Y\|Z=0]}{E[D\|Z=1]-E[D\|Z=0]}$ $= -0.6$	$\Pr(D = 1 \mid Z = 1) = 0.1088$
	$\Pr(D = 1 \mid Z = 0) = 0.0448$
	$E[Y_1 \mid D = 1] = 1.0$
	$E[Y_1 \mid D = 0] = 1.0$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = -0.6$	$E[Y_1] = 1.0$
$ATT = E[Y_1 - Y_0 \mid D = 1] = -0.6$	$E[Y_0 \mid D = 1] = 1.6$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = -0.6$	$E[Y_0 \mid D = 0] = 1.6$
$ATE = E[Y_1 - Y_0] = -0.6$	$E[Y_0] = 1.6$

Table 10.4: Tuebingen IV example treatment likelihoods for case 1b: uniformity fails

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D = 1 \mid s)$	0.32	0.0	0.08
compliers: $\Pr(s, D_0 = 0, D_1 = 1)$	0.0064	0.0	0.0256
never treated: $\Pr(s, D_0 = 0, D_1 = 0)$	0.02083	0.32	0.56323
always treated: $\Pr(s, D_0 = 1, D_1 = 1)$	0.00647	0.0	0.02567
defiers: $\Pr(s, D_0 = 1, D_1 = 0)$	0.0063	0.0	0.0255
$\Pr(Z = 1) = 0.3$			

Table 10.5: Tuebingen IV example treatment likelihoods for case 2: heterogeneous response

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D=1 \mid s)$	0.32	0.3	0.08
compliers: $\Pr(D_0=0, D_1=1)$	0.01	0.096	0.0512
never treated: $\Pr(D_0=0, D_1=0)$	0.0202	0.1568	0.55296
always treated: $\Pr(D_0=1, D_1=1)$	0.0098	0.0672	0.03584
defiers: $\Pr(D_0=1, D_1=0)$	0.0	0.0	0.0
$\Pr(Z=1)=0.3$			

the difficulty of identifying treatment effects when uniformity of selection with respect to the instrument fails even though in this ignorable treatment setting all treatment effects are equal. Uniformity failure means some individuals who were untreated when $Z=0$ opt for treatment when $Z=1$ but other individuals who were treated when $Z=0$ opt for no treatment when $Z=1$.

From the identification discussion, the difference in expected observed outcome when the instrument changes is

$$\begin{aligned} & E[Y_i \mid Z_i=1] - E[Y_i \mid Z_i=0] \\ = \; & \Pr(D_{1i}-D_{0i}=1)\, E[Y_{1i}-Y_{0i} \mid D_{1i}-D_{0i}=1] \\ & + \Pr(D_{1i}-D_{0i}=-1)\, E[-(Y_{1i}-Y_{0i}) \mid D_{1i}-D_{0i}=-1] \\ = \; & 0.032\,(-0.6) + 0.0318\,(0.6038) = 0.0 \end{aligned}$$

The effects

$$E[Y_{1i}-Y_{0i} \mid D_{1i}-D_{0i}=1] = -0.6$$

and

$$E[-(Y_{1i}-Y_{0i}) \mid D_{1i}-D_{0i}=-1] = 0.6038$$

are offsetting and seemingly hopelessly confounded. *2SLS-IV* estimates

$$\frac{E[Y_i \mid Z_i=1] - E[Y_i \mid Z_i=0]}{E[D_i \mid Z_i=1] - E[D_i \mid Z_i=0]} = \frac{0.0}{0.0002} = 0.0$$

which differs from $LATE = E[Y_{1i}-Y_{0i} \mid D_i(1) - D_i(0) = 1] = -0.6$. Therefore, we may be unable to identify *LATE*, the marginal treatment effect for compliers, via *2SLS-IV* when defiers are present in the sample.

Case 2

Case 2 perturbs the probabilities resulting in non-ignorable, inherently endogenous treatment and heterogeneous treatment effects. Treatment adoption likelihoods, assuming the likelihood an individual is a defier equals zero and $\Pr(Z=1) = 0.3$, are assigned in table 10.5. These treatment likelihoods imply the data structure in table 10.6. Various treatment effects including *LATE* and the *IV*-estimand

Table 10.6: Tuebingen IV example outcome likelihoods for case 2: heterogeneous response

state (s)	*one*		*two*		*three*	
$\Pr\begin{pmatrix} Y,D,s, \\ Z=0 \end{pmatrix}$	0.021728	0.006272	0.224	0.0	0.422912	0.025088
$\Pr\begin{pmatrix} Y,D,s, \\ Z=1 \end{pmatrix}$	0.005472	0.006528	0.096	0.0	0.165888	0.026112
D	0	1	0	1	0	1
Y	0	1	1	1	2	1
Y_0	0	0	1	1	2	2
Y_1	1	1	1	1	1	1

Table 10.7: Tuebingen IV example results for case 2: heterogeneous response

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1]$ $= -0.2621$	$p = \Pr(D=1) = 0.16$
$IV - estimand = \frac{E[Y\mid Z=1]-E[Y\mid Z=0]}{E[D\mid Z=1]-E[D\mid Z=0]}$ $= -0.2621$	$\Pr(D=1 \mid Z=1) = 0.270$
	$\Pr(D=1 \mid Z=0) = 0.113$
	$E[Y_1 \mid D=1] = 1.0$
	$E[Y_1 \mid D=0] = 1.0$
$OLS = \begin{matrix} E[Y_1 \mid D=1] \\ -E[Y_0 \mid D=0] \end{matrix} = -0.669$	$E[Y_1] = 1.0$
$ATT = E[Y_1 - Y_0 \mid D=1] = -0.24$	$E[Y_0 \mid D=1] = 1.24$
$ATUT = E[Y_1 - Y_0 \mid D=0] = -0.669$	$E[Y_0 \mid D=0] = 1.669$
$ATE = E[Y_1 - Y_0] = -0.6$	$E[Y_0] = 1.6$

Table 10.8: Tuebingen IV example treatment likelihoods for case 2b: LATE = ATT

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D=1 \mid s)$	0.3	0.3	0.08
compliers: $\Pr(s, D_0=0, D_1=1)$	0.04	0.32	0.17067
never treated: $\Pr(s, D_0=0, D_1=0)$	0.0	0.0	0.46933
always treated: $\Pr(s, D_0=1, D_1=1)$	0.0	0.0	0.0
defiers: $\Pr(s, D_0=1, D_1=0)$	0.0	0.0	0.0
$\Pr(Z=1)=0.3$			

Table 10.9: Tuebingen IV example outcome likelihoods for case 2b: LATE = ATT

state (s)	*one*		*two*		*three*	
$\Pr(Y, D, s, Z=0)$	0.028	0.0	0.224	0.0	0.448	0.0
$\Pr(Y, D, s, Z=1)$	0.0	0.012	0.0	0.096	0.1408	0.0512
D	0	1	0	1	0	1
Y	0	1	1	1	2	1
Y_0	0	0	1	1	2	2
Y_1	1	1	1	1	1	1

for case 2 are reported in table 10.7. In contrast to case 1, for case 2 all treatment effects (*ATE*, *ATT*, *ATUT*, and *LATE*) differ which, of course, means *OLS* cannot identify all treatment effects (though it does identify *ATUT* in this setting). Importantly, the *IV*-estimand identifies *LATE* for the subpopulation of compliers.

Case 2b

If we perturb the probability structure such that

$$\Pr(D=1 \mid Z=0)=0$$

then $LATE = ATT$.[9] For $\Pr(Z=1)=0.3$, treatment adoption likelihoods are assigned in table 10.8. Then, the data structure is as indicated in table 10.9. Various treatment effects including *LATE* and the *IV*-estimand for case 2b are reported in table 10.10. With this perturbation of likelihoods but maintenance of independence between Z and (Y_1, Y_0), *LATE=ATT* and *LATE* is identified via the *IV*-estimand but is not identified via *OLS*. Notice the evidence on counterfactuals draws from $Z=1$ as no one adopts treatment when $Z=0$.

Case 3

Case 3 maintains the probability structure of case 2 but adds some variation to outcomes with treatment Y_1. For $\Pr(Z=1)=0.3$, treatment adoption likelihoods

[9] We also perturbed $\Pr(D=1 \mid s=one)=0.3$ rather than 0.32 to maintain the exclusion restriction and a proper (non-negative) probability distribution.

Table 10.10: Tuebingen IV example results for case 2b: LATE = ATT

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1]$ $= -0.246$	$p = \Pr(D = 1) = 0.1592$
$IV - estimand = \frac{E[Y\|Z=1]-E[Y\|Z=0]}{E[D\|Z=1]-E[D\|Z=0]}$ $= -0.246$	$\Pr(D = 1 \mid Z = 1) = 0.5307$
	$\Pr(D = 1 \mid Z = 0) = 0.0$
	$E[Y_1 \mid D = 1] = 1.0$
	$E[Y_1 \mid D = 0] = 1.0$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = -0.667$	$E[Y_1] = 1.0$
$ATT = E[Y_1 - Y_0 \mid D = 1] = -0.246$	$E[Y_0 \mid D = 1] = 1.246$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = -0.667$	$E[Y_0 \mid D = 0] = 1.667$
$ATE = E[Y_1 - Y_0] = -0.6$	$E[Y_0] = 1.6$

Table 10.11: Tuebingen IV example treatment likelihoods for case 3: more heterogeneity

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D = 1 \mid s)$	0.32	0.3	0.08
compliers: $\Pr(s, D_0 = 0, D_1 = 1)$	0.01	0.096	0.0512
never treated: $\Pr(s, D_0 = 0, D_1 = 0)$	0.0202	0.1568	0.55296
always treated: $\Pr(s, D_0 = 1, D_1 = 1)$	0.0098	0.0672	0.03584
defiers: $\Pr(s, D_0 = 1, D_1 = 0)$	0.0	0.0	0.0
$\Pr(Z = 1) = 0.3$			

are assigned in table 10.11. Then, the data structure is defined in table 10.12 where Z_0 refers to $Z = 0$ and Z_1 refers to $Z = 1$. Various treatment effects including *LATE* and the *IV*-estimand for case 3 are reported in table 10.13. *OLS* doesn't identify any treatment effect but the *IV*-estimand identifies the discrete marginal treatment effect, *LATE,* for case 3.

Case 3b

Suppose the probability structure of case 3 is perturbed such that

$$\Pr(D = 1 \mid Z = 1) = 1$$

then *LATE*=*ATUT*.[10] For $\Pr(Z = 1) = 0.3$, treatment adoption likelihoods are assigned in table 10.14. Then, the data structure is as defined in table 10.15. Various treatment effects including *LATE* and the *IV*-estimand for case 3b are reported in table 10.16. The *IV*-estimand identifies *LATE* and $LATE = ATUT$ since treat-

[10] We assign $\Pr(D = 1 \mid s = three) = 0.6$ rather than 0.08 to preserve the exclusion restriction.

Table 10.12: Tuebingen IV example outcome likelihoods for case 3: more heterogeneity

state (s)	*one*		*two*		*three*	
$\Pr\begin{pmatrix} Y \\ D \\ s \\ Z_0 \end{pmatrix}$	0.02114	0.00686	0.17696	0.04704	0.422912	0.025088
$\Pr\begin{pmatrix} Y \\ D \\ s \\ Z_1 \end{pmatrix}$	0.00606	0.00594	0.04704	0.04896	0.165888	0.026112
D	0	1	0	1	0	1
Y	0	1	1	1	2	0
Y_0	0	0	1	1	2	2
Y_1	1	1	1	1	0	0

Table 10.13: Tuebingen IV example results for case 3: more heterogeneity

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1]$ $= -0.588$	$p = \Pr(D = 1) = 0.16$
$IV - estimand = \frac{E[Y\|Z=1]-E[Y\|Z=0]}{E[D\|Z=1]-E[D\|Z=0]}$ $= -0.588$	$\Pr(D = 1 \mid Z = 1) = 0.270$
	$\Pr(D = 1 \mid Z = 0) = 0.113$
	$E[Y_1 \mid D = 1] = 0.68$
	$E[Y_1 \mid D = 0] = 0.299$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = -0.989$	$E[Y_1] = 0.36$
$ATT = E[Y_1 - Y_0 \mid D = 1] = -0.56$	$E[Y_0 \mid D = 1] = 1.24$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = -1.369$	$E[Y_0 \mid D = 0] = 1.669$
$ATE = E[Y_1 - Y_0] = -1.24$	$E[Y_0] = 1.6$

ment is always selected when $Z = 1$. Also, notice *OLS* is close to *ATE* even though this is a case of inherent endogeneity. This suggests comparing *ATE* with *OLS* provide an inadequate test for the existence of endogeneity.

Case 4

Case 4 employs a richer set of outcomes but the probability structure for (D, Y, s) employed in case 1 and yields the Simpson's paradox result noted in chapter 8. For $\Pr(Z = 1) = 0.3$, assignment of treatment adoption likelihoods are described in table 10.17. Then, the data structure is identified in table 10.18. Various treatment effects including *LATE* and the *IV*-estimand for case 4 are reported in table 10.19. *OLS* estimates a negative effect while all the standard average treatment effects are positive. Identification conditions are satisfied and the *IV*-estimand identifies *LATE*.

Table 10.14: Tuebingen IV example treatment likelihoods for case 3b: LATE = ATUT

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D=1 \mid s)$	0.32	0.3	0.6
compliers: $\Pr(s, D_0=0, D_1=1)$	0.038857	0.32	0.365714
never treated: $\Pr(s, D_0=0, D_1=0)$	0.0	0.0	0.0
always treated: $\Pr(s, D_0=1, D_1=1)$	0.001143	0.0	0.274286
defiers: $\Pr(s, D_0=1, D_1=0)$	0.0	0.0	0.0
$\Pr(Z=1)=0.3$			

Table 10.15: Tuebingen IV example outcome likelihoods for case 3b: LATE = ATUT

state (s)	*one*		*two*		*three*	
$\Pr(Y, D, s, Z=0)$	0.0272	0.0008	0.224	0.0	0.256	0.192
$\Pr(Y, D, s, Z=1)$	0.0	0.0012	0.0	0.096	0.0	0.192
D	0	1	0	1	0	1
Y	0	1	1	1	2	0
Y_0	0	0	1	1	2	2
Y_1	1	1	1	1	0	0

Case 4b

For $Z = D$ and $\Pr(Z=1) = \Pr(D=1) = 0.16$, case 4b explores violation of the exclusion restriction. Assignment of treatment adoption likelihoods are described in table 10.20. However, as indicated earlier the exclusion restriction apparently can only be violated in this binary instrument setting if treatment alters the outcome distributions. To explore the implications of this variation, we perturb outcomes with treatment slightly as defined in table 10.21. Various treatment effects including *LATE* and the *IV*-estimand for case 4b are reported in table 10.22. Since the exclusion restriction is not satisfied the *IV*-estimand fails to identify *LATE*. In fact, *OLS* and *2SLS-IV* estimates are both negative while *ATE* and *LATE* are positive. As $Z = D$, the entire population consists of compliers, and it is difficult to assess the counterfactuals as there is no variation in treatment when either $Z = 0$ or $Z = 1$. Hence, it is critical to treatment effect identification that treatment not induce a shift in the outcome distributions but rather variation in the instruments produces a change in treatment status only.

Case 5

Case 5 involves $\Pr(z=1) = 0.3$, and non-overlapping support:

$$\Pr(s = one, D = 0) = 0.04$$

$$\Pr(s = two, D = 1) = 0.32$$

Table 10.16: Tuebingen IV example results for case 3b: LATE = ATUT

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0] = 1] = -0.9558$	$p = \Pr(D = 1) = 0.4928$
$IV - estimand = \frac{E[Y\|Z=1]-E[Y\|Z=0]}{E[D\|Z=1]-E[D\|Z=0]} = -0.9558$	$\Pr(D = 1 \mid Z = 1) = 1.0$
	$\Pr(D = 1 \mid Z = 0) = 0.2754$
	$E[Y_1 \mid D = 1] = 0.2208$
	$E[Y_1 \mid D = 0] = 0.4953$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = -1.230$	$E[Y_1] = 0.36$
$ATT = E[Y_1 - Y_0 \mid D = 1] = -1.5325$	$E[Y_0 \mid D = 1] = 1.7532$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = -0.9558$	$E[Y_0 \mid D = 0] = 1.4511$
$ATE = E[Y_1 - Y_0] = -1.24$	$E[Y_0] = 1.6$

Table 10.17: Tuebingen IV example treatment likelihoods for case 4: Simpson's paradox

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D = 1 \mid s)$	0.32	0.3	0.08
compliers: $\Pr(s, D_0 = 0, D_1 = 1)$	0.01	0.096	0.0512
never treated: $\Pr(s, D_0 = 0, D_1 = 0)$	0.0202	0.1568	0.55296
always treated: $\Pr(s, D_0 = 1, D_1 = 1)$	0.0098	0.0672	0.03584
defiers: $\Pr(s, D_0 = 1, D_1 = 0)$	0.0	0.0	0.0
$\Pr(Z = 1) = 0.3$			

and

$$\Pr(s = three, D = 0) = 0.64$$

as assigned in table 10.23.

There is no positive complier likelihood for this setting. The intuition for this is as follows. Compliers elect no treatment when the instrument takes a value of zero but select treatment when the instrument is unity. With the above likelihood structure there is no possibility for compliance as each state is singularly treatment or no treatment irrespective of the instrument as described in table 10.24.

Various treatment effects including *LATE* and the *IV*-estimand for case 5 are reported in table 10.25. Case 5 illustrates the danger of lack of common support. Common support concerns extend to other standard ignorable treatment and *IV* identification approaches beyond *LATE*. Case 5b perturbs the likelihoods slightly to recover *IV* identification of *LATE*.

Table 10.18: Tuebingen IV example outcome likelihoods for case 4: Simpson's paradox

state (s)	*one*		*two*		*three*	
$\Pr\begin{pmatrix} Y \\ D \\ s \\ Z_0 \end{pmatrix}$	0.02114	0.00686	0.17696	0.04704	0.422912	0.025088
$\Pr\begin{pmatrix} Y \\ D \\ s \\ Z_1 \end{pmatrix}$	0.00606	0.00594	0.04704	0.04896	0.165888	0.026112
D	0	1	0	1	0	1
Y	0.0	1.0	1.0	1.0	2.0	2.3
Y_0	0.0	0.0	1.0	1.0	2.0	2.0
Y_1	1.0	1.0	1.0	1.0	2.3	2.3

Table 10.19: Tuebingen IV example results for case 4: Simpson's paradox

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1] = 0.161$	$p = \Pr(D = 1) = 0.16$
$IV - estimand = \frac{E[Y\|Z=1]-E[Y\|Z=0]}{E[D\|Z=1]-E[D\|Z=0]} = 0.161$	$\Pr(D = 1 \mid Z = 1) = 0.27004$
	$\Pr(D = 1 \mid Z = 0) = 0.11284$
	$E[Y_1 \mid D = 1] = 1.416$
	$E[Y_1 \mid D = 0] = 1.911$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = -0.253$	$E[Y_1] = 1.832$
$ATT = E[Y_1 - Y_0 \mid D = 1] = 0.176$	$E[Y_0 \mid D = 1] = 1.24$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = 0.243$	$E[Y_0 \mid D = 0] = 1.669$
$ATE = E[Y_1 - Y_0] = 0.232$	$E[Y_0] = 1.6$

Case 5b

Case 5b perturbs the probabilities slightly such that

$$\Pr(s = two, D = 1) = 0.3104$$

and

$$\Pr(s = two, D = 0) = 0.0096$$

as depicted in table 10.26; everything else remains as in case 5. This slight perturbation accommodates treatment adoption likelihood assignments as defined in table 10.27. Various treatment effects including *LATE* and the *IV*-estimand for case 5b are reported in table 10.28. Even though there is a very small subpopulation of compliers, *IV* identifies *LATE*. The common support issue was discussed in

Table 10.20: Tuebingen IV example treatment likelihoods for case 4b: exclusion restriction violated

state (s)	*one*	*two*	*three*
$\Pr(s)$	0.04	0.32	0.64
$\Pr(D=1 \mid s)$	0.32	0.3	0.08
compliers: $\Pr(s, D_0=0, D_1=1)$	0.04	0.32	0.64
never treated: $\Pr(s, D_0=0, D_1=0)$	0.0	0.0	0.0
always treated: $\Pr(s, D_0=1, D_1=1)$	0.0	0.0	0.0
defiers: $\Pr(s, D_0=1, D_1=0)$	0.0	0.0	0.0
$\Pr(Z=1)=0.16$			

Table 10.21: Tuebingen IV example outcome likelihoods for case 4b: exclusion restriction violated

state (s)	*one*		*two*		*three*	
$\Pr\begin{pmatrix} Y,D,s, \\ Z=0 \end{pmatrix}$	0.0336	0.0	0.2688	0.0	0.5376	0.0
$\Pr\begin{pmatrix} Y,D,s, \\ Z=1 \end{pmatrix}$	0.0	0.0064	0.0	0.0512	0.0	0.1024
D	0	1	0	1	0	1
Y	0.0	3.0	1.0	1.0	2.0	1.6
Y_0	0.0	0.0	1.0	1.0	2.0	2.0
Y_1	1.0	1.0	1.0	1.0	2.3	1.6

the context of the asset revaluation regulation example in chapter 9 and comes up again in the discussion of regulated report precision example later in this chapter.

Discussion of *LATE*

Linear *IV* estimation of *LATE* has considerable appeal. Given the existence of instruments, it is simple to implement (*2SLS-IV*) and robust; it doesn't rely on strong distributional conditions and can accommodate unobservable heterogeneity. However, it also has drawbacks. We cannot identify the subpopulation of compliers due to unobservable counterfactuals. If the instruments change, it's likely that the treatment effect (*LATE*) and the subpopulation of compliers will change. This implies that different analysts are likely to identify different treatment effects — an issue of concern to Heckman and Vytlacil [2005]. Continuous or multi-level discrete instruments and/or regressors produce a complicated weighted average of marginal treatment effects that are again dependent on the particular instrument chosen as discussed in the next chapter. Finally, the treatment effect literature is asymmetric. Outcome heterogeneity can be accommodated but uniformity (or homogeneity) of treatment is fundamental. This latter limitation applies to all *IV* approaches including local *IV* (*LIV*) estimation of *MTE* which is discussed in chapter 11.

Table 10.22: Tuebingen IV example results for case 4b: exclusion restriction violated

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1]$ $= 0.160$	$p = \Pr(D = 1) = 0.16$
$IV - estimand = \frac{E[Y\|Z=1]-E[Y\|Z=0]}{E[D\|Z=1]-E[D\|Z=0]}$ $= -0.216$	$\Pr(D = 1 \mid Z = 1) = 1.0$
	$\Pr(D = 1 \mid Z = 0) = 0.0$
	$E[Y_1 \mid D = 1] = 1.192$
	$E[Y_1 \mid D = 0] = 1.911$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = -0.477$	$E[Y_1] = 1.796$
$ATT = E[Y_1 - Y_0 \mid D = 1] = -0.048$	$E[Y_0 \mid D = 1] = 1.24$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = 0.243$	$E[Y_0 \mid D = 0] = 1.669$
$ATE = E[Y_1 - Y_0] = 0.196$	$E[Y_0] = 1.6$

Table 10.23: Tuebingen IV example outcome likelihoods for case 5: lack of common support

state (s)	*one*		*two*		*three*	
$\Pr(Y, D, s, Z = 0)$	0.028	0.0	0.0	0.224	0.448	0.0
$\Pr(Y, D, s, Z = 1)$	0.012	0.0	0.0	0.096	0.192	0.0
D	0	1	0	1	0	1
Y	0	1	1	2	2	0
Y_0	0	0	1	1	2	2
Y_1	1	1	2	2	0	0

Censored regression and *LATE*

Angrist [2001] discusses identification of *LATE* in the context of censored regression.[11] He proposes a non-negative transformation $\exp(X\beta)$ combined with linear *IV* to identify a treatment effect. Like the discussion of *LATE* above, the approach is simplest and most easily interpreted when the instrument is binary and there are no covariates. Angrist extends the discussion to cover quantile treatment effects based on censored quantile regression combined with Abadie's [2000] causal *IV*.

[11] This is not to be confused with sample selection. Here, we refer to cases in which the observed outcome follows a switching regression that permits identification of counterfactuals.

Table 10.24: Tuebingen IV example treatment likelihoods for case 5: lack of common support

state	*one*	*two*	*three*
compliers: $\Pr(D_0 = 0, D_1 = 1)$	0.0	0.0	0.0
never treated: $\Pr(D_0 = 0, D_1 = 0)$	0.04	0.0	0.64
always treated: $\Pr(D_0 = 1, D_1 = 1)$	0.0	0.32	0.0
defiers: $\Pr(D_0 = 1, D_1 = 0)$	0.0	0.0	0.0
$\Pr(Z = 1) = 0.3$			

Table 10.25: Tuebingen IV example results for case 5: lack of common support

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1]$ $= NA$	$p = \Pr(D = 1) = 0.32$
$IV - estimand = \frac{E[Y\mid Z=1]-E[Y\mid Z=0]}{E[D\mid Z=1]-E[D\mid Z=0]}$ $= \frac{0}{0}$	$\Pr(D = 1 \mid Z = 1) = 0.32$
	$\Pr(D = 1 \mid Z = 0) = 0.32$
	$E[Y_1 \mid D = 1] = 2.0$
	$E[Y_1 \mid D = 0] = 0.0588$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = 0.118$	$E[Y_1] = 0.68$
$ATT = E[Y_1 - Y_0 \mid D = 1] = 1.0$	$E[Y_0 \mid D = 1] = 1.0$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = -1.824$	$E[Y_0 \mid D = 0] = 1.882$
$ATE = E[Y_1 - Y_0] = -0.92$	$E[Y_0] = 1.6$

For (Y_{0i}, Y_{1i}) independent of $(D_i \mid X_i, D_{1i} > D_{0i})$ Abadie defines the causal *IV* effect, *LATE*.

$$\begin{aligned} LATE &= E[Y_i \mid X_i, D_i = 1, D_{1i} > D_{0i}] \\ &\quad -E[Y_i \mid X_i, D_i = 0, D_{1i} > D_{0i}] \\ &= E[Y_{1i} - Y_{0i} \mid X_i, D_{1i} > D_{0i}] \end{aligned}$$

Then, for binary instrument Z, Abadie shows

$$\begin{aligned} & E\left[\left(E[Y_i \mid X_i, D_i, D_{1i} > D_{0i}] - X_i^T b - aD_i\right)^2 \mid D_{1i} > D_{0i}\right] \\ = \quad & \frac{E\left[\kappa_i \left(E[Y_i \mid X_i, D_i, D_{1i} > D_{0i}] - X_i^T b - aD_i\right)^2\right]}{\Pr(D_{1i} > D_{0i})} \end{aligned}$$

where

$$\kappa_i = 1 - \frac{D_i(1 - Z_i)}{\Pr(Z_i = 0 \mid X_i)} - \frac{(1 - D_i) Z_i}{\Pr(Z_i = 1 \mid X_i)}$$

Table 10.26: Tuebingen IV example outcome likelihoods for case 5b: minimal common support

state (s)	*one*		*two*		*three*	
$\Pr(Y, D, s, Z = 0)$	0.028	0.0	0.0082	0.21518	0.448	0.0
$\Pr(Y, D, s, Z = 1)$	0.012	0.0	0.00078	0.09522	0.192	0.0
D	0	1	0	1	0	1
Y	0	1	1	2	2	0
Y_0	0	0	1	1	2	2
Y_1	1	1	2	2	0	0

Table 10.27: Tuebingen IV example outcome likelihoods for case 5b: minimal common support

state	*one*	*two*	*three*
compliers: $\Pr(D_0 = 0, D_1 = 1)$	0.0	0.01	0.0
never treated: $\Pr(D_0 = 0, D_1 = 0)$	0.04	0.0026	0.64
always treated: $\Pr(D_0 = 1, D_1 = 1)$	0.0	0.3074	0.0
defiers: $\Pr(D_0 = 1, D_1 = 0)$	0.0	0.0	0.0
$\Pr(Z = 1) = 0.30$			

Since κ_i can be estimated from the observable data, one can employ minimum "weighted" least squares to estimate a and b. That is,

$$\underset{a,b}{min} E\left[\kappa_i \left(Y_i - X_i^T b - aD_i\right)^2\right]$$

Notice for compliers $Z_i = D_i$ (for noncompliers, $Z_i \neq D_i$) and κ_i always equals one for compliers and is unequal to one (in fact, negative) for noncompliers. Intuitively, Abadie's causal *IV* estimator weights the data such that the residuals are small for compliers but large (in absolute value) for noncompliers. The coefficient on D, a, is the treatment effect. We leave remaining details for the interested reader to explore. In chapter 11, we discuss a unified strategy, proposed by Heckman and Vytlacil [2005, 2007a, 2007b] and Heckman and Abbring [2007], built around marginal treatment effects for addressing means as well as distributions of treatment effects.

10.6 Continuous treatment

Suppressing covariates, the average treatment effect for continuous treatment can be defined as

$$ATE = E\left[\frac{\partial}{\partial d} Y\right]$$

Table 10.28: Tuebingen IV example results for case 5b: minimal common support

Results	Key components
$LATE = E[Y_1 - Y_0 \mid D_1 - D_0 = 1] = 1.0$	$p = \Pr(D = 1) = 0.3104$
$IV - estimand = \frac{E[Y\|Z=1]-E[Y\|Z=0]}{E[D\|Z=1]-E[D\|Z=0]} = 1.0$	$\Pr(D = 1 \mid Z = 1) = 0.3174$
	$\Pr(D = 1 \mid Z = 0) = 0.3074$
	$E[Y_1 \mid D = 1] = 2.0$
	$E[Y_1 \mid D = 0] = 0.086$
$OLS = \begin{matrix} E[Y_1 \mid D = 1] \\ -E[Y_0 \mid D = 0] \end{matrix} = 0.13$	$E[Y_1] = 0.68$
$ATT = E[Y_1 - Y_0 \mid D = 1] = 1.0$	$E[Y_0 \mid D = 1] = 1.0$
$ATUT = E[Y_1 - Y_0 \mid D = 0] = -1.784$	$E[Y_0 \mid D = 0] = 1.870$
$ATE = E[Y_1 - Y_0] = -0.92$	$E[Y_0] = 1.6$

Often, the more economically-meaningful effect, the average treatment effect on treated for continuous treatment is

$$ATT = E\left[\frac{\partial}{\partial d} Y \mid D = d\right]$$

Wooldridge [1997, 2003] provides conditions for identifying continuous treatment effects via 2*SLS-IV*. This is a classic correlated random coefficients setting (see chapter 3) also pursued by Heckman [1997] and Heckman and Vytlacil [1998] (denoted HV in this subsection). As the parameters or coefficients are random, the model accommodates individual heterogeneity. Further, correlation between the treatment variable and the treatment effect parameter accommodates unobservable heterogeneity.

Let y be the outcome variable and $\mathbf{D}$ be a vector of G treatment variables.[12] The structural model[13] written in expectation form is

$$E[y \mid a, b, D] = a + \mathbf{bD}$$

or in error form, the model is

$$y = a + \mathbf{bD} + e$$

where $E[e \mid a, b, \mathbf{D}] = 0$. It's instructive to rewrite the model in error form for random draw i

$$y_i = a_i + \mathbf{D}_i \mathbf{b}_i + e_i$$

The model suggests that the intercept, a_i, and slopes, b_{ij}, $j = 1, \ldots, G$, can be individual-specific and depend on observed covariates or unobserved heterogeneity. Typically, we focus on the average treatment effect, $\boldsymbol{\beta} \equiv E[\mathbf{b}] = E[\mathbf{b}_i]$, as $\mathbf{b}$

[12] For simplicity as well as clarity, we'll stick with Wooldridge's [2003] setting and notation.

[13] The model is structural in the sense that the partial effects of D_j on the mean response are identified after controlling for the factor determining the intercept and slope parameters.

is likely a function of unobserved heterogeneity and we cannot identify the vector of slopes, $\mathbf{b}_i$, for any individual i.

Suppose we have K covariates $\mathbf{x}$ and L instrumental variables $\mathbf{z}$. As is common with *IV* strategies, identification utilizes an exclusion restriction. Specifically, the identification conditions are

Condition 10.27 *The covariates* $\mathbf{x}$ *and instruments* $\mathbf{z}$ *are redundant for the outcome* y.

$$E[y \mid a, \mathbf{b}, \mathbf{D}, \mathbf{x}, \mathbf{z}] = E[y \mid a, \mathbf{b}, \mathbf{D}]$$

Condition 10.28 *The instruments* $\mathbf{z}$ *are redundant for* a *and* $\mathbf{b}$ *conditional on* $\mathbf{x}$.

$$\begin{aligned} E[a \mid \mathbf{x}, \mathbf{z}] &= E[a \mid \mathbf{x}] = \gamma_0 + \mathbf{x}\gamma \\ E[b_j \mid \mathbf{x}, \mathbf{z}] &= E[b_j \mid \mathbf{x}] = \beta_{0j} + (\mathbf{x} - E[\mathbf{x}])\boldsymbol{\delta}_j, j = 1, \ldots, G \end{aligned}$$

Let the error form of a and b be

$$\begin{array}{ll} a = \gamma_0 + \mathbf{x}\boldsymbol{\gamma} + c, & E[c \mid \mathbf{x}, \mathbf{z}] = 0 \\ b_j = \beta_{0j} + (\mathbf{x} - E[\mathbf{x}])\boldsymbol{\delta}_j + \upsilon_j, & E[\upsilon_j \mid \mathbf{x}, \mathbf{z}] = 0, \quad j = 1, \ldots, G \end{array}$$

When plugged into the outcome equation this yields

$$y = \gamma_0 + \mathbf{x}\gamma + \mathbf{D}\beta_0 + D_1(\mathbf{x} - E[\mathbf{x}])\boldsymbol{\delta}_1 + \ldots + D_G(\mathbf{x} - E[\mathbf{x}])\boldsymbol{\delta}_G + c + \mathbf{D}\mathbf{v} + e$$

where $\mathbf{v} = (\upsilon_1, \ldots, \upsilon_G)^T$. The composite error $\mathbf{D}\mathbf{v}$ is problematic as, generally, $E[\mathbf{D}\mathbf{v} \mid x, z] \neq 0$ but as discussed by Wooldridge [1997] and HV [1998], it is possible that the conditional covariances do not depend on $(\mathbf{x}, \mathbf{z})$. This is the third identification condition.

Condition 10.29 *The conditional covariances between* $\mathbf{D}$ *and* $\mathbf{v}$ *do not depend on* $(\mathbf{x}, \mathbf{z})$.

$$E[D_j\upsilon_j \mid \mathbf{x}, \mathbf{z}] = \alpha_j \equiv Cov(D_j, \upsilon_j) = E[D_j\upsilon_j], \quad j = 1, \ldots, G$$

Let $\alpha_0 = \alpha_1 + \cdots + \alpha_G$ and $r = \mathbf{D}v - E[\mathbf{D}v \mid x, z]$ and write the outcome equation as

$$y = (\gamma_0 + \alpha_0) + x\gamma + \mathbf{D}\beta_0 + D_1(x - E[x])\delta_1 + \ldots + D_G(x - E[x])\delta_G + c + r + e$$

Since the composite error $u \equiv c + r + e$ has zero mean conditional on (x, z), we can use any function of (x, z) as instruments in the outcome equation

$$y = \theta_0 + \mathbf{x}\boldsymbol{\gamma} + \mathbf{D}\beta_0 + D_1(\mathbf{x} - E[\mathbf{x}])\boldsymbol{\delta}_1 + \ldots + D_G(\mathbf{x} - E[\mathbf{x}])\boldsymbol{\delta}_G + u$$

Wooldridge [2003, p. 189] argues *2SLS-IV* is more robust than HV's plug-in estimator and the standard errors are simpler to obtain. Next, we revisit the third accounting setting from chapter 2, regulated report precision, and explore various treatment effect strategies within this richer accounting context.

10.7 Regulated report precision

Now, consider the report precision example introduced in chapter 2. Recall regulators set a target report precision as regulation increases report precision and improves the owner's welfare relative to private precision choice. However, regulation also invites transaction design (commonly referred to as earnings management) which produces deviations from regulatory targets. The owner's expected utility including the cost of transaction design, $\alpha_d \left(\hat{b} - \sigma_2^2\right)^2$, is

$$EU\left(\sigma_2\right) = \mu - \beta \frac{\sigma_1^2 \bar{\sigma}_2^2}{\sigma_1^2 + \bar{\sigma}_2^2} - \gamma \frac{\sigma_1^4 \left(\sigma_1^2 + \sigma_2^2\right)}{\left(\sigma_1^2 + \bar{\sigma}_2^2\right)^2} - \alpha \left(b - \sigma_2^2\right)^2 - \alpha_d \left(\hat{b} - \sigma_2^2\right)^2$$

Outcomes Y are reflected in exchange values or prices and accordingly reflect only a portion of the owner's expected utility.

$$Y = P\left(\bar{\sigma}_2\right) = \mu + \frac{\sigma_1^2}{\sigma_1^2 + \bar{\sigma}_2^2}\left(s - \mu\right) - \beta \frac{\sigma_1^2 \bar{\sigma}_2^2}{\sigma_1^2 + \bar{\sigma}_2^2}$$

In particular, cost may be hidden from the analysts' view; cost includes the explicit cost of report precision, $\alpha \left(b - \sigma_2^2\right)^2$, cost of any transaction design, $\alpha_d \left(\hat{b} - \sigma_2^2\right)^2$, and the owner's risk premia, $\gamma \frac{\sigma_1^4 \left(\sigma_1^2 + \sigma_2^2\right)}{\left(\sigma_1^2 + \bar{\sigma}_2^2\right)^2}$. Further, outcomes (prices) reflect realized draws from the accounting system, s, whereas the owner's expected utility is based on anticipated reports and her knowledge of the distribution for (s, EU). The causal effect of treatment (report precision choice) on outcomes is the subject under study and is almost surely endogenous. Our analysis entertains variations of treatment data including binary choice that is observed (by the analyst) binary, a continuum of choices that is observed binary, and continuous treatment that is observed from a continuum of choices.

10.7.1 Binary report precision choice

Suppose there are two types of owners, those with low report precision cost parameter α_d^L, and those with high report precision cost parameter α_d^H. An owner chooses report precision based on maximizing her expected utility, a portion of which is unobservable (to the analyst). For simplicity, we initially assume report precision is binary and observable to the analyst.

Base case

Focus attention on the treatment effect of report precision. To facilitate this exercise, we simulate data by drawing 200 samples of $2,000$ observations for normally distributed reports with mean μ and variance $\sigma_1^2 + \sigma_2^2$. Parameter values are tab-

ulated below

Base case parameter values
$\mu = 1,000$
$\sigma_1^2 = 100$
$\beta^L = \beta^H = \beta = 7$
$b = 150$
$\widehat{b} = 128.4$
$\gamma = 2.5$
$\alpha = 0.02$
$\alpha_d^L \sim N\left(0.02, 0.005^2\right)$
$\alpha_d^H \sim N\left(0.04, 0.01^2\right)$

The random α_d^j draws are not observed by firm owners until after their report precision choices are made.[14] On the other hand, the analyst observes α_d^j draws ex post but their mean is unknown.[15] The owner chooses inverse report precision (report variance) $\left\{\left(\sigma_2^L\right)^2 = 133.5, \left(\sigma_2^H\right)^2 = 131.7\right\}$ to maximize her expected utility given her type, $E\left[\alpha_d^L\right]$, or $E\left[\alpha_d^H\right]$.

The report variance choices described above are the Nash equilibrium strategies for the owner and investors. That is, for α_d^L, investors' conjecture $\left(\overline{\sigma}_2^L\right)^2 = 133.5$ and the owner's best response is $\left(\sigma_2^L\right)^2 = 133.5$. While for α_d^H, investors' conjecture $\left(\overline{\sigma}_2^H\right)^2 = 131.7$ and the owner's best response is $\left(\sigma_2^H\right)^2 = 131.7$. Hence, the owner's expected utility associated with low variance reports given α_d^L is $(EU_1 \mid D = 1) = 486.8$ while the owner's expected utility associated with high variance reports given α_d^L is lower, $(EU_0 \mid D = 1) = 486.6$. Also, the owner's expected utility associated with high variance reports given α_d^H is $(EU_0 \mid D = 0) = 487.1$ while the owner's expected utility associated with low variance reports given α_d^H is lower, $(EU_1 \mid D = 0) = 486.9$.

Even though treatment choice is driven by cost of transaction design, α_d, observable outcomes are traded values, P, and don't reflect cost of transaction design. To wit, the observed treatment effect on the treated is

$$\begin{aligned} TT &= \left(P^L \mid D = 1\right) - \left(P^H \mid D = 1\right) = (Y_1 \mid D = 1) - (Y_0 \mid D = 1) \\ &= \left(\mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\left(s^L - \mu\right) - \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\right) \\ &\quad - \left(\mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\left(s^H - \mu\right) - \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\right) \end{aligned}$$

Since $E\left[s^L - \mu\right] = E\left[s^H - \mu\right] = 0$,

$$E\left[TT\right] = ATT = 0$$

[14] For the simulation, type is drawn from a Bernoulli distribution with probability 0.5.

[15] Consequently, even if other parameters are observed by the analyst, there is uncertainty associated with selection due to α_d^j.

Also, the observed treatment effect on the untreated is

$$\begin{aligned} TUT &= \left(P^L \mid D=0\right) - \left(P^H \mid D=0\right) = \left(Y_1 \mid D=0\right) - \left(Y_0 \mid D=0\right) \\ &= \left(\mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}\left(s^L - \mu\right) - \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}\right) \\ &\quad - \left(\mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}\left(s^H - \mu\right) - \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}\right) \end{aligned}$$

and

$$E\left[TUT\right] = ATUT = 0$$

Therefore, the average treatment effect is

$$ATE = 0$$

However, the *OLS* estimand is

$$\begin{aligned} OLS &= E\left[\left(P^L \mid D=1\right) - \left(P^H \mid D=0\right)\right] \\ &= E\left[\left(Y_1 \mid D=1\right) - \left(Y_0 \mid D=0\right)\right] \\ &= \left(\mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}E\left[s^L - \mu\right] - \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\right) \\ &\quad - \left(\mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}E\left[s^H - \mu\right] - \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}\right) \\ &= \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2} - \beta\frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2} \end{aligned}$$

For the present example, the *OLS* bias is nonstochastic

$$\beta\left(\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2} - \frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\right) = -2.33$$

Suppose we employ a naive (unsaturated) regression model, ignoring the *OLS* bias,

$$E\left[Y \mid s, D\right] = \beta_0 + \beta_1 s + \beta_2 D$$

or even a saturated regression model that ignores the *OLS* bias

$$E\left[Y \mid s, D\right] = \beta_0 + \beta_1 s + \beta_2 Ds + \beta_3 D$$

where

$$D = \begin{array}{ll} 1 & \text{if } EU^L > EU^H \\ 0 & \text{if } EU^L < EU^H \end{array}$$

Table 10.29: Report precision OLS parameter estimates for binary base case

$statistic$	β_0	β_1	$\beta_2\,(estATE)$
$mean$	172.2	0.430	-2.260
$median$	172.2	0.430	-2.260
$std.dev.$	0.069	0.0001	0.001
$minimum$	172.0	0.430	-2.264
$maximum$	172.4	0.430	-2.257
$E[Y \mid D, s] = \beta_0 + \beta_1 s + \beta_2 D$			

Table 10.30: Report precision average treatment effect sample statistics for binary base case

$statistic$	ATT	$ATUT$	ATE
$mean$	0.024	-0.011	0.006
$median$	0.036	0.002	0.008
$std.dev.$	0.267	0.283	0.191
$minimum$	-0.610	-0.685	-0.402
$maximum$	0.634	0.649	0.516

$$\begin{aligned} EU^j &= \mu - \beta^j \frac{\sigma_1^2 \left(\bar{\sigma}_2^j\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^j\right)^2} - \gamma \frac{\sigma_1^4 \left(\sigma_1^2 + \left(\sigma_2^j\right)^2\right)}{\left(\sigma_1^2 + \left(\bar{\sigma}_2^j\right)^2\right)^2} \\ &\quad -\alpha \left(b - \left(\sigma_2^j\right)^2\right)^2 - E\left[\alpha_d^j\right] \left(\hat{b} - \left(\sigma_2^j\right)^2\right)^2 \end{aligned}$$

$$Y = DY^L + (1 - D)\, Y^H$$

$$Y^j = \mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^j\right)^2} \left(s^j - \mu\right) - \beta^j \frac{\sigma_1^2 \left(\bar{\sigma}_2^j\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^j\right)^2}$$

and

$$\begin{aligned} s &= Ds^L + (1 - D)\, s^H \\ s^j &\sim N\left(\mu, \sigma_1^2 + \left(\sigma_2^j\right)^2\right) \end{aligned}$$

for $j \in \{L, H\}$. Estimation results for the above naive regression are reported in table 10.29. Since this is simulation, we have access to the "missing" data and can provide sample statistics for average treatment effects. Sample statistics for standard average treatment effects, *ATE*, *ATT*, and *ATUT*, are reported in table 10.30. Estimation results for the above saturated regression are reported in table 10.31. As expected, the results indicate substantial *OLS* selection bias in both regressions. Clearly, to effectively estimate any treatment effect, we need to eliminate this *OLS* selection bias from outcome.

Table 10.31: Report precision saturated OLS parameter estimates for binary base case

$statistic$	β_0	β_1	β_2
$mean$	602.1	0.432	-0.003
$median$	602.1	0.432	0.003
$std.dev.$	0.148	0.000	0.000
$minimum$	601.7	0.432	-0.003
$maximum$	602.6	0.432	-0.003
$statistic$	$estATT$	$estATUT$	$\beta_3\,(estATE)$
$mean$	-2.260	-2.260	-2.260
$median$	-2.260	-2.260	-2.260
$std.dev.$	0.001	0.001	0.001
$minimum$	-2.264	-2.265	-2.264
$maximum$	-2.255	-2.256	-2.257
$E[Y \mid D, s] = \beta_0 + \beta_1 s + \beta_2 Ds + \beta_3 D$			

Adjusted outcomes

It's unusual to encounter *nonstochastic* selection bias.[16] Normally, nonstochastic bias is easily eliminated as it's captured in the intercept but here the selection bias is perfectly aligned with the treatment effect of interest. Consequently, we must decompose the two effects — we separate the selection bias from the treatment effect. Since the components of selection bias are proportional to the coefficients on the reports and these coefficients are consistently estimated when selection bias is nonstochastic, we can utilize the estimates from the coefficients on s^L and s^H. For example, the coefficient on s^L is $\omega_{s^L} = \frac{\sigma_1^2}{\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2}$. Then, $\left(\bar{\sigma}_2^L\right)^2 = \frac{\sigma_1^2\left(1-\omega_{s^L}\right)}{\omega_{s^L}}$ and $\frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2} = \omega_{s^L}\frac{\sigma_1^2\left(1-\omega_{s^L}\right)}{\omega_{s^L}} = \sigma_1^2\left(1-\omega_{s^L}\right)$. Hence, the *OLS* selection bias

$$bias = \beta\left(\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2} - \frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2}\right)$$

can be written

$$bias = \beta\sigma_1^2\left(\omega_{s^L} - \omega_{s^H}\right)$$

This decomposition suggests we work with adjusted outcome

$$Y' = Y - \beta\sigma_1^2\left(D\omega_{s^L} - (1-D)\,\omega_{s^H}\right)$$

[16] Like the asset revaluation setting (chapter 9), the explanation lies in the lack of common support for identifying counterfactuals. In this base case, cost of transaction design type (L or H) is a perfect predictor of treatment. That is, $\Pr(D = 1 \mid \text{type} = L) = 1$ and $\Pr(D = 1 \mid \text{type} = H) = 0$. In subsequent settings, parameter variation leads to common support and selection bias is resolved via more standard *IV* approaches.

The adjustment can be estimated as follows. Estimate ω_{s^L} and ω_{s^H} from the regression

$$E\left[Y \mid D, s^L, s^H\right] = \omega_0 + \omega_1 D + \omega_{s^L} D s^L + \omega_{s^H}\left(1 - D\right) s^H$$

Then, since

$$\begin{aligned} Y^j &= \mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^j\right)^2}\left(s^j - \mu\right) - \beta^j \frac{\sigma_1^2\left(\bar{\sigma}_2^j\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^j\right)^2} \\ &= \mu + \omega_{s^j}\left(s^j - \mu\right) - \beta^j \sigma_1^2\left(1 - \omega_{s^j}\right) \end{aligned}$$

we can recover the weight, $\omega = -\beta\sigma_1^2$, on $\left(1 - \omega_{s^j}\right)$ utilizing the "restricted" regression

$$\begin{aligned} & E\left[\begin{array}{c} Y - \omega_0 - \omega_{s^L} D\left(s^L - \mu\right) \\ -\omega_{s^H}\left(1 - D\right)\left(s^H - \mu\right) \end{array} \mid D, s^L, s^H, \omega_{s^L}, \omega_{s^H}\right] \\ = \;& \omega\left[D\left(1 - \omega_{s^L}\right) + \left(1 - D\right)\left(1 - \omega_{s^H}\right)\right] \end{aligned}$$

Finally, adjusted outcome is determined by plugging the estimates for ω, ω_{s^L}, and ω_{s^H} into

$$Y' = Y + \omega\left(D\omega_{s^L} - \left(1 - D\right)\omega_{s^H}\right)$$

Now, we revisit the saturated regression employing the adjusted outcome Y'.

$$E\left[Y' \mid D, s\right] = \beta_0 + \beta_1\left(s - \mu\right) + \beta_2 D\left(s - \mu\right) + \beta_3 D$$

The coefficient on D, β_2,estimates the average treatment effect. Estimation results for the saturated regression with adjusted outcome are reported in table 10.32.

As there is no residual uncertainty, response is homogeneous and the sample statistics for standard treatment effects, *ATE*, *ATT*, and *ATUT*, are of very similar magnitude — certainly within sampling variation. No residual uncertainty (in adjusted outcome) implies treatment is ignorable.

Heterogeneous response

Now, we explore a more interesting setting. Everything remains as in the base case except there is unobserved (by the analyst) variation in β the parameter controlling the discount associated with uncertainty in the buyer's ability to manage the assets. In particular, β^L, β^H are independent normally distributed with mean 7 and unit variance.[17] These β^L, β^H draws are observed by the owner in conjunction with the known mean for α_d^L, α_d^L when selecting report precision. In this setting, it is as if the owners choose equilibrium inverse-report precision, σ_2^L or σ_2^H, based on the combination of β^L and α_d^L or β^H and α_d^H with greatest expected utility.[18]

---

[17] Independent identically distributed draws of β for *L*-type and *H*-type firms ensure the variance-covariance matrix for the unobservables/errors is nonsingular.

[18] Notice the value of β does not impact the value of the welfare maximizing report variance. Therefore, the optimal inverse report precision choices correspond to $\left(\alpha, \gamma, E\left[\alpha_d^j\right]\right)$ as in the base case but the binary choice σ_2^L or σ_2^H does depend on β^j.

Table 10.32: Report precision adjusted outcome OLS parameter estimates for binary base case

$statistic$	β_0	β_1	β_2
$mean$	1000	0.432	−0.000
$median$	1000	0.432	0.000
$std.dev.$	0.148	0.000	0.001
$minimum$	999.6	0.432	−0.004
$maximum$	1001	0.432	0.003
$statistic$	$estATT$	$estATUT$	$\beta_3\,(estATE)$
$mean$	−0.000	−0.000	−0.000
$median$	0.000	−0.000	0.000
$std.dev.$	0.001	0.002	0.001
$minimum$	−0.004	−0.005	−0.004
$maximum$	0.005	0.004	0.003
$E[Y' \mid D, s] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 D$			

Therefore, unlike the base case, common support is satisfied, i.e., there are no perfect predictors of treatment, $0 < \Pr\left(D = 1 \mid \beta^j, \alpha_d^j\right) < 1$. Plus, the choice equation and price regressions have correlated, stochastic unobservables.[19] In fact, this correlation in the errors[20] creates a classic endogeneity concern addressed by Heckman [1974, 1975, 1978, 1979].

First, we define average treatment effect estimands for this heterogeneity setting, then we simulate results for various treatment effect identification strategies. The average treatment effect on the treated is

$$\begin{aligned} ATT &= E\left[Y_1 - Y_0 \mid D = 1, \beta^H, \beta^L\right] \\ &= E\left[\begin{array}{c} \mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\left(s^L - \mu\right) - \beta^L \frac{\sigma_1^2 \left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2} \\ - \left(\mu + \frac{\sigma_1^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\left(s^H - \mu\right) - \beta^H \frac{\sigma_1^2 \left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2}\right) \end{array}\right] \\ &= \left(\beta^H - \beta^L\right) \frac{\sigma_1^2 \left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2} \end{aligned}$$

[19] The binary nature of treatment may seem a bit forced with response heterogeneity. This could be remedied by recognizing that owners' treatment choice is continuous but observed by the analyst to be binary. In later discussions, we explore such a setting with a richer *DGP*.

[20] The two regression equations and the choice equation have trivariate normal error structure.

The average treatment effect on the untreated is

$$\begin{aligned} ATUT &= E\left[Y_1 - Y_0 \mid D=0, \beta^H, \beta^L\right] \\ &= E\left[\begin{array}{c} \mu + \frac{\sigma_1^2}{\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2}\left(s^L-\mu\right) - \beta^L \frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2} \\ -\left(\mu + \frac{\sigma_1^2}{\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2}\left(s^H-\mu\right) - \beta^H \frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2}\right) \end{array}\right] \\ &= \left(\beta^H - \beta^L\right)\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2} \end{aligned}$$

OLS

Our first simulation for this heterogeneous setting attempts to estimate average treatment effects via *OLS*

$$E\left[Y \mid s, D\right] = \beta_0 + \beta_1\left(s-\overline{s}\right) + \beta_2\left(s-\overline{s}\right)D + \beta_3 D$$

Following Wooldridge, the coefficient on D, β_3, is the model-based average treatment effect (under strong identification conditions). Throughout the remaining discussion $(s-\overline{s})$ is the regressor of interest (based on our structural model). The model-based average treatment effect on the treated is

$$estATT = \beta_3 + \frac{\sum_i D_i\left(s_i-\overline{s}\right)\beta_2}{\sum_i D_i}$$

and the model-based average treatment effect on the untreated is

$$estATUT = \beta_3 - \frac{\sum_i D_i\left(s_i-\overline{s}\right)\beta_2}{\sum_i \left(1-D_i\right)}$$

Simulation results, including model-based estimates and sample statistics for standard treatment effects, are reported in table 10.33. Average treatment effect sample statistics from the simulation for this binary heterogenous case are reported in table 10.34. Not surprisingly, *OLS* performs poorly. The key *OLS* identification condition is ignorable treatment but this is not sustained by the *DGP*. *OLS* model-based estimates of *ATE* are not within sampling variation of the average treatment effect. Further, the data are clearly heterogeneous and *OLS* (ignorable treatment) implies homogeneity.

IV approaches

Poor instruments

Now, we consider various *IV* approaches for addressing endogeneity. First, we explore various linear *IV* approaches. The analyst observes D and α_d^L if $D=1$

Table 10.33: Report precision adjusted outcome OLS parameter estimates for binary heterogeneous case

$statistic$	β_0	β_1	β_2
$mean$	634.2	0.430	-0.003
$median$	634.2	0.429	-0.007
$std.dev.$	1.534	0.098	0.137
$minimum$	629.3	0.197	-0.458
$maximum$	637.7	0.744	0.377
$statistic$	$\beta_3\,(estATE)$	$estATT$	$estATUT$
$mean$	-2.227	-2.228	-2.225
$median$	-2.236	-2.257	-2.207
$std.dev.$	2.208	2.210	2.207
$minimum$	-6.672	-6.613	-6.729
$maximum$	3.968	3.971	3.966
$E[Y \mid s, D] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) D + \beta_3 D$			

Table 10.34: Report precision average treatment effect sample statistics for binary heterogeneous case

$statistic$	ATE	ATT	$ATUT$
$mean$	0.189	64.30	-64.11
$median$	0.298	64.19	-64.10
$std.dev.$	1.810	1.548	1.462
$minimum$	-4.589	60.47	-67.80
$maximum$	4.847	68.38	-60.90

Table 10.35: Report precision poor 2SLS-IV estimates for binary heterogeneous case

$statistic$	β_0	β_1	β_2
$mean$	634.2	0.433	−0.010
$median$	634.4	0.439	−0.003
$std.dev.$	1.694	0.114	0.180
$minimum$	629.3	0.145	−0.455
$maximum$	638.2	0.773	0.507
$statistic$	$\beta_3\,(estATE)$	$estATT$	$estATUT$
$mean$	−2.123	−2.125	−2.121
$median$	−2.212	−2.217	−2.206
$std.dev.$	2.653	2.650	2.657
$minimum$	−7.938	−7.935	−7.941
$maximum$	6.425	6.428	6.423
$E[Y \mid s, D] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) D + \beta_3 D$			

or α_d^H if $D = 0$. Suppose the analyst employs $\alpha_d = D\alpha_d^L + (1 - D)\,\alpha_d^H$ as an "instrument." As desired, α_d is related to report precision selection, unfortunately α_d is not conditionally mean independent, $E\left[y^j \mid s, \alpha_d\right] \neq E\left[y^j \mid s\right]$. To see this, recognize the outcome errors are a function of β^j and while α_d^j and β^j are independent, only α_d and not α_d^j is observed. Since α_d and β^j are related through selection D, α_d is a poor instrument. Two stage least squares instrumental variable estimation (*2SLS-IV*) produces the results reported in table 10.35 where β_3 is the model estimate for *ATE*. These results differ little from the *OLS* results except the *IV* model-based interval estimates of the treatment effects are wider as is expected even of a well-specified *IV* model. The results serve as a reminder of how little consolation comes from deriving similar results from two or more poorly-specified models.

Weak instruments

Suppose we have a "proper" instrument z_α in the sense that z_α is conditional mean independent. For purposes of the simulation, we construct the instrument z_α as the residuals from a regression of α_d onto

$$U^L = -\left(\beta^L - E\left[\beta\right]\right)\left[D\frac{\sigma_1^2\left(\sigma_2^L\right)^2}{\sigma_1^2 + \left(\sigma_2^L\right)^2} + (1 - D)\,\frac{\sigma_1^2\left(\sigma_2^H\right)^2}{\sigma_1^2 + \left(\sigma_2^H\right)^2}\right]$$

and

$$U^H = -\left(\beta^H - E\left[\beta\right]\right)\left[D\frac{\sigma_1^2\left(\sigma_2^L\right)^2}{\sigma_1^2 + \left(\sigma_2^L\right)^2} + (1 - D)\,\frac{\sigma_1^2\left(\sigma_2^H\right)^2}{\sigma_1^2 + \left(\sigma_2^H\right)^2}\right]$$

But, we wish to explore the implications for treatment effect estimation if the instrument is only weakly related to treatment. Therefore, we create a noisy instrument by adding an independent normal random variable ε with mean zero and

Table 10.36: Report precision weak 2SLS-IV estimates for binary heterogeneous case

$statistic$	β_0	β_1	β_2
$mean$	628.5	-0.605	2.060
$median$	637.3	0.329	0.259
$std.dev.$	141.7	7.678	15.52
$minimum$	-856.9	-73.00	-49.60
$maximum$	915.5	24.37	153.0
$statistic$	$\beta_3\,(estATE)$	$estATT$	$estATUT$
$mean$	8.770	8.139	9.420
$median$	-6.237	-6.532	-6.673
$std.dev.$	276.8	273.2	280.7
$minimum$	-573.3	-589.4	-557.7
$maximum$	2769	2727	2818
$E[Y \mid s, D] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) D + \beta_3 D$			

standard deviation 0.1. This latter perturbation ensures the instrument is weak. This instrument $z_\alpha + \varepsilon$ is employed to generate model-based estimates of some standard treatment effects via *2SLS-IV*. Results are provided in table 10.36 where β_3 is the model estimate for *ATE*. The weak *IV* model-estimates are extremely noisy. Weak instruments frequently are suspected to plague empirical work. In a treatment effects setting, this can be a serious nuisance as evidenced here.

A stronger instrument

Suppose z_α is available and employed as an instrument. Model-based treatment effect estimates are reported in table 10.37 where β_3 is the model estimate for *ATE*. These results are far less noisy but nonetheless appear rather unsatisfactory. The results, on average, diverge from sample statistics for standard treatment effects and provide little or no evidence of heterogeneity. Why? As Heckman and Vytlacil [2005, 2007] discuss, it is very difficult to identify what treatment effect linear *IV* estimates and different instruments produce different treatment effects. Perhaps then, it is not surprising that we are unable to connect the *IV* treatment effect to *ATE*, *ATT*, or *ATUT*.

Propensity score as an instrument

A popular ignorable treatment approach implies homogeneous response[21] and uses the propensity score as an instrument. We estimate the propensity score via a probit regression of D onto instruments z_α and z_σ, where z_α is (as defined above) the residuals of $\alpha_d = D\alpha_d^L + (1 - D)\,\alpha_d^H$ onto U^L and U^H and z_σ is the residuals from a regression of $\sigma_2 = D\sigma_2^L + (1 - D)\,\sigma_2^H$ onto U^L and U^H. Now, use

[21] An exception, propensity score with heterogeneous response, is discussed in section 10.5.1. However, this *IV*-identification strategy doesn't accommodate the kind of unobservable heterogeneity present in this report precision setting.

Table 10.37: Report precision stronger 2SLS-IV estimates for binary heterogeneous case

$statistic$	β_0	β_1	β_2
$mean$	634.3	0.427	0.005
$median$	634.2	0.428	0.001
$std.dev.$	2.065	0.204	0.376
$minimum$	629.2	−0.087	−0.925
$maximum$	639.8	1.001	1.005
$statistic$	$\beta_3\,(estATE)$	$estATT$	$estATUT$
$mean$	−2.377	−2.402	−2.351
$median$	−2.203	−2.118	−2.096
$std.dev.$	3.261	3.281	3.248
$minimum$	−10.15	−10.15	−10.15
$maximum$	6.878	6.951	6.809
$E[Y \mid s, D] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) D + \beta_3 D$			

the estimated probabilities $m = \Pr(D = 1 \mid z_\alpha, z_\sigma)$ in place of D to estimate the treatment effects.

$$E[Y \mid s, D] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) m + \beta_3 m$$

Model-based estimates of the treatment effects are reported in table 10.38 with β_3 corresponding to *ATE*. These results also are very unsatisfactory and highly erratic. Poor performance of the propensity score *IV* for estimating average treatment effects is not surprising as the data are inherently heterogeneous and the key propensity score *IV* identification condition is ignorability of treatment.[22] Next, we explore propensity score matching followed by two *IV* control function approaches.

Propensity score matching

Propensity score matching estimates of average treatment effects are reported in table 10.39.[23] While not as erratic as the previous results, these results are also unsatisfactory. Estimated *ATT* and *ATUT* are the opposite sign of one another as expected but reversed of the underlying sample statistics (based on simulated counterfactuals). This is not surprising as ignorability of treatment is the key identifying condition for propensity score matching.

Ordinate IV control function

Next, we consider an ordinate control function *IV* approach. The regression is

$$E[Y \mid s, D, \phi] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 \phi (Z\theta) + \beta_4 D$$

[22] Ignorable treatment implies homogeneous response, $ATE = ATT = ATUT$, except for common support variations.

[23] Propensity scores within 0.02 are matched using Sekhon's [2008] matching **R** package.

Table 10.38: Report precision propensity score estimates for binary heterogeneous case

$statistic$	β_0	β_1	β_2	β_3
$mean$	634.4	0.417	0.024	−2.610
$median$	634.3	0.401	0.039	−2.526
$std.dev.$	1.599	0.151	0.256	2.075
$minimum$	630.9	−0.002	−0.617	−7.711
$maximum$	638.9	0.853	0.671	2.721
$statistic$	$estATE$	$estATT$	$estATUT$	
$mean$	−74.64	−949.4	−799.8	
$median$	7.743	−386.1	412.8	
$std.dev.$	1422	2400	1503	
$minimum$	−9827	−20650	57.75	
$maximum$	7879	−9.815	17090	
$E[Y \mid s, m] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) m + \beta_3 m$				

Table 10.39: Report precision propensity score matching estimates for binary heterogeneous case

$statistic$	$estATE$	$estATT$	$estATUT$
$mean$	−2.227	−39.88	35.55
$median$	−2.243	−39.68	35.40
$std.dev.$	4.247	5.368	4.869
$minimum$	−14.00	−52.00	23.87
$maximum$	12.43	−25.01	46.79

Table 10.40: Report precision ordinate control IV estimates for binary heterogeneous case

$statistic$	β_0	β_1	β_2	β_3
$mean$	598.6	0.410	0.030	127.6
$median$	598.5	0.394	0.049	127.1
$std.dev.$	3.503	0.139	0.237	12.08
$minimum$	590.0	0.032	−0.595	91.36
$maximum$	609.5	0.794	0.637	164.7
$statistic$	$\beta_4\,(estATE)$	$estATT$	$estATUT$	
$mean$	−2.184	33.41	−37.91	
$median$	−2.130	33.21	−37.83	
$std.dev.$	1.790	3.831	3.644	
$minimum$	−6.590	22.27	−48.56	
$maximum$	2.851	43.63	−26.01	
$E[Y \mid s, D, \phi] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 \phi (Z\theta) + \beta_4 D$				

and is estimated via *IV* where instruments $\{\iota, (s - \overline{s}), m (s - \overline{s}), \phi (Z\theta), m\}$ are employed and $m = \Pr\left(D = 1 \mid Z = \left[\begin{array}{cccc} \iota & z_\alpha & z_\sigma \end{array}\right]\right)$ is estimated via probit. *ATE* is estimated via β_4, the coefficient on D. Following the general *IV* identification of *ATT*, *ATT* is estimated as

$$estATT = \beta_4 + \frac{\sum D_i \beta_3 \phi (Z_i \theta)}{\sum D_i}$$

and *ATUT* is estimated as

$$estATUT = \beta_4 - \frac{\sum D_i \beta_3 \phi (Z_i \theta)}{\sum (1 - D_i)}$$

Simulation results are reported in table 10.40. The ordinate control function results are clearly the most promising so far but still underestimate the extent of heterogeneity. Further, an important insight is emerging. If we only compare *OLS* and *ATE* estimates, we might conclude endogeneity is a minor concern. However, estimates of *ATT* and *ATUT* and their support of self-selection clearly demonstrate the false nature of such a conclusion.

Inverse-Mills IV

Heckman's control function approach, utilizing inverse-Mills ratios as the control function for conditional expectations, employs the regression

$$\begin{aligned} E[Y \mid s, D, \lambda] &= \beta_0 + \beta_1 (1 - D)(s - \overline{s}) + \beta_2 D (s - \overline{s}) \\ &\quad + \beta_3 (1 - D) \lambda^H + \beta_4 D \lambda^L + \beta_5 D \end{aligned}$$

where $\overline{s}$ is the sample average of s, $\lambda^H = -\frac{\phi(Z\theta)}{1 - \Phi(Z\theta)}$, $\lambda^L = \frac{\phi(Z\theta)}{\Phi(Z\theta)}$, and θ is the estimated parameter vector from a probit regression of report precision choice D

Table 10.41: Report precision inverse Mills IV estimates for binary heterogeneous case

$statistic$	β_0	β_1	β_2	β_3	β_4
$mean$	603.2	0.423	0.433	−56.42	56.46
$median$	603.1	0.416	0.435	−56.72	56.63
$std.dev.$	1.694	0.085	0.089	2.895	2.939
$minimum$	598.7	0.241	0.188	−65.40	48.42
$maximum$	607.8	0.698	0.652	−47.53	65.59
$statistic$		$\beta_5\,(estATE)$	$estATT$	$estATUT$	
$mean$		−2.155	59.65	−64.14	
$median$		−2.037	59.59	−64.09	
$std.dev.$		1.451	2.950	3.039	
$minimum$		−6.861	51.36	−71.19	
$maximum$		1.380	67.19	−56.10	
$E[Y \mid s, D, \lambda] = \beta_0 + \beta_1 (1-D)(s-\overline{s}) + \beta_2 D (s-\overline{s})$ $+\beta_3 (1-D)\lambda^H + \beta_4 D\lambda^L + \beta_5 D$					

on $Z = \begin{bmatrix} \iota & z_\alpha & z_\sigma \end{bmatrix}$ (ι is a vector of ones). The coefficient on D, β_5, is the model-based estimate of the average treatment effect, *ATE*. The average treatment effect on the treated is estimated as

$$ATT = \beta_5 + (\beta_2 - \beta_1) E[s - \overline{s}] + (\beta_4 - \beta_3) E\left[\lambda^L\right]$$

While the average treatment effect on the untreated is estimated as

$$ATUT = \beta_5 + (\beta_2 - \beta_1) E[s - \overline{s}] + (\beta_4 - \beta_3) E\left[\lambda^H\right]$$

Simulation results including model-estimated average treatment effects on treated (*estATT*) and untreated (*estATUT*) are reported in table 10.41. The inverse-Mills treatment effect estimates correspond nicely with their sample statistics. Next, we explore a variation on treatment.

10.7.2 Continuous report precision but observed binary

Heterogeneous response

Now, suppose the analyst only observes high or low report precision but there is considerable variation across firms. In other words, wide variation in parameters across firms is reflected in a continuum of report precision choices.[24] Specifically, variation in the cost of report precision parameter α, the discount parameter associated with the buyer's uncertainty in his ability to manage the asset, β, and the

[24] It is not uncommon for analysts to observe discrete choices even though there is a richer underlying choice set. Any discrete choice serves our purpose here, for simplicity we work with the binary case.

owner's risk premium parameter γ produces variation in owners' optimal report precision $\frac{1}{\sigma_2}$.

Variation in α_d is again not observed by the owners prior to selecting report precision. However, α_d is observed ex post by the analyst where α_d^L is normally distributed with mean 0.02 and standard deviation 0.005, while α_d^H is normally distributed with mean 0.04 and standard deviation 0.01. There is unobserved (by the analyst) variation in β the parameter controlling the discount associated with uncertainty in the buyer's ability to manage the assets such that β is independent normally distributed with mean 7 and variance 0.2. Independent identically distributed draws of β are taken for *L*-type and *H*-type firms so that the variance-covariance matrix for the unobservables/errors is nonsingular. On the contrary, draws for "instruments" α (normally distributed with mean 0.03 and standard deviation 0.005) and γ (normally distributed with mean 5 and standard deviation 1) are not distinguished by type to satisfy *IV* assumptions. Otherwise, conditional mean independence of the outcome errors and instruments is violated.[25] For greater unobservable variation (that is, variation through the β term), the weaker are the instruments, and the more variable is estimation of the treatment effects. Again, endogeneity is a first-order consideration as the choice equation and price (outcome) regression have correlated, stochastic unobservables.

OLS

First, we explore treatment effect estimation via the following *OLS* regression

$$E\left[Y \mid s, D\right] = \beta_0 + \beta_1 \left(s - \overline{s}\right) + \beta_2 D \left(s - \overline{s}\right) + \beta_3 D$$

Simulation results are reported in table 10.42. Average treatment effect sample statistics from the simulation are reported in table 10.43. In this setting, *OLS* effectively estimates the average treatment effect, *ATE*, for a firm/owner drawn at random. This is readily explained by noting the sample statistic estimated by *OLS* is within sampling variation of the sample statistic for *ATE* but *ATE* is indistinguishable from zero. However, if we're interested in response heterogeneity and other treatment effects, *OLS*, not surprisingly, is sorely lacking. *OLS* provides inconsistent estimates of treatment effects on the treated and untreated and has almost no diagnostic power for detecting response heterogeneity — notice there is little variation in *OLS*-estimated *ATE*, *ATT*, and *ATUT*.

Propensity score as an instrument

Now, we estimate the propensity score via a probit regression of D onto instruments α and γ, and use the estimated probabilities

$$m = \Pr\left(D = 1 \mid z_\alpha, z_\sigma\right)$$

[25] As we discuss later, these conditions are sufficient to establish α and γ as instruments — though weak instruments.

Table 10.42: Continuous report precision but observed binary OLS parameter estimates

$statistic$	β_0	β_1	β_2
$mean$	634.3	0.423	0.004
$median$	634.3	0.425	0.009
$std.dev.$	1.486	0.096	0.144
$minimum$	630.7	0.151	−0.313
$maximum$	638.4	0.658	0.520
$statistic$	$\beta_3\,(estATE)$	$estATT$	$estATUT$
$mean$	−1.546	−1.544	−1.547
$median$	−1.453	−1.467	−1.365
$std.dev.$	2.083	2.090	2.078
$minimum$	−8.108	−8.127	−8.088
$maximum$	5.170	5.122	5.216
$E\left[Y \mid s, D\right] = \beta_0 + \beta_1\left(s - \overline{s}\right) + \beta_2 D\left(s - \overline{s}\right) + \beta_3 D$			

Table 10.43: Continuous report precision but observed binary average treatment effect sample statistics

$statistic$	ATE	ATT	$ATUT$
$mean$	0.194	64.60	−64.20
$median$	0.215	64.55	−64.18
$std.dev.$	1.699	1.634	1.524
$minimum$	−4.648	60.68	−68.01
$maximum$	4.465	68.70	−60.18

Table 10.44: Continuous report precision but observed binary propensity score parameter estimates

$statistic$	β_0	β_1	β_2	β_3
$mean$	612.2	0.095	0.649	42.80
$median$	619.9	0.309	0.320	24.43
$std.dev.$	248.2	4.744	9.561	499.2
$minimum$	-1693	-29.80	-46.64	-1644
$maximum$	1441	23.35	60.58	4661
$statistic$	$estATE$	$estATT$	$estATUT$	
$mean$	-1.558	-1.551	-1.565	
$median$	-1.517	-1.515	-1.495	
$std.dev.$	2.086	2.090	2.085	
$minimum$	-8.351	-8.269	-8.437	
$maximum$	5.336	5.300	5.370	
$E[Y \mid s,m] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) m + \beta_3 m$				

Table 10.45: Continuous report precision but observed binary propensity score matching parameter estimates

$statistic$	$estATE$	$estATT$	$estATUT$
$mean$	-1.522	-1.612	-1.430
$median$	-1.414	-1.552	-1.446
$std.dev.$	2.345	2.765	2.409
$minimum$	-7.850	-8.042	-8.638
$maximum$	6.924	9.013	4.906

in place of D to estimate the treatment effects.

$$E[Y \mid s,m] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) m + \beta_3 m$$

Model-based estimates of the treatment effects are reported in 10.44. These results again are very unsatisfactory and highly variable. As before, poor performance of the propensity score *IV* for estimating average treatment effects is not surprising as the data are inherently heterogeneous and the key propensity score *IV* identification condition is ignorability of treatment (conditional mean redundancy).

Propensity score matching

Propensity score matching estimates of average treatment effects are reported in table 10.45.[26] While not as erratic as the previous results, these results are also unsatisfactory. Estimated *ATT* and *ATUT* are nearly identical even though the data are quite heterogeneous. The poor performance is not surprising as ignorability

[26]Propensity scores within 0.02 are matched using Sekhon's [2008] **R** matching package. Other bin sizes (say, 0.01) produce similar results though fewer matches..

Table 10.46: Continuous report precision but observed binary ordinate control IV parameter estimates

$statistic$	β_0	β_1	β_2	β_3
$mean$	-11633	5.798	-10.68	30971
$median$	772.7	0.680	-0.497	-390.8
$std.dev.$	176027	36.08	71.36	441268
$minimum$	-2435283	-58.78	-663.3	-1006523
$maximum$	404984	325.7	118.6	6106127
$statistic$	$\beta_4\,(estATE)$	$estATT$	$estATUT$	
$mean$	-173.7	12181	-12505	
$median$	-11.21	-168.6	176.3	
$std.dev.$	1176	176015	175648	
$minimum$	-11237	-407049	-2431259	
$maximum$	2598	2435846	390220	
$E[Y \mid s, D, \phi] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 \phi (Z\theta) + \beta_4 D$				

of treatment (conditional stochastic independence, or at least, conditional mean independence) is the key identifying condition for propensity score matching.

Ordinate IV control

Now, we consider two *IV* approaches for addressing endogeneity. The ordinate control function regression is

$$E[Y \mid s, D, \phi] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 \phi (Z\theta) + \beta_4 D$$

and is estimated via *IV* where instruments

$$\{\iota, (s - \overline{s}), m (s - \overline{s}), \phi (Z\theta), m\}$$

are employed and

$$m = \Pr\left(D = 1 \mid Z = \left[\begin{array}{cccc} \iota & \alpha & \gamma \end{array}\right]\right)$$

is estimated via probit. *ATE* is estimated via β_4, the coefficient on D. Simulation results are reported in table 10.46. The ordinate control function results are inconsistent and extremely noisy. Apparently, the instruments, α and γ, are sufficiently weak that the propensity score is a poor instrument. If this conjecture holds, we should see similar poor results in the second *IV* control function approach as well.

Inverse-Mills IV

The inverse-Mills *IV* control function regression is

$$\begin{aligned} E[Y \mid s, D, \lambda] &= \beta_0 + \beta_1 (1 - D)(s - \overline{s}) + \beta_2 D (s - \overline{s}) \\ &\quad + \beta_3 D \lambda^H + \beta_4 (1 - D) \lambda^L + \beta_5 D \end{aligned}$$

Table 10.47: Continuous report precision but observed binary inverse Mills IV parameter estimates

$statistic$	β_0	β_1	β_2	β_3	β_4
$mean$	633.7	0.423	0.427	−0.926	−55.41
$median$	642.2	0.424	0.418	9.178	−11.44
$std.dev.$	198.6	0.096	0.106	249.9	407.9
$minimum$	−1141	0.152	0.164	−2228	−3676
$maximum$	1433	0.651	0.725	1020	1042

$statistic$	$\beta_5\,(estATE)$	$estATT$	$estATUT$
$mean$	43.38	−0.061	86.87
$median$	23.46	−16.03	17.39
$std.dev.$	504.2	399.1	651.0
$minimum$	−1646	−1629	−1663
$maximum$	12.50	3556	5867

$$E\left[Y \mid s, D, \lambda\right] = \beta_0 + \beta_1 \left(1 - D\right)\left(s - \overline{s}\right) + \beta_2 D\left(s - \overline{s}\right) + \beta_3 D\lambda^H + \beta_4 \left(1 - D\right)\lambda^L + \beta_5 D$$

where $\overline{s}$ is the sample average of s, $\lambda^H = -\frac{\phi(Z\theta)}{1-\Phi(Z\theta)}$, $\lambda^L = \frac{\phi(Z\theta)}{\Phi(Z\theta)}$, and θ is the estimated parameters from a probit regression of precision choice D on $Z = \begin{bmatrix} \iota & \alpha & \gamma \end{bmatrix}$ (ι is a vector of ones). The coefficient on D, β_5, is the estimate of the average treatment effect, *ATE*. The average treatment effect on the treated is estimated as

$$ATT = \beta_5 + \left(\beta_2 - \beta_1\right) E\left[s - \overline{s}\right] + \left(\beta_4 - \beta_3\right) E\left[\lambda^L\right]$$

While the average treatment effect on the untreated is estimated as

$$ATUT = \beta_5 + \left(\beta_2 - \beta_1\right) E\left[s - \overline{s}\right] + \left(\beta_4 - \beta_3\right) E\left[\lambda^H\right]$$

Simulation results including estimated average treatment effects on treated (*estATT*) and untreated (*estATUT*) are reported in table 10.47. While not as variable as ordinate control function model estimates, the inverse-Mills *IV* estimates are inconsistent and highly variable. It's likely, we are unable to detect endogeneity or diagnose heterogeneity based on this strategy as well.

The explanation for the problem lies with our supposed instruments, α and γ. Conditional mean independence may be violated due to variation in report precision or the instruments may be weak. That is, optimal report precision is influenced by variation in α and γ and variation in report precision is reflected in outcome error variation

$$U^L = -\left(\beta^L - E\left[\beta\right]\right)\left[D\frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2} + \left(1 - D\right)\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}\right]$$

and

$$U^H = -\left(\beta^H - E\left[\beta\right]\right)\left[D\frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^L\right)^2} + \left(1 - D\right)\frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2 + \left(\bar{\sigma}_2^H\right)^2}\right]$$

Table 10.48: Continuous report precision but observed binary sample correlations

$statistic$	$r(\alpha, U^L)$	$r(\alpha, U^H)$	$r(\gamma, U^L)$	$r(\gamma, U^H)$
$mean$	-0.001	-0.002	0.003	-0.000
$median$	-0.001	-0.004	0.003	0.001
$std.dev.$	0.020	0.024	0.023	0.024
$minimum$	-0.052	-0.068	-0.079	-0.074
$maximum$	0.049	0.053	0.078	0.060
$statistic$	$r(\alpha, D)$	$r(\gamma, D)$	$r(w_1, D)$	$r(w_2, D)$
$mean$	-0.000	0.001	-0.365	0.090
$median$	-0.001	0.003	-0.365	0.091
$std.dev.$	0.021	0.025	0.011	0.013
$minimum$	-0.046	-0.062	-0.404	0.049
$maximum$	0.050	0.075	-0.337	0.122

To investigate the poor instrument problem we report in table 10.48 sample correlation statistics $r(\cdot, \cdot)$ for α and γ determinants of optimal report precision with *unobservable* outcome errors U^L and U^H. We also report sample correlations between potential instruments, α, γ, w_1, w_2, and treatment D to check for weak instruments. The problem with the supposed instruments, α and γ, is apparently that they're weak and not that they're correlated with U^L and U^H. On the other hand, w_1 and w_2 (defined below) hold some promise. We experiment with these instruments next.

Stronger instruments

To further investigate this explanation, we employ stronger instruments, w_1 (the component of α_d independent of U^L and U^H) and w_2 (the component of $\sigma_2^D \equiv D\sigma_2^L + (1-D)\sigma_2^H$ independent of U^L and U^H),[27] and reevaluate propensity score as an instrument.[28]

Propensity score as an instrument. Now, we use the estimated probabilities

$$m = \Pr(D = 1 \mid w_1, w_2)$$

from the above propensity score in place of D to estimate the treatment effects.

$$E[Y \mid s, m] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s})\, m + \beta_3 m$$

Model-based estimates of the treatment effects are reported in table 10.49. These results again are very unsatisfactory and highly variable. As before, poor performance of the propensity score *IV* for estimating average treatment effects is not surprising as the data are inherently heterogeneous and the key propensity score

[27] For purposes of the simulation, these are constructed from the residuals of regressions of α_d and σ_2^D on unobservables U^H and U^L.

[28] A complementary possibility is to search for measures of nonpecuniary satisfaction as instruments. That is, measures which impact report precision choice but are unrelated to outcomes.

Table 10.49: Continuous report precision but observed binary stronger propensity score parameter estimates

$statistic$	β_0	β_1	β_2	β_3
$mean$	637.1	0.419	0.012	−7.275
$median$	637.1	0.419	−0.007	−7.215
$std.dev.$	2.077	0.203	0.394	3.455
$minimum$	631.8	−0.183	−0.820	−16.61
$maximum$	1441	23.35	60.58	4661
$statistic$	$estATE$	$estATT$	$estATUT$	
$mean$	−70.35	−99.53	−41.10	
$median$	−69.73	−97.19	−41.52	
$std.dev.$	12.92	21.04	7.367	
$minimum$	−124.0	−188.0	−58.59	
$maximum$	5.336	5.300	5.370	
$E[Y \mid s, m] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 (s - \overline{s}) m + \beta_3 m$				

Table 10.50: Continuous report precision but observed binary stronger propensity score matching parameter estimates

$statistic$	$estATE$	$estATT$	$estATUT$
$mean$	2.291	−7.833	13.80
$median$	2.306	−8.152	13.74
$std.dev.$	2.936	3.312	3.532
$minimum$	−6.547	−17.00	5.189
$maximum$	12.38	4.617	24.94

IV identification condition is ignorability of treatment (conditional mean independence).

Propensity score matching

Propensity score matching estimates of average treatment effects are reported in table 10.50.[29] While not as erratic as the previous results, these results are also unsatisfactory. Estimated *ATT* and *ATUT* are opposite their sample statistics. The poor performance is not surprising as ignorability of treatment is the key identifying condition for propensity score matching.

Ordinate IV control function. The ordinate control function regression is

$$E[Y \mid s, D, \phi] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 \phi (Z\theta) + \beta_4 D$$

and is estimated via *IV* where instruments

$$\{\iota, (s - \overline{s}), m (s - \overline{s}), \phi (Z\theta), m\}$$

[29] Propensity scores within 0.02 are matched.

Table 10.51: Continuous report precision but observed binary stronger ordinate control IV parameter estimates

$statistic$	β_0	β_1	β_2	β_3
$mean$	616.0	0.419	0.010	66.21
$median$	616.5	0.418	−0.006	65.24
$std.dev.$	7.572	0.202	0.381	24.54
$minimum$	594.0	−0.168	−0.759	1.528
$maximum$	635.5	0.885	1.236	147.3
$statistic$	$\beta_4\,(estATE)$	$estATT$	$estATUT$	
$mean$	−11.91	12.52	−36.35	
$median$	−11.51	12.31	−36.53	
$std.dev.$	4.149	7.076	$12,14$	
$minimum$	−24.68	−5.425	−77.47	
$maximum$	−2.564	32.37	−4.535	
$E[Y \mid s, D, \phi] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 \phi (Z\theta) + \beta_4 D$				

are employed and

$$m = \Pr\left(D = 1 \mid Z = \left[\begin{array}{ccc} \iota & w_1 & w_2 \end{array}\right]\right)$$

is estimated via probit. *ATE* is estimated via β_4, the coefficient on D. Simulation results are reported in table 10.51. The ordinate control function results are markedly improved relative to those obtained with poor instruments, α and γ. Model-estimated average treatment effects are biased somewhat toward zero. Nonetheless, the ordinate control *IV* approach might enable us to detect endogeneity via heterogeneity even though *OLS* and *ATE* are within sampling variation of one another. The important point illustrated here is that the effectiveness of *IV* control function approaches depend heavily on strong instruments. It's important to remember proper instruments in large part have to be evaluated ex ante — sample evidence is of limited help due to unobservability of counterfactuals.

Inverse-Mills IV

The inverse-Mills *IV* regression is

$$\begin{aligned} E[Y \mid s, D, \lambda] &= \beta_0 + \beta_1 (1 - D)(s - \overline{s}) + \beta_2 D (s - \overline{s}) \\ &\quad + \beta_3 D \lambda^H + \beta_4 (1 - D) \lambda^L + \beta_5 D \end{aligned}$$

where $\overline{s}$ is the sample average of s, $\lambda^H = -\frac{\phi(Z\theta)}{1-\Phi(Z\theta)}$, $\lambda^L = \frac{\phi(Z\theta)}{\Phi(Z\theta)}$, and θ is the estimated parameters from a probit regression of precision choice D on $Z = \left[\begin{array}{ccc} \iota & w_1 & w_2 \end{array}\right]$ (ι is a vector of ones). The coefficient on D, β_5, is the estimate of the average treatment effect, *ATE*. Simulation results including estimated average treatment effects on treated (*estATT*) and untreated (*estATUT*) are reported in table 10.52. While the inverse-Mills *IV* average treatment effect estimates come closest of any strategies (so far considered) to maintaining the

Table 10.52: Continuous report precision but observed binary stronger inverse Mills IV parameter estimates

$statistic$	β_0	β_1	β_2	β_3	β_4
$mean$	611.6	0.423	0.428	−32.03	80.04
$median$	611.5	0.431	0.422	−32.12	79.84
$std.dev.$	2.219	0.093	0.099	3.135	6.197
$minimum$	606.6	0.185	0.204	−41.47	62.39
$maximum$	617.5	0.635	0.721	−20.70	98.32

$statistic$	$\beta_5\,(estATE)$	$estATT$	$estATUT$
$mean$	−35.55	43.77	−114.8
$median$	−35.11	43.80	−114.7
$std.dev.$	3.868	4.205	8.636
$minimum$	−47.33	30.02	−142.0
$maximum$	−26.00	57.97	−90.55

$$E\left[Y \mid s, D, \lambda\right] = \beta_0 + \beta_1 \left(1 - D\right)\left(s - \overline{s}\right) + \beta_2 D\left(s - \overline{s}\right) + \beta_3 D\lambda^H + \beta_4 \left(1 - D\right)\lambda^L + \beta_5 D$$

spread between and direction of *ATT* and *ATUT*, all average treatment effect estimates are biased downward and the spread is somewhat exaggerated. Nevertheless, we are able to detect endogeneity and diagnose heterogeneity by examining estimated *ATT* and *ATUT*. Importantly, this derives from employing strong instruments, w_1 (the component of α_d independent of U^L and U^H) and w_2 (the component of $\sigma_2^D = D\sigma_2^L + (1 - D)\,\sigma_2^H$ independent of U^L and U^H). The next example reexamines treatment effect estimation in a setting where *OLS* and *ATE* differ markedly and estimates of *ATE* may help detect endogeneity.

Simpson's paradox

Suppose a firm's owner receives nonpecuniary and unobservable (to the analyst) satisfaction associated with report precision choice. This setting highlights a deep concern when analyzing data — perversely omitted, correlated variables which produce a Simpson's paradox result.

Consider α_d^L is normally distributed with mean 1.0 and standard deviation 0.25, while α_d^H is normally distributed with mean 0.04 and standard deviation 0.01.[30] As with β^j, these differences between *L* and *H*-type cost parameters are perceived or observed by the owner; importantly, β^L has standard deviation 2 while β^H has standard deviation 0.2 and each has mean 7. The unpaid cost of transaction design is passed on to the firm and its investors by *L*-type owners. Investors are aware of this (and price the firm accordingly) but the analyst is not (hence it's unobserved). *L*-type owners get nonpecuniary satisfaction from transaction design such that their personal cost is only 2% of $\alpha_d^L \left(\widehat{b} - \sigma_2^2\right)^2$, while *H*-type owners receive

[30] The labels seem reversed, but bear with us.

no nonpecuniary satisfaction — hence the labels.[31] Other features remain as in the previous setting. Accordingly, expected utility for *L*-type owners who choose treatment is

$$\begin{aligned} EU^L\left(\sigma_2^L\right) &= \mu - \beta^L \frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2} - \gamma \frac{\sigma_1^4\left(\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2\right)}{\left(\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2\right)^2} \\ &\quad -\alpha\left(b-\left(\sigma_2^L\right)^2\right)^2 - 0.02\alpha_d^L\left(\hat{b}-\left(\sigma_2^L\right)^2\right)^2 \end{aligned}$$

while expected utility for *H*-type owners who choose no treatment is

$$\begin{aligned} EU^H\left(\sigma_2^H\right) &= \mu - \beta^H \frac{\sigma_1^2\left(\bar{\sigma}_2^H\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2} - \gamma \frac{\sigma_1^4\left(\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2\right)}{\left(\sigma_1^2+\left(\bar{\sigma}_2^H\right)^2\right)^2} \\ &\quad -\alpha\left(b-\left(\sigma_2^H\right)^2\right)^2 - \alpha_d^H\left(\hat{b}-\left(\sigma_2^H\right)^2\right)^2 \end{aligned}$$

Also, outcomes or prices for owners who choose treatment include the cost of transaction design and accordingly are

$$Y^L = P\left(\bar{\sigma}_2^L\right) = \mu + \frac{\sigma_1^2}{\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2}\left(s^L-\mu\right) - \beta^L \frac{\sigma_1^2\left(\bar{\sigma}_2^L\right)^2}{\sigma_1^2+\left(\bar{\sigma}_2^L\right)^2} - \alpha_d^L\left(\hat{b}-\left(\sigma_2^L\right)^2\right)^2$$

OLS

An *OLS* regression is

$$E\left[Y \mid s, D\right] = \beta_0 + \beta_1\left(s-\overline{s}\right) + \beta_2 D\left(s-\overline{s}\right) + \beta_3 D$$

Simulation results are reported in table 10.53. The average treatment effect sample statistics from the simulation are reported in table 10.54. Clearly, *OLS* produces poor estimates of the average treatment effects. As other ignorable treatment strategies fair poorly in settings of rich heterogeneity, we skip propensity score strategies and move ahead to control function strategies.

Ordinate IV control

We consider two *IV* control function approaches for addressing endogeneity. An ordinate control function regression is

$$E\left[Y \mid s, D, \phi\right] = \beta_0 + \beta_1\left(s-\overline{s}\right) + \beta_2 D\left(s-\overline{s}\right) + \beta_3\phi\left(Z\theta\right) + \beta_4 D$$

[31] The difference in variability between β^L and β^H creates the spread between *ATE* and the effect estimated via *OLS* while nonpecuniary reward creates a shift in their mean outcomes such that *OLS* is positive and *ATE* is negative.

Table 10.53: Continuous report precision but observed binary OLS parameter estimates for Simpson's paradox DGP

$statistic$	β_0	β_1	β_2
$mean$	603.2	0.434	-0.014
$median$	603.2	0.434	-0.007
$std.dev.$	0.409	0.023	0.154
$minimum$	602.2	0.375	-0.446
$maximum$	604.4	0.497	0.443
$statistic$	$\beta_3\,(estATE)$	$estATT$	$estATUT$
$mean$	54.03	54.03	54.04
$median$	53.89	53.89	53.91
$std.dev.$	2.477	2.474	2.482
$minimum$	46.17	46.26	46.08
$maximum$	62.31	62.25	62.37
$E[Y \mid s, D] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 D$			

Table 10.54: Continuous report precision but observed binary average treatment effect sample statistics for Simpson's paradox DGP

$statistic$	ATE	ATT	$ATUT$
$mean$	-33.95	57.76	-125.4
$median$	-34.06	57.78	-125.4
$std.dev.$	2.482	2.386	2.363
$minimum$	-42.38	51.15	-131.3
$maximum$	-26.57	66.49	-118.5

Table 10.55: Continuous report precision but observed binary ordinate control IV parameter estimates for Simpson's paradox DGP

$statistic$	β_0	β_1	β_2	β_3
$mean$	561.0	0.441	−0.032	266.3
$median$	561.5	0.479	−0.041	263.7
$std.dev.$	9.703	0.293	0.497	31.41
$minimum$	533.5	−0.442	−1.477	182.6
$maximum$	585.7	1.305	1.615	361.5
$statistic$	$\beta_4\,(estATE)$	$estATT$	$estATUT$	
$mean$	−48.72	48.45	−145.6	
$median$	−49.02	47.97	−143.0	
$std.dev.$	8.190	10.43	16.58	
$minimum$	−71.88	21.53	−198.0	
$maximum$	−25.12	84.89	−99.13	
$E[Y \mid s, D, \phi] = \beta_0 + \beta_1 (s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 \phi (Z\theta) + \beta_4 D$				

and is estimated via *IV* where instruments

$$\{\iota, s, m (s - \overline{s}) m, \phi (Z\theta), m\}$$

are employed and

$$m = \Pr \left(D = 1 \mid Z = \left[\begin{array}{cccc} \iota & w_1 & w_2 \end{array} \right] \right)$$

is estimated via probit. *ATE* is estimated via β_4, the coefficient on D. Simulation results are reported in table 10.55. As expected, the ordinate control function fairs much better than *OLS*. Estimates of *ATUT* are biased somewhat away from zero and, as expected, more variable than the sample statistic, but estimates are within sampling variation. Nevertheless, the ordinate control *IV* model performs better than in previous settings. Next, we compare results with the inverse-Mills *IV* strategy.

Inverse-Mills IV

The inverse-Mills *IV* control function regression is

$$\begin{aligned} E[Y \mid s, D, \lambda] &= \beta_0 + \beta_1 (1 - D)(s - \overline{s}) + \beta_2 D (s - \overline{s}) \\ &\quad + \beta_3 (1 - D) \lambda^H + \beta_4 D \lambda^L + \beta_5 D \end{aligned}$$

where $\overline{s}$ is the sample average of s, $\lambda^H = -\frac{\phi(Z\theta)}{1-\Phi(Z\theta)}$, $\lambda^L = \frac{\phi(Z\theta)}{\Phi(Z\theta)}$, and θ is the estimated parameters from a probit regression of precision choice D on $Z = \left[\begin{array}{ccc} \iota & w_1 & w_2 \end{array} \right]$ (ι is a vector of ones). The coefficient on D, β_5, is the estimate of the average treatment effect, *ATE*. Simulation results including estimated average treatment effects on treated (*estATT*) and untreated (*estATUT*) are reported in table 10.56. As with the ordinate control function approach, inverse-Mills estimates of the treatment effects (especially *ATUT*) are somewhat biased

Table 10.56: Continuous report precision but observed binary inverse Mills IV parameter estimates for Simpson's paradox DGP

$statistic$	β_0	β_1	β_2	β_3	β_4
$mean$	603.3	0.434	0.422	0.057	182.8
$median$	603.2	0.434	0.425	0.016	183.0
$std.dev.$	0.629	0.023	0.128	0.787	11.75
$minimum$	601.1	0.375	0.068	−2.359	151.8
$maximum$	604.9	0.497	0.760	1.854	221.7

$statistic$	$\beta_5\,(estATE)$	$estATT$	$estATUT$
$mean$	−74.17	53.95	−201.9
$median$	−74.46	53.88	−201.3
$std.dev.$	8.387	2.551	16.58
$minimum$	−99.78	45.64	−256.7
$maximum$	−52.65	61.85	−159.1

$$E\left[Y \mid s, D, \lambda\right] = \beta_0 + \beta_1 (1 - D)(s - \overline{s}) + \beta_2 D (s - \overline{s}) + \beta_3 (1 - D)\lambda^H + \beta_4 D\lambda^L + \beta_5 D$$

away from zero and, as expected, more variable than the sample statistics. However, the model supplies strong evidence of endogeneity (*ATE* along with *ATT* and *ATUT* differ markedly from *OLS* estimates) and heterogeneous response ($ATE \neq ATT \neq ATUT$). Importantly, mean and median estimates reveal a Simpson's paradox result—*OLS* estimates a positive average treatment effect while endogeneity of selection produces a negative average treatment effect.[32]

10.7.3 Observable continuous report precision choice

Now we consider the setting where the analyst observes a continuum of choices based on the investors' (equilibrium) conjecture of the owner's report precision $\overline{\tau} = \frac{1}{\sigma_1^2 + \overline{\sigma}_2^2}$. This plays out as follows. The equilibrium strategy is the fixed point where the owner's expected utility maximizing report precision, $\frac{1}{\sigma^2} = \frac{1}{\sigma_1^2 + \sigma_2^2}$, equals investors' conjectured best response report precision, $\overline{\tau} = \frac{1}{\sigma_1^2 + \overline{\sigma}_2^2}$. Let conjectured report variance be denoted $\overline{\sigma}^2 \equiv \sigma_1^2 + \overline{\sigma}_2^2$. The owner's expected utility is

$$EU(\sigma_2) = \mu - \beta \frac{\sigma_1^2 \bar{\sigma}_2^2}{\sigma_1^2 + \bar{\sigma}_2^2} - \gamma \frac{\sigma_1^4 \left(\sigma_1^2 + \sigma_2^2\right)}{\left(\sigma_1^2 + \bar{\sigma}_2^2\right)^2} - \alpha \left(b - \sigma_2^2\right)^2 - \alpha_d \left(\hat{b} - \sigma_2^2\right)^2$$

[32] As noted previously, untabulated results using weak instruments (α and γ) reveal extremely erratic estimates of the treatment effects.

substitution of $\overline{\sigma}^2$ for $\sigma_1^2 + \overline{\sigma}_2^2$ yields

$$\begin{aligned} EU(\sigma_2) &= \mu - \beta\frac{\sigma_1^2\left(\bar{\sigma}^2 - \sigma_1^2\right)}{\bar{\sigma}^2} - \gamma\frac{\sigma_1^4\sigma^2}{\bar{\sigma}^4} \\ &\quad -\alpha\left(b - \sigma^2 + \sigma_1^2\right)^2 - \alpha_d\left(\hat{b} - \sigma^2 + \sigma_1^2\right)^2 \end{aligned}$$

The first order condition combined with the equilibrium condition is

$$\begin{aligned} \sigma^2 &= \frac{\alpha b + \alpha_d\widehat{b} - \gamma\frac{\sigma_1^4}{2\bar{\sigma}^4}}{\alpha + \alpha_d} \\ s.t.\ \sigma^2 &= \overline{\sigma}^2 \end{aligned}$$

As the outcome equation

$$\begin{aligned} Y &= P\left(\bar{\sigma}_2^2\right) = \mu + \frac{\sigma_1^2}{\sigma_1^2 + \bar{\sigma}_2^2}(s - \mu) - \beta\frac{\sigma_1^2\bar{\sigma}_2^2}{\sigma_1^2 + \bar{\sigma}_2^2} \\ &= P(\overline{\tau}) = \mu + \sigma_1^2(s - \mu)\overline{\tau} - \beta\sigma_1^2\left(1 - \sigma_1^2\overline{\tau}\right) \end{aligned}$$

is not directly affected by the owner's report precision choice (but rather by the conjectured report precision), we exploit the equilibrium condition to define an average treatment effect

$$ATE(\overline{\tau}) = E\left[\frac{\partial Y}{\partial\overline{\tau}}\right] = \beta\sigma_1^4$$

and an average treatment effect on the treated[33]

$$ATT(\overline{\tau}) = E\left[\frac{\partial Y}{\partial\overline{\tau}} \mid \overline{\tau} = \tau_j\right] = \beta_j\sigma_1^4$$

If β differs across firms, as is likely, the outcome equation

$$Y_j = \left[\mu - \beta_j\sigma_1^2\right] + \left[\sigma_1^2\right](s_j - \mu)\overline{\tau}_j + \left[\beta_j\sigma_1^4\right]\overline{\tau}_j$$

is a random coefficients model. And, if $\beta_j\sigma_1^4$ and $\overline{\tau}_j = \frac{1}{\sigma_1^2 + (\overline{\sigma}_{2j})^2}$ are related, then we're dealing with a correlated random coefficients model.

For our experiment, a simulation based on 200 samples of (balanced) panel data with $n = 200$ individuals and $T = 10$ periods (sample size, $nT = 2,000$) is employed. Three data variations are explored.

[33] As Heckman [1997] suggests the average treatment effect based on a random draw from the population of firms often doesn't address a well-posed economic question whether treatment is continuous or discrete.

Table 10.57: Continuous treatment OLS parameter estimates and average treatment effect estimates and sample statistics with only between individual variation

$statistic$	ω_0	ω_1	$\omega_2\,(estATE)$	ATE	$corr\,(\omega_{2i}, \overline{\tau}_i)$
$mean$	300.4	100.3	69916.	70002.	-0.001
$median$	300.4	100.3	69938.	70007.	0.002
$std.dev.$	7.004	1.990	1616	73.91	0.067
$minimum$	263.1	93.44	61945.	69779.	-0.194
$maximum$	334.9	106.2	78686.	70203.	0.140
$E\,[Y \mid s, \overline{\tau}] = \omega_0 + \omega_1\,(s - \overline{s})\,\overline{\tau} + \omega_2\overline{\tau}$					

Between individual variation

First, we explore a setting involving only variation in report precision between individuals. The following independent stochastic parameters characterize the data

Stochastic components	
parameters	number of draws
$\alpha \sim N\,(0.02, 0.005)$	n
$\alpha_d \sim N\,(0.02, 0.005)$	n
$\gamma \sim N\,(2.5, 1)$	n
$\beta \sim N\,(7, 0.1)$	n
$s \sim N\,(1000, \sigma)$	nT

where σ is the equilibrium report standard deviation; σ varies across firms but is constant through time for each firm.

First, we suppose treatment is ignorable and estimate the average treatment effect via *OLS*.

$$E\,[Y \mid s, \overline{\tau}] = \omega_0 + \omega_1\,(s - \mu)\,\overline{\tau} + \omega_2\overline{\tau}$$

Then, we accommodate unobservable heterogeneity (allow treatment and treatment effect to be correlated) and estimate the average treatment effect via *2SLS-IV*.

Hence, the *DGP* is

$$Y = 300 + 100\,(s - \mu)\,\overline{\tau} + (70,000 + \varepsilon_\beta)\,\overline{\tau}$$

where $\varepsilon_\beta = \beta_j - E\left[\beta_j\right] \sim N\,(0, 1), j = 1, \ldots, n.$

OLS

Results for *OLS* along with sample statistics for *ATE* and the correlation between treatment and treatment effect are reported in table 10.57 where ω_2 is the estimate of *ATE*. The *OLS* results correspond quite well with the *DGP* and the average treatment effect sample statistics. This is not surprising given the lack of correlation between treatment and treatment effect.

Table 10.58: Continuous treatment 2SLS-IV parameter and average treatment effect estimates with only between individual variation

$statistic$	ω_0	ω_1	$\omega_2\,(estATE)$
$mean$	300.4	100.3	69916.
$median$	300.4	100.2	69915.
$std.dev.$	7.065	1.994	1631
$minimum$	262.7	93.44	61308.
$maximum$	337.6	106.2	78781.
$E[Y \mid s, \overline{\tau}] = \omega_0 + \omega_1 (s - \overline{s}) \overline{\tau} + \omega_2 \overline{\tau}$			

2SLS-IV

On the other hand, as suggested by Wooldridge [1997, 2003], *2SLS-IV* consistently estimates *ATE* in this random coefficients setting. We employ the residuals from regressions of $(s - \mu)\,\overline{\tau}$ and $\overline{\tau}$ on U as instruments, z_1 and z_2; these are strong instruments. Results for *2SLS-IV* are reported in table 10.58. The *IV* results correspond well with the *DGP* and the sample statistics for *ATE*. Given the lack of correlation between treatment and treatment effect, it's not surprising that *IV* (with strong instruments) and *OLS* results are very similar.

Modest within individual variation

Second, we explore a setting involving within individual as well as between individuals report variation. Within individual variation arises through modest variation through time in the cost parameter associated with transaction design. The following independent stochastic parameters describe the data

Stochastic components	
parameters	number of draws
$\alpha \sim N(0.02, 0.005)$	n
$\alpha_d \sim N(0.02, 0.0005)$	nT
$\gamma \sim N(2.5, 1)$	n
$\beta \sim N(7, 0.1)$	n
$\beta_i = \beta + N(0, 0.0001)$	nT
$s \sim N(1000, \sigma)$	nT

where σ is the equilibrium report standard deviation; σ varies across firms and through time for each firm and unobserved β_i produces residual uncertainty.

OLS

This setting allows identification of ATE and ATT where $ATT\,(\overline{\tau} = median\,[\overline{\tau}])$. First, we estimate the average treatment effects via *OLS* where individual specific intercepts and slopes are accommodated.

$$E[Y \mid s_i, \overline{\tau}_i] = \sum_{i=1}^{n} \omega_{0i} + \omega_{1i} (s_i - \mu) \overline{\tau}_i + \omega_{2i} \overline{\tau}_i$$

Table 10.59: Continuous treatment OLS parameter and average treatment effect estimates for modest within individual report precision variation setting

$statistic$	$estATE$	$estATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$
$mean$	70306.	70152.
$median$	70193.	70368.
$std.dev.$	4625.	2211.
$minimum$	20419.	64722.
$maximum$	84891.	75192.
$E\left[Y \mid s_i, \overline{\tau}_i\right] = \sum_{i=1}^{n} \omega_{0i} + \omega_{1i}\left(s_i - \mu\right)\overline{\tau}_i + \omega_{2i}\overline{\tau}_i$		

Table 10.60: Continuous treatment ATE and ATT sample statistics and correlation between treatment and treatment effect for modest within individual report precision variation setting

$statistic$	ATE	$ATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$	$corr\left(\omega_{2it}, \overline{\tau}_{it}\right)$
$mean$	70014.	70026	-0.0057
$median$	70014.	69993	-0.0063
$std.dev.$	65.1	974.	0.072
$minimum$	69850.	67404	-0.238
$maximum$	70169.	72795	0.173

We report the simple average of ω_2 for $estATE$, and ω_{2i} for the median (of average $\overline{\tau}_i$ by individuals) as $estATT$ in table 10.59. That is, we average $\overline{\tau}_i$ for each individual, then select the median value of the individual averages as the focus of treatment on treated. Panel data allow us to focus on the average treatment effect for an individual but the median reported almost surely involves different individuals across simulated samples.

Sample statistics for *ATE* and $ATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$ along with the correlation between treatment and the treatment effect are reported in table 10.60. There is good correspondence between the average treatment effect estimates and sample statistics. The interval estimates for *ATT* are much tighter than those for *ATE*. Correlations between treatment and treatment effect suggest there is little to be gained from *IV* estimation. We explore this next.

2SLS-IV

Here, we follow Wooldridge [1997, 2003], and estimate average treatment effects via *2SLS-IV* in this random coefficients setting. We employ the residuals from regressions of $\left(s - \mu\right)\overline{\tau}$ and $\overline{\tau}$ on U as strong instruments, z_1 and z_2. Results for *2SLS-IV* are reported in table 10.61. The *IV* results correspond well with the *DGP* and the sample statistics for the average treatment effects. Also, as expected given the low correlation between treatment and treatment effect, *IV* produces similar results to those for *OLS*.

Table 10.61: Continuous treatment 2SLS-IV parameter and average treatment effect estimates for modest within individual report precision variation setting

$statistic$	$estATE$	$estATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$
$mean$	69849.	70150.
$median$	70096.	70312.
$std.dev.$	5017	2210
$minimum$	35410.	64461.
$maximum$	87738.	75467.
$E\left[Y \mid s_i, \overline{\tau}_i\right] = \sum_{i=1}^{n} \omega_{0i} + \omega_{1i}\left(s_i - \mu\right)\overline{\tau}_i + \omega_{2i}\overline{\tau}_i$		

More variation

Finally, we explore a setting with greater between individuals report variation as well as continued within individual variation. The independent stochastic parameters below describe the data

Stochastic components	
parameters	number of draws
$\alpha \sim N\left(0.02, 0.005\right)$	n
$\alpha_d \sim N\left(0.02, 0.0005\right)$	nT
$\gamma \sim N\left(2.5, 1\right)$	n
$\beta \sim N\left(7, 1\right)$	n
$\beta_i = \beta + N\left(0, 0.001\right)$	nT
$s \sim N\left(1000, \sigma\right)$	nT

where σ is the equilibrium report standard deviation; σ varies across firms and through time for each firm and greater unobserved β_i variation produces increased residual uncertainty.

OLS

This setting allows identification of ATE and ATT where $ATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$. First, we estimate the average treatment effects via *OLS* where individual specific intercepts and slopes are accommodated.

$$E\left[Y \mid s_i, \overline{\tau}_i\right] = \sum_{i=1}^{n} \omega_{0i} + \omega_{1i}\left(s_i - \mu\right)\overline{\tau}_i + \omega_{2i}\overline{\tau}_i$$

We report the simple average of ω_{2i} for $estATE$ and ω_{2i} for the median of average $\overline{\tau}_i$ by individuals as $estATT$ in table 10.62.

Sample statistics for *ATE* and $ATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$ along with the correlation between treatment and the treatment effect are reported in table 10.63. As expected with greater residual variation, there is weaker correspondence between the average treatment effect estimates and sample statistics. Correlations between treatment and treatment effect again suggest there is little to be gained from *IV* estimation. We explore *IV* estimation next.

Table 10.62: Continuous treatment OLS parameter and average treatment effect estimates for the more between and within individual report precision variation setting

$statistic$	$estATE$	$estATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$
$mean$	71623.	67870.
$median$	70011.	68129.
$std.dev.$	34288.	22360.
$minimum$	$-20220.$	$-8934.$
$maximum$	223726.	141028.
$E\left[Y \mid s_i, \overline{\tau}_i\right] = \sum_{i=1}^{n} \omega_{0i} + \omega_{1i}\left(s_i - \mu\right)\overline{\tau}_i + \omega_{2i}\overline{\tau}_i$		

Table 10.63: Continuous treatment ATE and ATT sample statistics and correlation between treatment and treatment effect for the more between and within individual report precision variation setting

$statistic$	ATE	$ATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$	$corr\left(\omega_{2it}, \overline{\tau}_{it}\right)$
$mean$	69951.	69720.	-0.0062
$median$	69970.	70230.	-0.0129
$std.dev.$	709.	10454.	0.073
$minimum$	67639.	34734	-0.194
$maximum$	71896.	103509	0.217

2SLS-IV

Again, we follow Wooldridge's [1997, 2003] random coefficients analysis, and estimate average treatment effects via *2SLS-IV*. We employ the residuals from regressions of $(s - \mu)\,\overline{\tau}$ and $\overline{\tau}$ on U as strong instruments, z_1 and z_2. Results for *2SLS-IV* are reported in table 10.64. The *IV* results are similar to those for *OLS* as expected given the near zero correlation between treatment and treatment effect.

Table 10.64: Continuous treatment 2SLS-IV parameter and average treatment effect estimates for the more between and within individual report precision variation setting

$statistic$	$estATE$	$estATT\left(\overline{\tau} = median\left[\overline{\tau}\right]\right)$
$mean$	66247.	67644.
$median$	68998.	68004.
$std.dev.$	36587	22309.
$minimum$	$-192442.$	$-9387.$
$maximum$	192722.	141180.
$E\left[Y \mid s_i, \overline{\tau}_i\right] = \sum_{i=1}^{n} \omega_{0i} + \omega_{1i}\left(s_i - \mu\right)\overline{\tau}_i + \omega_{2i}\overline{\tau}_i$		

10.8 Summary

This chapter has surveyed some *IV* approaches for identifying and estimating average treatment effects and illustrated them in a couple of ways. The Tuebingen-style examples illustrate critical features for *IV* identification then we added accounting context. The endogenous selection of report precision examples highlight several key features in the econometric analysis of accounting choice. First, reliable results follow from carefully linking theory and data. For instance, who observes which data is fundamental. When the analysis demands instruments (ignorable treatment conditions are typically not satisfied by the data in this context), their identification and collection is critical. Poor instruments (exclusion restriction fails) or weak instruments (weakly associated with selection) can lead to situations where the "cure" is worse than the symptom. *IV* results can be less reliable (more prone to generate logical inconsistencies) than *OLS* when faced with endogeneity if we employ faulty instruments. Once again, we see there is no substitute for task-appropriate data. Finally, two (or more) poor analyses don't combine to produce one satisfactory analysis.

10.9 Additional reading

Wooldridge [2002] (chapter 18 is heavily drawn upon in these pages), Amemiya [1985, chapter 9], and numerous other econometric texts synthesize *IV* treatment effect identification strategies. Recent volumes of *Handbook of Econometrics* (especially volumes 5 and 6b) report extensive reviews as well as recent results.