University of California, Berkeley
Department of Economics

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ECON 142

SKETCH OF SOLUTIONS FOR APPLIED EXERCISE #3

A. The difference in mean birth weights of infants of smoking and non-smoking mothers provides the average treatment effect if maternal smoking is "randomly assigned". That is, smoking and non-smoking mothers do not differ in other ways that affect the birth weight of their child.[1]

More formally, let y_{1i} be the birth weight for infant i if his mother smoked during pregnancy and y_{0i} be his birth weight if his mother didn't smoke. Then the average treatment effect (ATE) of maternal smoking is defined to be $ATE \equiv E(y_{1i}-y_{0i})$. The fundamental problem of causal inference is that one cannot observe both the factual and "counterfactual" states for infant i at the same time. Thus, the observed difference in average birth weights provides a consistent estimate of ATE if and only if nonsmoking moms provide a valid counterfactual for smoking moms. That is, the potential outcomes are independent of smoking status, and the effect of smoking is additive and homogeneous. This will be true if smoking is randomly assigned.

Evidence against the random assignment assumption is found in the fact that the other characteristics of the mother on which we have data also differ by smoking habits, particularly things that we think might directly or indirectly affect the health of the child. Here is the table of sample means by smoking status:

Table 1: Sample Means by Smoking Status

VARIABLE	Mean of Non-Smokers	Mean of Smokers	Difference	T-STAT
dmage	27.23	25.37	-1.86	49.85
dmeduc	13.12	11.87	-1.25	85.42
dmar	0.18	0.41	0.23	83.52
dlivord	1.93	2.10	0.17	20.71
nprevist	11.16	10.25	-0.91	37.29
disllb	24.54	29.40	4.87	21.32
dfage	29.54	28.35	-1.18	27.48
dfeduc	13.25	12.04	-1.22	78.14
anemia	0.01	0.02	0.01	11.21
diabete	0.02	0.02	0.00	1.89
phyper	0.03	0.02	-0.01	9.63
pre4000	0.01	0.01	-0.01	8.54
preterm	0.01	0.03	0.02	18.44
alcohol	0.01	0.05	0.04	49.49
drink	0.02	0.18	0.17	36.92

[1] Another implicit assumption is that the effect of smoking is constant and additive (i.e., homogeneous treatment effects). This sample is the universe of Pennsylvania mothers giving birth in 1989.

foreignb	0.05	0.02	-0.03	23.75
plural	0.02	0.01	0.00	2.95
deadkids	0.33	0.44	0.11	22.42
mblack	0.11	0.14	0.03	13.43
motherr	0.02	0.00	-0.01	16.13
mhispan	0.03	0.02	-0.01	8.75
fblack	0.12	0.15	0.04	16.80
fotherr	0.02	0.00	-0.01	15.90
fhispan	0.03	0.02	0.00	4.03
tripre1	0.84	0.72	-0.12	45.06
tripre2	0.13	0.21	0.08	34.43
tripre3	0.03	0.05	0.02	16.61
tripre0	0.01	0.02	0.02	23.97
first	0.44	0.38	-0.06	17.82

Tobacco-smoking mothers have 0.9 fewer pre-natal care visits, 1.2 fewer years of education, consume 0.4 more alcoholic drinks per week and are more than twice as likely to be single mothers. All of these differences are highly significant.

Notwithstanding this evidence, the estimate of the average treatment effect given by the assumption of random assignment is minus 258 grams. Smoking mothers have babies that weigh 258 grams less than the babies of non-smoking mothers, on average.

B. If smoking were randomly assigned to mothers conditional on our observables, then unobserved determinants of birth weight would be independent of (uncorrelated with) maternal smoking conditional on these observables.

(1) $Y_i = \theta T_i + X_i'\beta + u_i$

Using the linear specification for the covariates as written in (1), we are saying $\text{cov}(u_i, T_i) = 0$. This identifies the true average treatment effect only when the conditional mean of birth weight is linear and additive in our covariates and when unobserved determinants of birth weight are uncorrelated with smoking (T_i) conditional on the linear regression function. That is, $E(T_i u_i | X_i'\beta)=0$.

Linear specification (1) produces an estimate of the impact of maternal smoking of minus 218g.

```
. reg dbirwt dmage-dfeduc  anemia-first

Source       SS        df       MS          Number of obs =  139149
                                             F( 29,139119) =  955.87
Model    7.8142e+09 29     269456433        Prob > F      =  0.0000
Residual 3.9217e+10 139119 281897.905       R-squared     =  0.1661
                                            Adj R-squared =  0.1660
Total    4.7032e+10 139148 337996.89        Root MSE      =  530.94

dbirwt       Coef.    Std. Err.      t    P>|t|     [95% Conf. Interval]

dmage     -2.833211   .4700512    -6.03   0.000    -3.754503    -1.91192
dmeduc     5.567311   .9308191     5.98   0.000     3.742923    7.391699
dmar      -38.41732   4.402929    -8.73   0.000    -47.04698   -29.78766
```

dlivord	33.65949	1.900515	17.71	0.000	29.93452	37.38446
nprevist	31.9865	.4873836	65.63	0.000	31.03124	32.94176
disllb	-.1827473	.0617356	-2.96	0.003	-.3037479	-.0617466
dfage	-.231785	.346988	-0.67	0.504	-.9118748	.4483049
dfeduc	4.046349	.8357826	4.84	0.000	2.408231	5.684467
anemia	-37.52513	13.48443	-2.78	0.005	-63.95437	-11.09589
diabete	45.29752	11.22951	4.03	0.000	23.28789	67.30716
phyper	-171.1969	8.591369	-19.93	0.000	-188.0359	-154.358
pre4000	468.4053	13.348	35.09	0.000	442.2435	494.5672
preterm	-522.4339	11.57709	-45.13	0.000	-545.1248	-499.743
tobacco	-218.2253	3.888441	-56.12	0.000	-225.8466	-210.6041
alcohol	-24.48929	13.77647	-1.78	0.075	-51.49091	2.512326
drink	-7.889139	2.679653	-2.94	0.003	-13.14121	-2.63707
foreignb	-12.48429	8.599005	-1.45	0.147	-29.33818	4.369599
plural	-954.0265	11.55229	-82.58	0.000	-976.6688	-931.3842
deadkids	-12.17768	2.021694	-6.02	0.000	-16.14016	-8.215199
mblack	-155.3402	12.17387	-12.76	0.000	-179.2007	-131.4796
motherr	-79.95507	19.28692	-4.15	0.000	-117.7571	-42.15308
mhispan	-60.51458	13.76448	-4.40	0.000	-87.49269	-33.53647
fblack	-55.73426	11.91465	-4.68	0.000	-79.08674	-32.38178
fotherr	-107.7197	19.13711	-5.63	0.000	-145.2281	-70.21135
fhispan	-72.80703	12.77849	-5.70	0.000	-97.85262	-47.76144
tripre1	-191.3085	9.165144	-20.87	0.000	-209.272	-173.345
tripre2	-116.2427	9.24364	-12.58	0.000	-134.3601	-98.12536
tripre0	-230.2495	16.08804	-14.31	0.000	-261.7818	-198.7173
first	-85.2064	5.046445	-16.88	0.000	-95.09734	-75.31546
_cons	3242.559	15.18014	213.61	0.000	3212.806	3272.312

C. Even if we are willing to make the (strong) assumption that smoking is randomly assigned conditional on the variables we have, a remaining source of bias in the estimate in part (B) is that the conditional mean of birth weight isn't necessarily *linear* in these controls. Excluded nonlinear terms may influence maternal smoking and birth weight, producing biased estimates of the effect of maternal smoking. In particular, random assignment conditional on the observables implies that $E(T_i u_i | X_i)=0$, which is much more general than the assumption underlying linear regression, $E(T_i u_i | X_i'\beta)=0$.

   Since we don't know the form of the conditional mean, a more general specification that uses higher order terms (squares, cubes, etc.) and interactions between variables may capture some of these missing effects. As we add terms to our model, we effectively compare outcomes of smokers and non-smokers with more and more similar observable characteristics. (In the most extreme form of multivariate matching, only mothers with *identical* covariates[2] are compared.) The potential benefit of this approach is a reduction in the bias in our estimated average treatment effect. By comparing mothers with very similar observable characteristics, we hope also to reduce the influence of unobserved factors that may affect maternal smoking and birth weight.

   A drawback to this approach is that how one allows the X's to enter is somewhat arbitrary and involves an aspect of data mining. Further, as we absorb residual variation with more covariates, we also reduce variation in our treatment (maternal smoking), rendering our estimates less precise. As the number of covariates, hence the number of things we must match on, increases, this problem becomes worse (the "curse of dimensionality.") If the additional control variables are unimportant, the net result may be to increase the sampling errors without changing the coefficient estimate. There would be additional concern if we had reason to believe that some mothers misreport their smoking status – overfitting models or comparing small numbers of observations with similar characteristics in this case could exacerbate bias due to measurement error.

[2] covariate is another term for control.

Since we don't know the correct functional form, it is probably most appropriate to try a number of models and compare estimates. Estimates may vary depending on which control terms are included in the regression. An estimate that controls for a similar set of covariates as used to estimate the p-score in part E is minus 208g.

D. **The propensity score method** summarizes the effect of the observable controls in a single index of the probability of being a maternal smoker conditional on the observables. This method is suggested by the propensity score theorem which states (in this case) that under the assumption that maternal smoking is independent of the child's birth weight (conditional on observables), the conditional probability of smoking is a sufficient control for these other determinants of birth weight.

More formally, if smoking is random conditional on the observables, then smoking will also be independent of the potential outcomes conditional on the propensity score.
$(y_{0i}, y_{1i}) \perp\!\!\!\perp T_i|X_i \Rightarrow (y_{0i}, y_{1i}) \perp\!\!\!\perp T_i|p(X_i)$,
where $p(X_i) = P(T_i=1|X_i) = E(T_i|X_i)$ is the probability of smoking conditional on the observables. It reduces the dimensionality problem of multivariate matching since instead of matching on all of the K covariates in "K-dimensional space", one just needs to match on the single variable $p(X_i)$.

The p-score approach can be implemented in two steps. In the first step, a logit, usually, is used to estimate the relationship between the probability of smoking and the observables. In the second step, the effect of smoking is estimated controlling for the estimated propensity score. The purpose of this approach is to "balance" the control variables in the treatment and control groups -- that is, smoking and non-smoking mothers with similar p-scores should have similar means values for their covariates. This suggests an algorithm for estimating the propensity score in the first stage:

(i) start with a parsimonious logit (e.g., just the additive terms of the control variables and highly significant nonlinear terms) and save the predicted probabilities from the logit.
(ii) stratify the data into quantile blocks of the estimated propensity score from the logit.
(iii) compare the means of the X's in the treatment and control groups by quantile block using t-tests or F-tests of the significance of the difference in means between the two groups.
- if the covariates are balanced between the two groups for each block, stop.
- if covariate k is not balanced for all blocks, modify the logit by adding higher order terms or interactions of covariate k and reevaluate the balance of the covariates in each quintile block.

We then control for this estimated propensity score either linearly or non-linearly:

(2) $Y_i = \theta T_i + f(\hat{p}_i \mid X_i) + u_i$

where $\hat{p}_i \mid X_i$ is the p-score estimated from the logit and f() is some unknown function. This method reduces all of the information in the covariates to a single index – the propensity score. However, a priori we know neither the correct specification of the logit nor the correct specification for the p-score in the second-stage estimation equation, f().

E.

i. <u>The Model</u>. Results will vary by your choice of model for the p-score. Justin used the following model:

```
. logit tobacco dmage-dfeduc anemia-preterm alcohol-first
```

```
dmage2-dlivord5 disllb2-deadkids2  dmelivord-dmadblack

Iteration 0:   log likelihood = -66979.129
Iteration 1:   log likelihood = -58362.273
Iteration 2:   log likelihood = -57213.79
Iteration 3:   log likelihood = -57136.665
Iteration 4:   log likelihood = -57134.566
Iteration 5:   log likelihood = -57134.561
Iteration 6:   log likelihood = -57134.561

Logit estimates                           Number of obs   =     139149
                                          LR chi2(67)     =    19689.13
                                          Prob > chi2     =     0.0000
Log likelihood = -57134.561               Pseudo R2       =     0.1470

------------------------------------------------------------------------------
tobacco       Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
dmage        7.975046   1.287367     6.19   0.000     5.451853    10.49824
dmeduc      -4.587142   .4319276   -10.62   0.000    -5.433704   -3.740579
dmar         .7125635   .1848358     3.86   0.000      .350292    1.074835
dlivord     -1.104287   .7058892    -1.56   0.118    -2.487805    .2792305
nprevist    -.0176662   .0025338    -6.97   0.000    -.0226323   -.0127001
disllb      -.0209374   .0051904    -4.03   0.000    -.0311103   -.0107645
dfage        1.316713   .2494535     5.28   0.000     .8277929    1.805633
dfeduc      -1.400281   .3103197    -4.51   0.000    -2.008497   -.7920658
anemia       .3080614   .061466      5.01   0.000     .1875903    .4285325
diabete      .0490041   .0605422     0.81   0.418    -.0696564    .1676647
phyper      -.4560633   .0510239    -8.94   0.000    -.5560682   -.3560583
pre4000     -.7851804   .0880387    -8.92   0.000     -.957733   -.6126278
preterm      .512701    .0516189     9.93   0.000     .4115297    .6138723
alcohol      .8795961   .1526641     5.76   0.000     .5803799    1.178812
drink        .6735509   .124851      5.39   0.000     .4288476    .9182543
foreignb    -.639313    .0592298   -10.79   0.000    -.7554012   -.5232248
plural      -.1600877   .0644401    -2.48   0.013     -.286388   -.0337873
deadkids     .253575    .0181218    13.99   0.000     .2180568    .2890932
mblack      -.171247    .0724286    -2.36   0.018    -.3132045   -.0292895
motherr     -.3560628   .1352845    -2.63   0.008    -.6212156     -.09091
mhispan     -.8318565   .0771644   -10.78   0.000    -.9830959   -.6806171
fblack      -.1629476   .0559045    -2.91   0.004    -.2725184   -.0533767
fotherr     -.7789486   .1395033    -5.58   0.000     -1.05237   -.5055271
fhispan     -.3502288   .0667771    -5.24   0.000    -.4811095   -.2193481
tripre1     -.3708134   .066066     -5.61   0.000    -.5003004   -.2413265
tripre2     -.1867279   .0645224    -2.89   0.004    -.3131896   -.0602663
tripre3     -.2677017   .0700712    -3.82   0.000    -.4050388   -.1303645
first       -1.017526   .2369723    -4.29   0.000    -1.481984   -.5530694
dmage2      -.524003    .0961962    -5.45   0.000     -.712544   -.3354619
dmage3       .0167751   .0035188     4.77   0.000     .0098785    .0236718
dmage4      -.0002628   .000063     -4.17   0.000    -.0003863   -.0001393
dmage5       1.61e-06   4.43e-07     3.64   0.000     7.44e-07    2.48e-06
dmeduc2      1.396263   .1341838    10.41   0.000     1.133267    1.659258
dmeduc3     -.1592871   .0162713    -9.79   0.000    -.1911784   -.1273959
dmeduc4      .0079669   .0008739     9.12   0.000     .0062541    .0096797
dmeduc5     -.000147    .0000174    -8.44   0.000    -.0001811   -.0001128
dlivord2     .29881     .2867482     1.04   0.297    -.2632062    .8608262
dlivord3    -.0635876   .0525077    -1.21   0.226    -.1665008    .0393255
dlivord4     .0056264   .0043628     1.29   0.197    -.0029245    .0141772
dlivord5    -.0001785   .000133     -1.34   0.179    -.0004391    .0000821
disllb2      .0004761   .000122      3.90   0.000      .000237    .0007151
```

disllb3	-3.38e-06	1.18e-06	-2.87	0.004	-5.70e-06	-1.07e-06
disllb4	9.89e-09	4.87e-09	2.03	0.042	3.52e-10	1.94e-08
disllb5	-1.00e-11	7.05e-12	-1.42	0.154	-2.39e-11	3.77e-12
dfage2	-.0687335	.0135837	-5.06	0.000	-.095357	-.04211
dfage3	.0017245	.0003529	4.89	0.000	.0010328	.0024163
dfage4	-.0000205	4.37e-06	-4.70	0.000	-.0000291	-.0000119
dfage5	9.14e-08	2.05e-08	4.45	0.000	5.12e-08	1.32e-07
dfeduc2	.5089613	.0992035	5.13	0.000	.3145259	.7033966
dfeduc3	-.0546737	.0121852	-4.49	0.000	-.0785563	-.0307911
dfeduc4	.0025427	.0006598	3.85	0.000	.0012496	.0038359
dfeduc5	-.0000438	.0000132	-3.31	0.001	-.0000697	-.0000178
drink2	-.0816761	.023267	-3.51	0.000	-.1272786	-.0360735
drink3	.0036845	.0015012	2.45	0.014	.0007423	.0066267
drink4	-.000066	.0000369	-1.79	0.073	-.0001383	6.27e-06
drink5	3.98e-07	2.87e-07	1.39	0.166	-1.65e-07	9.60e-07
deadkids2	-.0213968	.0051654	-4.14	0.000	-.0315208	-.0112728
dmelivord	.0495299	.0048692	10.17	0.000	.0399864	.0590734
dmefirst	.0592447	.0133347	4.44	0.000	.0331092	.0853801
dmedfeduc	-.0302435	.002516	-12.02	0.000	-.0351748	-.0253122
dmadmeduc	-.0161816	.0130217	-1.24	0.214	-.0417036	.0093403
dmadlivord	.059957	.0207079	2.90	0.004	.0193702	.1005437
dmdisllb	-.0016122	.0006164	-2.62	0.009	-.0028202	-.0004041
dmadfage	.0285812	.0030328	9.42	0.000	.0226369	.0345254
dmdfeduc	-.0254136	.0120058	-2.12	0.034	-.0489445	-.0018827
dmafirst	-.1540835	.0587776	-2.62	0.009	-.2692854	-.0388815
dmadblack	-.6090008	.0568116	-10.72	0.000	-.7203496	-.497652
_cons	-56.08907	6.913881	-8.11	0.000	-69.64002	-42.53811

The predicted values from this logit regression were used to divide the sample into 20 quantiles. It is then possible to test whether smokers and non-smokers have similar mean observable characteristics within each of these groups. The results of this testing are shown in Table 2:

Table 2 – T-Test results for equality of covariate means between smokers and non-smokers
(at a 5% significance level)

Quantile of P-Score	% of X's "unbalanced" (% of T-stats > 1.96)	Quantile of P-Score	% of X's "unbalanced" (% of T-stats > 1.96)
1	20.7%	11	3.4%
2	31.0%	12	10.3%
3	34.5%	13	6.9%
4	20.7%	14	6.9%
5	27.6%	15	17.2%
6	6.9%	16	10.3%
7	3.4%	17	31.0%
8	6.9%	18	17.2%
9	10.3%	19	10.3%
10	10.3%	20	51.7%
		Average:	16.9%

Note that the observations in the extreme quantiles are least balanced. It is perhaps not that surprising that it was more difficult to find a comparable group of non-smokers among high-propensity smokers and vice-versa. Overall covariates are balanced over 80% of the time, but balance in the extreme quantile is questionable (particularly 20). If these quantile are not balanced, our estimate of the treatment effect may be biased even if conditional random assignment holds. Inspection of the data reveals that in this case the 20^{th} quantile contain observations with p-scores ranging from .49-.99 (this is due to the relatively small number of observations with high p-scores). Thus even if the estimated p-score was balancing, we would expect difference in the observables between the treatment and control groups in this quantile as we are comparing observations with very different estimated p-scores (i.e., not controlling adequately for the p-score). In a case like this, the questionable quantile should be broken into sub-quantiles, and the same difference in means test performed on the sub-quantiles. An adequate number of sub-quantiles will allow us to control for the estimated p-score, but if we add too many sub-quantiles we may fail to reject a difference in means, even though the estimated estimated p-score is not balancing, because our test has weak power.

This estimate of the p-score produces some overlap between non-smokers and smokers, as seen in the box plot (figure 1) at the end of these solutions. It does appear, however, that non-smokers are concentrated in the low propensity part of the graph.

ii. The Estimated Treatment Effect

One method of estimating the treatment effect is to control linearly for the p-score:

(3) $Y_i = \theta T_i + \delta_1 \hat{P}_i + \delta_2 T_i\left(\hat{P}_i - \hat{\mu}_p\right) + u_i$

Results will vary depending on your p-score model, but this model produces:

```
Source       SS       df       MS              Number of obs = 139149
                                                F(  3,139145) = 1718.41
Model    1.6802e+09     3   560079215          Prob > F      = 0.0000
Residual 4.5351e+10 139145  325928.733         R-squared     = 0.0357
                                                Adj R-squared = 0.0357
Total 4.7032e+10139148   337996.89             Root MSE      = 570.90

dbirwt       Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
```

```
tobacco   -207.603   4.565888   -45.47   0.000     -216.552   -198.6539
pscore    -315.5137  13.66672   -23.09   0.000     -342.3002  -288.7272
tdmpscore -25.69618  23.41467    -1.10   0.272     -71.58849   20.19614
_cons      3475.703  2.756464  1260.93   0.000      3470.3     3481.105
```

The estimated average treatment effect is –208 g. The significant coefficient on the p-score suggests that smokers and non-smokers are not randomly selected from the population. It says that smokers – who tend to have large p-scores – tend to have characteristics that make the birth weights of their babies lower anyway. Non-smokers – who tend to have low p-scores – tend to have characteristics that make the birth weight of their babies heavier. The interaction term (tdmpscore) asks whether this selection is stronger for smokers. The coefficient also has the interpretation of how the effect of the treatment varies with the p-score. The estimate on the interaction is in this case is small and statistically insignificant.

Another way to estimate the treatment effect, which does not require that the outcome be linear in the p-score, is to estimate the difference in mean birth weight for the groups created above that were supposedly comparable in their observable characteristics. This separately estimates the average treatment effect for each group.

Quantile	Estimated TE	Standard Error	Treated	% of All Treated
1	-102	50	132	1%
2	-229	38	207	1%
3	-186	34	256	1%
4	-206	31	332	1%
5	-193	27	479	2%
6	-232	25	535	2%
7	-226	22	705	3%
8	-197	21	796	3%
9	-247	21	862	3%
10	-196	19	995	4%
11	-217	18	1138	4%
12	-232	18	1152	4%
13	-233	16	1376	5%
14	-215	16	1460	6%
15	-190	16	1598	6%
16	-235	16	1842	7%
17	-203	16	2155	8%
18	-187	15	2583	10%
19	-183	15	3131	12%
20	-236	16	4240	16%

Note that for the lowest quantile the effect of smoking appears to be smaller. This may mean that low-propensity women also smoke fewer cigarettes, or that there are other omitted factors that make the babies of these smoking mothers relatively healthier. Other than this, the effect seems to be fairly uniform across quantiles, and not that different than the conditional difference in birth weights from parts (B) – (C).

We can get an estimate of the overall average treatment effect by averaging these, weighted by the fraction of all smokers in each group. In this case these average to negative 212g. (look at your class notes for the appropriate equation)

F. Nonparametric Estimates.
See figures 2 & 3 at the end of the handout. This breaks smokers and non-smokers into finer groups based on their p-score. The purpose again is to abstract from a particular functional form for the p-score in the outcome equation (in (3), for example, we specified that the outcome was linear in the p-score.) The treatment effect in these pictures is the vertical distance between the outcomes of smokers and non-smokers with identical propensity scores.

The figure that breaks the population into 100 groups shows a smoker/non-smoker birth weight difference of about 200 grams, similar to previous estimates. The graph appears fairly linear, so (3) may not have been too bad for estimating the treatment effect. Note again that there are very few non-smokers in the high-propensity range.

The results when breaking the data into 200 groups are a bit noisier, especially for smokers. This is expected since there are fewer observations per group. The gap between smokers and non-smokers outcomes appears to be slightly smaller in this picture.

G. Infant Death. This question asks us to substitute for the outcome of interest an indicator for infant death (within one year of being born). This does not require us to re-estimate the p-score.

Notice that the dependent variable is now binary. Justin used the linear probability model (i.e., reg) due to its simplicity. Using the linear probability model I obtain the unadjusted mean difference as in part (A.):

```
  Source |       SS       df       MS                  Number of obs =  139149
---------+------------------------------               F(  1,139147) =   22.62
   Model |   .15684942     1   .15684942               Prob > F      =  0.0000
Residual |  965.053422139147  .006935496               R-squared     =  0.0002
---------+------------------------------               Adj R-squared =  0.0002
   Total |  965.210271139148  .006936573               Root MSE      =  .08328

------------------------------------------------------------------------------
   death |      Coef.   Std. Err.       t     P>|t|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
 tobacco |   .0027248    .000573      4.756   0.000       .0016018    .0038478
   _cons |   .0064767   .0002476     26.163   0.000       .0059915    .0069619
------------------------------------------------------------------------------
```

This says maternal smoking is associated with a .0027 increased probability of infant death. Controlling linearly for the other covariates (results not shown) lowers the estimated effect to 0.000032, which is statistically indistinguishable from 0. These estimates suggest that smoking has little impact on infant death.

Nonparametric estimates that use the p-score as a control concur with this result. Figure 4, attached to the end of these solutions, displays no distinguishable difference between the outcomes of smoking and non-smoking mothers' babies when breaking the data into 100 p-score groups.

H. These results suggest that maternal smoking during pregnancy has a negative impact on the birth weight of infants. A variety of estimation techniques, including OLS and matching propensity scores in fine cells, produce a stable estimate of around 200 grams. This effect, however, does not appear to translate into a higher infant morality rate for the children of smokers.

There remains some question of whether it is possible to interpret these results as truly causal or not. In particular, it appears on average that non-smoking mothers tend to have other observable characteristics that lead them to have higher birth-weight babies. It was possible with this large dataset to control for a long list of such observable characteristics, but the overlap between smokers and non-smokers in these characteristics appears to be sparse in some places, particularly among people who are predicted to be very likely to smoke and very unlikely to smoke.

Figure 1. Box and Whiskers

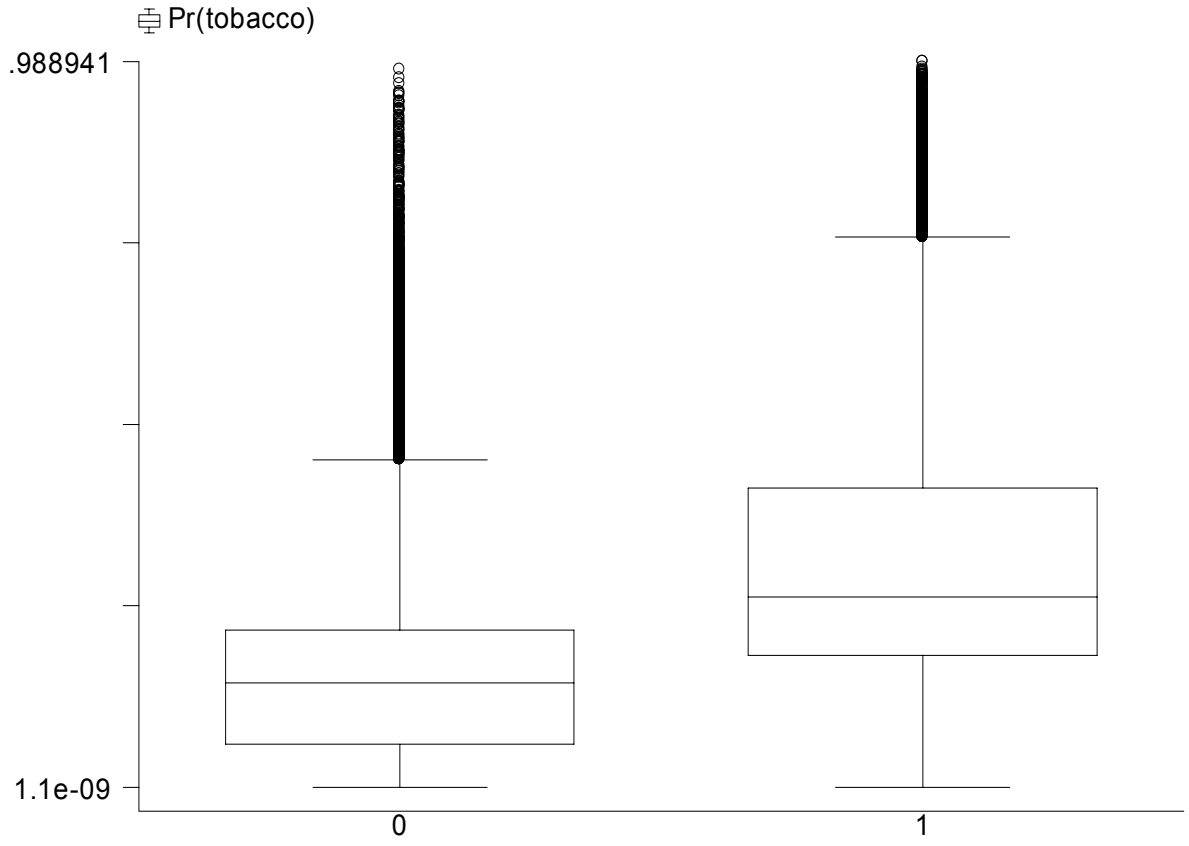

Figure 2. Sample Mean Birth Weights for Smokers and Non-Smokers (100 Quantiles)

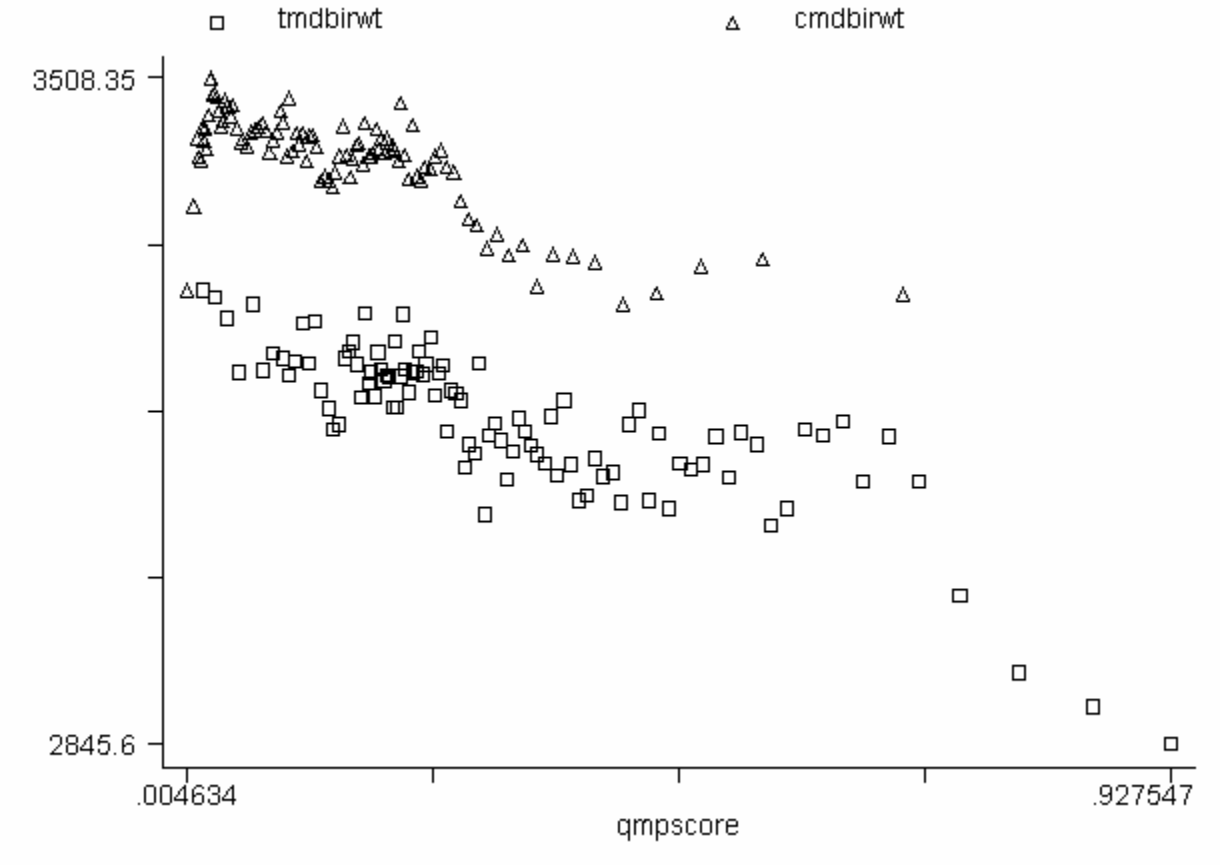

Figure 3. Sample Mean Birth Weights for Smokers and Non-Smokers (200 Quantiles)

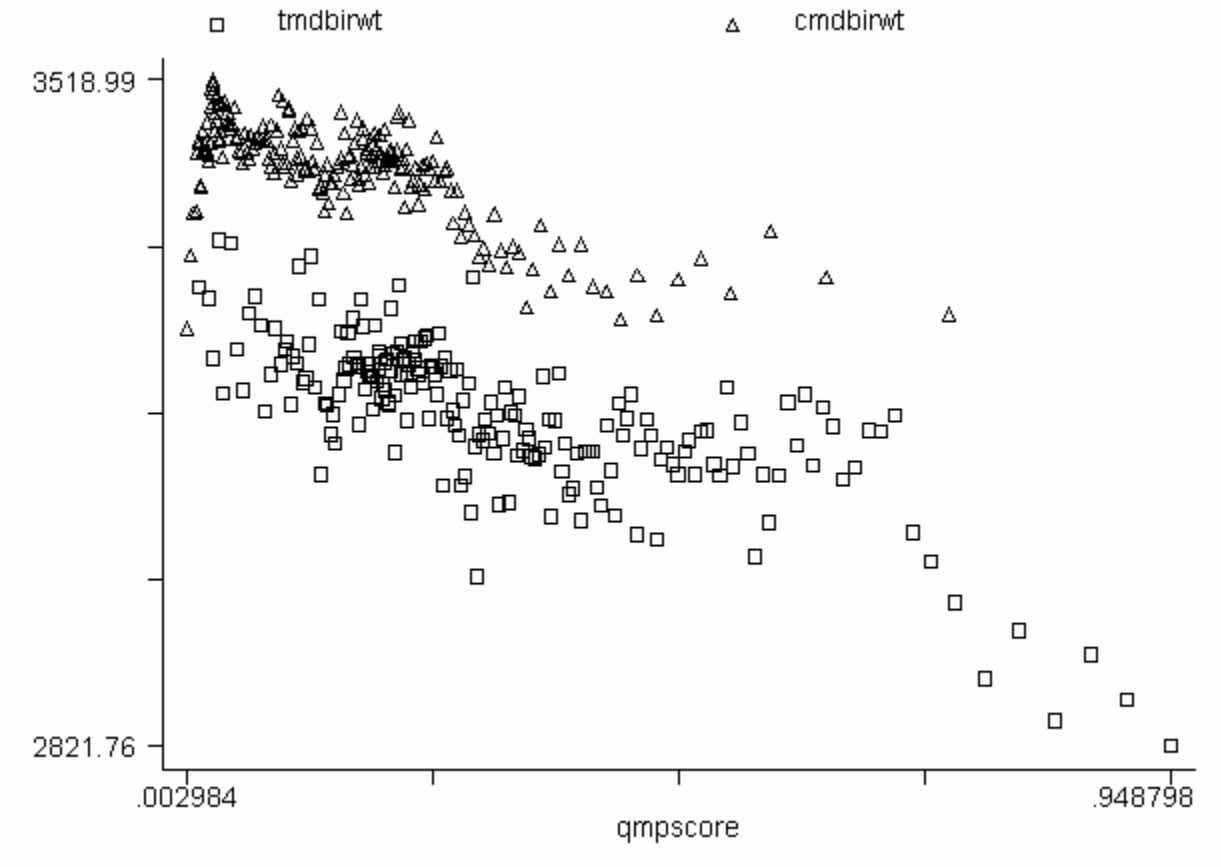

Figure 4. Sample Mean Infant Mortality for Smokers and Non-Smokers (100 Quantiles)