# Web Material

# Causal mediation analysis with observational data: considerations and illustration examining mechanisms linking neighborhood poverty to adolescent substance use

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## Contents

# Web Appendix 1

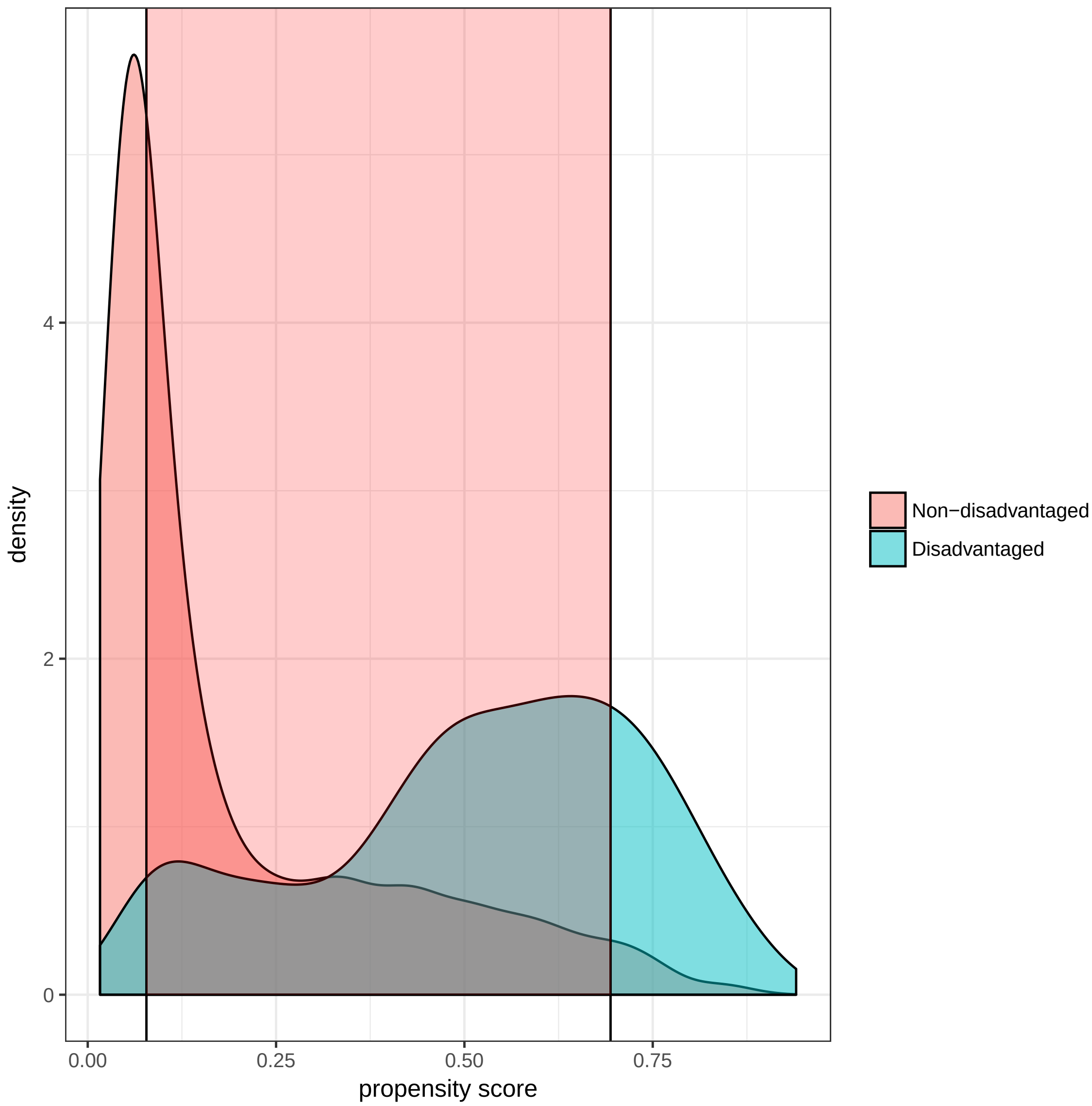
Adolescence is a critical time for social and biological development (1, 2), as well as a period of heightened risk for experimentation with, and initial onset of drug, alcohol, and tobacco use (3). For example, among 17-18 year-olds in the U.S., 78% have used alcohol, 43% have used illicit drugs (4), and nearly 24% have a history of substance use disorder by age 18 (5). Studies have indicated that earlier and adolescent onset of tobacco, alcohol, and drug use is associated with greater risk of substance use problems, abuse, and dependence during adulthood (6–9). Substance use disorders, in turn, are associated with considerable morbidity, mortality, and societal and economic costs (10–12). Likewise, drug and alcohol use during adolescence is associated with negative educational, economic, mental health outcomes that may have long-term consequences (13–17). Cigarette smoking during the critical period of adolescence may lead to delays in development, long-term tobacco addiction and use, and increased risk of anxiety disorders (18). Accordingly, a wealth of research has been devoted to identifying risk factors for drug, alcohol, and tobacco use, as well as problematic substance use and substance abuse and dependence, during adolescence.

# Web Appendix 2

## Sample

The National Comorbidity Survey - Adolescent Supplement (NCS-A) is a nationally-representative survey of adolescents living in the contiguous U.S. conducted from 2001-2004. Details of the sampling design and procedures have been published previously (19–21). Briefly, a dual-frame sampling design was used that included household (n=879) and school (n=9,244) subsamples. A total of 10,123 13-18 year-olds participated in the survey, with an overall response rate of 75.6% (20). Adolescents were interviewed in their homes by trained lay interviewers using a modified version of the World Health Organization Composite International Diagnostic Interview Version 3.0 (CIDI), a fully-structured interview that elicits information about the presence of mental disorders, adolescent and family characteristics, and risk factors (19). Written informed consent was provided by parents and assent by adolescents. Study procedures were approved by the human subjects committees of Harvard Medical School and the University of Michigan. This analysis, which only used de-identified data, was determined to be nonhuman subjects research by the University of California, Berkeley and the University of California, Davis.

We restricted our analysis to adolescents who were part of the NCS-A school subsample and whose principals completed a paper-and-pencil questionnaire that was sent to the principals of all participating schools (N=7,442 from 271 schools). We further restricted our analysis to those living in urban areas (N=3,064), as previous research demonstrated effect modification of the neighborhood disadvantage-mental health relationships by urbanicity (22). Finally, we limited our analysis to the area of support in terms of propensity to live in a disadvantaged neighborhood (N=1,829) (23) by limiting the sample to adolescents whose probability of living in a disadvantaged neighborhood conditional on covariates was greater than 3rd percentile of those who actually lived in a disadvantaged neighborhood and less than the 97th percentile of those who actually lived in a nondisadvantaged neighborhood, in accordance with prior recommendations (24). Web Figure 1 shows this restriction. Restricting to this area of support ensures that each participant has at least one counterpart in the other exposure group with a comparable propensity to live in a disadvantaged neighborhood, which guards against model extrapolation and satisfies the assumption of positivity (25). This weighted subsample roughly corresponds to the 2000 population of urban, US adolescents whose residence in a disadvantaged versus nondisadvantaged neighborhood cannot be nearly perfectly predicted by his/her covariates (21).




Web Figure 1: Distribution of propensity scores by neighborhood disadvantage status. Area of overlap used for the sample restriction is highlighted.

## Measures

The exposure of neighborhood disadvantage was defined as living in the lowest tertile of neighborhood socioeconomic status, measured using data from the 2000 U.S. Census. This measure has been used previously in studies using the NCS-A (22) and widely used in other epidemiologic studies (26–28). Residential addresses were geocoded to Census tract, which was the neighborhood geography considered here. The measure (29) was created by summing the z-scores of six US Census indicators: 1) log median household income, 2) percent households with interest, dividend, or rental income, 3) log median value of housing units, 4) percent persons over age 25 with high school degree, 5) percent persons over age 25 with college degree, and 6) percent persons in executive, managerial, or professional specialty occupations.

We considered four binary mediators related to the school and peer environments that had low levels of missingness. Two hypothesized mediators were aspects of the school environment and were reported by principals: 1) high rates of violent crime, defined as greater than 3 violent crimes per 100 students (corresponding to the 75th percentile), and 2) security presence. Two hypothesized mediators were aspects of the adolescent's peer environment and were reported by adolescents in modules of the CIDI: 3) whether most or all of his/her friends and siblings ever use marijuana or other drugs and 4) never having participated in an after-school sport or club himself/herself.

We considered six binary substance use outcomes: 1) lifetime cigarette use, 2) lifetime alcohol use, 3) problematic alcohol use, defined as reporting that drinking ever caused problems at school, work or at home, 4) lifetime marijuana use, 5) problematic drug use, defined as ever using hard drugs or prescription drugs not prescribed by a doctor or reporting that use of drug(s) ever caused problems at school, work or home, and 6) past-year *DSM-IV* substance use abuse or dependence. The first five outcomes were based on adolescent self-report, while substance abuse or dependence was based on CIDI diagnoses (20).

Baseline covariates included the adolescent's sex, age, race/ethnicity (black, white, Hispanic/latino, or other), whether or not English was his/her primary language, citizenship status, immigration generation (1st, 2nd, or $\geq$ 3rd), region of the country (Northeast, Midwest, South, or West), religion (protestant, catholic, other religion, and no religion), whether or not he/she lived his or her whole life with his/her 1) mother and 2) father, family dynamics (presence of psychological abuse, moderate forms of physical abuse, and severe forms of physical abuse separately for 1) the adolescent and a parent and 2) the parents), whether the adolescent was employed or a student, family income (log-transformed), and maternal age at birth of the adolescent (centered at 35). All covariates were adolescent-reported.

# Web Appendix 3

Targeted minimum loss-based estimation (TMLE) is a doubly robust substitution-based estimation strategy. We invite the interested reader to learn more about TMLE in general (30) and about the particular TMLE we employed in estimating stochastic direct and indirect effects (31). Annotated R code for estimating these effects is included in Web Appendix 4.

We describe how to obtain the TMLE estimate for $E(Y_{a,\hat{g}_{M|a^*,W}})$. One can follow the same procedure to obtain the estimates for $E(Y_{a,\hat{g}_{M|a,W}})$ and $E(Y_{a^*,\hat{g}_{M|a^*,W}})$ and then take the appropriate contrasts to obtain the stochastic direct and indirect effects.

We assume a specified stochastic intervention on $M$, $\hat{g}_{M|a^*,W}$. First, generate predicted values of $Y$, conditional on $m, z, w$. Target these predicted values with weights $\frac{\hat{g}_{M|a^*,W}}{P(a|W)g_{M|Z,W}}$ by fitting a weighted logistic regression model of $Y$ with the logit of the predicted $Y$ values as an offset. The intercept of that model is added to the initial predicted values to give updated values.

We then integrate out M and generate predicted values of this new quantity setting $A$ to $a$. The resulting empirical mean is the TMLE estimate of $E(Y_{a,\hat{g}_{M|a^*,W}})$.

The variance is estimated by the sample variance of the efficient influence curve, which is outside the scope of this paper, but is detailed elsewhere (31).

# Web Appendix 4

```
#################################################################
### Code for:                                               ###
### Causal mediation analysis with                          ###
### observational data: considerations and illustration ###
### examining mechanisms linking neighborhood poverty to###
### adolescent substance use                  ###
#################################################################

# The following code estimates direct and indirect effects
# for the 1) Baron and Kenny approach and 2) the stochastic
# mediation TMLE approach for a given dataset, mediator, and outcome.

library(MASS)
library(glmnet)
library(doParallel)
library(sandwich)

set.seed(42394)

mediator<-"highviolentcrime"
outcome<-"d_substance_NIMH2"
covlist<-c("SEXF", "lninc", "racecat2", "racecat3", "racecat4", "age_cent")
wcol<-c("SEXF", "emp", "ImgGen", "Language", "citizen", "lninc","CH33","pc_psych_minor", "pp_pa_minor","pp_pa_severe","pc_pa_minor" , "pc_pa_severe" , "age_cent","fath","moth", "cmage", "cmage2" ,"racecat2" , "racecat3" , "racecat4" , "religion2", "religion3" ,"religion4" ,"region2","region3","region4" )

#load data
load("dat.RData")

#vector of covariates to include
wcolbig<-as.character(read.csv("wcolbig.csv")[,2])
#vector of 2nd order interactions of W with mediator to include
```

```r
wcolmedonly<-as.character(read.csv("wcolmedonly.csv")[,2])
wintmcols<-wcolmedonly[seq(1,147,7)]

###############################
## Baron and Kenny Approach
###############################

tmpdatm<-dat[,c("tertscore", wcolbig)]

#use lasso for model fitting
    pfac<-rep(1, ncol(tmpdatm))
    pfac[which( colnames(tmpdatm) %in% c("tertscore", covlist) )]<-0

    cl<-makePSOCKcluster(4)
    registerDoParallel(cl)

    cvfit<-cv.glmnet(x=data.matrix(tmpdatm), y=dat[,mediator], weights=dat$sbwt,  penalty.factor=pfac, parallel=TRUE)
    tmp_coeffs <- coef(cvfit, s = "lambda.1se")

    a<-colnames(dat)[colnames(dat) %in% tmp_coeffs@Dimnames[[1]][tmp_coeffs@i + 1][-1]]

  #fit M model
    fit<-glm(formula=paste0(mediator, "~ . "), data=dat[,c(a, mediator)], weights=dat$sbwt)
  #get treatment coefficient from M model
  acoefmmodel<-summary(fit)$coef[rownames(summary(fit)$coef)=="tertscore",1]

  tmpdaty<-dat[,c("tertscore", mediator, wcolbig, wintmcols)]

    pfac<-rep(1, ncol(tmpdaty))
    pfac[which( colnames(tmpdaty) %in% c("tertscore", mediator, covlist) )]<-0
    cvfit<-cv.glmnet(x=data.matrix(tmpdaty), y=dat[,outcome],  weights=dat$sbwt, penalty.factor=pfac, parallel=TRUE)
    stopCluster(cl)
    tmp_coeffs <- coef(cvfit, s = "lambda.1se")
    a<-colnames(dat)[colnames(dat) %in% tmp_coeffs@Dimnames[[1]][tmp_coeffs@i + 1][-1]]

  #fit Y model
    fit<-glm(formula=paste0(outcome, "~ . "), data=dat[,c(a, outcome)], weights=dat$sbwt)
    #get treatment coefficient from Y model and robust variance estimate
  acoefymodel<-c(summary(fit)$coef[rownames(summary(fit)$coef)=="tertscore",1], diag(vcovHC(fit, type="HC0"))["tertscore"])
  #get mediator coefficient from M model and robust variance estimate
  mcoefymodel<-c( summary(fit)$coef[rownames(summary(fit)$coef)==mediator,1], diag(vcovHC(fit, type="HC0"))[mediator])

    nde<-acoefymodel[1]
    varnde<-acoefymodel[2]

    nie<-mcoefymodel[1]*acoefmmodel[1]
```

```r
    varnie<-(mcoefymodel[2] * acoefmmodel[2]) + (mcoefymodel[2]* acoefmmodel[1]^2) + (acoefmmodel[2] * mcoefymodel[1]^2)

    #SDE = stochastic direct effect
    #SDEVAR = variance of the stochastic direct effects
    #SIE = stochastic indirect effect
    #SIEVAR = variance of the stochastic indirect effect
    resbk<-list("sde"=nde, "sdevar"=varnde, "sie"=nie, "sievar"=varnie)

##############################
## TMLE Approach
##############################
wcolbig<-as.character(read.csv("wcolbig.csv")[,2])
#vector of 2nd order interactions of W with mediator to include
wcolmedonly<-as.character(read.csv("wcolmedonly.csv")[,2])
wintmcols<-wcolmedonly[seq(1,147,7)]

#get the data-dependent stochastic mediator draws from observed data
      tmpdatmz1<-tmpdatmz0<-tmpdatm<-data.frame(cbind(dat[,wcolbig], tertscore=dat$tertscore))
      tmpdatmz1$tertscore<-1
      tmpdatmz0$tertscore<-0

      pfac<-rep(1, ncol(tmpdatm))
      pfac[which( colnames(tmpdatm) %in% c("tertscore", covlist) )]<-0

      cvfit<-cv.glmnet(x=data.matrix(tmpdatm), y=dat[,mediator], family="binomial", weights=bigdat$sbwt,
                       penalty.factor=pfac)
# This is the data-dependent stochastic draw from g_{M|a,W} for all observations
     gma1<-predict(cvfit, type="response", newx=data.matrix(tmpdatmz1), s="lambda.1se")
# This is the data-dependent stochastic draw from g_{M|a*,W} for all observations
      gm<-predict(cvfit, type="response", newx=data.matrix(tmpdatmz0), s="lambda.1se")

#covariates to include for the M and Y models, respectively
wformmodel<-as.character(na.omit(read.csv("wformmodel.csv")[,-1]))
wforymodel<-as.character(na.omit(read.csv("wforymodel.csv")[,-1]))

#covariates to include for the Qz model
q2a1a1model<-as.character(na.omit(read.csv("q2a1a1model.csv")[,-1]))
q2a1a0model<-as.character(na.omit(read.csv("q2a1a1model.csv")[,-1]))
q2a0a0model<-as.character(na.omit(read.csv("q2a0a0model.csv")[,-1]))

#fit M model
fitm<-glm(formula=paste(mediator, ".", sep="~"), data=dat, family="quasibinomial", weights=dat$sbwt)

mz<-predict(fitm, type="response", newdata=dat)

psm<-(mz*dat[,mediator]) + ((1-mz)*(1-dat[,mediator]))
```

```
#clever covariate
dat$ha1a1<-((dat[,mediator]*gma1 + (1-dat[,mediator])*(1-gma1))/psm) * (I(dat$tertscore==1)/dat$pscore) * dat$sbwt
dat$ha1a0<-((dat[,mediator]*gm + (1-dat[,mediator])*(1-gm))/psm) * (I(dat$tertscore==1)/dat$pscore) * dat$sbwt

dat$ha0a0<-((dat[,mediator]*gm + (1-dat[,mediator])*(1-gm))/psm) * (I(dat$tertscore==0)/(1-dat$pscore))* dat$sbwt

tmpdatym0<-tmpdatym1<-tmpdaty<-dat[,c("tertscore", mediator, wcolbig, wintmcols)]
tmpdatym0[,mediator]<-0
tmpdatym1[,mediator]<-1

#fit Y model
fity<-glm(formula=paste(outcome, ".", sep="~"), data=dat[,c(wforymodel[!is.na(wforymodel)], outcome)], family="quasibinomial", weights=dat$sbwt)
#get initial Qy
ym1<-predict(fity, type="response", newdata=tmpdatym1)
ym0<-predict(fity, type="response", newdata=tmpdatym0)

#integrate out m
dat$q1<-(ym1*gma1) + (ym0*(1-gma1))
dat$q1a0<-(ym1*gm) + (ym0*(1-gm))

#get update
epsilona1a1<-coef(glm(formula= paste(outcome, "1", sep="~") , weights=dat$ha1a1, offset=(qlogis(q1)), family="quasibinomial", data=dat[,c(outcome, "q1")]))
epsilona1a0<-coef(glm(formula= paste(outcome, "1", sep="~") , weights=dat$ha1a0, offset=(qlogis(q1a0)), family="quasibinomial", data=dat[,c(outcome, "q1a0")]))
epsilona0a0<-coef(glm(formula= paste(outcome, "1", sep="~") , weights=dat$ha0a0, offset=(qlogis(q1a0)), family="quasibinomial", data=dat[,c(outcome, "q1a0")]))

#updated Qm
dat$q1upa1a1<-plogis(qlogis(dat$q1) + epsilona1a1)
dat$q1upa1a0<-plogis(qlogis(dat$q1a0) + epsilona1a0)
dat$q1upa0a0<-plogis(qlogis(dat$q1a0) + epsilona0a0)

#get initial Qz
fitq2a1a1<-glm(qlogis(q1upa1a1) ~ . , data=dat[dat$tertscore==1, c(q2a1a1model[!is.na(q2a1a1model)], "q1upa1a1")], weights=dat[dat$tertscore==1, "sbwt"])
dat$q2preda1a1<-predict(fitq2a1a1, newdata=dat[, c(mediator, wcolbig, wintmcols)])

fitq2a1a0<-glm(qlogis(q1upa1a0) ~ . , data=dat[dat$tertscore==1, c(q2a1a0model[!is.na(q2a1a0model)], "q1upa1a0")], weights=dat[dat$tertscore==1, "sbwt"])
dat$q2preda1a0<-predict(fitq2a1a0, newdata=dat[, c(mediator, wcolbig, wintmcols)])
```

```r

fitq2a0a0<-glm(qlogis(q1upa0a0) ~ . , data=dat[dat$tertscore==0, c(q2a0a0model[!is.na(q2a0a0model)], "q1upa0a0")], weights=dat[dat$tertscore==0, "sbwt"])
dat$q2preda0a0<-predict(fitq2a0a0, newdata=dat[, c(mediator, wcolbig, wintmcols)])

#estimate
tmlea1m1<-sum(plogis(dat$q2preda1a1)*dat$sbwt)/sum(dat$sbwt)
tmlea1m0<-sum(plogis(dat$q2preda1a0)*dat$sbwt)/sum(dat$sbwt)
tmlea0m0<-sum(plogis(dat$q2preda0a0)*dat$sbwt)/sum(dat$sbwt)

#get update
epsilon2<-coef(glm(q1upa1a1~ 1 , weights=(I(dat$tertscore==1)/dat$pscore)*dat$sbwt, offset=q2preda1a1, family="quasibinomial", data=dat))
epsilon2a1m0<-coef(glm(q1upa1a0~ 1 , weights=(I(dat$tertscore==1)/dat$pscore)*dat$sbwt, offset=q2preda1a0, family="quasibinomial", data=dat))
epsilon2a0m0<-coef(glm(q1upa0a0~ 1 , weights=(I(dat$tertscore==0)/(1-dat$pscore))*dat$sbwt, offset=q2preda0a0, family="quasibinomial", data=dat))

#updated Qz
q2up<-plogis(dat$q2preda1a1 + epsilon2)
q2upa1m0<-plogis(dat$q2preda1a0 + epsilon2a1m0)
q2upa0m0<-plogis(dat$q2preda0a0 + epsilon2a0m0)

#components of eic
eic1<-dat$ha1a1 * (dat[,outcome] - dat$q1upa1a1)
eic2<-(I(dat$tertscore==1)/dat$pscore)*dat$sbwt*(dat$q1upa1a1 - q2up)
eica1a1<-eic1 + eic2

eic0<-dat$ha1a0 * (dat[,outcome] - dat$q1upa1a0)
eic2a1m0<-(I(dat$tertscore==1)/dat$pscore)*dat$sbwt*(dat$q1upa1a0 - q2upa1m0)
eica1a0<-eic0 + eic2a1m0

eic00<-dat$ha0a0 * (dat[,outcome] - dat$q1upa0a0)
eic2a0m0<-(I(dat$tertscore==0)/(1-dat$pscore))*dat$sbwt*(dat$q1upa0a0 - q2upa0m0)
eica0a0<-eic00 + eic2a0m0

ndeeic<-eica1a0 - eica0a0
vareic<-var(ndeeic)/nrow(tmpdaty)

nieeic<-eica1a1 - eica1a0
varnieeic<-var(nieeic)/nrow(dat)

#results
#SDE = stochastic direct effect
#SDEVAR = variance of the stochastic direct effects
#SIE = stochastic indirect effect
#SIEVAR = variance of the stochastic indirect effect
restmle<-list("sde"=tmlea1m0-tmlea0m0, "sdevar"=vareic, "sie"=tmlea1m1-tmlea1m0, "sievar"=varnieeic)
```

implementationcoder1.R

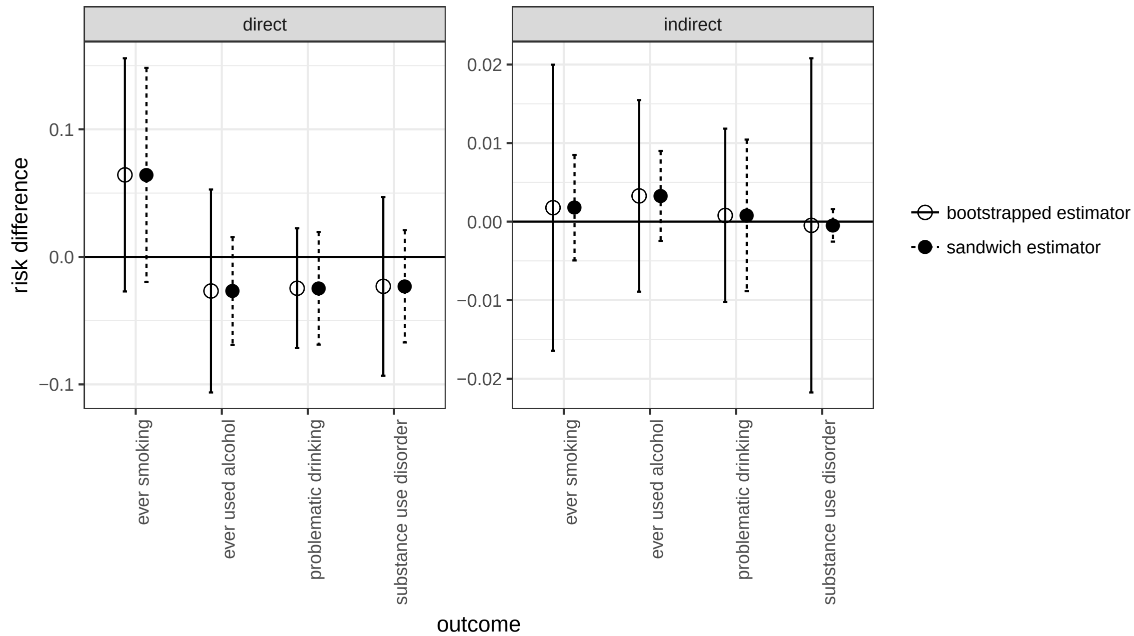
# Web Appendix 5

## Results

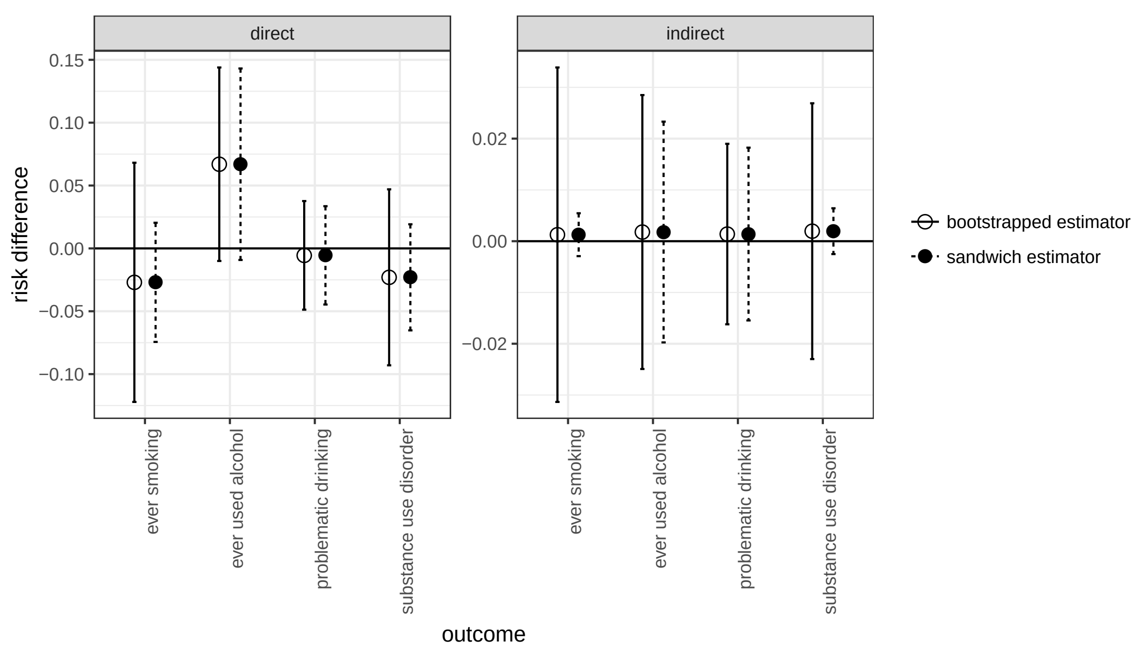
The analytic sample is shown in Web Table 1. Baseline characteristics by neighborhood disadvantage status are shown in the top portion of Web Table 1. The distribution of most characteristics are similar across the exposure groups with the possible exceptions of race/ethnicity and region. Mediators and outcomes by exposure group are shown in the bottom portion of Web Table 1. Those living in disadvantaged neighborhoods were more likely to have high violent crime at school, have a security presence at school, and not to engage in after-school sports or clubs. Those living in disadvantaged neighborhoods were also more likely to ever have smoked, ever have used marijuana and less likely to engage in problematic drinking.

Web Table 1: Characteristics by neighborhood disadvantage status, National Comorbidity Survey Adolescent Supplement, 2001-2004. Numbers are percentages unless otherwise specified. Descriptive statistics are survey weighted and combined across 30 imputed datasets.

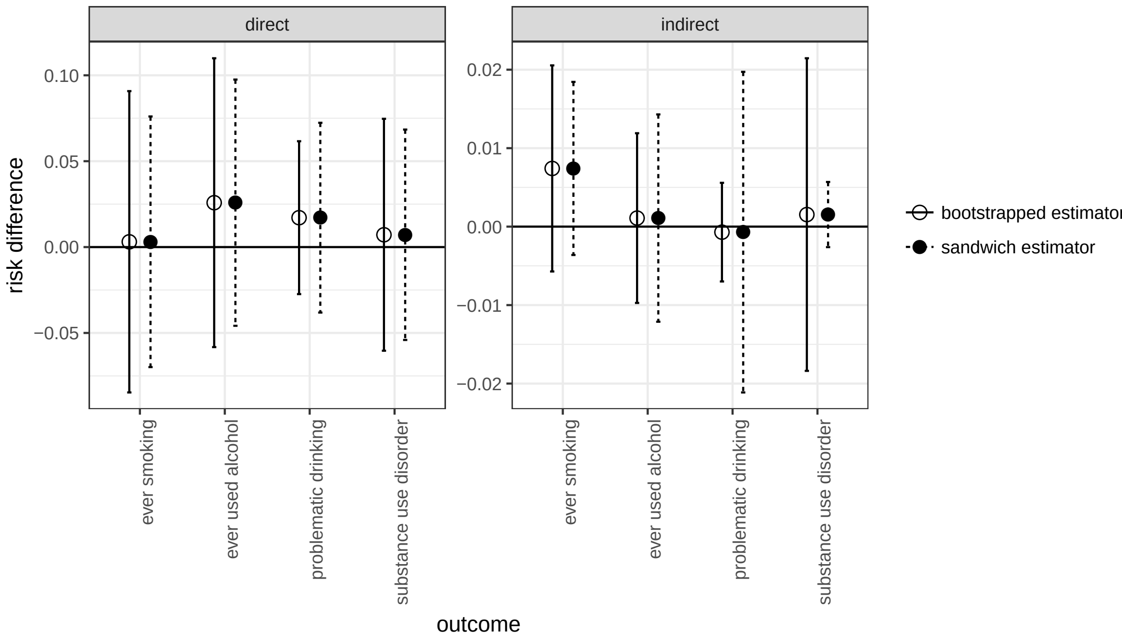
| Characteristic | Neighborhood | |
|---|---|---|
| | Nondisadvantaged N=1183 | Disadvantaged N=646 |
| Female | 50.85 | 53.14 |
| Age (mean, (SE)) | 15.42 (0.07) | 15.24 (0.10) |
| Race/ethnicity | | |
| Hispanic/Latino | 20.87 | 22.19 |
| Black | 17.02 | 27.23 |
| Other | 12.54 | 6.03 |
| White | 49.57 | 33.55 |
| Student | 94.51 | 93.16 |
| English as a second language | 30.47 | 37.32 |
| Citizen | 92.06 | 89.40 |
| Region | | |
| Northeast | 14.93 | 31.60 |
| Midwest | 14.14 | 7.07 |
| South | 17.31 | 24.11 |
| West | 53.62 | 37.22 |
| Household income (log, mean (SE)) | 11.09 (0.05) | 10.86 (0.10) |
| Maternal age at birth of child (mean, (SE)) | 34.63 (0.25) | 33.63 (0.38) |
| Lived whole life with father | 42.91 | 47.12 |
| Lived whole life with mother | 84.62 | 83.13 |
| Family conflict tactics | | |
| Parent-parent psychological aggression (5-point scale, mean (SE)) | 3.02 (0.05) | 2.92 (0.08) |
| Parent-child psychological aggression (5-point scale, mean (SE)) | 3.42 (0.04) | 3.30 (0.07) |
| Parent-parent minor physical assault | 25.35 | 29.74 |
| Parent-parent severe physical assault | 7.11 | 9.64 |
| Parent-child minor physical assault | 34.22 | 44.40 |
| Parent-child severe physical assault | 18.62 | 20.32 |
| Religion | | |
| Protestant | 33.95 | 30.63 |
| Catholic | 29.08 | 37.16 |
| No religion | 12.28 | 13.29 |
| Other | 24.69 | 18.92 |
| Mediators | | |
| High violent crime at school | 23.43 | 40.37 |
| Most peers use marijuana | 23.04 | 23.17 |
| Security presence at school | 61.79 | 73.93 |
| No engagement in after-school sports or clubs | 22.47 | 28.03 |
| Outcomes | | |
| Ever smoked | 24.43 | 29.85 |
| Ever used alcohol | 67.41 | 68.16 |
| Problematic drinking | 9.44 | 6.22 |
| Ever used marijuana | 31.87 | 35.21 |
| Problematic drug use | 9.64 | 8.86 |
| Past-year DSM-IV substance use disorder | 16.58 | 16.49 |




Web Figure 2: Direct and indirect effect estimates and 95% confidence intervals considering the mediator of high violent crime at school by outcome and variance estimation approach. Data from the National Comorbidity Survey, Adolescent Supplement.




Web Figure 3: Direct and indirect effect estimates and 95% confidence intervals considering the mediator of security presence at school by outcome and variance estimation approach. Data from the National Comorbidity Survey, Adolescent Supplement.




Web Figure 4: Direct and indirect effect estimates and 95% confidence intervals considering the mediator of no participation in after-school sports or clubs by outcome and variance estimation approach. Data from the National Comorbidity Survey, Adolescent Supplement.

## Comparison of results with results from the MTO experiment

In using the Baron and Kenny and TMLE approaches to examine mediation of neighborhood disadvantage and adolescent substance use by aspects of the school and peer environments, we found no evidence of mediation. This was similar to a related analysis using data from the Moving to Opportunity study that found largely null results (32); in that study, where non-null mediation results were identified, they were weak. Differences between the null results found using the NCS-A and the weak non-null results using the MTO could be explained by 1) residual confounding in the NCS-A analysis since the exposure was not randomized where as the experimental design of MTO addressed both observed and unobserved confounding, and 2) differences in the exposures, the NCS-A exposure being neighborhood disadvantage and the MTO exposure being randomization to receive a housing voucher (which was hypothesized to subsequently affect neighborhood disadvantage).

However, despite generally null mediation effects, both studies demonstrated evidence of similar, significant first-stage effects. In both studies, the exposures related to a more positive neighborhood environment were associated with aspects of safer school environments (Table 3 from the main text and (32)). However, results differed between the two studies in terms of the exposure's effect on peer drug use and the adolescent's participation in sports or clubs. This could be both because of residual confounding and because the MTO's exposure of housing voucher receipt, if utilized, resulted in moving out of the original neighborhood, which would likely have impacts on the peer environment over and above those due to neighborhood disadvantage status.

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