Biostatistics II Exam

2025-10-30

Question 1

The Missingness in Cannabis Use does not depend on the outcome or the covariates in the Figure 1A. There is no path going from $Miss_{cu}$ to $Outcome$, $Exposure$ and the $covariates$, hence we can say that $Miss_{cu}$ is d-separated from them and is independent of them. So, the probability of missingness does not depend on any variable in the Figure 1A, therefore, missingness in cannabis use is missing completely at random (MCAR).

Question 2

By definition, Missing at Random (MAR) is when the missingness in Cannabis Use can be explained by associations with the observed data, specifically fully observed data of covariates or outcome or both. Which is what we observe in Figure 1B, 1F and 1D respectively. In terms of d-separation, in the Figure 1B, there are multiple open paths from $Miss_{cu}$ to $Outcome$:

1. $$Miss_{cu} \leftarrow MaternalSubstanceUse \rightarrow Outcome$$
2. $$Miss_{cu} \leftarrow MaternalSubstanceUse \rightarrow CannabisUse \rightarrow Outcome$$
3. $$Miss_{cu} \leftarrow MaternalSubstanceUse \rightarrow CannabisUse \leftarrow Sex \rightarrow Outcome$$

and can all be blocked by adjusting for $MaternalSubstanceUse$ alone, and it is possible as $MaternalSubstanceUse$ is fully observed. Then, $Miss_{cu}$ and $Outcome$ are independent conditioned on $MaternalSubstanceUse$. Hence, as missingness can be explained with observed data, this is MAR.

Secondly in the Figure 1D, there is an additional open path compared to Figure 1B:

$$Miss_{cu} \leftarrow Outcome$$

which means that $Outcome$ is causing the missingness in Cannabis Use. This path can be blocked by conditioning on $Outcome$, which is fully observed as well. Hence, the Missingness in Cannabis Use and $Outcome$ are d-separated conditioned on $Maternal\ Substance\ Use$ and $Outcome$. Therefore, missingness is MAR.

Lastly, in the Figure 1F, the only open path existing is:

$$Miss_{cu} \leftarrow Outcome$$

Hence, the Missingness in Cannabis Use and $Outcome$ are d-separated conditioned on $Outcome$ and is MAR.

Question 3

By definition, Missingness Not At Random (MNAR) is when missingness cannot be explained by the observed data. For example, when Missingness in Cannabis Use is explained by the Cannabis Use itself, we can say that the missingness is not at random. In terms of d-separation, firstly in Figure 1C, there are multiple open paths from $Miss_{cu}$ to $Outcome$:

1. $$Miss_{cu} \leftarrow Cannabis\ Use \rightarrow Outcome$$
2. $$Miss_{cu} \leftarrow Cannabis\ Use \leftarrow Sex \rightarrow Outcome$$
3. $$Miss_{cu} \leftarrow Maternal\ Substance\ Use \rightarrow Outcome$$
4. $$Miss_{cu} \leftarrow Maternal\ Substance\ Use \rightarrow Cannabis\ Use \rightarrow Outcome$$
5. $$Miss_{cu} \leftarrow Maternal\ Substance\ Use \rightarrow Cannabis\ Use \leftarrow Sex \rightarrow Outcome$$

Here, the first two paths can only be blocked by conditioning on $CannabisUse$, which is not fully observed. Hence, $Miss_{cu}$ and $Outcome$ cannot be d-separated and are not independent of each other. Therefore, is MNAR.

Similarly in Figure 1E, the open path of

$$Miss_{cu} \leftarrow Outcome$$

is added to the previous list of Figure 1C, which can be blocked by adjusting for $Outcome$. Still, the $Miss_{cu}$ and $Outcome$ cannot be d-separated, even after adjusting for all observed variables and is MNAR.

Question 4

The linear regression model is:

$\mathbf{Outcome} \sim \mathbf{MaternalSubstanceUse} + \mathbf{Sex} + \mathbf{CannabisUse}$

$\Rightarrow \mathbf{Y} = \beta_{\mathbf{0}} + \beta_{\mathbf{M}} \times \mathbf{M} + \beta_{\mathbf{S}} \times \mathbf{S} + \beta_{\mathbf{X}} \times \mathbf{X}$

where Y stands for the $Outcome$, X denotes the exposure, which is $CannabisUse$, M is $MaternalSubstanceUse$, S is Sex and R is Missingness In Cannabis Use, $Miss_{Cu}$.

Here, in both Figure 1C and 1D, we know that the exposure: $\mathbf{X} \sim \mathbf{M} + \mathbf{S}$.

Although,

in Figure 1C : $\mathbf{R} \sim \mathbf{M} + \mathbf{X}$,

in Figure 1D : $\mathbf{R} \sim \mathbf{M} + \mathbf{Y}$.

In this question, we try to calculate the Maximum Likelihood estimators of parameters $\beta_0, \beta_X, \beta_M, \beta_S$ in the above mentioned linear regression model of Y. Maximum likelihood here, tries to estimate the regression coefficients that makes the observable data most likely under the model we assume.

First, we simulate data based on the knowledge we know from the above mentioned causal diagrams. Since, there is missingness in the exposure, we need to factor in the marginal distribution of $CannibasUse$ into the likelihood. Then, we use the `frm_em()` function to carry out the actual estimation. This function assumes that the missingness is **MAR**, that is, after we account for the observed variables, the missingness does not depend on the unobserved data.

```
beta0_true <- 1
betaX_true <- 1
betaM_true <- 1
betaS_true <- 1

generate_data <- function(n, dag) {

  pX <- function(M, S)
```

```
    plogis(M + S)

  M <- rbinom(n, 1, 0.5)
  S <- rbinom(n, 1, 0.5)
  X_continuous <- 1 + 0.8*M + 0.5*S + rnorm(n, 0, 1)
  # Convert to values 0,1,2 (categorical cannabis use), but still numeric
  X <- pmin(pmax(round(X_continuous), 0), 2)
  Y <- rnorm(n, mean = beta0_true + betaM_true*M + betaS_true*S + betaX_true*X, sd=1)


  # chosen the parameters in a way to show bias evidently
  if (dag == "fig1D") {
    # scale the continuous value Y so logit doesn't explode
    R <- rbinom(n, 1, pX(0.5*M, 0.5*scale(Y)[,1]))
  } else if(dag == "fig1C") {
        R <- rbinom(n, 1, pX(0.5*M, 4*X))
  }
  Xobs <- ifelse(R==1, X, NA_integer_)
  dat <- data.frame(Y=Y, X=Xobs, M=M, S=S, R=R, X_true=X)
  return(dat)
}

# Maximum Likelihood Estimation with Missing data
fit_frm_em_once <- function(n, dag) {
  dat <- generate_data(n, dag)
  dep <- list(model="linreg", formula=Y ~ X + M + S)
  ind <- list(X = list(model="linreg", formula=X ~ M + S))
  sink(tempfile()) # to suppress the progress bar output
  fit <- frm_em(dat=dat, dep=dep, ind=ind, verbose=FALSE)
  sink()
  cf  <- coef(fit)
  beta_0 <- unname(cf["Y ON (Intercept)"])
  se_0 <- unname(fit$se["Y ON (Intercept)"])
  beta_X  <- unname(cf["Y ON X"])
  se_X  <- unname(fit$se["Y ON X"])
  beta_M  <- unname(cf["Y ON M"])
  se_M  <- unname(fit$se["Y ON M"])
  beta_S  <- unname(cf["Y ON S"])
  se_S  <- unname(fit$se["Y ON S"])

  c(beta_0=beta_0, se_0=se_0, beta_X=beta_X, se_X=se_X, beta_M=beta_M, se_M=se_M, beta_S=beta_S, se_S=se
}

eval_ML_grid <- function(n_vec, reps, dag){
  out <- lapply(n_vec, function(n){
    M <- replicate(reps, fit_frm_em_once(n, dag))
    beta_0 <- M["beta_0",]; se_0 <- M["se_0",]
    beta_X <- M["beta_X",]; se_X <- M["se_X",]
    beta_M <- M["beta_M",]; se_M <- M["se_M",]
    beta_S <- M["beta_S",]; se_S <- M["se_S",]
    coverage_0 <- mean((beta_0 - 1.96*se_0) <= beta0_true & (beta_0 + 1.96*se_0) >= beta0_true)
    coverage_X <- mean((beta_X - 1.96*se_X) <= betaX_true & (beta_X + 1.96*se_X) >= betaX_true)
    coverage_M <- mean((beta_M - 1.96*se_M) <= betaM_true & (beta_M + 1.96*se_M) >= betaM_true)
```

```
    coverage_S <- mean((beta_S - 1.96*se_S) <= betaS_true & (beta_S + 1.96*se_S) >= betaS_true)
    data.frame(
      n=n,
      mean_Intercept = mean(beta_0), bias_Intercept = mean(beta_0)-beta0_true, coverage_Intercept = cov
      mean_X = mean(beta_X), bias_X = mean(beta_X)-betaX_true, coverage_X = coverage_X,
      mean_M = mean(beta_M), bias_M = mean(beta_M)-betaM_true, coverage_M = coverage_M,
      mean_S = mean(beta_S), bias_S = mean(beta_S)-betaS_true, coverage_S = coverage_S
    )
  })
  rbindlist(out)
}
```

Now, we compare the ML estimates with the true value $\beta_{\mathbf{0}} = \mathbf{1}$, $\beta_{\mathbf{X}} = \mathbf{1}$, $\beta_{\mathbf{M}} = \mathbf{1}$ and $\beta_{\mathbf{S}} = \mathbf{1}$. I am repeating the simulation for increasing values of n reaching towards infinity to discover the asymptotic behavior accurately.

```
# should be unbiased for Fig 1D
n_vec <- c(1000, 2000, 5000, 8000, 10000)
reps  <- 100

res_1D  <- eval_ML_grid(n_vec, reps, "fig1D")
print(res_1D)
```

```
##        n mean_Intercept bias_Intercept coverage_Intercept   mean_X     bias_X
##    <num>          <num>          <num>              <num>    <num>      <num>
## 1:  1000      0.9413996    -0.05860041               0.85 1.029078 0.02907830
## 2:  2000      0.9285465    -0.07145351               0.78 1.031134 0.03113446
## 3:  5000      0.9290823    -0.07091765               0.60 1.031765 0.03176501
## 4:  8000      0.9371569    -0.06284312               0.54 1.027078 0.02707833
## 5: 10000      0.9364791    -0.06352093               0.42 1.024411 0.02441098
##    coverage_X   mean_M     bias_M coverage_M   mean_S      bias_S coverage_S
##         <num>    <num>      <num>      <num>    <num>       <num>      <num>
## 1:       0.87 1.010887 0.01088740       0.95 1.001364 0.001364479       0.92
## 2:       0.87 1.018889 0.01888927       0.94 1.019498 0.019498377       0.96
## 3:       0.80 1.015710 0.01571023       0.95 1.016274 0.016274490       0.92
## 4:       0.75 1.014906 0.01490622       0.91 1.014726 0.014726498       0.91
## 5:       0.74 1.020694 0.02069388       0.89 1.015847 0.015846913       0.91
```

As n increases, we see that the mean of ML estimates converges towards the true values, the biases converges towards 0, the coverage for β_M, β_S is above 95% and β_X is mostly above 75%, even though β_0 coverage goes below 50%. Hence, we can conclude that the maximum likelihood estimation of the regression coefficients is asymptotically unbiased for Figure 1D. We know that the missingness is MAR according to Figure 1D, hence, the ML estimates produced by `frm_em()` function match the true values.

Next for Figure 1C, we run ML estimation:

```
# should be biased for Fig 1C
res_1C  <- eval_ML_grid(n_vec, reps, "fig1C")
print(res_1C)
```

```
##        n mean_Intercept bias_Intercept coverage_Intercept   mean_X     bias_X
##    <num>          <num>          <num>              <num>    <num>      <num>
## 1:  1000      0.8107997     -0.1892003               0.41 1.052670 0.05267039
## 2:  2000      0.8228480     -0.1771520               0.14 1.044082 0.04408188
## 3:  5000      0.8148672     -0.1851328               0.00 1.044460 0.04446020
## 4:  8000      0.8152364     -0.1847636               0.00 1.045955 0.04595547
```

```
## 5: 10000      0.8132272     -0.1867728               0.00 1.047489 0.04748889
##    coverage_X   mean_M     bias_M coverage_M   mean_S     bias_S coverage_S
##         <num>    <num>      <num>      <num>    <num>      <num>      <num>
## 1:       0.81 1.058232 0.05823196       0.91 1.021714 0.02171447       0.95
## 2:       0.80 1.062411 0.06241083       0.67 1.023946 0.02394570       0.93
## 3:       0.54 1.062039 0.06203924       0.52 1.030248 0.03024828       0.80
## 4:       0.32 1.064171 0.06417087       0.23 1.026879 0.02687911       0.78
## 5:       0.23 1.061752 0.06175214       0.15 1.027241 0.02724081       0.73
```

Here, as n increases, the mean of the ML estimates don't converge to their true values, their biases don't converge towards 0 and the coverage for $\beta_X, \beta_M, \beta_0$ goes well below 30% and goes below 75% for β_S. Hence, we can say that the ML estimation of these regression coefficients is biased for Figure 1C where the missingness of the exposure depends on the exposure itself. Hence, ML estimates couldn't truly represent the true values as the un-explainable missingness was causing the estimates to converge to a pseudo value instead.

Question 5

Now, for the same linear regression model, let us check whether the complete case analysis estimates are asymptotically unbiased or biased for the causal diagram in Figure 1C and 1D.

In Complete Case Analysis (CCA), we only consider the data that is observed for all variables in the main analysis. To calculate the CCA estimates of the regression coefficients, we simply fit the `lm` model. We reuse the same parameters and function to generate the data from Question 4.

```
fit_lm_once <- function(n, dag) {
  dat <- generate_data(n, dag)
  fit <- lm(formula=Y ~ X + M + S, data=dat)
  cf  <- coef(fit)
  beta_0  <- unname(cf["(Intercept)"])
  beta_X  <- unname(cf["X"])
  beta_M  <- unname(cf["M"])
  beta_S  <- unname(cf["S"])

  c(beta_0=beta_0, beta_X=beta_X, beta_M=beta_M, beta_S=beta_S)
}

eval_CCA_grid <- function(n_vec, reps, dag){
  out <- lapply(n_vec, function(n){
    M <- replicate(reps, fit_lm_once(n, dag))
    beta_0 <- M["beta_0",]
    beta_X <- M["beta_X",]
    beta_M <- M["beta_M",]
    beta_S <- M["beta_S",]
    data.frame(
      n=n,
      mean_0 = mean(beta_0), bias_0 = mean(beta_0)-beta0_true,
      mean_X = mean(beta_X), bias_X = mean(beta_X)-betaX_true,
      mean_M = mean(beta_M), bias_M = mean(beta_M)-betaM_true,
      mean_S = mean(beta_S), bias_S = mean(beta_S)-betaS_true
    )
  })
  rbindlist(out)
}
```

Now, we compare the CCA estimates with the true values, $\beta_{\mathbf{0}} = \mathbf{1}$, $\beta_{\mathbf{X}} = \mathbf{1}$, $\beta_{\mathbf{M}} = \mathbf{1}$ and $\beta_{\mathbf{S}} = \mathbf{1}$.

```r
# should be unbiased for Fig 1C
res_1C  <- eval_CCA_grid(n_vec, reps, "fig1C")
print(res_1C)
```

```
##        n    mean_0       bias_0    mean_X        bias_X    mean_M        bias_M
##    <num>     <num>        <num>     <num>         <num>     <num>         <num>
## 1:  1000 1.0186098  0.018609790 0.9856513 -0.0143486867 1.0085006  0.0085005886
## 2:  2000 1.0024223  0.002422303 1.0009019  0.0009019425 0.9934204 -0.0065796040
## 3:  5000 1.0035945  0.003594489 0.9985570 -0.0014430469 1.0002286  0.0002285886
## 4:  8000 0.9963598 -0.003640214 1.0009847  0.0009846515 1.0003259  0.0003259403
## 5: 10000 1.0060398  0.006039810 0.9942979 -0.0057020952 1.0014938  0.0014937961
##       mean_S        bias_S
##        <num>         <num>
## 1: 1.0093546  9.354606e-03
## 2: 1.0030847  3.084707e-03
## 3: 0.9999393 -6.070272e-05
## 4: 0.9995387 -4.612980e-04
## 5: 1.0025133  2.513282e-03
```

As n increases, we see that the mean of CCA estimates converges towards their true values and their biases converges towards 0. Hence, we can conclude that the complete case analysis estimation of the regression coefficients is asymptotically unbiased for Figure 1C. In such cases where the missingness of the exposure depends on the exposure itself, discarding the incomplete data is more beneficial towards being unbiased.

Next for Figure 1D, we run CCA estimation:

```r
# should be biased for Fig 1D
res_1D  <- eval_CCA_grid(n_vec, reps, "fig1D")
print(res_1D)
```

```
##        n   mean_0    bias_0    mean_X      bias_X    mean_M      bias_M
##    <num>    <num>     <num>     <num>       <num>     <num>       <num>
## 1:  1000 1.207863 0.2078630 0.9767942 -0.02320580 0.9388055 -0.06119447
## 2:  2000 1.230103 0.2301029 0.9696315 -0.03036849 0.9389726 -0.06102739
## 3:  5000 1.219186 0.2191864 0.9738943 -0.02610566 0.9313804 -0.06861958
## 4:  8000 1.211317 0.2113174 0.9793701 -0.02062988 0.9406836 -0.05931640
## 5: 10000 1.217168 0.2171682 0.9746661 -0.02533388 0.9406012 -0.05939876
##       mean_S      bias_S
##        <num>       <num>
## 1: 0.9743542 -0.02564582
## 2: 0.9677601 -0.03223989
## 3: 0.9792456 -0.02075436
## 4: 0.9741469 -0.02585314
## 5: 0.9802368 -0.01976320
```

Here, as n increases, the mean of the CCA estimates doesn't converge to their true values and the bias doesn't converge towards 0. Hence, we can say that the CCA estimation of these regression coefficients is biased for Figure 1D where the missingness of the exposure depends on observable and explainable variables, in this case, the outcome and a covariate. Here, we are potentially discarding an entire set of outcome data as they are missing due to their outcome, leading to a bias in the CCA estimates.

The results above also confirm the potential bias of exposure regression coefficient in CCA based on linear regression mentioned in the Table 1.

Question 6

Now, let us look at the mathematical argument as to why CCA is unbiased for Figure 1C, as seen with the help of estimates above. As we know that the missingness of the exposure in Figure 1C is dependent on a covariate and the exposure itself, hence, **MNAR**.

Hence, the missingness of the exposure in Figure 1C is independent of the outcome given exposure and the covariate $MaternalSubstanceUse$.

We can say that:

$\mathbf{R} \perp \mathbf{Y}|(\mathbf{X}, \mathbf{M}) \Leftrightarrow \mathbf{R} \perp \mathbf{Y}|(\mathbf{X}, \mathbf{M}, \mathbf{S}) \Leftrightarrow \mathbf{Y} \perp \mathbf{R}|(\mathbf{X}, \mathbf{M}, \mathbf{S})$

where **S** is Sex which also is not associated with the missingness in Cannabis Use.

To prove that CCA is unbiased, we can look at the true estimator and the CCA estimator of Y and check if they are identical.

The true estimator is $\mathbf{E}[\mathbf{Y} \mid \mathbf{X}, \mathbf{M}, \mathbf{S}]$.

The CCA estimator is $\mathbf{E}[\mathbf{Y} \mid \mathbf{X}, \mathbf{M}, \mathbf{S}, \mathbf{R} = \mathbf{1}]$.

But, for the figure 1C, due to the conditional independence of R and Y given (X, M, S), we can say that:

$\mathbf{P}(\mathbf{Y} \mid \mathbf{X}, \mathbf{M}, \mathbf{S}, \mathbf{R} = \mathbf{1}) = \mathbf{P}(\mathbf{Y} \mid \mathbf{X}, \mathbf{M}, \mathbf{S})$

Hence, the CCA estimator for figure 1C is deduced to:

$\mathbf{E}[\mathbf{Y} \mid \mathbf{X}, \mathbf{M}, \mathbf{S}, \mathbf{R} = \mathbf{1}] \Rightarrow \mathbf{E}[\mathbf{Y} \mid \mathbf{X}, \mathbf{M}, \mathbf{S}]$.

Therefore, we can say that the CCA is unbiased when the missingness pattern is conditionally independent of the outcome given exposure and covariates as seen in Figure 1C.

Question 7

The NPSEM corresponding to the causal diagram in the figure 1C looks like this:

$$\left.\begin{aligned} S &:= f_S(\varepsilon_S) \\ M &:= f_M(\varepsilon_M) \\ X &:= f_X(M, S, \varepsilon_X) \\ Y &:= f_Y(X, M, S, \varepsilon_Y) \\ R &:= f_R(M, X, \varepsilon_R) \end{aligned}\right\}$$

We can also rewrite this as:

$$\left.\begin{aligned} S &:= f_S(\varepsilon_S) \\ M &:= f_M(\varepsilon_M) \\ X &:= f_X(f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_X) \\ Y &:= f_Y(f_X(f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_X), f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_Y) \\ R &:= f_R(f_M(\varepsilon_M), f_X(f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_X), \varepsilon_R) \end{aligned}\right\}$$

Since, the auxiliary variables are not included in the main analysis and the absence of missing data the main analysis is assumed to give unbiased results, I have not added any additional factor U contributing to the variables. Hence, we can assume that the error terms ε_S, ε_M, ε_X, ε_R and ε_Y are independent.

Similarly, the NPSEM of figure 1D is:

$$\left.\begin{array}{l} S := f_S(\varepsilon_S) \\ M := f_M(\varepsilon_M) \\ X := f_X(M, S, \varepsilon_X) \\ Y := f_Y(X, M, S, \varepsilon_Y) \\ R := f_R(M, Y, \varepsilon_R) \end{array}\right\}$$

also, represented as:

$$\left.\begin{array}{l} S := f_S(\varepsilon_S) \\ M := f_M(\varepsilon_M) \\ X := f_X(f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_X) \\ Y := f_Y(f_X(f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_X), f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_Y) \\ R := f_R(f_M(\varepsilon_M), f_Y(f_X(f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_X), f_M(\varepsilon_M), f_S(\varepsilon_S), \varepsilon_Y), \varepsilon_R) \end{array}\right\}$$

The error terms are assumed to be independent here as well.

Question 8

Let us check if we see a statistical association between X and Y when adjusted for $M, S, R = 1$ (as it is a complete case analysis) prove a causal effect from X on the Y in the case of Figure 1C and 1D. There are several open paths from X to Y in both the figures. Once we block all these open paths passing through various covariates, and assume that there is no direct arrow from X to Y, we can then say that they are d-separated conditional on the covariates. And, if we still see a statistically significant association (after adjusting for these covariates), then we can prove that is a causal effect from X to Y.

First, for Figure 1C:

The open paths from X to Y, assuming that there is no direct arrow from X to Y:

1. $$X \leftarrow S \rightarrow Y$$

where S is a fork and it needs to be conditioned for to block the path by the first rule.

2. $$X \leftarrow M \rightarrow Y$$

where M is a fork as well and blocks the open path when conditioned for.

3. $$X \rightarrow R \leftarrow M \rightarrow Y$$

where R is an inverted fork and need not be conditioned for to block the path by the second rule. But, Conditioning the fork M is enough to block this path even when conditioned on $R = 1$ for CCA.

Hence,

$$X \perp_d Y \mid M, S, R = 1$$

meaning there are X and Y are statistically independent when adjusted for $M, S, R = 1$. And if we see a statistical association in the complete case analysis, we can say that CCA is a valid test to prove that there is a direct causal effect from X to Y.

Now, for Figure 1D:

The open paths from X to Y, assuming that there is no direct arrow from X to Y:

1. $$X \leftarrow S \rightarrow Y$$

2. $$X \leftarrow M \rightarrow Y$$

3.

$$X \leftarrow M \rightarrow R \leftarrow Y$$

and they can all be blocked by conditioning on M, S, even when conditioning on $R = 1$ for CCA. Hence, again

$$X \perp_d Y \mid M, S, R = 1$$

meaning they are statistically independent when adjusted for M, S, R. And if we see a statistical association in the complete case analysis we can say that there is a direct causal effect from X to Y. Even though, CCA is biased for Figure 1D, I would say that it can be used to prove the causal effect by statistical association, but I would think maybe not for estimating the causal effect.

Question 9

Now, we will actually look at estimating the causal effect of X on the Y, given $M, S, R = 1$. We want to see if CCA can provide us an unbiased estimate of this causal effect for Figure 1C and 1D.

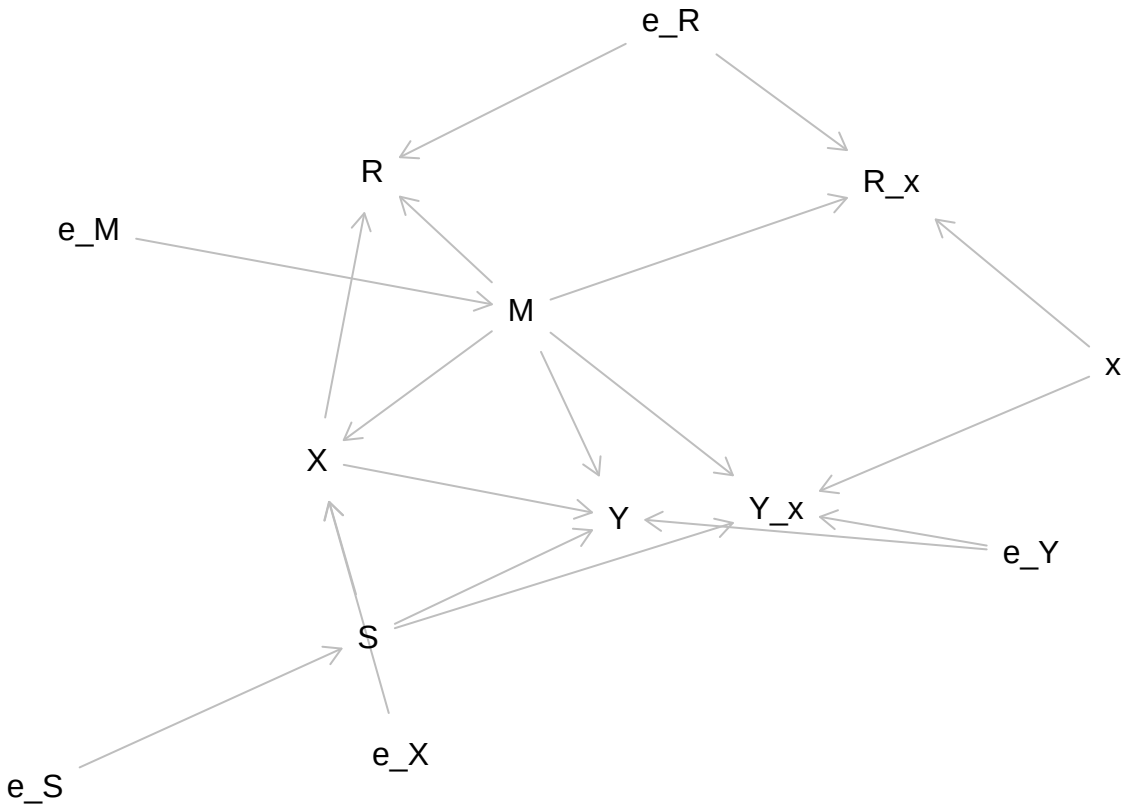
Let us check the counterfactual diagrams for Figure 1C and 1D, where we fix the exposure to a constant x. In these counterfactual diagrams, we will check if X is conditionally d-separated to Y_x, which can prove that conditional association is identical to conditional causation.

Figure 1C:

As R and Y both are associated directly with X, we introduce both Y_x and R_x to the counterfactual diagram:

The NPSEM for the counterfactual diagram looks like this:

$$\left.\begin{aligned} S &:= f_S(\varepsilon_S) \\ M &:= f_M(\varepsilon_M) \\ X &:= f_X(M, S, \varepsilon_X) \\ Y &:= f_Y(X, M, S, \varepsilon_Y) \\ R &:= f_R(M, X, \varepsilon_R) \\ Y_x &:= f_Y(x, M, S, \varepsilon_Y) \\ R_x &:= f_R(M, x, \varepsilon_R) \end{aligned}\right\}$$



Now, let us look at the open paths between X and Y_x:

1. $$X \leftarrow M \rightarrow Y_x$$
   where M is a fork and needs to be conditioned on to block the path.
2. $$X \leftarrow S \rightarrow Y_x$$
   where S is a fork and needs to be conditioned on to block the path.
3. $$X \rightarrow R \leftarrow M \rightarrow Y_x$$
   where R is an inverted fork and shouldn't be conditioned on to block the path. The path is blocked by conditioning on M, even when conditioning on R.
4. $$X \rightarrow Y \leftarrow e_Y \rightarrow Y_x$$
   where Y is an inverted fork and shouldn't be conditioned on to block the path. Hence, this path is blocked by not conditioning on Y.
5. $$X \leftarrow M \rightarrow Y \leftarrow e_Y \rightarrow Y_x$$
   also blocked by conditioning on fork M and not conditioning on Y.
6. $$X \leftarrow S \rightarrow Y \leftarrow e_Y \rightarrow Y_x$$
   also blocked by conditioning on fork S and not conditioning on Y.
7. $$X \rightarrow R \leftarrow e_R \rightarrow R_x \leftarrow M \rightarrow Y_x$$
   also blocked by conditioning on fork M alone.

8.
$$X \leftarrow M \rightarrow R_x \leftarrow x \rightarrow Y_x$$
also blocked by conditioning on fork M alone.

9.
$$X \rightarrow R \leftarrow M \rightarrow Y \leftarrow e_Y \rightarrow Y_x$$
also blocked by conditioning on fork M alone and not conditioning on inverted fork Y.

and other open paths with more similar loops are all blocked by conditioning on $M, S, R = 1$ and not conditioning on Y.

Hence, we can say that:

$$X \perp_d Y_x \mid M, S, R = 1$$

.

This proves that the associational risk ratio between cannabis use and the outcome when adjusting for maternal substance use and sex is identical to the conditional causal risk ratio in complete case analysis for Figure 1C.

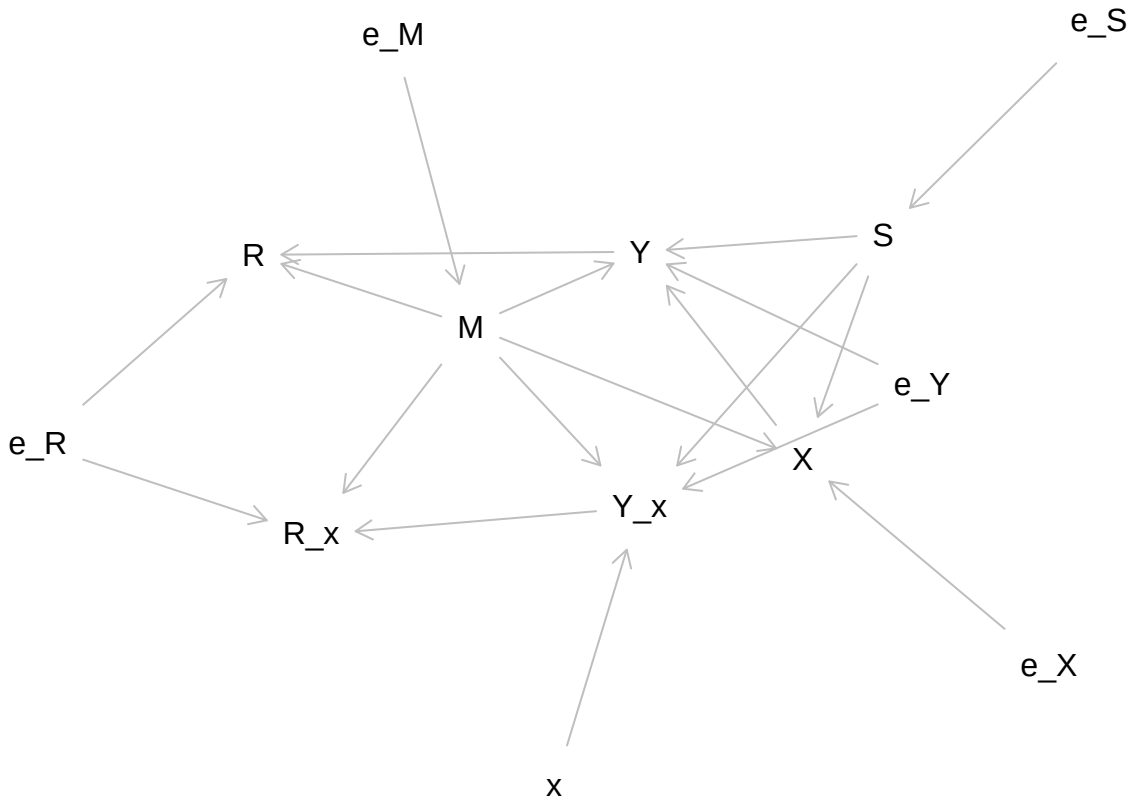
$$\frac{P(Y = 1|X = 1, M, S, R = 1)}{P(Y = 1|X = 0, M, S, R = 1)} = \frac{P(Y_1 = 1|M, S, R = 1)}{P(Y_0 = 1|M, S, R = 1)}$$

Figure 1D:

As only Y is associated directly with X, we introduce Y_x to the diagram, and then by association of R with Y_x, we also introduce R_x.

The NPSEM for the counterfactual diagram looks like this:

$$\left.\begin{array}{l} S := f_S(\varepsilon_S) \\ M := f_M(\varepsilon_M) \\ X := f_X(M, S, \varepsilon_X) \\ Y := f_Y(X, M, S, \varepsilon_Y) \\ R := f_R(M, Y, \varepsilon_R) \\ Y_x := f_Y(x, M, S, \varepsilon_Y) \\ R_x := f_R(M, Y_x, \varepsilon_R) \end{array}\right\}$$



Now, let us look at the open paths between X and Y_x and if they can be blocked by conditioning on $M, S, R = 1$:

One path stands out in this counterfactual diagram in contrast with the one from Figure 1C:

$$X \rightarrow Y \rightarrow R \leftarrow e_R \rightarrow R_x \leftarrow Y_x$$

Here, conditioning on R will open up this path and it cannot be blocked.

Hence, we can say that not all paths from X to Y_x are blocked when conditioned on $M, S, R = 1$.

Therefore,

$$X \not\perp_d Y_x \mid M, S, R = 1$$

.

This proves that the associational risk ratio between cannabis use and the outcome when adjusting for maternal substance use and sex is not identical to the conditional causal risk ratio in complete case analysis for Figure 1D.

$$\frac{P(Y = 1|X = 1, M, S, R = 1)}{P(Y = 1|X = 0, M, S, R = 1)} \neq \frac{P(Y_1 = 1|M, S, R = 1)}{P(Y_0 = 1|M, S, R = 1)}$$

The assignment took 24 hours to complete.