6 The Haunted DAG & The Causal Terror

It seems like the most newsworthy scientific studies are the least trustworthy. The more likely it is to kill you, if true, the less likely it is to be true. The more boring the topic, the more rigorous the results. How could this widely believed negative correlation exist? There doesn't seem to be any reason for studies of topics that people care about to produce less reliable results. Maybe popular topics attract more and worse researchers, like flies drawn to the smell of honey?

Actually all that is necessary for such a negative correlation to arise is that peer reviewers care about both newsworthiness and trustworthiness. Whether it is grant review or journal review, if editors and reviewers care about both, then the act of selection itself is enough to make the most newsworthy studies the least trustworthy. In fact, it's hard to imagine how scientific peer review could avoid creating this negative correlation. And, dear reader, this fact will help us understand the perils of multiple regression.

Here's a simple simulation to illustrate the point[85]. Suppose a grant review panel receives 200 proposals for scientific research. Among these proposals, there is no correlation at all between trustworthiness (rigor, scholarship, plausibility of success) and newsworthiness (social welfare value, public interest). The panel weighs trustworthiness and newsworthiness equally. Then they rank the proposals by their combined scores and select the top 10% for funding.

At the end of this section, I show the code to simulate this thought experiment. FIGURE 6.1 displays the full sample of simulated proposals, with those selected in blue. I've drawn a simple linear regression line through the selected proposals. There's the negative correlation, -0.77 in this example. Strong selection induces a negative correlation among the criteria used in selection. Why? If the only way to cross the threshold is to score high, it is more common to score high on one item than on both. Therefore among funded proposals, the most newsworthy studies can actually have less than average trustworthiness (less than 0 in the figure). Similarly the most trustworthy studies can actually be less newsworthy than average.

This general phenomenon has been recognized for a long time. It is sometimes called BERKSON'S PARADOX[86]. But it is easier to remember if we call it the *selection-distortion effect*. Once you appreciate this effect, you'll see it everywhere. Why do so many restaurants in good locations have bad food? The only way a restaurant with less-than-good food can survive is if it is in a nice location. Similarly, restaurants with excellent food can survive even in bad locations. Selection-distortion ruins your city.

What does this have to do with multiple regression? Unfortunately, everything. The previous chapter demonstrated some amazing powers of multiple regression. It can smoke

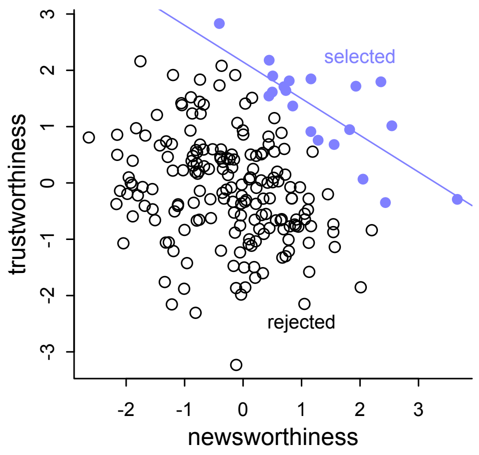


FIGURE 6.1. Why the most newsworthy studies might be the least trustworthy. 200 research proposals are ranked by combined trustworthiness and newsworthiness. The top 10% are selected for funding. While there is no correlation before selection, the two criteria are strongly negatively correlated after selection. The correlation here is -0.77.

out spurious correlations and clear up masking effects. This may encourage the view that, when in doubt, just add everything to the model and let the oracle of regression sort it out.

Regression will not sort it out. Regression is indeed an oracle, but a cruel one. It speaks in riddles and delights in punishing us for asking bad questions. The selection-distortion effect can happen inside of a multiple regression, because the act of adding a predictor induces statistical selection within the model, a phenomenon that goes by the unhelpful name **COLLIDER BIAS**. This can mislead us into believing, for example, that there is a negative association between newsworthiness and trustworthiness in general, when in fact it is just a consequence of conditioning on some variable. This is both a deeply confusing fact and one that is important to understand in order to regress responsibly.

This chapter and the next are both about terrible things that can happen when we simply add variables to a regression, without a clear idea of a causal model. In this chapter, we'll explore three different hazards: multicollinearity, post-treatment bias, and collider bias. We'll end by tying all of these examples together in a framework that can tell us which variables we must and must not add to a model in order to arrive at valid inferences. But this framework does not do the most important step for us: It will not give us a valid model.

Overthinking: Simulated science distortion. Simulations like this one are easy to do in R, or in any other scripting language, once you have seen a few examples. In this simulation, we just draw some random Gaussian criteria for a sample of proposals and then select the top 10% combined scores.

R code 6.1

```
set.seed(1914)
N <- 200 # num grant proposals
p <- 0.1 # proportion to select
# uncorrelated newsworthiness and trustworthiness
nw <- rnorm(N)
tw <- rnorm(N)
# select top 10% of combined scores
s <- nw + tw  # total score
q <- quantile( s , 1-p ) # top 10% threshold
selected <- ifelse( s >= q , TRUE , FALSE )
cor( tw[selected] , nw[selected] )
```

I chose a specific seed so you can replicate the result in Figure 6.1, but if you rerun the simulation without the `set.seed` line, you'll see there is nothing special about the seed I used.

6.1. Multicollinearity

It is commonly true that there are many potential predictor variables to add to a regression model. In the case of the primate milk data, for example, there are 7 variables available to predict any column we choose as an outcome. Why not just fit a model that includes all 7? There are several hazards. The one we'll focus on here is **MULTICOLLINEARITY**. Multicollinearity means very strong correlation between two or more predictor variables. The consequence of it is that the posterior distribution will seem to suggest that none of the variables is reliably associated with the outcome, even if all of the variables are in reality strongly associated with the outcome. This frustrating phenomenon arises from the details of how multiple regression works. In fact, there is nothing wrong with multicollinearity. The model will work fine for prediction. You will just be frustrated trying to understand it. The hope is that once you understand multicollinearity, you will better understand regression models in general.

Let's begin with a simple simulation. Then we'll turn to the primate milk data again and see multicollinearity in a real data set.

6.1.1. Multicollinear legs. The simulation example is predicting an individual's height using the length of his or her legs as predictor variables. Surely height is positively associated with leg length, or at least the simulation will assume it is. Nevertheless, once you put both leg lengths into the model, something vexing will happen.

The code below will simulate the heights and leg lengths of 100 individuals. For each, first a height is simulated from a Gaussian distribution. Then each individual gets a simulated proportion of height for their legs, ranging from 0.4 to 0.5. Finally, each leg is salted with a little measurement or developmental error, so the left and right legs are not exactly the same length, as is typical in real populations. At the end, the code puts height and the two leg lengths into a common data frame.

R code 6.2
```r
N <- 100                          # number of individuals
set.seed(909)
height <- rnorm(N,10,2)           # sim total height of each
leg_prop <- runif(N,0.4,0.5)      # leg as proportion of height
leg_left <- leg_prop*height +     # sim left leg as proportion + error
    rnorm( N , 0 , 0.02 )
leg_right <- leg_prop*height +    # sim right leg as proportion + error
    rnorm( N , 0 , 0.02 )
                                  # combine into data frame
d <- data.frame(height,leg_left,leg_right)
```

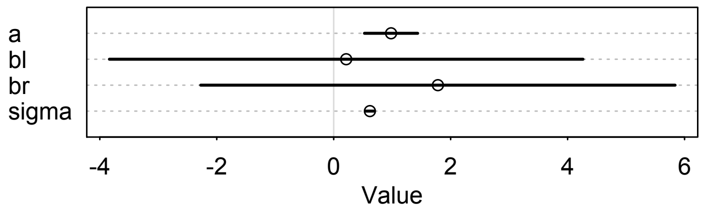
Now let's analyze these data, predicting the outcome `height` with both predictors, `leg_left` and `leg_right`. Before approximating the posterior, however, consider what we expect. On average, an individual's legs are 45% of their height (in these simulated data). So we should expect the beta coefficient that measures the association of a leg with height to end up around the average height (10) divided by 45% of the average height (4.5). This is $10/4.5 \approx 2.2$. Now let's see what happens instead. I'll use very vague, bad priors here, just so we can be sure that the priors aren't responsible for what is about to happen.

R code 6.3
```
m6.1 <- quap(
    alist(
        height ~ dnorm( mu , sigma ) ,
        mu <- a + bl*leg_left + br*leg_right ,
        a ~ dnorm( 10 , 100 ) ,
        bl ~ dnorm( 2 , 10 ) ,
        br ~ dnorm( 2 , 10 ) ,
        sigma ~ dexp( 1 )
    ) ,
    data=d )
precis(m6.1)
```

```
      mean   sd  5.5% 94.5%
a     0.98 0.28  0.53  1.44
bl    0.21 2.53 -3.83  4.25
br    1.78 2.53 -2.26  5.83
sigma 0.62 0.04  0.55  0.69
```

Those posterior means and standard deviations look crazy. This is a case in which a graphical view of the `precis` output is more useful, because it displays the posterior means and 89% intervals in a way that allows us with a glance to see that something has gone wrong here:

R code 6.4
```
plot(precis(m6.1))
```



Go ahead and try the simulate a few more times, omitting the `set.seed` line. If both legs have almost identical lengths, and height is so strongly associated with leg length, then why is this posterior distribution so weird? Did the posterior approximation work correctly?

It did work correctly, and the posterior distribution here is the right answer to the question we asked. The problem is the question. Recall that a multiple linear regression answers the question: *What is the value of knowing each predictor, after already knowing all of the other predictors?* So in this case, the question becomes: *What is the value of knowing each leg's length, after already knowing the other leg's length?*

The answer to this weird question is equally weird, but perfectly logical. The posterior distribution is the answer to this question, considering every possible combination of the parameters and assigning relative plausibilities to every combination, conditional on this model and these data. It might help to look at the bivariate posterior distribution for `bl` and `br`:

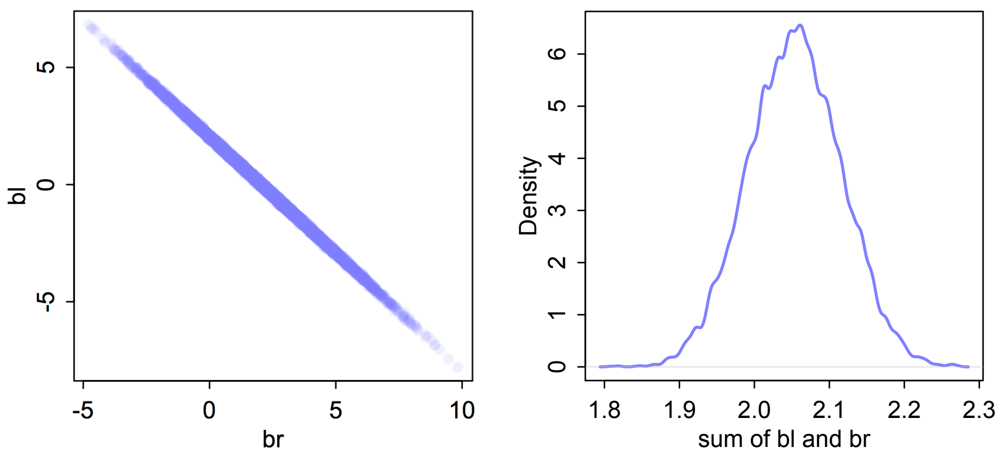


FIGURE 6.2. Left: Posterior distribution of the association of each leg with height, from model `m6.1`. Since both variables contain almost identical information, the posterior is a narrow ridge of negatively correlated values. Right: The posterior distribution of the sum of the two parameters is centered on the proper association of either leg with height.

R code 6.5

```
post <- extract.samples(m6.1)
plot( bl ~ br , post , col=col.alpha(rangi2,0.1) , pch=16 )
```

The resulting plot is shown on the left of FIGURE 6.2. The posterior distribution for these two parameters is very highly correlated, with all of the plausible values of `bl` and `br` lying along a narrow ridge. When `bl` is large, then `br` must be small. What has happened here is that since both leg variables contain almost exactly the same information, if you insist on including both in a model, then there will be a practically infinite number of combinations of `bl` and `br` that produce the same predictions.

One way to think of this phenomenon is that you have approximated this model:

$$\begin{aligned} y_i &\sim \text{Normal}(\mu_i, \sigma) \\ \mu_i &= \alpha + \beta_1 x_i + \beta_2 x_i \end{aligned}$$

The variable y is the outcome, like height in the example, and x is a single predictor, like the leg lengths in the example. Here x is used twice, which is a perfect example of the problem caused by using the almost-identical leg lengths. From the computer's perspective, this model is simply:

$$\begin{aligned} y_i &\sim \text{Normal}(\mu_i, \sigma) \\ \mu_i &= \alpha + (\beta_1 + \beta_2) x_i \end{aligned}$$

All I've done is factor x_i out of each term. The parameters β_1 and β_2 cannot be pulled apart, because they never separately influence the mean μ. Only their sum, $\beta_1 + \beta_2$, influences μ. So this means the posterior distribution ends up reporting the very large range of combinations of β_1 and β_2 that make their sum close to the actual association of x with y.

And the posterior distribution in this simulated example has done exactly that: It has produced a good estimate of the sum of `bl` and `br`. Here's how you can compute the posterior distribution of their sum, and then plot it:

R code 6.6
```
sum_blbr <- post$bl + post$br
dens( sum_blbr , col=rangi2 , lwd=2 , xlab="sum of bl and br" )
```

And the resulting density plot is shown on the right-hand side of FIGURE 6.2. The posterior mean is in the right neighborhood, a little over 2, and the standard deviation is much smaller than it is for either component of the sum, `bl` or `br`. If you fit a regression with only one of the leg length variables, you'll get approximately the same posterior mean:

R code 6.7
```
m6.2 <- quap(
    alist(
        height ~ dnorm( mu , sigma ) ,
        mu <- a + bl*leg_left,
        a ~ dnorm( 10 , 100 ) ,
        bl ~ dnorm( 2 , 10 ) ,
        sigma ~ dexp( 1 )
    ) ,
    data=d )
precis(m6.2)
```

```
      mean   sd 5.5% 94.5%
a     1.00 0.28 0.54  1.45
bl    1.99 0.06 1.89  2.09
sigma 0.62 0.04 0.55  0.69
```

That 1.99 is almost identical to the mean value of `sum_blbr`.

You'll get slightly different results in your own simulation, due to random variation across simulations. But the basic lesson remains intact across different simulations: *When two predictor variables are very strongly correlated, including both in a model may lead to confusion*. The posterior distribution isn't wrong, in such cases. It's telling you that the question you asked cannot be answered with these data. And that's a great thing for a model to say, that it cannot answer your question. And if you are just interested in prediction, you'll find that this leg model makes fine predictions. It just doesn't make any claims about which leg is more important.

This leg example is clear and cute. But it is also purely statistical. We aren't asking any serious causal questions here. Let's try a more causally interesting example next.

6.1.2. Multicollinear milk. In the leg length example, it's easy to see that including both legs in the model is a little silly. But the problem that arises in real data sets is that we may not anticipate a clash between highly correlated predictors. And therefore we may mistakenly read the posterior distribution to say that neither predictor is important. In this section, we look at an example of this issue with real data.

Let's return to the primate milk data from earlier in the chapter. Let's get back the original data again:

R code 6.8
```
library(rethinking)
data(milk)
d <- milk
d$K <- scale( d$kcal.per.g )
d$F <- scale( d$perc.fat )
d$L <- scale( d$perc.lactose )
```

In this example, we are concerned with the variables `perc.fat` (percent fat) and `perc.lactose` (percent lactose) that we might use to model the total energy content, `kcal.per.g`. The code above has already standardized these three variables. You're going to use these three variables to explore a natural case of multicollinearity. Note that there are no missing values, `NA`, in these columns, so there's no need here to extract complete cases. But you can rest assured that `quap`, unlike reckless functions like `lm`, would never silently drop cases.

Start by modeling `kcal.per.g` as a function of `perc.fat` and `perc.lactose`, but in two bivariate regressions. Look back in Chapter 5 (page 151), for a discussion of these priors.

R code 6.9
```
# kcal.per.g regressed on perc.fat
m6.3 <- quap(
    alist(
        K ~ dnorm( mu , sigma ) ,
        mu <- a + bF*F ,
        a ~ dnorm( 0 , 0.2 ) ,
        bF ~ dnorm( 0 , 0.5 ) ,
        sigma ~ dexp( 1 )
    ) , data=d )

# kcal.per.g regressed on perc.lactose
m6.4 <- quap(
    alist(
        K ~ dnorm( mu , sigma ) ,
        mu <- a + bL*L ,
        a ~ dnorm( 0 , 0.2 ) ,
        bL ~ dnorm( 0 , 0.5 ) ,
        sigma ~ dexp( 1 )
    ) , data=d )

precis( m6.3 )
precis( m6.4 )
```

```
      mean   sd  5.5% 94.5%
a     0.00 0.08 -0.12  0.12
bF    0.86 0.08  0.73  1.00
sigma 0.45 0.06  0.36  0.54

       mean   sd  5.5% 94.5%
a      0.00 0.07 -0.11  0.11
bL    -0.90 0.07 -1.02 -0.79
sigma  0.38 0.05  0.30  0.46
```

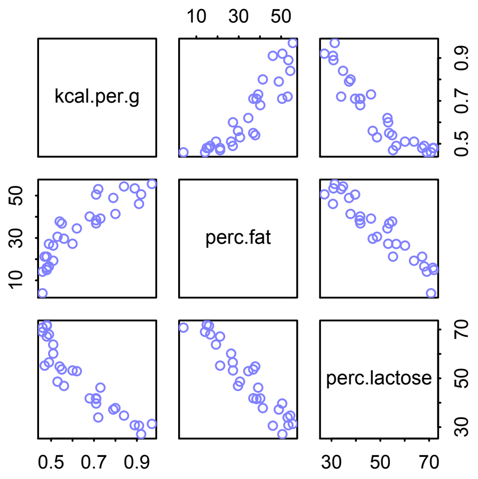


FIGURE 6.3. A pairs plot of the total energy, percent fat, and percent lactose variables from the primate milk data. Percent fat and percent lactose are strongly negatively correlated with one another, providing mostly the same information.

The posterior distributions for bF and bL are essentially mirror images of one another. The posterior mean of bF is as positive as the mean of bL is negative. Both are narrow posterior distributions that lie almost entirely on one side or the other of zero. Given the strong association of each predictor with the outcome, we might conclude that both variables are reliable predictors of total energy in milk, across species. The more fat, the more kilocalories in the milk. The more lactose, the fewer kilocalories in milk. But watch what happens when we place both predictor variables in the same regression model:

R code 6.10

```
m6.5 <- quap(
    alist(
        K ~ dnorm( mu , sigma ) ,
        mu <- a + bF*F + bL*L ,
        a ~ dnorm( 0 , 0.2 ) ,
        bF ~ dnorm( 0 , 0.5 ) ,
        bL ~ dnorm( 0 , 0.5 ) ,
        sigma ~ dexp( 1 )
    ) ,
    data=d )
precis( m6.5 )
```

```
       mean   sd  5.5% 94.5%
a      0.00 0.07 -0.11  0.11
bF     0.24 0.18 -0.05  0.54
bL    -0.68 0.18 -0.97 -0.38
sigma  0.38 0.05  0.30  0.46
```

Now the posterior means of both bF and bL are closer to zero. And the standard deviations for both parameters are twice as large as in the bivariate models (m6.3 and m6.4).

This is the same statistical phenomenon as in the leg length example. What has happened is that the variables perc.fat and perc.lactose contain much of the same information.

They are almost substitutes for one another. As a result, when you include both in a regression, the posterior distribution ends up describing a long ridge of combinations of `bF` and `bL` that are equally plausible. In the case of the fat and lactose, these two variables form essentially a single axis of variation. The easiest way to see this is to use a pairs plot:

R code 6.11

```
pairs( ~ kcal.per.g + perc.fat + perc.lactose , data=d , col=rangi2 )
```

I display this plot in FIGURE 6.3. Along the diagonal, the variables are labeled. In each scatterplot off the diagonal, the vertical axis variable is the variable labeled on the same row and the horizontal axis variable is the variable labeled in the same column. For example, the two scatterplots in the first row in FIGURE 6.3 are `kcal.per.g` (vertical) against `perc.fat` (horizontal) and then `kcal.per.g` (vertical) against `perc.lactose` (horizontal). Notice that percent fat is positively correlated with the outcome, while percent lactose is negatively correlated with it. Now look at the right-most scatterplot in the middle row. This plot is the scatter of percent fat (vertical) against percent lactose (horizontal). Notice that the points line up almost entirely along a straight line. These two variables are negatively correlated, and so strongly so that they are nearly redundant. Either helps in predicting `kcal.per.g`, but neither helps much *once you already know the other*.

In the scientific literature, you might encounter a variety of dodgy ways of coping with multicollinearity. Few of them take a causal perspective. Some fields actually teach students to inspect pairwise correlations before fitting a model, to identify and drop highly correlated predictors. This is a mistake. Pairwise correlations are not the problem. It is the conditional associations—not correlations—that matter. And even then, the right thing to do will depend upon what is causing the collinearity. The associations within the data alone are not enough to decide what to do.

What is likely going on in the milk example is that there is a core tradeoff in milk composition that mammal mothers must obey. If a species nurses often, then the milk tends to be watery and low in energy. Such milk is high in sugar (lactose). If instead a species nurses rarely, in short bouts, then the milk needs to be higher in energy. Such milk is very high in fat. This implies a causal model something like this:

$$\begin{tikzcd}
L \arrow[dr] & \textcircled{D} \arrow[l] \arrow[r] & F \arrow[dl] \\
& K &
\end{tikzcd}$$

The central tradeoff decides how dense, *D*, the milk needs to be. We haven't observed this variable, so it's shown circled. Then fat, *F*, and lactose, *L*, are determined. Finally, the composition of *F* and *L* determines the kilocalories, *K*. If we could measure *D*, or had an evolutionary and economic model to predict it based upon other aspects of a species, that would be better than stumbling through regressions. We'd just regression *K* on *D*, ignoring the mediating *L* and *F*, to estimate the causal influence of density on energy.

The problem of multicollinearity is a member of a family of problems with fitting models, a family sometimes known as **NON-IDENTIFIABILITY**. When a parameter is non-identifiable, it means that the structure of the data and model do not make it possible to estimate the parameter's value. Sometimes this problem arises from mistakes in coding a model, but many important types of models present non-identifiable or weakly identifiable parameters, even when coded completely correctly. Nature does not owe us easy inference, even when the model is correct.

In general, there's no guarantee that the available data contain much information about a parameter of interest. When that's true, your Bayesian machine will return a posterior distribution very similar to the prior. Comparing the posterior to the prior can therefore be a good idea, a way of seeing how much information the model extracted from the data. When the posterior and prior are similar, it doesn't mean the calculations are wrong—you got the right answer to the question you asked. But it might lead you to ask a better question.

Rethinking: Identification guaranteed; comprehension up to you. Technically speaking, *identifiability* is not a concern for Bayesian models. The reason is that as long as the posterior distribution is proper—which just means that it integrates to 1—then all of the parameters are identified. But this technical fact doesn't also mean that you can make sense of the posterior distribution. So it's probably better to speak of *weakly identified* parameters in a Bayesian context. There will be several examples as the book progresses.

Overthinking: Simulating collinearity. The code to produce Figure ?? involves writing a function that generates correlated predictors, fits a model, and returns the standard deviation of the posterior distribution for the slope relating `perc.fat` to `kcal.per.g`. Then the code repeatedly calls this function, with different degrees of correlation as input, and collects the results.

R code 6.12
```
library(rethinking)
data(milk)
d <- milk
sim.coll <- function( r=0.9 ) {
    d$x <- rnorm( nrow(d) , mean=r*d$perc.fat ,
        sd=sqrt( (1-r^2)*var(d$perc.fat) ) )
    m <- lm( kcal.per.g ~ perc.fat + x , data=d )
    sqrt( diag( vcov(m) ) )[2] # stddev of parameter
}
rep.sim.coll <- function( r=0.9 , n=100 ) {
    stddev <- replicate( n , sim.coll(r) )
    mean(stddev)
}
r.seq <- seq(from=0,to=0.99,by=0.01)
stddev <- sapply( r.seq , function(z) rep.sim.coll(r=z,n=100) )
plot( stddev ~ r.seq , type="l" , col=rangi2, lwd=2 , xlab="correlation" )
```

So for each correlation value in `r.seq`, the code generates 100 regressions and returns the average standard deviation from them. This code uses implicit flat priors, which are bad priors. So it does exaggerate the effect of collinear variables. When you use informative priors, the inflation in standard deviation can be much slower.

6.2. Post-treatment bias

It is routine to worry about mistaken inferences that arise from omitting predictor variables. Such mistakes are often called **omitted variable bias**, and the examples from the previous chapter illustrate it. It is much less routine to worry about mistaken inferences arising from including variables that are consequences of other variables. We'll call this **post-treatment bias**.[87] Being aware of post-treatment bias is important in all types of studies.

Carefully controlled experiments can be ruined just as easily as uncontrolled observational studies. Blindly tossing variables into the causal salad is never a good idea, no matter how the data were collected.

The language "post-treatment" comes in fact from thinking about experimental designs. Suppose for example that you are growing some plants in a greenhouse. You want to know the difference in growth under different anti-fungal soil treatments, because fungus on the plants tends to reduce their growth. Plants are initially seeded and sprout. Their heights are measured. Then different soil treatments are applied. Final measures are the height of the plant and the presence of fungus. There are four variables of interest here: initial height, final height, treatment, and presence of fungus. Final height is the outcome of interest. But which of the other variables should be in the model? If your goal is to make a causal inference about the treatment, you shouldn't include the presence of fungus, because it is a *post-treatment* effect.

Let's simulate some data, to make the example more transparent and see what exactly goes wrong when we include a post-treatment variable.

R code 6.13

```
set.seed(71)
# number of plants
N <- 100

# simulate initial heights
h0 <- rnorm(N,10,2)

# assign treatments and simulate fungus and growth
treatment <- rep( 0:1 , each=N/2 )
fungus <- rbinom( N , size=1 , prob=0.5 - treatment*0.4 )
h1 <- h0 + rnorm(N, 5 - 3*fungus)

# compose a clean data frame
d <- data.frame( h0=h0 , h1=h1 , treatment=treatment , fungus=fungus )
precis(d)
```

```
           mean   sd  5.5% 94.5%    histogram
h0         9.96 2.10  6.57 13.08  ▁▂▂▂▇▃▂▃▁▁▁▁
h1        14.40 2.69 10.62 17.93     ▁▁▃▇▇▇▁▁
treatment  0.50 0.50  0.00  1.00   ▇▁▁▁▁▁▁▁▁▇
fungus     0.23 0.42  0.00  1.00   ▇▁▁▁▁▁▁▁▁▂
```

Now you should have a data frame `d` with the simulated plant experiment data.

6.2.1. A prior is born.

When designing the model, it helps to pretend you don't have the data generating process just above. In real research, you will not know the real data generating process. But you will have a lot of scientific information to guide model construction. So let's spend some time taking this mock analysis seriously.

We know that the plants at time $t = 1$ should be taller than at time $t = 0$, whatever scale they are measured on. So if we put the parameters on a scale of *proportion* of height at time $t = 0$, rather than on the absolute scale of the data, we can set the priors more easily. To make this simpler, let's focus right now only on the height variables, ignoring the predictor variables. We might have a linear model like:

$$
\begin{aligned}
h_{1,i} &\sim \text{Normal}(\mu_i, \sigma) \\
\mu_i &= h_{0,i} \times p
\end{aligned}
$$

where $h_{0,i}$ is plant i's height at time $t = 0$, $h_{1,i}$ is its height at time $t = 1$, and p is a parameter measuring the proportion of $h_{0,i}$ that $h_{1,i}$ is. More precisely, $p = h_{1,i}/h_{0,i}$. If $p = 1$, the plant hasn't changed at all from time $t = 0$ to time $t = 1$. If $p = 2$, it has doubled in height. So if we center our prior for p on 1, that implies an expectation of no change in height. That is less than we know. But we should allow p to be less than 1, in case the experiment goes horribly wrong and we kill all the plants. We also have to ensure that $p > 0$, because it is a proportion. Back in Chapter 4 (page 4.4.1.3), we used a Log-Normal distribution, because it is always positive. Let's use one again. If we use $p \sim \text{Log-Normal}(0, 0.25)$, the prior distribution looks like:

R code 6.14

```r
sim_p <- rlnorm( 1e4 , 0 , 0.25 )
precis( data.frame(sim_p) )
```

```
'data.frame': 10000 obs. of 1 variables:
      mean   sd 5.5% 94.5%   histogram
sim_p 1.03 0.26 0.67  1.48 ▁▃▇▇▃▁▁▁▁▁▁
```

So this prior expects anything from 40% shrinkage up to 50% growth. Let's fit this model, so you can see how it just measures the average growth in the experiment.

R code 6.15

```r
m6.6 <- quap(
    alist(
        h1 ~ dnorm( mu , sigma ),
        mu <- h0*p,
        p ~ dlnorm( 0 , 0.25 ),
        sigma ~ dexp( 1 )
    ), data=d )
precis(m6.6)
```

```
      mean   sd 5.5% 94.5%
p     1.43 0.02 1.40  1.45
sigma 1.79 0.13 1.59  1.99
```

About 40% growth, on average. Now to include the treatment and fungus variables. We'll include both of them, following the notion that we'd like to measure the impact of both the treatment and the fungus itself. The parameters for these variables will also be on the proportion scale. They will be *changes* in proportion growth. So we're going to make a linear model of p now.

$$\begin{aligned}
h_{1,i} &\sim \text{Normal}(\mu_i, \sigma) \\
\mu_i &= h_{0,i} \times p \\
p &= \alpha + \beta_T T_i + \beta_F F_i \\
\alpha &\sim \text{Log-Normal}(0, 0.25) \\
\beta_T &\sim \text{Normal}(0, 0.5) \\
\beta_F &\sim \text{Normal}(0, 0.5) \\
\sigma &\sim \text{Exponential}(1)
\end{aligned}$$

The proportion of growth p is now a function of the predictor variables. It looks like any other linear model. The priors on the slopes are almost certainly too flat. They place 95% of the prior mass between -1 (100% reduction) and $+1$ (100% increase) and two-thirds of the prior mass between -0.5 and $+0.5$. After we finish this section, you may want to loop back and try simulating from these priors. Here's the code to approximate the posterior:

R code 6.16

```
m6.7 <- quap(
    alist(
        h1 ~ dnorm( mu , sigma ),
        mu <- h0 * p,
        p <- a + bt*treatment + bf*fungus,
        a ~ dlnorm( 0 , 0.2 ) ,
        bt ~ dnorm( 0 , 0.5 ),
        bf ~ dnorm( 0 , 0.5 ),
        sigma ~ dexp( 1 )
    ), data=d )
precis(m6.7)
```

```
       mean   sd  5.5% 94.5%
a      1.48 0.02  1.44  1.52
bt     0.00 0.03 -0.05  0.05
bf    -0.27 0.04 -0.33 -0.21
sigma  1.41 0.10  1.25  1.57
```

That `a` parameter is the same as `p` before. And it has nearly the same posterior. The marginal posterior for `bt`, the effect of treatment, is solidly zero, with a tight interval. The treatment is not associated with growth. The fungus seems to have hurt growth, however. Given that we know the treatment matters, because we built the simulation that way, what happened here?

6.2.2. Blocked by consequence. The problem is that `fungus` is mostly a consequence of `treatment`. This is to say that `fungus` is a post-treatment variable. So when we control for `fungus`, the model is implicitly answering the question: *Once we already know whether or not a plant developed fungus, does soil treatment matter?* The answer is "no," because soil treatment has its effects on growth through reducing fungus. But we actually want to know, based on the design of the experiment, is the impact of treatment on growth. To measure this properly, we should omit the post-treatment variable `fungus`. Here's what the inference looks like in that case:

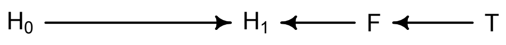
R code 6.17
```
m6.8 <- quap(
    alist(
        h1 ~ dnorm( mu , sigma ),
        mu <- h0 * p,
        p <- a + bt*treatment,
        a ~ dlnorm( 0 , 0.2 ),
        bt ~ dnorm( 0 , 0.5 ),
        sigma ~ dexp( 1 )
    ), data=d )
precis(m6.8)
```

```
      mean   sd 5.5% 94.5%
a     1.38 0.03 1.34  1.42
bt    0.08 0.03 0.03  0.14
sigma 1.75 0.12 1.55  1.94
```

Now the impact of treatment is clearly positive, as it should be. It makes sense to control for pre-treatment differences, like the initial height `h0`, that might mask the causal influence of treatment. But including post-treatment variables can actually mask the treatment itself. This doesn't mean you don't want the model that includes both treatment and fungus. The fact that including `fungus` zeros the coefficient for `treatment` suggests that the treatment works for exactly the anticipated reasons. It tells us about mechanism. But a correct inference about the treatment still depends upon omitting the post-treatment variable.

6.2.3. Fungus and *d*-separation. It helps to look at this problem in terms of a DAG. In this case, I'll show you how to draw it using the `dagitty` R package, because we are going to use that package now to do some graph analysis.

R code 6.18
```
library(dagitty)
plant_dag <- dagitty( "dag {
    H_0 -> H_1
    F -> H_1
    T -> F
}")
coordinates( plant_dag ) <- list( x=c(H_0=0,T=2,F=1.5,H_1=1) ,
                                  y=c(H_0=0,T=0,F=0,H_1=0) )
drawdag( plant_dag )
```


$$\begin{tikzcd} H_0 \arrow[r] & H_1 & F \arrow[l] & T \arrow[l] \end{tikzcd}$$

So the treatment T influences the presence of fungus F which influences plant height at time 1, H_1. Plant height at time 1 is also influenced by plant height at time 0, H_0. That's our DAG. When we include F, the post-treatment effect, in the model, we end up blocking the path from the treatment to the outcome. This is the DAG way of saying that learning the treatment tells us nothing about the outcome, once we know the fungus status.

An even more DAG way to say this is that conditioning on F induces **D-SEPARATION**. The "d" stands for *directional*.[88] *d*-separation means that some variables on a directed graph are independent of others. There is no path connecting them. In this case, H_1 is *d*-separated from

T, but only when we condition on F. Conditioning on F effectively blocks the directed path $T \rightarrow F \rightarrow H_1$, making T and H_1 independent (d-separated). In the previous chapter, you saw the notation $H_1 \perp\!\!\!\perp T|F$ for this kind of statement, when we discussed implied **CONDITIONAL INDEPENDENCIES**. Why does this happen? There is no information in T about H_1 that is not also in F. So once we know F, learning T provides no additional information about H_1. You can query the implied conditional independencies for this DAG:

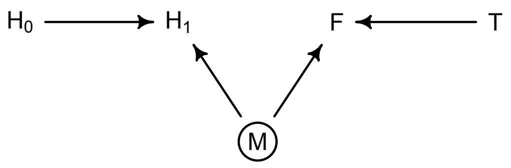
R code 6.19
```
impliedConditionalIndependencies(plant_dag)
```

```
F _||_ H0
H0 _||_ T
H1 _||_ T | F
```

There are three. The third one is the focus of our discussion. But the other two implications provide ways to test the DAG. What $F \perp\!\!\!\perp H_0$ and $H_0 \perp\!\!\!\perp T$ say is that the original plant height, H_0, should not be associated with the treatment T or fungus F, provided we do not condition on anything.

Obviously the problem of post-treatment variables applies just as well to observational studies as it does to experiments. But in experiments, it can be easier to tell which variables are pre-treatment, like `h0`, and which are post-treatment, like `fungus`. In observational studies, it is harder to know. But there are some subtle traps in experiments as well.

For example, conditioning on a post-treatment variable can not only fool you into thinking the treatment doesn't work. It can also fool you into thinking it does work. Consider the DAG below:



In this graph, the treatment T influences fungus F, but fungus doesn't influence plant growth. Maybe the plant species just isn't bothered by this particular fungus. The new variable M is moisture. It influences both H_1 and F. M is circled to indicate that it is unobserved. Any unobserved common cause of H_1 and F will do—it doesn't have to be moisture of course. A regression of H_1 on T will show no association between the treatment and plant growth. But if we include F in the model, suddenly there will be an association. Let's try it. I'll just modify the plant growth simulation so that fungus has no influence on growth, but moisture M influences both H_1 and F:

R code 6.20
```
set.seed(71)
N <- 1000
h0 <- rnorm(N,10,2)
treatment <- rep( 0:1 , each=N/2 )
M <- rbern(N)
fungus <- rbinom( N , size=1 , prob=0.5 - treatment*0.4 + 0.4*M )
h1 <- h0 + rnorm( N , 5 + 3*M )
d2 <- data.frame( h0=h0 , h1=h1 , treatment=treatment , fungus=fungus )
```

Rerun the models from earlier, models `m6.7` and `m6.8`, using the data in `d2` now. You'll see that including fungus again confounds inference about the treatment, this time by making it seem like it helped the plants, even though it had no effect.

This result is rather mysterious. Why should *M* have this effect? The next section is all about effects like this.

Rethinking: Model selection doesn't help. In the next chapter, you'll learn about model selection using information criteria. Like other model comparison and selection schemes, these criteria help in contrasting and choosing model structure. But such approaches are no help in the example presented just above, since the model that includes `fungus` both fits the sample better and would make better out-of-sample predictions. Model `m6.7` misleads because it asks the wrong question, not because it would make poor predictions. As argued in Chapter 1, prediction and causal inference are just not the same task. No statistical procedure can substitute for scientific knowledge and attention to it. We need multiple models because they help us understand causal paths, not just so we can choose one or another for prediction.

6.3. Collider bias

At the start of the chapter, I argued that all that is necessary for scientific studies to show a negative association between trustworthiness and newsworthiness is that selection processes—grant and journal review—care about both. Now I want to explain how this same selection phenomenon can happen inside a statistical model. When it does, it can seriously distort our inferences, a phenomenon known as **COLLIDER BIAS**.

Let's consider a DAG for this example. The model is that trustworthiness (*T*) and newsworthiness (*N*) are statistically independent in the population research proposals submitted to grant review panels. Both of them influence selection (*S*) for funding. This is the graph:

$$\begin{tikzcd} T \arrow[r] & S & N \arrow[l] \end{tikzcd}$$

The fact that two arrows enter *S* means it is a **COLLIDER**. The core concept is easy to understand: When you condition on a collider, it creates statistical—but not necessarily causal—associations among its causes. In this case, once you learn that a proposal has been selected (*S*), then learning its trustworthiness (*T*) also provides information about its newsworthiness (*N*). Why? Because if, for example, a selected proposal has low trustworthiness, then it must have high newsworthiness. Otherwise it wouldn't have been funded. The same works in reverse: If a proposal has low newsworthiness, we'd infer that it must have higher than average trustworthiness. Otherwise it would not have been selected for funding.

This is the informational phenomenon that generates the negative association between *T* and *N* in the population of selected proposals. And it means we have to pay attention to processes that select our sample of observations and may distort associations among variables. But the same phenomenon will also generate a misleading association inside a statistical model, when you include the collider as a predictor variable. If you are not careful, you can make an erroneous causal inference. Let's consider an extended example.

6.3.1. Collider of false sorrow. Consider the question of how aging influences happiness. If we have a large survey of people rating how happy they are, is age associated with happiness?

If so, is that association causal? Here, I want to show you how controlling for a plausible confound of happiness can actually bias inference about the influence of age.[89]

Suppose, just to be provocative, that an individual's average happiness is a trait that is determined at birth and does not change with age. However, happiness does influence events in one's life. One of those events is marriage. Happier people are more likely to get married. Another variable that causally influences marriage is age: The more years you are alive, the more likely you are to eventually get married. Putting these three variables together, this is the causal model:

$$\begin{tikzcd} H \arrow[r] & M & A \arrow[l] \end{tikzcd}$$

Happiness (H) and age (A) both cause marriage (M). Marriage is therefore a collider. Even though there is no causal association between happiness and age, if we condition on marriage—which means here, if we include it as a predictor in a regression—then it will induce a statistical association between age and happiness. And this can mislead us to think that happiness changes with age, when in fact it is constant.

To convince you of this, let's do another simulation. Simulations are useful in these examples, because these are the only times when we know the true causal model. If a procedure cannot figure out the truth in a simulated example, we shouldn't trust it in a real one. We're going to do a fancier simulation this time, using an agent-based model of aging and marriage to produce a simulated data set to use in a regression. Here is the simulation design:

(1) Each year, 20 people are born with uniformly distributed happiness values.
(2) Each year, each person ages one year. Happiness does not change.
(3) At age 18, individuals can become married. The odds of marriage each year are proportional to an individual's happiness.
(4) Once married, an individual remains married.
(5) After age 65, individuals leave the sample. (They move to Spain.)

I've written this algorithm into the `rethinking` package. You can run it out for 1000 years and collect the resulting data:

R code 6.21

```
library(rethinking)
d <- sim_happiness( seed=1977 , N_years=1000 )
precis(d)
```

```
'data.frame': 1300 obs. of 3 variables:
           mean    sd  5.5% 94.5%     histogram
age        33.0 18.77  4.00 62.00 ▇▇▇▇▇▇▇▇▇▇▇▇▇
married     0.3  0.46  0.00  1.00    ▇▁▁▁▁▁▁▁▁▂
happiness   0.0  1.21 -1.79  1.79      ▇▅▇▅▅▇▅▇
```

These data comprise 1300 people of all ages from birth to 65 years old. The variables correspond to the variables in the DAG above, and the simulation itself obeys the DAG.

I've plotted these data in Figure 6.4, showing each individual as a point. Filled points are married individuals. Age is on the horizontal, and happiness the vertical, with the happiest individuals at the top. At age 18, they become able to marry, and then gradually more individuals are married each year. So at older ages, more individuals are married. But at all ages, the happiest individuals are more likely to be married.

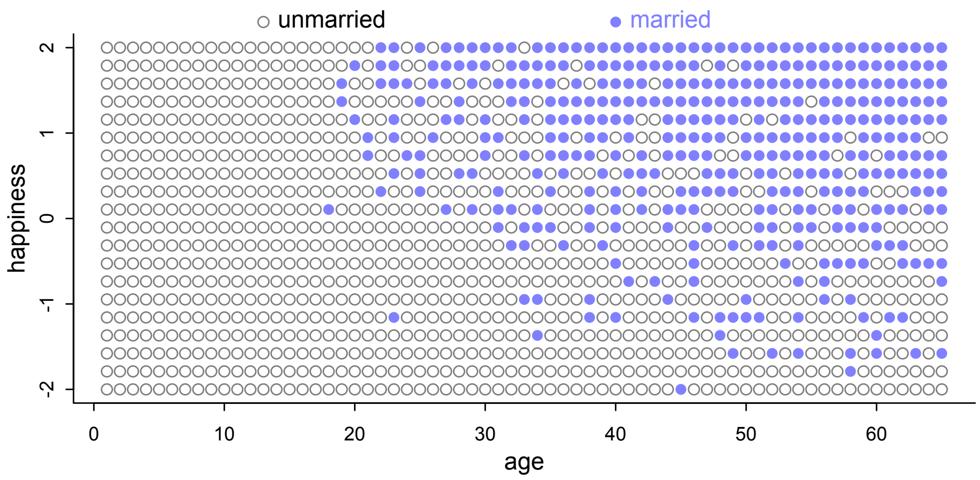


FIGURE 6.4. Simulated data, assuming that happiness is uniformly distributed and never changes. Each point is a person. Married individuals are shown with filled blue points. At each age after 18, the happiest individuals are more likely to be married. At later ages, more individuals tend to be married. Marriage status is a collider of age and happiness: A $\rightarrow$ M $\leftarrow$ H. If we condition on marriage in a regression, it will mislead us to believe that happiness declines with age.

Suppose you come across these data and want to ask whether age is related to happiness. You don't know the true causal model. But you reason, reasonably, that marriage status might be an important confound. If married people are more or less happy, on average, then you need to condition on marriage status in order to infer the relationship between age and happiness.

So let's consider a multiple regression model aimed at inferring the influence of age on happiness, while controlling for marriage status. This is just a plain multiple regression, like the others in this and the previous chapter. The linear model is this:

$$\mu_i = \alpha_{\text{MID}[i]} + \beta_A A_i$$

where MID$[i]$ is an index for the marriage status of individual i, with 1 meaning single and 2 meaning married. This is just the categorical variable strategy from Chapter 4. It's easier to make priors, when we use multiple intercepts, one for each category, than when we use indicator variables.

Now we should do our duty and think about the priors. Let's consider the slope β_A first, because how we scale the predictor A will determine the meaning of the intercept. We'll focus only on the adult sample, those 18 or over. Imagine a very strong relationship between age and happiness, such that happiness is at its maximum at age 18 and its minimum at age 65. It'll be easier if we rescale age so that the range from 18 to 65 is one unit. This will do it:

R code 6.22
```
d2 <- d[ d$age>17 , ] # only adults
d2$A <- ( d2$age - 18 ) / ( 65 - 18 )
```

Now this new variable A ranges from 0 to 1, where 0 is age 18 and 1 is age 65. Happiness is on an arbitrary scale, in these data, from -2 to $+2$. So our imaginary strongest relationship, taking happiness from maximum to minimum, has a slope with rise over run of $(2-(-2))/1 = 4$. Remember that 95% of the mass of a normal distribution is contained within 2 standard deviations. So if we set the standard deviation of the prior to half of 4, we are saying that we expect 95% of plausible slopes to be less than maximally strong. That isn't a very strong prior, but again, it at least helps bound inference to realistic ranges. Now for the intercepts. Each α is the value of μ_i when $A_i = 0$. In this case, that means at age 18. So we need to allow α to cover the full range of happiness scores. Normal$(0, 1)$ will put 95% of the mass in the -2 to $+2$ interval.

Finally, let's approximate the posterior. We need to construct the marriage status index variable, as well. I'll do that, and then immediate present the quap code.

R code 6.23
```
d2$mid <- d2$married + 1
m6.9 <- quap(
    alist(
        happiness ~ dnorm( mu , sigma ),
        mu <- a[mid] + bA*A,
        a[mid] ~ dnorm( 0 , 1 ),
        bA ~ dnorm( 0 , 2 ),
        sigma ~ dexp(1)
    ) , data=d2 )
precis(m6.9,depth=2)
```

```
       mean   sd  5.5% 94.5%
a[1]  -0.23 0.06 -0.34 -0.13
a[2]   1.26 0.08  1.12  1.40
bA    -0.75 0.11 -0.93 -0.57
sigma  0.99 0.02  0.95  1.03
```

The model is quite sure that age is negatively associated with happiness. We'd like to compare the inferences from this model to a model that omits marriage status. Here it is, followed by a comparison of the marginal posterior distributions:

R code 6.24
```
m6.10 <- quap(
    alist(
        happiness ~ dnorm( mu , sigma ),
        mu <- a + bA*A,
        a ~ dnorm( 0 , 1 ),
        bA ~ dnorm( 0 , 2 ),
        sigma ~ dexp(1)
    ) , data=d2 )
precis(m6.10)
```

```
      mean   sd  5.5% 94.5%
a     0.00 0.08 -0.12  0.12
bA    0.00 0.13 -0.21  0.21
sigma 1.21 0.03  1.17  1.26
```

This model, in contrast, finds no association between age and happiness.

The pattern above is exactly what we should expect when we condition on a collider. The collider is marriage status. It a common consequence of age and happiness. As a result, when we condition on it, we induce a spurious association between the two causes. So it looks like, to model `m6.9`, that age is negatively associated with happiness. But this is just a statistical association, not a causal association. Once we know whether someone is married or not, then their age does provide information about how happy they are.

You can see this in Figure 6.4. Consider only the blue points, the married people. Among only the blue points, older individuals have lower average happiness. This is because more people get marriage at time goes on, so the mean happiness among married people approaches the population average of zero. Now consider only the open points, the unmarried people. Here it is also true that mean happiness declines with age. This is because happier individuals migrate over time into the married sub-population. So in both the married and unmarried sub-populations, there is a negative relationship between age and happiness. But in neither sub-population does this accurately reflect causation.

It's easy to plead with this example. Shouldn't marriage also influence happiness? What if happiness does change with age? But this misses the point. If you don't have a causal model, you can't make inferences from a multiple regression. And the regression itself does not provide the evidence you need to justify a causal model. Instead, you need some science.

6.3.2. The haunted DAG.

6.3.2. The haunted DAG. Collider bias arises from conditioning on a common consequence, as in the previous example. If we can just get our graph sorted, we can avoid it. But it isn't always so easy to see a potential collider, because there may be unmeasured causes. Unmeasured causes can still induce collider bias. So I'm sorry to say that we also have to consider the possibility that our DAG may be haunted.

Suppose for example that we are interested in inferring the direct influence of both parents (P) and grandparents (G) on the educational achievement of children (C).[90] Since grandparents also presumably influence their own children's education, there is an arrow $G \rightarrow P$. This sounds pretty easy, so far. It's similar in structure to our divorce rate example from last chapter:

$$\begin{tikzcd}
G \arrow[r] \arrow[dr] & P \arrow[d] \\
& C
\end{tikzcd}$$

But suppose there are unmeasured, common influences on parents and their children, such as neighborhoods, that are not shared by grandparents (who live on the south coast of Spain now). Then our DAG becomes haunted by the unobserved U:

$$\begin{tikzcd}
G \arrow[r] \arrow[ddr] & P \arrow[dd] & \\
& & \textcircled{U} \arrow[ul] \arrow[dl] \\
& C &
\end{tikzcd}$$

Now P is a common consequence of G and U, so if we condition on P, it will bias inference about $G \to C$, *even if we never get to measure U*. I don't expect that fact to be immediately obvious. So let's crawl through a quantitative example.

First, let's simulate 200 triads of grandparents, parents, and children. This simulation will be simple. We'll just project our DAG as a series of implied functional relationships. The DAG above implies that:

(1) P is some function of G and U
(2) C is some function of G, P, and U
(3) G and U are not functions of any other known variables

We can make these implications into a simple simulation, using `rnorm` to generate simulated observations. But to do this, we need to be a bit more precise than "some function of." So I'll invent some strength of association:

R code 6.25
```
N <- 200  # number of grandparent-parent-child triads
b_GP <- 1 # direct effect of G on P
b_GC <- 0 # direct effect of G on C
b_PC <- 1 # direct effect of P on C
b_U <- 2  # direct effect of U on P and C
```

These parameters are like slopes in a regression model. Notice that I've assumed that grandparents G have zero effect on their grandkids C. The example doesn't depend upon that effect being exactly zero, but it will make the lesson clearer. Now we use these slopes to draw random observations:

R code 6.26
```
set.seed(1)
U <- 2*rbern( N , 0.5 ) - 1
G <- rnorm( N )
P <- rnorm( N , b_GP*G + b_U*U )
C <- rnorm( N , b_PC*P + b_GC*G + b_U*U )
d <- data.frame( C=C , P=P , G=G , U=U )
```

I've made the neighborhood effect, U, binary. This will make the example easier to understand. But the example doesn't depend upon that assumption. The other lines are just linear models embedded in `rnorm`.

Now what happens when we try to infer the influence of grandparents? Since some of the total effect of grandparents passes through parents, we realize we need to control for parents. Here is a simple regression of C on P and G. Normally I would advise standardizing the variables, because it makes establishing sensible priors a lot easier. But I'm going to keep the simulated data on its original scale, so you can see what happens to inference about the slopes above. If we changed the scale, we shouldn't expect to get those values back. But if we leave the scale alone, we should be able to recover something close to those values. So I apologize for using vague priors here, just to push forward in the example.

R code 6.27
```
m6.11 <- quap(
    alist(
        C ~ dnorm( mu , sigma ),
        mu <- a + b_PC*P + b_GC*G,
```

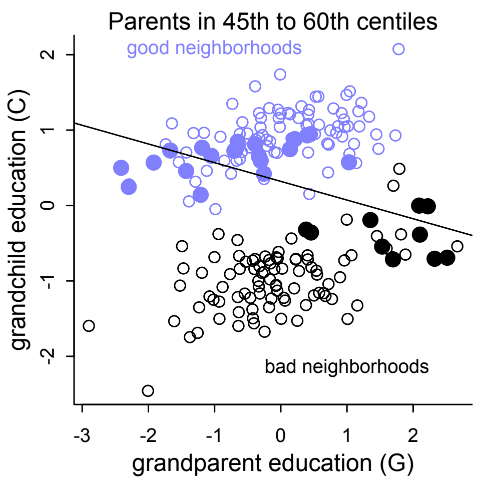


FIGURE 6.5. Unobserved confounds and collider bias. In this example, grandparents influence grandkids only indirectly, through parents. However, unobserved neighborhood effects on parents and their children create the illusion that grandparents harm their grandkids education. Parental education is a collider: Once we condition on it, grandparental education becomes negatively associated with grandchild education.

```
        a ~ dnorm( 0 , 1 ),
        c(b_PC,b_GC) ~ dnorm( 0 , 1 ),
        sigma ~ dexp( 1 )
    ), data=d )
precis(m6.11)
```

```
       mean   sd  5.5% 94.5%
a     -0.12 0.10 -0.28  0.04
b_PC   1.79 0.04  1.72  1.86
b_GC  -0.84 0.11 -1.01 -0.67
sigma  1.41 0.07  1.30  1.52
```

The inferred effect of parents looks too big, almost twice as large as it should be. That isn't surprising. Some of the correlation between *P* and *C* is due to *U*, and the model doesn't know about *U*. That's a simple confound. More surprising is that the model is confident that the direct effect of grandparents is to hurt their grandkids. The regression is not wrong. But a causal interpretation of that association would be.

How does collider bias arise in this case? Consider FIGURE 6.5. Note that I did standardize the variables to make this plot. So the units on the axes are standard deviations. The horizontal axis is grandparent education. The vertical is grandchild education. There are two clouds of points. The blue cloud comprises children who live in good neighborhoods ($U = 1$). The black cloud comprises children who live in bad neighborhoods ($U = -1$). Notice that both clouds of points show positive associations between *G* and *C*. More educated grandparents have more educated grandkids, but this effect arises entirely through parents. Why? Because we assumed it is so. The direct effect of *G* in the simulation is zero.

So how does the negative association arise, when we condition on parents? Conditioning on parents is like looking within sub-populations of parents with similar education. So let's try that. In FIGURE 6.5, I've highlighted in filled points those parents between the 45th and 60th centiles of education. There is nothing special of this range. It just makes the phenomenon easier to see. Now if we draw a regression line through only these points, regressing *C* on *G*, the slope is negative. There is the negative association that our multiple regression finds. But why does it exist?

It exists because, once we know *P*, learning *G* invisibly tells us about the neighborhood *U*, and *U* is associated with the outcome *C*. I know this is confusing. As I keep saying, if you are confused, it is only because you are paying attention. So consider two different parents with the same education level, say for example at the median 50th centile. One of these parents has a highly educated grandparent. The other has a poorly educated grandparent. The only probable way, in this example, for these parents to have the same education is if they live in different types of neighborhoods. We can't see these neighborhood effects—we haven't measured them, recall—but the influence of neighborhood is still transmitted to the children *C*. So for our mythical two parents with the same education, the one with the highly educated grandparent ends up with a less well educated child. The one with the less educated grandparent ends up with the better educated child. *G* predicts lower *C*.

The unmeasured *U* makes *P* a collider, and conditioning on *P* produces collider bias. So what can we do about this? You have to measure *U*. Here's the regression that conditions also on *U*:

R code 6.28

```
m6.12 <- quap(
    alist(
        C ~ dnorm( mu , sigma ),
        mu <- a + b_PC*P + b_GC*G + b_U*U,
        a ~ dnorm( 0 , 1 ),
        c(b_PC,b_GC,b_U) ~ dnorm( 0 , 1 ),
        sigma ~ dexp( 1 )
    ), data=d )
precis(m6.12)
```

```
       mean   sd  5.5% 94.5%
a     -0.12 0.07 -0.24 -0.01
b_PC   1.01 0.07  0.91  1.12
b_GC  -0.04 0.10 -0.20  0.11
b_U    2.00 0.15  1.76  2.23
sigma  1.02 0.05  0.94  1.10
```

And those are the slopes we simulated with.

Rethinking: Statistical paradoxes and causal explanations. The grandparents example serves as an example of SIMPSON'S PARADOX: Including another predictor (*P* in this case) can reverse the direction of association between some other predictor (*G*) and the outcome (*C*). Usually, Simpson's paradox is presented in cases where adding the new predictor helps us. But in this case, it misleads us. Simpson's paradox is a statistical phenomenon. To know whether the reversal of the association correctly reflects causation, we need something more than just a statistical model.[91]

6.4. Confronting confounding

In this chapter and in the previous one, there have been several examples of how we can use multiple regression to deal with confounding. But we have also seen how multiple regression can *cause* confounding—controlling for the wrong variables ruins inference. Hopefully I have succeeded in scaring you aware from just adding everything to a model and hoping regression will sort it out, as well as inspired you to believe that effective inference is possible, if we are careful enough and knowledgable enough.

But which principles explain why sometimes leaving out variables and sometimes adding them can produce the same phenomenon? Are there other causal monsters lurking out there, haunting our graphs? We require some coherence.

Let's define **CONFOUNDING** as any context in which the association between an outcome Y and a predictor of interest X is not the same as it would be, if we had experimentally determined the values of X.[92] For example, suppose we are interested in the association between education E and wages W. The problem is that in a typical population there are many unobserved variables U that influence both E and W. Examples include where a person lives, who their parents are, and who their friends are. This is what the DAG looks like:

$$\begin{tikzcd}
& U \arrow[dl] \arrow[dr] & \\
E \arrow[rr] & & W
\end{tikzcd}$$

If we regress W on E, the estimate of the causal effect will be confounded by U. It is confounded, because there are two paths connecting E and W: (1) $E \rightarrow W$ and (2) $E \leftarrow U \rightarrow W$. A "path" here just means any series of variables you could walk through to get from one variable to another, ignoring the directions of the arrows. Both of these paths create a statistical association between E and W. But only the first path is causal. The second path is non-causal. Why? Because if only the second path existed, and we changed E, it would not change W. Any causal influence of E on W operates only on the first path.

How can we isolate the causal path? The most famous solution is to run an experiment. If we could assign education levels at random, it changes the graph:

$$\begin{tikzcd}
& U \arrow[dr] & \\
E \arrow[rr] & & W
\end{tikzcd}$$

Manipulation removes the influence of U on E. The unobserved variables do not influence education when we ourselves determine education. With the influence of U removed from E, this then removes the path $E \leftarrow U \rightarrow W$. It blocks the second path. Once the path is blocked, there is only one way for information to go between E and W, and then measuring the association between E and W would yield a useful measure of causal influence. Manipulation removes the confounding, because it blocks the other path between E and W.

Luckily, there are statistical ways to achieve the same result, without actually manipulating E. How? The most obvious is to add U to the model, to *condition* on U. Why does this also remove the confounding? Because it also blocks the flow of information between E and W through U. It blocks the second path.

To understand why conditioning on U blocks the path $E \leftarrow U \rightarrow W$, think of this path in isolation, as a complete model. Once you learn U, also learning E will give you no additional information about W. Suppose for example that U is the average wealth in a region. Regions with high wealth have better schools, resulting in more education E, as well as better paying jobs, resulting in higher wages W. If you don't know the region a person lives in, learning the person's education E will provide information about their wages W, because E and W are correlated across regions. But after you learn which region a person lives in, assuming there is no other path between E and W, then learning E tells you nothing about W. This is the

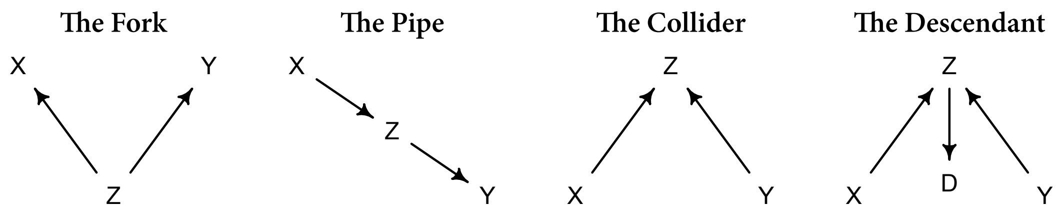


FIGURE 6.6. The four elemental confounds. Any directed acyclic graph is built from these elementary relationships.

sense in which condition on U blocks the path—it makes E and W independent, conditional on U.

6.4.1. Shutting the backdoor. Blocking all confounding paths between some predictor X and some outcome Y is known as shutting the **BACKDOOR**. The metaphor in play is that we don't want any spurious correlation sneaking in through a non-causal path, which is one that enters the back of the predictor X. In the example above, the path $E \leftarrow U \rightarrow W$ is a backdoor path, because it enters E with an arrow and also connects E to W. This path is confounding. Now for some good news. Given a causal DAG, it is always possible to say which, if any, variables one must control for in order to shut all the backdoor paths. It is also possible to say which variables one must *not* control for, in order to leave the path of interest open.

And—some more good news—there are only four types of variable relations that combine to form all possible paths. So you really only need to understand four things and how information flows in each of them. I'll define the four types of relations. Then we'll work through some examples.

FIGURE 6.6 shows DAGs for each elemental relation. Every DAG, no matter how big and complicated, is built out of these four relations. Let's consider each, going left to right.

(1) The first type of relation is the one we worked with just above, a **FORK**: $X \leftarrow Z \rightarrow Y$. This is the classic confounder. In a fork, some variable Z is a common cause of X and Y, generating a correlation between them. If we condition on Z, then learning X tells us nothing about Y. X and Y are independent, conditional on Z.

(2) The second type of relation is a **PIPE**: $X \rightarrow Z \rightarrow Y$. We saw this when we discussed the plant growth example and post-treatment bias: The treatment X influences fungus Z which influences growth Y. If we condition on Z now, we also block the path from X to Y. So in both a fork and a pipe, conditioning of the middle variable blocks the path.

(3) The third type of relation is a **COLLIDER**: $X \rightarrow Z \leftarrow Y$. You met colliders earlier in this chapter. Unlike the other two types of relations, in a collider there is no association between X and Y unless you condition on Z. Conditioning on Z, the collider variable, opens the path. Once the path is open, information flows between X and Y.

(4) The fourth bit of knowledge you need is that conditioning on a **DESCENDENT** variable is like conditioning on the variable itself, but weaker. A descendent is a variable influenced by another variable. In the far right DAG in FIGURE 6.6, controlling for

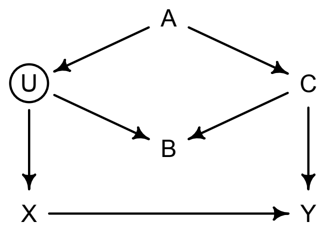
D will also control, to a lesser extent, for *Z*. The reason is that *D* has some information about *Z*. This will (partially) open the path from *X* to *Y*, because *Z* is a collider. The same holds for non-colliders. If you condition on a descendent of *Z* in the pipe, it'll still be like (weakly) closing the pipe.

No matter how complicated a causal DAG appears, it is always built out of these four types of relations. And since you know how to open and close each, you (or your computer) can figure out which variables you need to control—or not—in order to shut the backdoor. Here's the recipe:

(1) List all of the paths connecting *X* (the potential cause of interest) and *Y* (the outcome).
(2) Classify each path by whether it is open or closed. A path is open unless it contains a collider.
(3) Classify each path by whether it is a backdoor path. A backdoor path has an arrow entering *X*.
(4) If there are any backdoor paths that are also open, decide which variable(s) to condition on to close it.

Let's consider some examples.

6.4.2. Two roads. The DAG below contains an exposure of interest *X*, an outcome of interest *Y*, an unobserved variable *U*, and three observed covariates (*A*, *B*, and *C*).



We are interested in the blue path, the causal effect of *X* on *Y*. Which of the observed covariates do we need to add to the model, in order to correctly infer it? To figure this out, look for backdoor paths. Aside from the direct path, there are two paths from *X* to *Y*:

(1) $X \leftarrow U \leftarrow A \rightarrow C \rightarrow Y$
(2) $X \leftarrow U \rightarrow B \leftarrow C \rightarrow Y$

These are both backdoor paths that could confound inference. Now ask which of these paths is open. If a backdoor path is open, then we must close it. If a backdoor path is closed already, then we must not accidentally open it and create a confound.

Consider the first path, passing through *A*. This path is open, because there is no collider within it. There is just a fork at the top and two pipes, one on each side. Information will flow through this path, confounding $X \rightarrow Y$. It is a backdoor. To shut this backdoor, we need to condition on one of its variables. We can't condition on *U*, since it is unobserved. That leaves *A* or *C*. Either will shut the backdoor. You can ask your computer to reproduce this analysis, to analyze the graph and find the necessary variables to control for in order to block the backdoor. The `dagitty` R package provides `adjustmentSets` for this purpose:

R code 6.29
```
library(dagitty)
dag_6.1 <- dagitty( "dag {
    U [unobserved]
    X -> Y
    X <- U <- A -> C -> Y
    U -> B <- C
}")
adjustmentSets( dag_6.1 , exposure="X" , outcome="Y" )
```

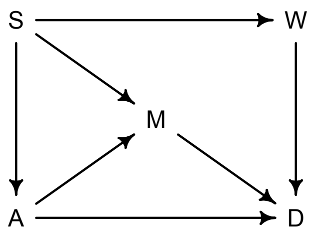
```
 { C }
 { A }
```

Conditioning on either C or A would suffice. Conditioning on C is the better idea, from the perspective of efficiency, since it could also help with the precision of the estimate of $X \rightarrow Y$. Notice that conditioning on U would also work. But since we told `dagitty` that U is unobserved (see the code above), it didn't suggest it in the adjustment sets.

Now consider the second path, passing through B. This path does contain a collider, $U \rightarrow B \leftarrow C$. It is therefore already closed. It is not a backdoor, and that is why `adjustmentSets` above did not mention B. You don't need to control for B. But if we do condition on B, it is not harmless. It will open the path, creating a confound. Then our inference about $X \rightarrow Y$ will change, but without the DAG, we won't know whether that change is helping us or rather misleading us. The fact that including a variable changes the $X \rightarrow Y$ coefficient does not always mean that the coefficient is better now. You could have just conditioned on a collider.

6.4.3. Backdoor waffles. As a final example, let's return to the Waffle House and divorce rate correlation from the introduction to Chapter 5. We'll make a DAG, use it to find a minimal set of covariates, and use it as well to derive the testable implications of the DAG. This is important, because sometimes you really can test whether your DAG is consistent with the evidence. The data alone can never tell us when a DAG is right. But the data can tell us when a DAG is wrong.

We're interested in the total causal effect of the number of Waffle Houses on divorce rate in each State. Presumably, the naive correlation between these two variables is spurious. What is the minimal adjustment set that will block backdoor paths from Waffle House to divorce? Let's make a graph:



In this graph, S is whether or not a State is in the southern United States, A is median age at marriage, M is marriage rate, W is number of Waffle Houses, and D is divorce rate. This graph assumes that southern States have lower ages of marriage ($S \rightarrow A$), higher rates of marriage both directly ($S \rightarrow M$) and mediated through age of marriage ($S \rightarrow A \rightarrow M$), as well as more waffles ($S \rightarrow W$). Age of marriage and marriage rate both influence divorce.

There are three open backdoor paths between W and D. Just trace backwards, starting at W and ending up at D. But notice that all of them pass first through S. So we can close them all by conditioning on *S*. That's all there is to it. You can get your computer to confirm this answer:

R code 6.30
```
library(dagitty)
dag_6.2 <- dagitty( "dag {
    A -> D
    A -> M -> D
    A <- S -> M
    S -> W -> D
}")
adjustmentSets( dag_6.2 , exposure="W" , outcome="D" )
```

```
 { A, M }
 { S }
```

We could control for either *A* and *M* or for *S* alone. If you don't have to add something to the model, then don't.

This DAG is obviously not satisfactory—it assumes there are no unobserved confounds, which is very unlikely for this sort of data. But we can still learn something by analyzing it. While the data cannot tell us whether a graph is correct, it can sometimes suggest how a graph is wrong. Earlier in chapter, we discussed **CONDITIONAL INDEPENDENCIES**, which are some of a model's testable implications. Condition independencies are pairs of variables that are not associated, once we condition on some set of other variables. By listing these implied conditional independencies and assessing each, we can at least test some of the features of a graph.

Now that you know the elemental confounds, you are ready to derive any DAG's conditional independencies on your own. You can find conditional independencies using the same path logic you learned for finding and closing backdoors. You just have to focus on a pair of variables, find all paths connecting them, and figure out if there is any set of variables you could condition on to close them all. In a large graph, this is quite a chore, because there are many pairs of variables and possibly many paths. But your computer is good at such chores. In this case, there are three implied conditional independencies:

R code 6.31
```
impliedConditionalIndependencies( dag_6.2 )
```

```
A _||_ W | S
D _||_ S | A, M, W
M _||_ W | S
```

Read the first as "median age of marriage should be independent of (_||_) Waffle Houses, conditioning on (|) a State being in the south." In the second, divorce and being in the south should be independent when we simultaneously condition on all of median age of marriage, marriage rate, and Waffle Houses. Finally, marriage rate and Waffle Houses should be independent, conditioning on being in the south.

In the problems at the end of this chapter, I'll ask you to evaluate these implications, as well as try to assess the causal influence of Waffle Houses on divorce.

Rethinking: DAGs are not enough. If you don't have a real, mechanistic model of your system, DAGs are fantastic tools. If nothing else, they caution against the commonplace approach of using multiple regression as a substitute for theory. But DAGs are not a destination. Once you have a dynamical model of your system, you don't need a DAG. In fact, many dynamical systems cannot be usefully represented by DAGs, because they have complex behavior that is sensitive to initial conditions. But these models can still be analyzed and causal interventions designed from them. The fact that DAGs are not useful for everything is no argument against them. All theory tools have limitations. I have yet to see a better tool than DAGs for teaching the mechanics of and obstacles to causal inference.

Overthinking: A smooth operator. To define confounding with precise notation, we need to adopt something called the DO-OPERATOR.[93] Confounding occurs when:

$$\Pr(Y|X) \neq \Pr(Y|\text{do}(X))$$

That $\text{do}(X)$ means to cut all of the backdoor paths into X, as if we did a manipulative experiment. The do-operator changes the graph, closing the backdoors. The do-operator defines a causal relationship, because $\Pr(Y|\text{do}(X))$ tells us the expected result of manipulating X on Y, given a causal graph. We might say that some variable X is a cause of Y when $\Pr(Y|\text{do}(X)) > \Pr(Y|\text{do}(\text{not-}X))$. The ordinary conditional probability comparison, $\Pr(Y|X) > \Pr(Y|\text{not-}X)$, is not the same. It does not close the backdoor. Note that what the do-operator gives you is not just the *direct* causal effect. It is the *total* causal effect through all forward paths. To get a direct causal effect, you might have to close more doors. The do-operator can also be used to derive causal inference strategies even when some back doors cannot be closed. We'll look at a couple in a later chapter.

6.5. Summary

Multiple regression is no oracle. It is logical, but the relationships it describes are conditional associations, not causal influences. Therefore additional information, from outside the model, is needed to make sense of it. This chapter presented introductory examples of some common frustrations: multicollinearity, post-treatment bias, and collider bias. Solutions to these frustrations can be organized under a coherent framework in which hypothetical causal relations among variables are analyzed to locate and cope with confounding. In all cases, causal models exist outside the statistical model and can be difficult to test. However, it is possible to reach valid causal inferences in the absence of experiments. This is good news, because we often cannot perform experiments, both for practical and ethical reasons.

6.6. Practice

Easy. x

Medium.

6M1. Modify the DAG on page 190 to include the variable V, an unobserved cause of C and Y: $C \leftarrow V \rightarrow Y$. Reanalyze the DAG. How many paths connect X to Y? Which must be closed? Which variables should you condition on now?

Hard.

6H1. Use the Waffle House data, `data(WaffleDivorce)`, to find the total causal influence of number of Waffle Houses on divorce rate. Justify your model or models with a causal graph.

6H2. Build a series of models to test the implied conditional independencies of the causal graph you used in the previous problem. If any of the tests fail, how do you think the graph needs to be amended? Does the graph need more or fewer arrows? Feel free to nominate variables that aren't in the data.

6H3. x