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who don't). The second is known as the false positive rate. According to the BCSC, the false positive rate for forty-year-old women is about 12 percent.

Why a weighted average? Because there are many more healthy women (~*D*) than women with cancer (*D*). In fact, only 1 in 700 women has cancer, and the other 699 do not, so the probability of a positive test for a randomly chosen woman should be much more strongly influenced by the 699 women who don't have cancer than by the one woman who does.

Mathematically, we compute the weighted average as follows: $P(T) = (1/700) \times (73\text{ percent}) + (699/700) \times (12\text{ percent}) \approx 12.1$ percent. The weights come about because only 1 in 700 women has a 73 percent chance of a positive test, and the other 699 have a 12 percent chance. Just as you might expect, $P(T)$ came out very close to the false positive rate.

Now that we know $P(T)$, we finally can compute the updated probability—the woman's chances of having breast cancer after the test comes back positive. The likelihood ratio is 73 percent/12.1 percent ≈ 6. As I said before, this is the factor by which we augment her prior probability to compute her updated probability of having cancer. Since her prior probability was one in seven hundred, her updated probability is 6 × 1/700 × 1/116. In other words, she still has less than a 1 percent chance of having cancer.

multiplication symbol should be an approx sign

≈

The conclusion is startling. I think that most forty-year-old women who have a positive mammogram would be astounded to learn that they still have less than a 1 percent chance of having breast cancer. Figure 3.3 might make the reason easier to understand: the tiny number of true positives (i.e., women with breast cancer) is overwhelmed by the number of false positives. Our sense of surprise at this result comes from the common cognitive confusion between the forward probability, which is well studied and thoroughly documented, and the inverse probability, which is needed for personal decision making.

The conflict between our perception and reality partially explains the outcry when the US Preventive Services Task Force, in 2009, recommended that forty-year-old women should not get annual mammograms. The task force understood what many women

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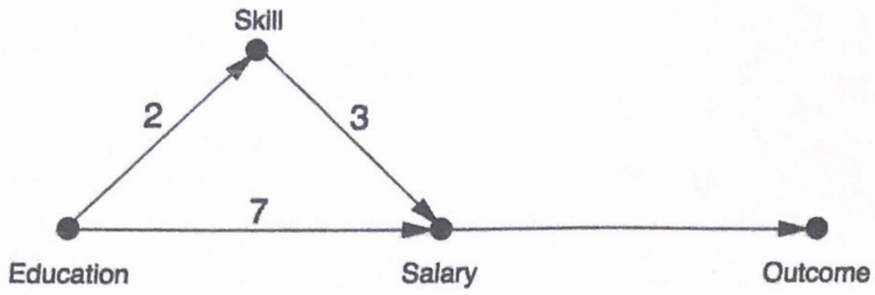

Figure 9.8. Mediation combined with a threshold effect.

set Skill at the level it would take if we had increased Education by one. It's easy to see that in this case, Salary goes from zero to 2 × 3 = 6. This is below the threshold of ten, so the applicant will turn the offer down. Thus NIE = 0.

Now what about the direct effect? As mentioned before, we have the problem of figuring out what value to hold the mediator at. If we hold Skill at the level it had before we changed Education, then Salary will increase from zero to seven, making Outcome = 0. Thus, CDE(0) = 0. On the other hand, if we hold Skill at the level it attains after the change in Education (namely two), Salary will increase from six to thirteen. This changes the Outcome from zero to one, because thirteen is above the applicant's threshold for accepting the job offer. So CDE(2) = 1.

Thus, the direct effect is either zero or one depending on the constant value we choose for the mediator. Unlike in Linear Wonderland, the choice of a value for the mediator makes a difference, and we have a dilemma. If we want to preserve the additive principle, Total Effect = Direct Effect + Indirect Effect, we need to use CDE(2) as our definition of the causal effect. But this seems arbitrary and even somewhat unnatural. If we are contemplating a change in Education and we want to know its direct effect, we would most likely want to keep Skill at the level it already has. In other words, it makes more intuitive sense to use CDE(0) as our direct effect. Not only that, this agrees with the natural direct effect in this example. But then we lose additivity: Total Effect π Direct Effect + Indirect Effect.

replace greek pi symbol "π" with not-equal sign "≠ "

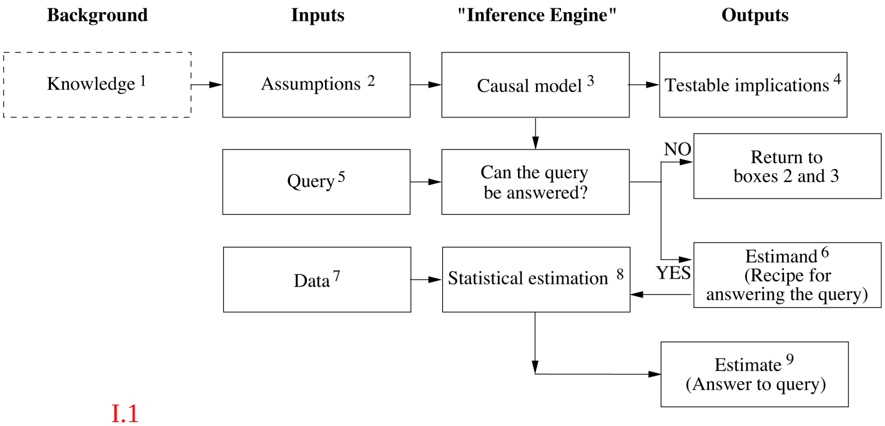

FIGURE ~~1.~~ I.1 How an "inference engine" combines data with causal knowledge to produce answers to queries of interest. The dashed box is not part of the engine but is required for building it. Arrows could also be drawn from boxes 4 and 9 to box 1, but I have opted to keep the diagram simple.

the Data input, it will use the recipe to produce an actual Estimate for the answer, along with statistical estimates of the amount of uncertainty in that estimate. This uncertainty reflects the limited size of the data set as well as possible measurement errors or missing data.

To dig more deeply into the chart, I have labeled the boxes 1 through 9, which I will annotate in the context of the query "What is the effect of Drug *D* on Lifespan *L*?"

1. "Knowledge" stands for traces of experience the reasoning agent has had in the past, including past observations, past actions, education, and cultural mores, that are deemed relevant to the query of interest. The dotted box around "Knowledge" indicates that it remains implicit in the mind of the agent and is not explicated formally in the model.
2. Scientific research always requires simplifying assumptions, that is, statements which the researcher deems worthy of making explicit on the basis of the available Knowledge. While most of the researcher's knowledge remains implicit in his or her brain, only Assumptions see the light of day and

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of people among those with Lifespan *L* who took the Drug *D*, we simply write this query as $P(D \mid L)$. The same quantity would be our estimand. This already specifies what proportions in the data need to be estimated and requires no causal knowledge. For this reason, some statisticians to this day find it extremely hard to understand why some knowledge lies outside the province of statistics and why data alone cannot make up for lack of scientific knowledge.

8. The estimate is what comes out of the oven. However, it is only approximate because of one other real-world fact about data: they are always only a finite sample from a theoretically infinite population. In our running example, the sample consists of the patients we choose to study. Even if we choose them at random, there is always some chance that the proportions measured in the sample are not representative of the proportions in the population at large. Fortunately, the discipline of statistics, nowadays empowered by advanced techniques of machine learning, gives us many, many ways to manage this uncertainty—parametric and semi-parametric models, maximum likelihood ~~estimators,~~ methods, and propensity scores~~, confidence intervals, significance tests, and so forth.~~ are often used to smooth the sparse data.
9. In the end, if our model is correct and our data are sufficient, we get an answer to our causal query, such as "Drug *D* increases the Lifespan *L* of diabetic Patients *Z* by 30 percent, plus or minus 20 percent." Hooray! The answer will also add to our scientific knowledge (box 1) and, if things did not go the way we expected, might suggest some improvements to our causal model (box 3).

This flowchart may look complicated at first, and you might wonder whether it is really necessary. Indeed, in our ordinary lives, we are somehow able to make causal judgments without consciously going through such a complicated process and certainly without resorting to the mathematics of probabilities and proportions. Our causal intuition alone is usually sufficient for handling the kind of uncertainty we find in household routines or even in our

They can call upon a tradition of thought about causation that goes back at least to Aristotle, and they can talk about causation without blushing or hiding it behind the label of "association."

However, in their effort to mathematize the concept of causation—itself a laudable idea—philosophers were too quick to commit to the only uncertainty-handling language they knew, the language of probability. They have for the most part gotten over this blunder in the past decade or so, but unfortunately similar ideas are being pursued in econometrics even now, under names like "Granger causality" and "vector autoregression ~~autocorrelation~~."

Now I have a confession to make: I made the same mistake. I did not always put causality first and probability second. Quite the opposite! When I started working in artificial intelligence, in the early 1980s, I thought that uncertainty was the most important thing missing from AI. Moreover, I insisted that uncertainty be represented by probabilities. Thus, as I explain in Chapter 3, I developed an approach to reasoning under uncertainty, called Bayesian networks, that mimics how an idealized, decentralized brain might incorporate probabilities into its decisions. Given that we see certain facts, Bayesian networks can swiftly compute the likelihood that certain other facts are true or false. Not surprisingly, Bayesian networks caught on immediately in the AI community and even today are considered a leading paradigm in artificial intelligence for reasoning under uncertainty.

Though I am delighted with the ongoing success of Bayesian networks, they failed to bridge the gap between artificial and human intelligence. I'm sure you can figure out the missing ingredient: causality. True, causal ghosts were all over the place. The arrows invariably pointed from causes to effects, and practitioners often noted that diagnostic systems became unmanageable when the direction of the arrows was reversed. But for the most part we thought that this was a cultural habit, or an artifact of old thought patterns, not a central aspect of intelligent behavior.

At the time, I was so intoxicated with the power of probabilities that I considered causality a subservient concept, merely a convenience or a mental shorthand for expressing probabilistic dependencies and distinguishing relevant variables from irrelevant ones.

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explain why it is harder; he took that as self-evident, proved that it is doable, and showed us how.

To appreciate the nature of the problem, let's look at ~~the~~ a slightly simplified version of an example he suggested ~~himself~~ in his posthumous paper of 1763. Imagine that we shoot a billiard ball on a table, making sure that it bounces many times so that we have no idea where it will end up. What is the probability that it will stop within *x* feet of the left-hand end of the table? If we know the length of the table and it is perfectly smooth and flat, this is a very easy question (Figure 3.2, top). For example, on a twelve-foot snooker table, the probability of the ball stopping within a foot of the end would be 1/12. On an eight-foot billiard table, the probability would be 1/8.

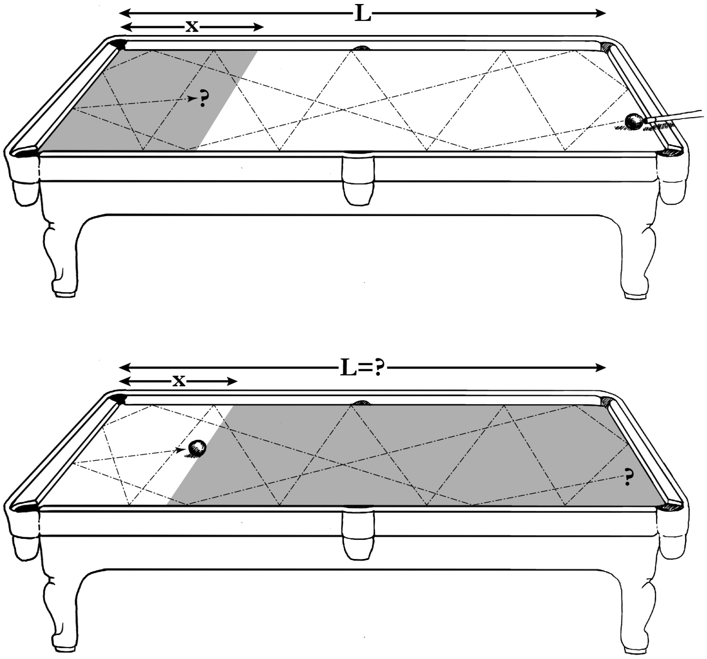

FIGURE 3.2. Thomas Bayes's pool table example. In the first version, a forward-probability question, we know the length of the table and want to calculate the probability of the ball stopping within *x* feet of the end. In the second, an inverse-probability question, we observe that the ball stopped *x* feet from the end and want to estimate the likelihood that the table's length is *L*. (*Source:* Drawing by Maayan Harel.)

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to be independent, conditional on *B*, then we can safely conclude that the chain model is incompatible with the data and needs to be discarded (or repaired). Second, the graphical properties of the diagram dictate which causal models can be distinguished by data and which will forever remain indistinguishable, no matter how large the data. For example, we cannot distinguish the fork $A \leftarrow B \rightarrow C$ from the chain $A \rightarrow B \rightarrow C$ by data alone, because, with *C* listening to *B* only, the two ~~diagrams~~ imply the same independence conditions.

Another convenient way of thinking about the causal model is in terms of hypothetical experiments. Each arrow can be thought of as a statement about the outcome of a hypothetical experiment. An arrow from *A* to *C* means that if we could wiggle only *A*, then we would expect to see a change in the probability of *C*. A missing arrow from *A* to *C* means that in the same experiment we would not see any change in *C*, once we held constant the parents of *C* (in other words, *B* in the example above). Note that the probabilistic expression "once we know the value of *B*" has given way to the causal expression "once we hold *B* constant," which implies that we are physically preventing *B* from varying and disabling the arrow from *A* to *B*.

The causal thinking that goes into the construction of the causal network will pay off, of course, in the type of questions the network can answer. Whereas a Bayesian network can only tell us how likely one event is, given that we observed another (rung-one information), causal diagrams can answer interventional and counterfactual questions. For example, the causal fork $A \leftarrow B \rightarrow C$ tells us in no uncertain terms that wiggling *A* would have no effect on *C*, no matter how intense the wiggle. On the other hand, a Bayesian network is not equipped to handle a "wiggle," or to tell the difference between seeing and doing, or indeed to distinguish a fork from a chain. In other words, both a chain and a fork would predict that observed changes in *A* are associated with changes in *C*, making no prediction about the effect of "wiggling" *A*.

Now we come to the second, and perhaps more important, impact of Bayesian networks on causal inference. The relationships that were discovered between the graphical structure of the

the causal effect of X on Y. It is a disaster to control for Z if you are trying to find the causal effect of X on Y. If you look only at those individuals in the treatment and control groups for whom $Z = 0$, then you have completely blocked the effect of X, because it works by changing Z. So you will conclude that X has no effect on Y. This is exactly what Ezra Klein meant when he said, “Sometimes you end up controlling for the thing you’re trying to measure.”

In example (ii), Z is a proxy for the mediator M. Statisticians very often control for proxies when the actual causal variable can’t be measured; for instance, party affiliation might be used as a proxy for political beliefs. Because Z isn’t a perfect measure of M, some of the influence of X on Y might “leak through” if you control for Z. Nevertheless, controlling for Z is still a mistake. While the bias might be less than if you controlled for M, it is still there.

For this reason later statisticians, notably David Cox in his textbook *The Design of Experiments* (1958), warned that you should only control for Z if you have a ~~“~~strong prior reason~~”~~ to believe that it is "quite unaffected" ~~not affected~~ by X. This "quite unaffected" condition is of course ~~“strong prior reason” is nothing more or less than~~ a causal assumption. He adds, “Such hypotheses may be perfectly in order, but the scientist should always be aware when they are being appealed to.” Remember that it’s 1958, in the midst of the great prohibition on causality. Cox is saying that you can go ahead and take a swig of causal moonshine when adjusting for confounders, but don’t tell the preacher. A daring suggestion! I never fail to commend him for his bravery.

By 1980, Simpson’s and Cox’s conditions had been combined into the three-part test for confounding that I mentioned above. It is about as trustworthy as a canoe with only three leaks. Even though it does make a halfhearted appeal to causality in part (3), each of the first two parts can be shown to be both unnecessary and insufficient.

Greenland and Robins drew that conclusion in their landmark 1986 paper. The two took a completely new approach to confounding, which they called “exchangeability.” They went back to the original idea that the control group ($X = 0$) should be comparable to the treatment group ($X = 1$). But they added a counterfactual twist. (Remember from Chapter 1 that counterfactuals are at rung

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to assist in distinguishing confounders from deconfounders. She is the only person I know of who managed this feat. Later, in 2012, she collaborated on an updated version that analyzes the same examples with causal diagrams and verifies that all her conclusions from 1993 were correct.

In both of Weinberg's papers, the medical application was to estimate the effect of smoking (*X*) on miscarriages, or "spontaneous abortions" (*Y*). In Game 1, *A* represents an underlying abnormality that is induced by smoking; this is not an observable variable because we don't know what the abnormality is. *B* represents a history of previous miscarriages. It is very, very tempting for an epidemiologist to take previous miscarriages into account and adjust for them when estimating the probability of future miscarriages. But that is the wrong thing to do here! By doing so we are partially inactivating the mechanism through which smoking acts, and we will thus underestimate the true effect of smoking.

Game 2 is a more complicated version where there are two different smoking variables: *X* represents whether the mother smokes now (at the beginning of the second pregnancy), while *A* represents whether she smoked during the first pregnancy. *B* and *E* are underlying abnormalities caused by smoking, which are unobservable, and *D* represents other physiological causes of those abnormalities. Note that this diagram allows for the fact that the mother could have changed her smoking behavior between pregnancies, but the other physiological causes would not change. Again, many epidemiologists would adjust for prior miscarriages (*C*), but this is a bad idea unless you also adjust for smoking behavior in the first pregnancy (*A*).

Games 4 and 5 come from a paper published in 2014 by Andrew Forbes~~, a biostatistician at~~ of Monash University and Elizabeth Williamson, now at the London ~~in Australia, along~~ School of Hygiene and Tropical Medicine. They are ~~with several collaborators. He is~~ interested in the effect of smoking on adult asthma. In Game 4, *X* represents an individual's smoking behavior, and *Y* represents whether the person has asthma as an adult. *B* represents childhood asthma, which is a collider because it is affected by both *A*, parental smoking, and *C*, an underlying (and unobservable) predisposition toward asthma. In Game 5 the variables have the same meanings, but they ~~Forbes~~ added two arrows

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for greater realism. (Game 4 was only meant to introduce the *M*-graph.)

In fact, the full model in their ~~Forbes'~~ paper has a few more variables and looks like the diagram in Figure 4.7. Note that Game 5 is embedded in this model in the sense that the variables *A*, *B*, *C*, *X*, and *Y* have exactly the same relationships. So we can transfer our conclusions over and conclude that we have to control for *A* and *B* or for *C*; but *C* is an unobservable and therefore uncontrollable variable. In addition we have four new confounding variables: *D* = parental asthma, *E* = chronic bronchitis, *F* = sex, and *G* = socioeconomic status. The reader might enjoy figuring out that we must control for *E*, *F*, and *G*, but there is no need to control for *D*. So a sufficient set of variables for deconfounding is *A*, *B*, *E*, *F*, and *G*.

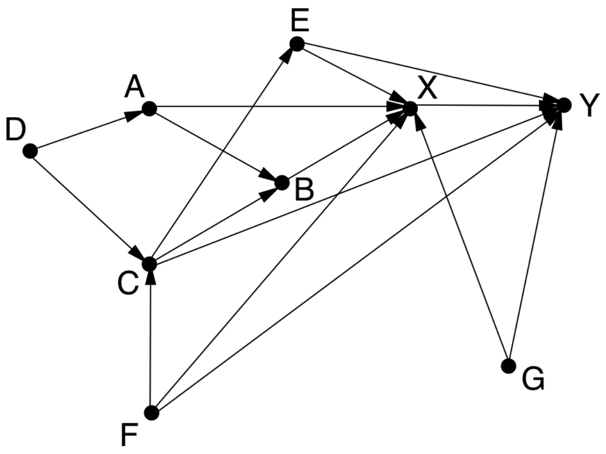

FIGURE 4.7. Andrew Forbes~~'s~~ and Elizabeth Williamson's model of smoking (*X*) and asthma (*Y*).

In the end, Forbes and Williamson's found that smoking had a small and statistically insignificant association with adult asthma in the raw data, and the effect became even smaller and more insignificant after adjusting for the confounders. The null result should not detract, however, from the fact that their ~~his~~ paper is a model for the "skillful interrogation of Nature."

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patient chooses to take Drug *D*. In the study, women clearly had a preference for taking Drug *D* and men preferred not to. Thus Gender is a confounder of Drug and Heart Attack. For an unbiased estimate of the effect of Drug on Heart Attack, we must adjust for the confounder. We can do that by looking at the data for men and women separately, then taking the average:

- For women, the rate of heart attacks was 5 percent without Drug *D* and 7.5 percent with Drug *D*.
- For men, the rate of heart attacks was 30 percent without Drug *D* and 40 percent with.
- Taking the average (because men and women are equally frequent in the general population), the rate of heart attacks without Drug *D* is 17.5 percent (the average of 5 and 30), and the rate with Drug *D* is 23.75 percent (the average of 7.5 and 40).

This is the clear and unambiguous answer we were looking for. Drug *D* isn't BBG, it's BBB: bad for women, bad for ~~w~~omen, and bad for people.

I don't want you to get the impression from this example that aggregating the data is always wrong or that partitioning the data is always right. It depends on the process that generated the data. In the Monty Hall paradox, we saw that changing the rules of the game also changed the conclusion. The same principle works here. I'll use a different story to demonstrate when pooling the data would be appropriate. Even though the data will be precisely the same, the role of the "lurking third variable" will differ and so will the conclusion.

Let's begin with the assumption that blood pressure is known to be a possible cause of heart attack, and Drug *B* is supposed to reduce blood pressure. Naturally, the Drug *B* researchers wanted to see if it might also reduce heart attack risk, so they measured their patients' blood pressure after treatment, as well as whether they had a heart attack.

Table 6.6 shows the data from the study of Drug *B*. It should look amazingly familiar: the numbers are the same as in Table

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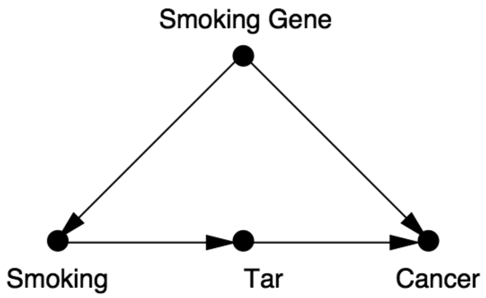

FIGURE 7.1. Hypothetical causal diagram for smoking and cancer, suitable for front-door adjustment.

no direct arrow points from Smoking to Cancer, and there are no other indirect pathways.

Suppose we are doing an observational study and have collected data on Smoking, Tar, and Cancer for each of the participants. Unfortunately, we cannot collect data on the Smoking Gene because we do not know whether such a gene exists. Lacking data on the confounding variable, we cannot block the back-door path Smoking ← Smoking Gene → Cancer. Thus we cannot use back-door adjustment to control for the effect of the confounder.

So we must look for another way. Instead of going in the back door, we can go in the front door! In this case, the front door is the direct causal path Smoking → Tar → Cancer, for which we do have data on all three variables. Intuitively, the reasoning is as follows. First, we can estimate the average causal effect of Smoking on Tar, because there is no unblocked back-door path from Smoking to ~~Cancer,~~ Tar, as the Smoking ← Smoking Gene → Cancer ← Tar path is already blocked by the collider at Cancer. Because it is blocked already, we don't even need back-door adjustment. We can simply observe $P(\mathit{tar} \mid \mathit{smoking})$ and $P(\mathit{tar} \mid \mathit{no\ smoking})$, and the difference between them will be the average causal effect of Smoking on Tar.

Likewise, the diagram allows us to estimate the average causal effect of Tar on Cancer. To do this we can block the back-door path from Tar to Cancer, Tar ← Smoking ← Smoking Gene → Cancer,

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by adjusting for Smoking. Our lessons from Chapter 4 come in handy: we only need data on a sufficient set of deconfounders (i.e., Smoking). Then the back-door adjustment formula will give us *P*(*cancer* | *do*(*tar*)) and *P*(*cancer* | *do*(*no tar*)). The difference between these is the average causal effect of Tar on Cancer.

Now we know the average increase in the likelihood of tar deposits due to smoking and the average increase of cancer due to tar deposits. Can we combine these somehow to obtain the average increase in cancer due to smoking? Yes, we can. The reasoning goes as follows. Cancer can come about in two ways: in the presence of Tar or in the absence of Tar. If we force a person to smoke, then the probabilities of these two states are *P*(*tar* | *do*(*smoking*)) and → *P*(*no tar* | *do*(~~*no*~~ *smoking*)), respectively. If a Tar state evolves, the likelihood of causing Cancer is *P*(*cancer* | *do*(*tar*)). If, on the other hand, a No-Tar state evolves, then it would result in a Cancer likelihood of *P*(*cancer* | *do*(*no tar*)). We can weight the two scenarios by their respective probabilities under *do*(*smoking*) and in this way compute the total probability of cancer due to smoking. The same argument holds if we prevent a person from smoking, *do*(*no smoking*). The difference between the two gives us the average causal effect on cancer of smoking versus not smoking.

As I have just explained, we can estimate each of the *do*-probabilities discussed from the data. That is, we can write them mathematically in terms of probabilities that do not involve the *do*-operator. In this way, mathematics does for us what ten years of debate and congressional testimony could not: quantify the causal effect of smoking on cancer—provided our assumptions hold, of course.

The process I have just described, expressing *P*(*cancer* | *do* (*smoking*)) in terms of *do*-free probabilities, is called the front-door adjustment. It differs from the back-door adjustment in that we adjust for two variables (Smoking and Tar) instead of one, and these variables lie on the front-door path from Smoking to Cancer rather than the back-door path. For those readers who "speak mathematics," I can't resist showing you the formula (Equation 7.1), which cannot be found in ordinary statistics textbooks. Here *X* stands for Smoking, *Y* stands for Cancer, *Z* stands for Tar, and

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U (which is conspicuously absent from the formula) stands for the unobservable variable, the Smoking Gene.

$$P(Y \mid do(X)) = \Sigma_z\, P(Z = z \mid X)\ \Sigma_x\, P(Y \mid X = x, Z = z)\ P(X = x) \qquad (7.1)$$

Readers with an appetite for mathematics might find it interesting to compare this to the formula for the back-door adjustment, which looks like Equation 7.2.

$$P(Y \mid do(X)) = \Sigma_z\, P(Y \mid X, Z = z)\ P(Z = z) \qquad (7.2)$$

Even for readers who do not speak mathematics, we can make several interesting points about Equation 7.1. First and most important, you don't see *U* (the Smoking Gene) anywhere. This was the whole point. We have successfully deconfounded *U* even without possessing any data on it. Any statistician of Fisher's generation would have seen this as an utter miracle. Second, way back in the Introduction I talked about an estimand as a recipe for computing the quantity of interest in a query. Equations 7.1 and 7.2 are the most complicated and interesting estimands that I will show you in this book. The left-hand side represents the query "What is the effect of *X* on *Y*?" The right-hand side is the estimand, a recipe for answering the query. Note that the estimand contains no *do*'s, only *see*'s, represented by the vertical bars, and this means it can be estimated from data.

At this point, I'm sure that some readers are wondering how close this fictional scenario is to reality. Could the smoking-cancer controversy have been resolved by one observational study and one causal diagram? If we assume that Figure 7.1 accurately reflects the causal mechanism for cancer, the answer is absolutely yes. However, we now need to discuss whether our assumptions are valid in the real world.

David Freedman, a longtime friend and a Berkeley statistician, took me to task over this issue. He argued that the model in Figure 7.1 is unrealistic in three ways. First, if there is a smoking gene, it might also affect how the body gets rid of foreign matter in the

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benchmarks by hundreds or thousands of dollars. This is exactly what you would expect to see if there is an unobserved confounder, such as Motivation. The back-door criterion cannot adjust for it.

On the other hand, the front-door estimates succeeded in removing almost all of the Motivation effect. For males, the front-door estimates were well within the experimental error of the randomized controlled trial, even with the small positive bias that Glynn and Kashin predicted. For females, the results were even better: The front-door estimates matched the experimental benchmark almost perfectly, with no apparent bias. Glynn and Kashin's work gives both empirical and methodological proof that as long as the effect of *C* on *M* (in Figure 7.2) is weak, front-door adjustment can give a reasonably good estimate of the effect of *X* on *Y*. It is much better than not controlling for *C*.

Glynn and Kashin's results show why the front-door adjustment is such a powerful tool: it allows us to control for confounders that we cannot observe (like Motivation), including those that we can't even name. RCTs are considered the "gold standard" of causal effect estimation for exactly the same reason. Because front-door estimates do the same thing, with the additional virtue of observing people's behavior in their own natural habitat instead of a laboratory, I would not be surprised if this method eventually becomes a useful alternative ~~serious competitor~~ to randomized controlled trials.

THE *DO*-CALCULUS, OR MIND OVER MATTER

In both the front- and back-door adjustment formulas, the ultimate goal is to calculate the effect of an intervention, $P(Y \mid do(X))$, in terms of data such as $P(Y \mid X, A, B, Z, \ldots)$ that do not involve a *do*-operator. If we are completely successful at eliminating the *do*'s, then we can use observational data to estimate the causal effect, allowing us to leap from rung one to rung two of the Ladder of Causation.

The fact that we were successful in these two cases (front- and back-door) immediately raises the question of whether there are other doors through which we can eliminate all the *do*'s. Thinking more generally, we can ask whether there is some way to decide in

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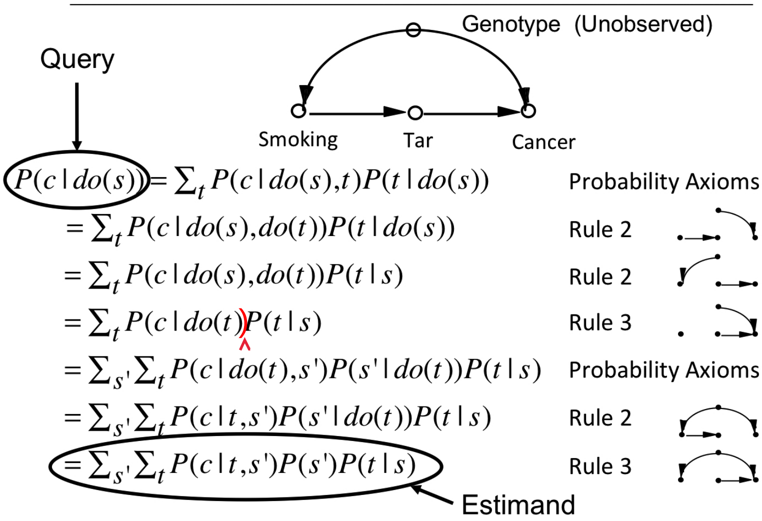
DO-CALCULUS AT WORK



$$\begin{aligned}
\boxed{P(c \mid do(s))} &= \textstyle\sum_t P(c \mid do(s),t)P(t \mid do(s)) && \text{Probability Axioms}\\
&= \textstyle\sum_t P(c \mid do(s),do(t))P(t \mid do(s)) && \text{Rule 2}\\
&= \textstyle\sum_t P(c \mid do(s),do(t))P(t \mid s) && \text{Rule 2}\\
&= \textstyle\sum_t P(c \mid do(t))P(t \mid s) && \text{Rule 3}\\
&= \textstyle\sum_{s'}\sum_t P(c \mid do(t),s')P(s' \mid do(t))P(t \mid s) && \text{Probability Axioms}\\
&= \textstyle\sum_{s'}\sum_t P(c \mid t,s')P(s' \mid do(t))P(t \mid s) && \text{Rule 2}\\
&\boxed{= \textstyle\sum_{s'}\sum_t P(c \mid t,s')P(s')P(t \mid s)} && \text{Rule 3}
\end{aligned}$$

FIGURE 7.4. Derivation of the front-door adjustment formula from the rules of *do*-calculus.

for confounders. I believed no one could do this without the *do*-calculus, so I presented it as a challenge in a statistics seminar at Berkeley in 1993 and even offered a $100 prize to anyone who could solve it. Paul Holland, who attended the seminar, wrote that he had assigned the problem as a class project and would send me the solution when ripe. (Colleagues tell me that he eventually presented a long solution at a conference in 1995, and I may owe him $100 if I could only find his proof.) Economists James Heckman and Rodrigo Pinto made the next attempt to prove the front-door formula using "standard tools" in 2015. They succeeded, albeit at the cost of eight pages of hard labor.

In a restaurant the evening before the talk, I had written the proof (very much like the one in Figure 7.4) on a napkin for David Freedman. He wrote me later to say that he had lost the napkin. He could not reconstruct the argument and asked if I had kept a copy. The next day, Jamie Robins wrote to me from Harvard, saying that he had heard about the "napkin problem" from Freedman, and he straightaway offered to fly to California to check the

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me, this historical detective work makes the story more beautiful. It shows that Philip took the trouble to understand his son's theory and articulate it in his own language.

Now let's move forward from the 1850s and 1920s to look at a present-day example of instrumental variables in action, one of literally dozens I could have chosen.

GOOD AND BAD CHOLESTEROL

Do you remember when your family doctor first started talking to you about "good" and "bad" cholesterol? It may have happened in the 1990s, when drugs that lowered blood levels of "bad" cholesterol, low-density lipoprotein (LDL), first came on the market. These drugs, called statins, have turned into multibillion-dollar revenue generators for pharmaceutical companies.

An early ~~The first~~ cholesterol-modifying drug subjected to a randomized controlled trial was cholestyramine. The Coronary Primary Prevention Trial, begun in 1973 and concluded in 1984, showed a 12.6 percent reduction in cholesterol among men given the drug cholestyramine and a 19 percent reduction in the risk of heart attack.

Because this was a randomized controlled trial, you might think we wouldn't need any of the methods in this chapter, because they are specifically designed to replace RCTs in situations where you only have observational data. But that is not true. This trial, like many RCTs, faced the problem of noncompliance, when subjects randomized to receive a drug don't actually take it. This will reduce the apparent effectiveness of the drug, so we may want to adjust the results to account for the noncompliers. But as always, confounding rears its ugly head. If the noncompliers are different from the compliers in some relevant way (maybe they are sicker to start with?), we cannot predict how they would have responded had they adhered to instructions.

In this situation, we have a causal diagram that looks like Figure 7.11. The variable Assigned (Z) will take the value 1 if the patient is randomly assigned to receive the drug and 0 if he is randomly assigned a placebo. The variable Received will be 1 if the patient actually took the drug and 0 otherwise. For convenience, we'll also

This definition is perfectly legitimate for someone in possession of a probability function over counterfactuals. But how is a biologist or economist with only scientific knowledge for guidance supposed to assess whether this is true or not? More concretely, how is a scientist to assess whether ignorability holds in any of the examples discussed in this book?

To understand the difficulty, let us attempt to apply this explanation to our example. To determine if *ED* is ignorable (conditional on *EX*), we are supposed to judge whether employees who would have one potential salary, say $S_1 = s$, are just as likely to have one level of education as the employees who would have a different potential salary, say $S_1 = s'$. If you think that this sounds circular, I can only agree with you! We want to determine Alice's potential salary, and even before we start—even before we get a hint about the answer—we are supposed to speculate on whether the result is dependent or independent of *ED*, in every stratum of *EX*. It is quite a cognitive nightmare.

As it turns out, *ED* in our example is not ignorable with respect to *S*, conditional on *EX*, and this is why the matching approach (setting Bert and Caroline equal) would yield the wrong answer for their potential salaries. In fact, their estimates should differ by an amount S_1(Bert) − S_1(Caroline) = –$9,500. The reader should be able to show this from the numbers in Table 8.1 and the three-step procedure.) I will now show that with the help of a causal diagram, a student could see immediately that *ED* is not ignorable and would not attempt matching here. Lacking a diagram, a student would be tempted to assume that ignorability holds by default and would fall into this trap. (This is not a speculation. I borrowed the idea for this example from an article in *Harvard Law Review* where the story was essentially the same as in Figure 8.3 and the author did use matching.)

Here is how we can use a causal diagram to test for (conditional) ignorability. To determine if *X* is ignorable relative to outcome *Y*, conditional on a set *Z* of matching variables, we need only test to see if *Z* blocks all the back-door paths between *X* and *Y* and no member of *Z* is a descendant of *X*. It is as simple as that! In our example, the proposed matching variable (Experience) blocks

Joe is legally responsible for her death even though he did not light the fire.

How can we express necessary or but-for causes in terms of potential outcomes? If we let the outcome Y be "Judy's death" (with $Y = 0$ if Judy lives and $Y = 1$ if Judy dies) and the treatment X be "Joe's blocking the fire escape" (with $X = 0$ if he does not block it and $X = 1$ if he does), then we are instructed to ask the following question:

> Given that we know the fire escape was blocked ($X = 1$) and Judy died ($Y = 1$), what is the probability that Judy would have lived ($Y = 0$) if X had been 0?

Symbolically, the probability we want to evaluate is $P(Y_{X=0} = 0 \mid X = 1, Y = 1)$. Because this expression is rather cumbersome, I will later abbreviate it as "*PN*," the *probability of necessity* (i.e., the probability that $X = 1$ is a necessary or but-for cause of $Y = 1$).

→ italicize

Note that the probability of necessity involves a contrast between two different worlds: the actual world where $X = 1$ and the counterfactual world where $X = 0$ (expressed by the subscript $X = 0$). In fact, hindsight (knowing what happened in the actual world) is a critical distinction between counterfactuals (rung three of the Ladder of Causation) and interventions (rung two). Without hindsight, there is no difference between $P(Y_{X=0} = 0)$ and $P(Y = 0 \mid do(X = 0))$. Both express the probability that, under normal conditions, Judy will be alive if we ensure that the exit is not blocked; they do not mention the fire, Judy's death, or the blocked exit. But hindsight may change our estimate of the probabilities. Suppose we observe that $X = 1$ and $Y = 1$ (hindsight). Then $P(Y_{X=0} = 0 \mid X = 1, Y = 1)$ is not the same as $P(Y_{X=0} = 0 \mid X = 1)$. Knowing that Judy died ($Y = 1$) gives us information on the circumstances that we would not get just by knowing that the door was blocked ($X = 1$). For one thing, it is evidence of the strength of the fire.

In fact, it can be shown that there is no way to capture $P(Y_{X=0} = 0 \mid X = 1, Y = 1)$ in a *do*-expression. While this may seem like a rather arcane point, it does give mathematical proof for confounders between mediator and outcome. Yet those who eschew the language of diagrams (some economists still do) complain and confess that it is a torture to explain what this warning means.

Thankfully, the problem that Kruskal once called "perhaps insoluble" was solved two decades ago. I have this strange feeling that Kruskal would have enjoyed the solution, and in my fantasy I imagine showing him the power of the *do*-calculus and the algorithmization of counterfactuals. Unfortunately, he retired in 1990, just when the rules of *do*-calculus were being shaped, and he died in 2005.

I'm sure that some readers are wondering: What finally happened in the Berkeley case? The answer is, nothing. Hammel and Bickel were convinced that Berkeley had nothing to worry about, and indeed no lawsuits or federal investigations ever materialized. The data hinted at reverse discrimination against males, and in fact there was explicit evidence of this: "In most of the cases involving favored status for women it appears that the admissions committees were seeking to overcome long-established shortages of women in their fields," Bickel wrote. Just three years later, a lawsuit over affirmative action on another campus of the University of California went all the way to the Supreme Court. Had the Supreme Court struck down affirmative action, such "favored status for women" might have become illegal. However, the Supreme Court upheld affirmative action, and the Berkeley case became a historical footnote.

A wise man leaves the final word not with the Supreme Court but with his wife. Why did mine have such a strong intuitive conviction that it is utterly impossible for a school to discriminate while each of its departments acts fairly? It is a theorem of causal calculus similar to the sure-thing principle. The sure-thing principle, as Leonard ~~Jimmie~~ Savage originally stated it, pertains to total effects, while this theorem holds for direct effects. The very definition of a direct effect on a global level relies on aggregating direct effects in the subpopulations.

To put it succinctly, local fairness everywhere implies global fairness. My wife was right.

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it can have a blueprint summary of its major software components. Other components can then reason about that blueprint and mimic a state of self-awareness.

To create the perception of agency, we must also equip this software package with a memory to record past activations, to which it can refer when asked, "Why did you do that?" Actions that pass certain patterns of path activation will receive reasoned explanations, such as "Because the alternative proved less attractive." Others will end up with evasive and useless answers, such as "I wish I knew why" or "Because that's the way you programmed me."

In summary, I believe that the software package that can give a thinking machine the benefits of agency would consist of at least three parts: a causal model of the world; a causal model of its own software, however superficial; and a memory that records how intents in its mind correspond to events in the outside world.

This may even be how our own causal education as infants begins. We may have something like an "intention generator" in our minds, which tells us that we are supposed to take action $X = x$. But children love to experiment—to defy their parents', their teachers', even their own initial intentions—and to do something different, just for fun. Fully aware that we are supposed to do $X = x$, we playfully do $X = x'$ instead. We watch what happens, repeat the process, and keep a record of how good our intention generator is. Finally, when we start to adjust our own software, that is when we begin to take moral responsibility for our actions. This responsibility may be an illusion at the level of neural activation but not at the level of self-awareness software.

Encouraged by these possibilities, I believe that strong AI with causal understanding and agency capabilities is a realizable promise, and this raises the question that science fiction writers have been asking since the 1950s: Should we be worried? Is strong AI a Pandora's box that we should not open?

Recently public figures like Elon Musk and Stephen Hawking have gone on record saying that we should be worried. On Twitter, Musk said that AIs were "potentially more dangerous than nukes." In 2015, John Brockman's website Edge.org posed as its annual question, that year asking, "What do you think about machines

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CHAPTER 6. PARADOXES GALORE!

Annotated Bibliography

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2014, p. 201). The equivalent "three prisoners dilemma" was used to demonstrate the inadequacy of non-Bayesian approaches in Pearl (1988, pp. 58–62).

Tierney (July 21, 1991) and Crockett (2015) tell the amazing story of vos Savant's column on the Monty Hall paradox; Crockett gives several other entertaining and embarrassing comments that vos Savant received from so-called experts. Tierney's article tells what Monty Hall himself thought of the fuss—an interesting human-interest angle!

An extensive account of the history of Simpson's paradox is given in Pearl (2009, pp. 174–182), including many attempts by statisticians and philosophers to resolve it without invoking causation. A more recent account, geared for educators, is given in Pearl (2014).

Savage (2009), Julious and Mullee (1994), and Appleton, French, and Vanderpump (1996) give the three real-world examples of Simpson's paradox mentioned in the text (relating to baseball, kidney stones, and smoking, respectively).

Savage's sure-thing principle (Savage, 1954) is treated in Pearl (2016b), and its corrected causal version is derived in Pearl (2009, pp. 181–182).

Versions of Lord's paradox (Lord, 1967) are described in Glymour (2006); Hernández-Díaz, Schisterman, and Hernán (2006); Senn (2006); Wainer (1991); Wainer and Brown (2007). A comprehensive analysis can be found in Pearl (2016a).

Paradoxes invoking counterfactuals are not included in this chapter but are no less intriguing. For a sample, see Pearl (2013).

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