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Probability and Causality

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Preface

What can we do to reduce global warming? How can we prevent another global financial crisis? How to fight AIDS? How can we reduce hunger in the world? These questions ask about causal effects of interventions. Obviously, interventions based on the wrong causal theories and hypotheses will cost the life of many and huge amounts of money that could be spent more appropriately. Even if our daily problems are less dramatic, they are of the same nature. Just think about your own actions that you have to chose in your responsibilities as a student, scientist, teacher, physician, psychologist, politician, or just as a parent! Whatever you do has effects, and these effects might be different if you take one action instead of another one. It is these kind of thoughts that make us believe that there is no other issue in the methodology of empirical sciences that deserves and needs more attention and effort than causality. And because the dependencies we are investigating are of a nondeterministic nature, we need a *probabilistic theory of causality*. In other words, we need to understand *probability* and *causality*.

What This Book is About

Empirical causal research involves several inferences and substantive interpretations. Among these are:

(a) statistical inference, i. e., the inference from sample data to parameters characterizing the distributions of random variables,
(b) causal inference, i. e., the inference from parameters characterizing the distributions of random variables to causal effects and/or dependencies,
(c) substantive interpretation of the putative cause,
(d) substantive interpretation of the outcome variable,
(e) substantive interpretation of the random experiment considered.

This book does not deal with all these points. We will neither discuss the mathematics of statistical inference nor the substantive issues of construct validity or external validity (Campbell & Stanley, 1963; Cook & Campbell, 1979; Shadish, Cook, & Campbell, 2002) involved in points (c) to (e). Instead we will focus on the second point: causal inference, i. e., the inference from parameters (such as the expectations of an outcome variable in two treatment conditions) to causal effects and/or causal dependencies. This is what the probabilistic theory of causality presented in this book is about. As will be shown in this book, causal effects are

also parameters that characterize the joint distributions of the random variables considered in a random experiment. However, their definitions are less obvious than 'ordinary' expectations and their differences.

Basic Idea

In order to get a first impression of what this means, let us briefly formulate the basic idea that can most easily be explained if the putative (or presumed) cause is a treatment variable. Suppose an individual, or in more general terms, an observational unit, could be treated by condition 1 or it could be treated by condition 0, *everything else being invariant*. If there is a difference in the outcome considered (some measure of success of the treatment), then this difference is due to the difference in the two treatment conditions. This conception goes back at least to Mill (1843/1865).

Multiple Determinacy

The problem with this first version of the basic idea is that most outcomes are *multiply determined*, i. e., they are not only influenced by the treatment variable, but by many other variables as well. In the field of agricultural research, e. g., the *yield* (*outcome*) of a *variety* not only depends on the variety (*treatment*) itself, but it also depends on the quality of the *plot* (*observational unit*), such as the average hours of sunshine on the plot per day, the amount of water reaching the plot, and the number of microbes in the plot, etc. Although Mill's idea sounds perfect, it is not immediately clear which implications it has for practice, because the number of other causes is often too large for keeping constant all of them. Furthermore Mill's idea fails to distinguish between covariates and intermediate variables. Holding constant all intermediate as well — and not only all covariates — would imply that there is no treatment effect any more, if we assume that all treatment effects have to be transmitted by some intermediate(s) (see section 3.3 for a more detailed discussion).

Because of the problem of multiple determinacy, Mills conception has been complemented by Sir Ronald A. Fisher (1925/1946) and by Jerzy S. Neyman (1923/1990) in the second and third decades of the last century. Simply speaking, introducing the randomized experiment, Fisher replaced the *ceteris paribus* clause ('everything else invariant') by the *ceteris paribus distributionibus* clause: *all other possible causes (the 'covariates') having the same distribution*. This is what random assignment of units to treatment conditions secures.

A Metaphor — The Invisible Man and his Shadow

Imagine an invisible man. Although we cannot see him, suppose we know that he is there, because we can see his shadow. Furthermore, suppose we would like to measure his size. Doing that, we have two problems, a theoretical and a practical one. The *theoretical problem* is to define *size*. We have to clarify that we do not

mean 'volume' or 'weight', but 'height' — without shoes, and without hat and hair. Unfortunately, actual height varies slightly in the course of a day. Hence, we define *size* to be the average of the actual heights at the different times of the day. This solves the theoretical problem; now we know what we want to measure.

However, because the man is invisible, we cannot measure his *size* directly — and this is not only because his size slightly varies over the day. The crucial problem is that we can only observe his shadow. And this is the *practical problem*: How to determine his size from his shadow? Sometimes, there is almost no shadow at all, sometimes it is huge. Some geometrical reflection yields a first simple solution: measuring the shadow when the sun has an angle of 45°. But what if it is winter and the sun does not reach this angle and if traveling to another point of the earth is too expensive? Now we need more geometrical knowledge, taking into account the actual angle of the sun and the observed length of the shadow. This will yield an exact measure of the *size* of the invisible man as well.

Determining a causal effect we face the same kind of problems. First, we have to define a *causal effect*, and second, we have to find out how to determine it from empirical estimable parameters such as true means, i. e., from expectations. The simple solution — corresponding to the 45° angle of the sun in the metaphor — is the perfect randomized experiment. The sample mean differences we see in a randomized experiment only randomly deviate from the causal effect (due to random sample variation). In contrast, in quasi-experiments and observational studies, solutions to the practical problem are more sophisticated. They are also more sophisticated than in the problem of the invisible man, because it is not only *one* other variable (the angle) that determines the length of the shadow; instead there often are *many* other variables systematically determining the sample means as well as the true means that are estimated by these sample means. This is again the problem of multiple determinacy.

This book presents a solution to the theoretical and the practical problems mentioned above. Unfortunately, both solutions are not as simple and obvious as in our metaphor. Furthermore, there is not only one single kind of causal effects. (In the paragraphs above we referred to total causal effects.) To our knowledge, the first pioneer tackling the theoretical *and* the practical problems was Jerzy S. Neyman (1923/1990).

Individual and Average Causal Effects

While Fisher introduced the design technique of randomization, Neyman introduced the concepts of individual and average causal effects, thus attempting a first solution to the theoretical problem mentioned above. (Note, however, that he used different terms for these concepts). He assumed that, for each individual plot, there is an intra-individual (plot-specific) distribution of the outcome variable, say Y, under each treatment. He then simply defined the *individual causal effect of treatment x compared to treatment x'* to be the difference between the intra-individual (plot-specific) expectation of Y (the "true yield") given treatment ("variety") x and the intra-individual (plot-specific) expectation of Y given

treatment ("variety") x'. Having defined the individual causal effect, the *average treatment effect* is simply the expectation of the corresponding individual (plot-specific) causal effects in the population of observational units (plots). Similarly, several kinds of *conditional effects* can be defined, conditioning, for instance, on covariates, i. e ., on other causes of Y that cannot be affected by X, such as measures of the *quality of the soil*, *average hours of sunshine*, *average hours of rain*, etc.

Total, Direct, and Indirect Effects

At about the same time as Neyman and Fisher developed their ideas, Sewall Wright (Wright, 1918, 1921, 1923, 1934, 1960a, 1960b) developed his ideas on path analysis and the concepts of total, direct, and indirect effects. While his *total effect* aims at the same idea as the average causal effect, his *direct* and *indirect effects* were new. Simply speaking, in the context of an experiment or quasi-experiment, a direct effect of the treatment is the effect that is not transmitted through an intermediate variable; it is the conditional effect of the treatment variable holding constant the intermediate variable on one of its values. In contrast, the *indirect effect* is the difference between the total effect and the direct effect.

Fundamental Problem of Causal Inference

Whereas the basic ideas outlined above are relatively simple and straightforward, trying to put them into practice — i. e., solving the practical problem mentioned above — is often difficult and needs considerable sophistication. The "fundamental problem of causal inference" (Holland, 1986) is that we cannot expose an observational unit to treatment 1 and, at the same time, to treatment 0. However, this is exactly what is necessary if we want to be sure that 'everything else is invariant', a clause that is also an implicit idea in the solution proposed by Neyman.

Pre-Post Designs

If we choose to first observe a unit under 'no treatment' and then observe it again after 'treatment', we may be tempted to interpret the pre-post differences as estimates of the individual causal effects of the treatment given in between. However, this interpretation might be wrong, because the unit may have developed (maturated, learned), may have suffered from critical life events, may have experienced historical change, etc. (see, e. g., Campbell & Stanley, 1963; Cook & Campbell, 1979; Shadish et al., 2002). Hence, in these *pre-post designs* or synonymously, *within-group designs*, we have to make assumptions on the nature of these possible alternative interpretations of the pre-post comparisons, e. g., that they do not hold in the application considered or that they have a certain structure that can be taken into account when making causal inferences based on pre-post comparisons.

Between-Group Designs

If, instead of making comparisons within a unit, we compare different units to each other in *between-group experiments*, we certainly lose the possibility of estimating the *individual* causal effects. However, what we can hope for is that we are still able to estimate the *average causal effect* and certain conditional causal effects. But how to estimate the average of the individual causal effects if the individual causal effects are not estimable? Both, between-group experiments and quasi-experiments, have a set of (observational) units, at least two experimental conditions ('treatment conditions', 'expositions', 'interventions', etc.), and at least one outcome variable ('response', 'criterion', 'dependent variable') Y. In the medical sciences, the units are usually patients. In psychology the observational units are often persons, but it could be persons-in-a-situation, or groups as well. In economics it could be subjects, companies, or countries, for instance. In educational sciences the units might be school classes, schools, communities, districts, or countries. In sociology and the political sciences, the units could be persons, but also communities, countries, etc.

Scope of the Theory

In order to delineate the scope of the theory, consider the following kind of *random experiment*: Draw an observational unit u (e. g., a person) out of a set of units, observe the value z of a (possibly multivariate qualitative or quantitative) covariate Z for this unit, assign the unit or observe its assignment to one of several experimental conditions, observe the value m of an intermediate variable M, and record the numerical value y of the outcome variable Y. We will use U to denote the random variable representing with its value u the unit drawn. Note that many observations can be made additional to observing U, Z, X, M, and Y. Although this simple single-unit trial is a prototype of the kind of empirical phenomena the theory is dealing with, there are other single-unit trials in which the theory can be applied as well (see ch. 2). In fact, the theory is applicable far beyond the true experiment and the quasi-experiment. This includes applications in which the putative causes are *not* manipulable and in which the putative cause is a continuous random variable. The theory has its limitations only if there is no clear ordering of the random variables considered as putative causes or outcomes.

True Experiments and Quasi-Experiments

The single-unit trial described above is a random experiment, but not necessarily a randomized experiment. A *randomized experiment* is a special random experiment in which the unit drawn is *randomly assigned* to one of the treatment conditions, e. g., depending on the outcome of a coin toss. (In empirical applications, the single-unit trials are repeated n times, where n denotes the sample size.) Referring to single-unit trials, we can distinguish the *true experiment* from

the *quasi-experiment* as follows: In the *true experiment*, there are at least two treatment conditions and the assignment to one of the treatment conditions is randomized, e. g., by flipping a coin. In a traditional *randomized experiment*, for instance, the treatment probabilities are chosen to be equal for all units. However, equal treatment probabilities for all units are neither essential for the definition of the true experiment nor for drawing valid causal inferences. We may as well have treatment probabilities depending on the units and/or on another covariate (see section 7.5), as long as these treatment probabilities are fixed or known by the researcher. Note, however, that in designs, in which different units have different treatment probabilities, standard data analysis techniques such as t-tests or analysis of variance do not test the correct hypotheses any more.

For between-group designs, the *quasi-experiment* may be defined such that there are at least two treatment conditions; however, in contrast to the true experiment, the treatment probabilities are unknown. Nevertheless, valid causal inferences can be drawn in quasi-experiments *provided that we can rely on certain assumptions*. In specific applications these assumptions might be wrong. If they are actually wrong, causal inferences can be completely wrong as well.

Beyond Experiments and Quasi-Experiments

As it turns out, formalizing the ideas outlined above in probabilistic terms results in a theory of probabilistic causality that is applicable far beyond experiments and quasi-experiments, thus bringing together the experimental tradition of Fisher and Neyman on one side and Wright's observational studies tradition on the other side. Furthermore, causal dependencies of manifest variables measuring latent variables as well as causal dependencies between latent variables can be treated in the framework presented in this book. Hence, the scope of the theory also includes what in the past has been addressed only within structural equation modeling (see, e. g., Bentler & Wu, 2002; Jöreskog & Sörbom, 1996/2001; Muthén & Muthén, 1998-2007) and/or graphical modeling (see, e. g., Pearl, 2009; Spirtes, Glymour, & Scheines, 2000). Furthermore, specific psychometric problems such as 'differential item functioning' and 'measurement invariance' turn out to be problems of causal modeling that can be treated within the same theoretical framework as the analysis of causal effects in experimental and quasi-experimental designs.

Who Should Study This Book?

The Methodologist

In the first place, we would like to address the *methodologist*, i. e., the expert in empirical research methodology, especially in the social, economic, behavioral, cognitive, medical, agricultural, and biological sciences. This book provides answers to some of the most important and fundamental questions of these empirical sciences: What do we mean by terms like 'X affects Y', 'X has an effect on Y',

'X influences Y', 'X leads to Y', etc. used in our informal theories and hypotheses? How can we translate these terms into a language that is compatible with the statistical analysis of empirical data? How to design a study and how to look at the resulting data if we want to probe our theories empirically and learn about the causal dependencies postulated in these theories and hypotheses? And last but not least: How to evaluate interventions, treatments, or expositions to (possibly detrimental) environments and learn about how effective they are for which kind of subjects or observational-units, and under which circumstances?

The Statistician

Many statisticians believe that causality is beyond their horizon. Causality might be a matter of substantive researchers and philosophers, they say, but not their own. They think that it cannot be treated mathematically and therefore a statistician cannot be helpful. As a consequence, they ignore the issue of causality. Reading this book will prove that all these beliefs should be abandoned. Probabilistic causality, as presented here, is a branch of probability theory, which itself, at least since Kolmogorov (1956), is a part of pure mathematics — although with an enormous potential for applications in many empirical sciences and even beyond. The main purpose of this book is to translate the informal concepts about causality shared by many methodologists and applied statisticians into the well-defined terms of mathematical probability theory. The principle is not to use any undefined term, and the result is a pure mathematical theory of probabilistic causality. Of course, this will make it harder for the methodologist and those not yet trained in probability theory. However, the reward is a much deeper understanding of what is essential and a much better grasp of the nature of our theories about the real world.

Of course, undefined terms are still used in this book, but only in the examples, in the substantive interpretations, and in the motivations of the definitions. The theory itself is pure mathematics, just in the same way as Kolmogorov's probability theory presented in 1933, which explicated the mathematical, measure-theoretical structure of probabilistic concepts. Substantive meaning only results if we interpret the core components of the formal structure in a specific random experiment considered. And this is also true for the theory of probabilistic causality presented in this book.

The Substantive Scientist

The substantive scientist in the fields mentioned above has at least two good reasons to study this book. The *first* is that some crucial parts of his theories and hypotheses are explicated, at least when it comes to considering a concrete experiment or study. The ambiguity in causal language such as 'X affects Y', 'X has an effect on Y', 'X influences Y', 'X leads to Y' are not necessary any more. Reading this book will make it possible to replace these ambiguous terms by well-

understood and well-defined terms, improving quality of empirical research and theories.

The *second* motivation of the substantive scientist is that even if he knows his own theoretical concepts and hypotheses, he still has to know how to design experiments and studies that enable him to test them. Furthermore, the standard ways of analyzing data offered in the textbooks of applied statistics and in the available computer programs often do not estimate and test the correct causal effects and dependencies. And this is not only bad for the substantive scientist but also for all those relying on the validity of his inferences and his expertise. Just think about all the harmful consequences of wrong causal theories in various substantive fields, if they are applied to solving concrete problems!

The Experimental Scientist

This book has two messages for those who do their research with experiments, a good one and a bad one. The good news is that, in perfect randomized experiments, the average causal total treatment effect is indeed estimated when comparing means between two different treatment conditions. The bad news is that *we can not rely on randomized assignment of units to treatment conditions* when it comes to estimating *direct* and *indirect* effects. More specifically, in such an analysis it is usually not sufficient to consider intermediates, treatment and outcome variables. Instead we also have to include in our analysis *pre-treatment variables* such as a pre-test of the intermediate and a pre-test of the outcome variable and apply adjustment methods, very much in the same way as we have to use these techniques in quasi-experiments — *even though we have randomized*! Hence if you want to look into the black box between the treatment and the outcome variables, you have to adopt the techniques of causal modeling that are far beyond traditional comparisons of means and analysis of variance.

The Philosopher of Science

Philosophers of science study and teach the methodology of empirical sciences. In that respect, their task is very similar to that of the methodologist, perhaps only more general and less specific for a certain discipline. Therefore, it is not surprising that probabilistic causality has also been tackled by philosophers of science (see, e. g., Cartwright, 1979; Spohn, 1980; Stegmüller, 1983; Suppes, 1970). Compared to these approaches, our emphasis is more on those parts of the theory that have implications for the *design* of empirical studies and the *analysis of data* resulting from such studies.

The Students in These Fields

We believe that probabilistic causality is the most rewarding topic in methodology. Although it is tough to get into it, you will get insights why all this methodology stuff was useful and what it was good for. At least this is what our students

say at the end of our curriculum, even if they did not have the choice whether or not to take our course on probabilistic causality.

Research Traditions in Stochastic Causality

Several research traditions have been contributing to the theory probabilistic causality in various ways. From the *Neyman-Rubin tradition*, we adopted the idea that it is important to define various causal effects such as individual, conditional, and average causal effects, even though we modified and extended these concepts in important aspects. Defining causal effects is important for proving that certain methods of data analysis yield estimates of these effects if certain assumptions can be made. Are there conditions under which the analysis of change scores (between pre- and post-tests) and repeated-measures analysis of variance yield causal effects? Under which conditions do we test causal effects in the analysis of covariance? Which are the assumptions under which propensity score methods yield estimates of causal effects? Which are the assumptions under which an instrumental variable analysis estimates a causal effect? All these questions and their answers presuppose that we have a clear definition of causal effects and/or of causal probabilistic dependencies.

From the *Campbellian tradition* (see, e. g., Campbell & Stanley, 1966; Cook & Campbell, 1979; Shadish et al., 2002) we learned that there are questions and problems beyond stochastic causality itself that are relevant in empirical causal research, such as: How to generalize beyond the study? What does the treatment variable mean? What is the meaning of the outcome variable? And, perhaps the most important question: Are there alternative explanations for the effect? The vast majority of social scientists (including ourselves) have been educated in this research tradition to some degree. Although this training is still very useful as a general methodology framework, it lacks precision and clarity in a number of issues — and causality is one of these.

From the *graphical modeling tradition* (see, e. g., Cox & Wermuth, 2004; Pearl, 2009; Spirtes et al., 2000), we learned that conditional independence plays an important role in causal modeling. This research tradition has also been developing techniques to estimate causal effects and to search for causal models if specific assumptions can be made. The fact that randomization in a true experiment in no way guarantees the validity of causal inferences on *direct* effects has been brought up by this research tradition.

Structural equation modeling and *psychometrics* have been teaching us how to use latent variables and structural equation modeling in testing causal hypotheses. Due to a number of statistical programs such as AMOS (Arbuckle, 2006), EQS (Bentler, 1995), lavaan (?, ?), LISREL (Jöreskog & Sörbom, 1996/2001), Mplus (Muthén & Muthén, 1998-2007), OpenMx (OpenMx, 2009), RAMONA (Browne & Mels, 1998), structural equation modeling became extremely popular in the Social Sciences. Although many users of these programs hope to find causal answers, it should be clearly stated that structural equation modeling — and this is true for all kinds of statistical models (including analysis of variance) — does

neither automatically estimate and test causal effects, nor does it provide a satisfactory *theory* of causal effects and dependencies. Nevertheless, this research tradition contributes — just like other areas of statistics — a number of statistical techniques that can be very useful in causal modeling.

In this book, we also aim at embedding — and, where necessary, extending — conventional statistical procedures such as analysis of covariance, nonorthogonal analysis of variance, and latent variable modeling, but also more recent techniques based on propensity scores, or on instrumental variables into a coherent theory of probabilistic causality.

How to Use This Book

This book is self-contained. It is written such that standard mathematical probability theory is sufficient for a complete understanding, provided one takes the time that these topics require. In many parts, this is not a book one can just *read*; instead it is a book to be *studied*. This includes working on the questions and exercises. We presume that the reader is familiar with — or learns while studying this book — the essentials of probability theory, including conditional expectations, as well as conditional independence and conditional distributions. These essentials of probability theory are dealt with in Steyer and Nagel (in press-a).

We devoted this book almost entirely to the *theory* of causal effects and probabilistic causality, although, in chapter 13, we outline the implications of the theory for *design* and for *data analysis in experiments and quasi-experiments*. We also developed the PC program *Causal Effects Explorer* (Nagengast, Kröhne, Bauer, & Steyer, 2007) that can be used for exploring prima facie effects, conditional and average total effects given certain parameters. We believe that this program is useful for teaching and learning the fundamentals of the theory. Furthermore, the program *EffectLiteR* (?, ?), can be used to estimate total, direct, and indirect effects from empirical data in experiments and quasi-experiments. Both programs, which are available at *www.causal-effects.de*, may be used together with this book in a course on causal modeling. In fact, this is the content of our workshops on the analysis of total, direct, and indirect causal effects, which are available both as videos-on-demand on the internet and on DVDs, again at *www.causal-effects.de*.

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Chapter 1
Introductory Examples

For more than a century there have been examples in the statistical literature showing that comparing means or comparing probabilities (e. g., of success of a treatment) between a group exposed to a treatment and a comparison group (unexposed or exposed to a different treatment) does not necessarily answer our substantive questions: 'Which treatment is better overall?' or 'Which treatment is better for which kind of person?' Differences between means and differences between probabilities (or any other comparison between probabilities such as odds ratios, log odds ratios, or relative risk) are usually not the treatment effects we are looking for (see, e. g., Pearson, Lee, & Bramley-Moore, 1899; Yule, 1903; Simpson, 1951). They are just *effects at first sight* or "prima facie effects" (Holland, 1986).

Just like the shadow in the metaphor of the invisible man (see the preface), prima facie effects reflect the effects of the treatment (the size of the invisible man), but also of other causes (the angle of the sun). The goal of analyzing *causal* effects is to estimate the effect of the treatment alone, isolating it from other potential influences, e. g., of sex, educational background, socio-economic status, etc. The general idea is to compute a treatment effect that is not biased by differences between treatment groups that would also exist *without treatment*.

Overview

We will illustrate systematic bias in determining *total* treatment effects in quasi-experiments by two examples. The first one deals with a dichotomous outcome variable, the second with a quantitative one. While the problems described in these two examples cannot occur in a randomized experiment, our third example will show that the randomized assignment of units to treatment conditions does not help to prevent systematic bias in determining *direct* treatment effects with respect to an intermediate variable that may transmit the effects of the treatment on the outcome variable.

1.1 Example 1 — Simpson's Paradox

In our first example, the prima facie effect reverses if we switch from comparing $P(Y{=}1|X{=}1)$ to $P(Y{=}1|X{=}0)$, the conditional probabilities of success between

Table 1.1. Joint Probabilities of Treatment and Success

	Treatment		
Success	No (X=0)	Yes (X=1)	
No (Y=0)	.240	.232	.472
Yes (Y=1)	.360	.168	.528
	.600	.400	1.000

treatment and control, to comparing $P(Y{=}1|X{=}1,Z{=}z)$ to $P(Y{=}1|X{=}0,Z{=}z)$, the corresponding probabilities additionally controlling for Z = *sex* with values m (males) and f (females). This kind of phenomenon, which is already known at least since Yule (1903), is called *Simpson's paradox* (Simpson, 1951), and it is still being debated (see, e. g., Hernán, Clayton, & Keiding, 2011).

1.1.1 Prima Facie Effect

Table 1.1 shows the joint distribution of treatment and success, i. e., the joint probabilities $P(X{=}x,Y{=}y)$ of treatment and success, as well as the marginal probabilities $P(X{=}x)$ and $P(Y{=}y)$ of treatment x and success y, respectively. Comparing the conditional probability of success (Y=1) given the *treatment condition* (X=1) to the conditional probability of success given the *control condition* (X=0) would lead us to the conclusion that the *treatment is harmful*. These two conditional probabilities can be computed by

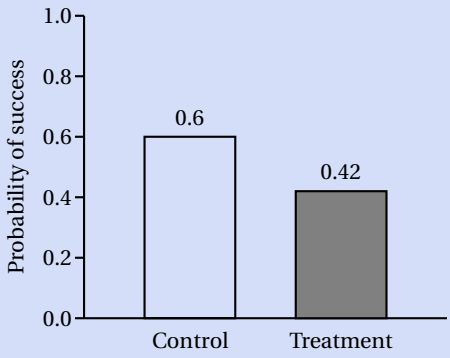

Figure 1.1. Probability of success given treatment

$$P(Y{=}1\,|\,X{=}1) \;=\; \frac{P(Y{=}1, X{=}1)}{P(X{=}1)} \;=\; \frac{.168}{.168+.232} \;=\; .42$$

and

$$P(Y{=}1\,|\,X{=}0) \;=\; \frac{P(Y{=}1, X{=}0)}{P(X{=}0)} \;=\; \frac{.360}{.360+.240} \;=\; .60,$$

respectively (see, e. g., Steyer & Nagel, in press-a, section 4.2). Figure 1.1 displays both conditional probabilities in a histogram.

These two conditional probabilities can be compared to each other in different ways. The simplest one is looking at the *difference* $P(Y{=}1\,|\,X{=}1) - P(Y{=}1\,|\,X{=}0)$. This is a particular case of the difference $E(Y\,|\,X{=}1) - E(Y\,|\,X{=}0)$ between two conditional expectation values, in which the outcome variable Y is dichotomous with values 0 and 1. Following Holland (1986), we will call this difference the (unconditional) *prima facie effect* and use the notation PFE_{10}. Other possibilities of comparing the two conditional probabilities are to look at the odds ratio, or the logarithm of the odds ratio (see chapter 4 of Rothman, Greenland, & Lash, 2008, for a detailed discussion of these and other effect parameters).

1.1.2 Prima Facie Effects Controlling for Sex

The conclusion about the effect of the treatment is completely different if we look at the dependencies separately for males and females. Table 1.2 (p. 4) shows the joint distributions of treatment, success and $Z :=$ *sex* with values 0 (*male*) and 1 (*female*). The probabilities of the two values are $P(Z{=}0) = P(Z{=}1) = .50$. According to this table, the probability of success for the males in the treatment condition is

$$P(Y{=}1\,|\,X{=}1, Z{=}0) \;=\; \frac{.016}{.016+.004} \;=\; .80$$

(see Exercise 1-7), whereas the probability of success in the control condition is

$$P(Y{=}1\,|\,X{=}0, Z{=}0) \;=\; \frac{.336}{.336+.144} \;=\; .70.$$

Hence, the difference

$$P(Y{=}1\,|\,X{=}1, Z{=}0) \;-\; P(Y{=}1\,|\,X{=}0, Z{=}0) \tag{1.1}$$

is $.80 - .70 = .10$, which may lead us to conclude that *the treatment is beneficial for males*. Again, because Y is dichotomous with values 0 and 1, this difference is a particular case of the difference $PFE_{10;\,Z=0} := E(Y\,|\,X{=}1, Z{=}0) - E(Y\,|\,X{=}0, Z{=}0)$, which we call the *conditional prima facie effect* given $Z{=}0$.

What about the treatment effects for females? Table 1.2 shows that the probability of success for the females in the treatment condition is $.152/(.152+.228) = .40$, whereas it is $.024/(.024+.096) = .20$ in the control condition. Figure 1.2 shows these conditional probabilities in a histogram. Considering the difference $.40 - .20 = .20$ may lead us to conclude that *the treatment is also beneficial for females*.

Table 1.2. Joint Probabilities of Treatment, Sex and Success

Males ($Z=0$); $P(Z=0)=0.50$

	Treatment		
Success	No ($X=0$)	Yes ($X=1$)	
No ($Y=0$)	.144	.004	.148
Yes ($Y=1$)	.336	.016	.352
	.480	.020	.500

Females ($Z=1$); $P(Z=1)=0.50$

	Treatment		
Success	No ($X=0$)	Yes ($X=1$)	
No ($Y=0$)	.096	.228	.324
Yes ($Y=1$)	.024	.152	.176
	.120	.380	.500

Hence, we can conclude that the treatment seems to be *beneficial for both, males and females*. This, however, seems to contradict our finding ignoring sex. Just considering the difference $E(Y|X=1)-E(Y|X=0)$, the *treatment seemed to be harmful*.

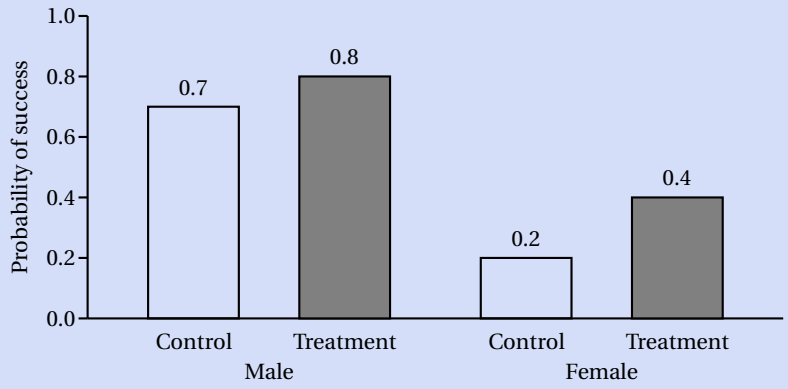

Figure 1.2. Probabilities of success given treatment (and sex)

1.1.3 Prima Facie Effect vs. Average of the Prima Facie Effects

In contrast to our intuition, the *prima facie effect* $E(Y|X=1) - E(Y|X=0)$ is *neither* the *simple average nor any weighted average of the corresponding prima facie effects* $E(Y|X=1, Z=z) - E(Y|X=0, Z=z)$ controlling for Z = *sex*. This is now studied in more detail.

Prima Facie Effect

The probability $P(Y=1|X=0)$ of success in the control condition is the sum of the corresponding probabilities, $P(Y=1|X=0, Z=0)$ and $P(Y=1|X=0, Z=1)$, *weighted by the conditional probabilities* $P(Z=0|X=0)$ and $P(Z=1|X=0)$, respectively, i. e.,

$$\begin{aligned} P(Y=1\,|\,X=0) \;&= P(Y=1\,|\,X=0, Z=0)\cdot P(Z=0\,|\,X=0) + \\ &\quad\; P(Y=1\,|\,X=0, Z=1)\cdot P(Z=1\,|\,X=0) \\ &= .70\cdot\frac{.48}{.60} \;+\; .20\cdot\frac{.12}{.60} \;=\; .60 \end{aligned}$$

[see Box 9.2 (ii) of Steyer & Nagel, in press-a, and Exercise 1-8]. Because the difference between the conditional probabilities $P(Z=0\,|\,X=0) = .48/.60$ and $P(Z=1\,|\,X=0) = .12/.60$ is large, the probability of success in treatment 0 is much closer to .70 than to .20 (see the dots above $X=0$ in Fig. 1.3).

Similarly, the probability $P(Y=1\,|\,X=1)$ of success in the treatment condition ($X=1$) is the sum of the two corresponding probabilities, $P(Y=1\,|\,X=1, Z=0)$ and $P(Y=1\,|\,X=1, Z=1)$, *weighted by the conditional probabilities* $P(Z=0\,|\,X=1)$ and $P(Z=1\,|\,X=1)$, respectively, i. e.,

$$\begin{aligned} P(Y=1\,|\,X=1) \;&= P(Y=1\,|\,X=1, Z=0)\cdot P(Z=0\,|\,X=1) + \\ &\quad\; P(Y=1\,|\,X=1, Z=1)\cdot P(Z=1\,|\,X=1) \\ &= .80\cdot\frac{.02}{.40} + .40\cdot\frac{.38}{.40} \;=\; .42. \end{aligned}$$

Hence, the *prima facie effect* is $P(Y=1\,|\,X=1) - P(Y=1\,|\,X=0) = .42 - .60. = -.18$. Because the two conditional probabilities $P(Z=0\,|\,X=1) = .02/.40$ and $P(Z=1\,|\,X=1) = .38/.40$ are very different, the probability of success in treatment 1 is much closer to .40 than to .80 (see the dots above $X=1$ in Fig. 1.3). (The size of the area of the dotted circles represent the joint probabilities $P(X=x, Z=z)$. For $X=1$ and $Z=0$, this probability is very small such that the circle is not visible. This kind of graphics has been adopted from Agresti, 2007).

Average of the Conditional Prima Facie Effects

In contrast to the prima facie effect, the *average of the conditional prima facie effects* is the expectation of the function $PFE_{10;Z}$, the values of which are the two prima facie effects $PFE_{10;Z=0}$ and $PFE_{10;Z=1}$ for males and females, i. e.,

$$E(PFE_{10;Z}) \;=\; \sum_z PFE_{10;Z=z}\cdot P(Z=z). \tag{1.2}$$

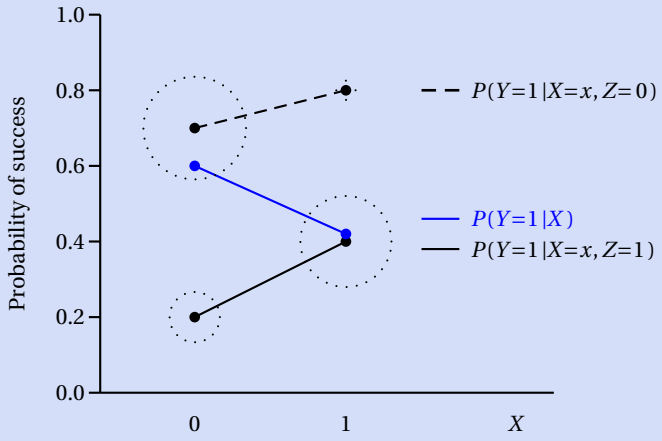

Figure 1.3. Probabilities of success given treatment and sex

Because the conditional prima facie effect of the treatment is $PFE_{10;Z=0} = .10$ for males and $PFE_{10;Z=1} = .20$ for females, the average prima facie effect is simply:

$$E(PFE_{10;Z}) \;=\; .10 \cdot P(Z=0) + .20 \cdot P(Z=1) \;=\; .10 \cdot \frac{1}{2} + .20 \cdot \frac{1}{2} \;=\; .15\,.$$

Hence, whereas the *prima facie effect* $E(Y|X=1) - E(Y|X=0)$ is *negative*, namely $-.18$, the *average of the* $(Z=z)$*-conditional prima facie effects* $E(Y|X=1,Z=z) - E(Y|X=0,Z=z)$ is *positive*, namely .15.

1.1.4 How to Evaluate the Treatment?

Because the conclusions drawn from the differences $E(Y|X=1) - E(Y|X=0)$ and $E(Y|X=1,Z=z) - E(Y|X=0,Z=z)$ are contradictory, which of these comparisons should we trust? Is the treatment harmful — as $E(Y|X=1) - E(Y|X=0)$ suggests? Or is it beneficial as suggested by the differences $E(Y|X=1,Z=z) - E(Y|X=0,Z=z)$? Which of these comparisons are meaningful for evaluating the causal effect of the treatment? Before we come back to these questions, let us consider another example.

1.2 Example 2 — Nonorthogonal Two-Factorial Experiment

In this section, we treat an example with three treatment conditions, three values of a discrete covariate, and a quantitative outcome variable. In this example, we use a 3×3 factorial design with crossed, non-orthogonal factors. The analysis of

Table 1.3. Conditional Expectations Given Treatment

Treatment	Expectation of Y in treatment conditions $E(Y \mid X{=}x)$	Treatment probabilities $P(X{=}x)$
$X{=}0$ (Control)	111.25	1/3
$X{=}1$ (Treatment 1)	100.00	1/3
$X{=}2$ (Treatment 2)	114.25	1/3
$E(Y)$	108.50	

such designs has been puzzling many statisticians (see, e. g., Aitkin, 1978; Appelbaum & Cramer, 1974; Carlson & Timm, 1974; Gosslee & Lucas, 1965; Jennings & Green, 1984; Keren & Lewis, 1976; Kramer, 1955; Overall & Spiegel, 1969, 1973b, 1973a; Overall, Spiegel, & Cohen, 1975; Williams, 1972), and it continues to do so (see, e. g., Langsrud, 2003; Nelder & Lane, 1995).[1]

1.2.1 Prima Facie Effects

In the example presented in Table 1.3, there are *three treatment conditions* representing two treatments and a control. The outcome variable Y is now a quantitative measure of success. The expectations of the outcome variable Y in the three treatment conditions are displayed in Table 1.3. The ratios in the last column are the treatment probabilities $P(X{=}x)$ which are, in this example, the same for all three treatment conditions. However, although the probabilities $P(X{=}x)$ are the same for all three groups, this is *not* a randomized design as will become obvious if we look at the second factor and the 'cell probabilities' (see Table 1.4). Discussing the example at the level of conditional expectation values will again make clear that the contradictory inferences are not due to errors in *statistical inference* (from sample statistics to true parameters), but due to errors in *causal inference*, i. e., they are due to the misinterpretation of the differences between the expectations $E(Y \mid X{=}x)$ of the outcome variable Y in the three treatment conditions as causal effects.

If our evaluation of the treatment effects were based on these differences between the expectations of Y in the three treatment conditions, we would conclude that there are two treatment effects: a *negative effect* (namely, 100.00 –

[1] In fact, none of the statistical packages such as SAS, SysStat, or SPSS with their Type I, II, III or IV sums of squares provide correct estimates and tests of the average effects (or main effects) for such a design unless the covariate (the second factor) has a uniform distribution, with equal probabilities for all values of the covariate. In this case Type III analysis yields correct results, at least, if the second factor is assumed to be fixed. However, in most applications in the Social Sciences, the covariate (second factor) is not fixed but stochastic with varying sample means, etc. In chapter 13, we will outline a correct analysis including the average total effects.

Table 1.4. Conditional Expectations Given Treatment and Neediness

	Neediness						
Treatment	Low (Z=0)		Medium (Z=1)		High (Z=2)		
X=0	120	(20/120)	110	(17/120)	60	(3/120)	(40/120)
X=1	100	(7/120)	100	(26/120)	100	(7/120)	(40/120)
X=2	80	(3/120)	90	(17/120)	140	(20/120)	(40/120)
		(30/120)		(60/120)		(30/120)	

Note. Probabilities $P(X{=}x, Z{=}z)$, $P(Z{=}z)$, and $P(X{=}x)$ in parentheses.

111.25 = −11.25) of treatment 1 compared to the control, and a *positive effect* (namely, 114.25 − 111.25 = 3.00) of treatment 2 compared to the control.

1.2.2 Prima Facie Effects Controlling for Neediness

A second way to evaluate the 'effects' of the three *treatment conditions* is to look at the differences between the expectations of Y in the three treatment conditions *within each of the three classes of neediness* for the therapy: low, medium, and high. Table 1.4 displays the expectations of the outcome variable Y in the nine cells of the 3 × 3 design. The ratios in parentheses are the probabilities that the pairs (x, z) of values of X and Z are observed. Hence, this table contains the conditional expectation values (true cell means) of the outcome variable Y, and the probabilities $P(X{=}x, Z{=}z)$ determining the true joint distribution of X and Z.

In the *low neediness condition* ($Z{=}0$), there are large negative effects, both of treatment 1 and of treatment 2 compared to the control:

$$PFE_{10;Z=0} := E(Y|X{=}1, Z{=}0) - E(Y|X{=}0, Z{=}0) = 100 - 120 = -20$$

and

$$PFE_{20;Z=0} := E(Y|X{=}2, Z{=}0) - E(Y|X{=}0, Z{=}0) = 80 - 120 = -40.$$

In the *medium neediness condition* ($Z{=}1$), there are also negative effects of treatment 1 and of treatment 2 compared to the control:

$$PFE_{10;Z=1} := E(Y|X{=}1, Z{=}1) - E(Y|X{=}0, Z{=}1) = 100 - 110 = -10$$

and

$$PFE_{20;Z=1} := E(Y|X{=}2, Z{=}1) - E(Y|X{=}0, Z{=}1) = 90 - 110 = -20.$$

Finally, in the *high neediness condition* ($Z{=}2$), the effects of treatment 1 and treatment 2 are both positive:

$$PFE_{10;Z=2} := E(Y|X=1,Z=2) - E(Y|X=0,Z=2) = 100-60 = 40$$

and

$$PFE_{20;Z=2} := E(Y|X=2,Z=2) - E(Y|X=0,Z=2) = 140-60 = 80.$$

Based on these comparisons, we can conclude that the 'effects' of the treatments depend on the neediness of the subjects: the differences between the expectations of Y are negative for subjects with low and medium neediness, and they are positive for the subjects with high neediness.

1.2.3 Prima Facie Effects vs. Average of the Prima Facie Effects

There is no doubt that the conditional effects given neediness, which are sometimes also called *simple effects*, are more informative than average treatment effects if we want to know which treatment is the best for which level of neediness. Nevertheless, we might ask: What are the 'treatment effects' on average? Or, in other words which are the 'main effects'? In fact, all major statistical programs compute 'main effects' (see Langsrud, 2003 for a list on which program suggests what solution to this problem). Note that we have two average effects in this example, because we can compare treatment 1 *and* treatment 2 to the control. Because we already looked at the corresponding conditional effects, we just have to compute their averages, i. e., the expectations of these conditional effects over the distribution of neediness:

$$E(PFE_{10;Z}) = \sum_z PFE_{10;Z=z} \cdot P(Z=z) = -20 \cdot \frac{1}{4} + (-10) \cdot \frac{1}{2} + 40 \cdot \frac{1}{4} = 0.$$

Hence, the average effect of treatment 1 compared to the control is zero.

Comparing treatment 2 to the control yields on average:

$$E(PFE_{20;Z}) = \sum_z PFE_{20;Z=z} \cdot P(Z=z) = -40 \cdot \frac{1}{4} + (-20) \cdot \frac{1}{2} + 80 \cdot \frac{1}{4} = 0.$$

According to this result, the average effect of treatment 2 compared to the control is zero as well.

1.2.4 How to Evaluate the Treatment?

To summarize, we discussed three ways that may, at first sight, be used to evaluate the treatment effects: *First*, we may compare the differences between the expectations $E(Y|X=x)$ of the outcome variable in the three treatment conditions $X=0$, $X=1$, and $X=2$. *Second*, we may consider the corresponding differences between the conditional expectations $E(Y|X=x,Z=z)$ *within each of the three values* $Z=0$, $Z=1$, *and* $Z=2$ *of neediness. Third*, we may compare the averages of these differences between the conditional expectations over the distribution of

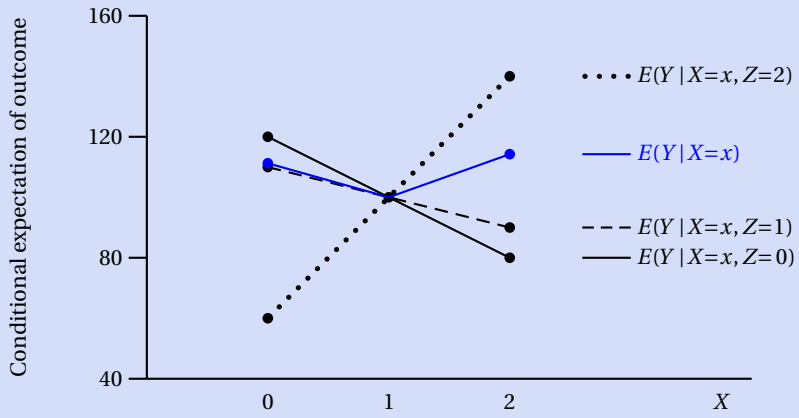

Figure 1.4. Conditional expectation values of Y given treatment and neediness

Z (see Box 1.1 for a summary of these effects).[2] All these comparisons yield different results. Which of them are meaningful for the evaluation of the treatment effects? All three of them, or only two, just one, or none at all?

1.3 Example 3 — Direct Effect in a Randomized Experiment

The problems described in the examples treated in the preceding sections occur because there are covariates (in the examples, *sex* and *neediness*) that are related to the treatment variable *and* the outcome variable. Hence, these problems can *not* occur in a randomized experiment, in which, by definition, all covariates and the treatment are (stochastically) independent. Hence, if in a randomized experiment, we are only interested in the *total effects* of the treatment on the outcome variable, the effects that are estimated by the differences between means in the treatment groups are the total effects of the treatment. However, often we are also interested in the mediation processes producing these total effects. A typical question in educational research is: 'Is there a direct effect of the treatment that is not transmitted through *motivation after treatment*?' In medical research we may ask: 'Is there a direct effect of the treatment that is not transmitted through the *amount of antibodies*?'

[2] In fact, there are even more than three ways. Types II and III of computing the sums of squares in nonorthogonal ANOVA are not yet considered in our discussion. In chapter 13, we show that all four types of computing sums of squares in such a design yield wrong results in our example (see also Exercise 1-14).

Table 1.5. Covariances, Correlations, and Expectations (Omitting Pre-Tests)

		X	M	Y
Treatment (yes = 1, no = 0)	X	0.25	*.727*	*.597*
Post-test motivation	M	5.00	189.00	*.893*
Post-test achievement	Y	5.00	205.70	280.45
Expectations		0.50	90.00	140.00

Note. Correlations (in italics) are rounded.

1.3.1 Conditional Expectation of Y Given Treatment and Intermediate Variables

Suppose that Table 1.5 displays the true means, variances, covariances, and correlations of a treatment variable X with values 0 and 1, an intermediate variable M, and an outcome variable Y. (This example is adopted from Mayer, Thoemmes, Rose, Steyer, & West, 2014.)

First of all, let us consider the conditional expectation $E(Y \mid X, M)$, assuming that it can be written as a linear function of X and M (see Fig. 1.5). In fact, the covariance matrix presented in Table 1.5 has been constructed such that this linearity assumption holds. Using the covariances and expectations displayed in this table, we receive

$$E(Y \mid X, M) \approx 34.9924 - 3.7528 \cdot X + 1.1876 \cdot M \tag{1.3}$$

(see Exercise 1-12). According to textbook wisdom (see, e. g., MacKinnon, 2008, but also Baron & Kenny, 1986), the direct effect of X on Y, controlling for M, is approximately -3.75.

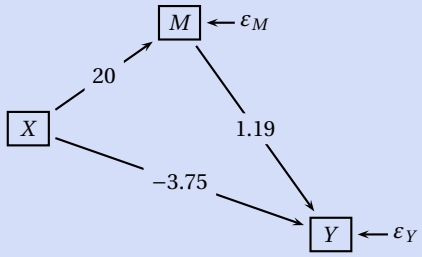

Figure 1.5. Path diagram of $E(M \mid X)$ and $E(Y \mid X, M)$

Table 1.6. Covariances, Correlations, and Expectations (Including Pre-Tests)

		W	Z	X	M	Y
Pre-test achievement	W	100.00	*.850*	*.000*	*.495*	*.740*
Pre-test motivation	Z	85.00	100.00	*.000*	*.582*	*.696*
Treatment (yes=1, no=0)	X	0.00	0.00	0.25	*.727*	*.597*
Post-test motivation	M	68.00	80.00	5.00	189.00	*.893*
Post-test achievement	Y	124.00	116.50	5.00	205.70	280.45
Expectations		100.00	100.00	0.50	90.00	140.00

Note. Correlations (in italics) are rounded.

1.3.2 Conditional Expectation of Y Given Treatment, Intermediate, and Pre-Test Variables

Suppose M represents *post-test motivation* in a randomized experiment designed to evaluate two teaching methods represented by $(X=0)$ and $(X=1)$, respectively. In this case, even if not observed, there will be a variable, say Z representing *pre-test motivation* with respect to which students will differ before treatment. Furthermore, there will be a variable, say W, representing *pre-test achievement* with respect to which students will differ prior to treatment as well. Furthermore, the two pre-test variables Z and W will be correlated. This is a plausible scenario for such a teaching experiment, and this is how the complete variance-covariance matrix and the expectations presented in Table 1.6 have been generated.

Hence, if instead of $E(Y|X,M)$, we consider the conditional expectation of Y given X, M, Z, and W, again assuming linearity — and this is how the parameters presented in Table 1.6 have been generated — we receive

$$E(Y|X,M,Z,W) = .00 + 10\cdot X + 0.50\cdot M + 0.00\cdot Z + .90\cdot W \tag{1.4}$$

(see Exercise 1-13). Now the coefficient 10 of X might be interpreted to be the direct treatment effect, 'direct' with respect to the intermediate variable M. It is the effect of X controlling for the intermediate variable M *and* for all covariates, in this example, the two pre-test variables Z and W.

How can we explain this seemingly paradoxical result? How can there be confounding in a perfect randomized experiment? The answer is that even though X and the bivariate random variable (W,Z) are independent, *conditional independence* of X and (W,Z) given M does *not* hold. Instead, conditioning on M induces conditional *dependence* of X and Z, if both Z and X are related to M. Intuitively speaking, because both Z and X affect M, a high value of *post-test motivation* M means that both, X and Z tend to be high, whereas a low value of M means that both, X and Z tend to be low (see Fig. 1.6). Hence, conditioning on M, the treatment variable X and the *pre-test motivation* Z will be dependent, even though X and Z are unconditionally independent, due to randomization (see also Pearl,

2009, ch. 1, p. 17, or Spirtes et al., 2000). This conditional dependence between X and Z given M is also reflected by a non-zero partial correlation $Corr(X, Z; M)$ (see section 11.6 of Steyer & Nagel, in press-b).

1.3.3 Conditional Expectation of Y Given Treatment Variable

Finally, let us consider the *average total treatment effect*. In this example, in which X and all covariates are independent, the average total treatment effect is the coefficient of X in the equation

$$E(Y|X) = 130 + 20 \cdot X, \tag{1.5}$$

where the intercept $\alpha_0 = 130$ is obtained by $\alpha_0 = E(Y) - \alpha_1 \cdot E(X) = 140 - 20 \cdot 0.50 = 130$ and the slope by $\alpha_1 = Cov(X, Y)/Var(X) = 5.00/0.25 = 20$ [see Steyer & Nagel, in press-a, Eqs. (12.58) and (12.59)]. Therefore, in this example, the *indirect treatment effect* is the difference $20 - 10 = 10$. In this model with no interaction, this indirect effect is also equal to the product $20 \cdot .50$ (see Fig. 1.6), which is in accordance with the rules of path analysis developed by Sewall Wright in the twenties of last century (see, e. g., Wright, 1918, 1921, 1923).

1.3.4 How to Analyze Direct Effects?

We discussed two different ways to analyze the direct effect of the treatment variable on the outcome variable. The first one is recommended in traditional textbooks such as MacKinnon (2008) and in one of the most frequently cited papers Baron and Kenny (1986). It yields the negative direct effect of −3.75. The second one also controls for the pre-tests of the intermediate variable and the outcome variables. This second analysis yields a direct treatment effect of 10. Hence, the effect is reversed as compared to the first analysis. Which is the correct direct effect? Or are both wrong?

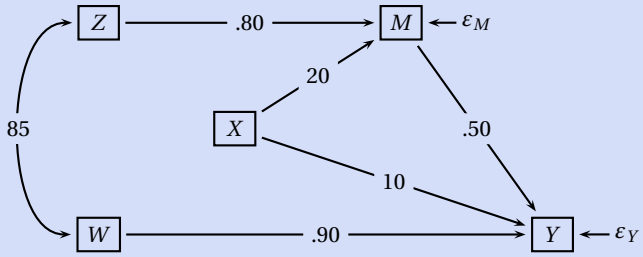

Figure 1.6. Path diagram of $E(M|X, Z, W)$ and $E(Y|X, M, Z, W)$

1.4 Summary and Conclusions

In this chapter, we treated three examples. In the first example, a dichotomous treatment variable X has a negative 'effect' $E(Y|X=1)-E(Y|X=0)$ on a *dichotomous outcome variable* Y ('success'), although the corresponding treatment 'effects' $E(Y|X=1,Z=z)-E(Y|X=0,Z=z)$ are positive if we condition on males ($Z=m$) and females ($Z=f$). Taking the expectation of these two conditional effects also yielded a positive 'effect'. In the second example, there are nonzero differences $E(Y|X=1) - E(Y|X=0)$ and $E(Y|X=2) - E(Y|X=0)$, where Y is a *quantitative outcome variable*, and nonzero conditional 'effects' $E(Y|X=1,Z=z) - E(Y|X=0,Z=z)$ and $E(Y|X=2,Z=z) - E(Y|X=0,Z=z)$ for the different values of *neediness*. The expectations of these conditional 'effects' over the three neediness conditions, i. e., the average 'effects', are zero. In the third example, we discussed two different ways of analyzing the direct treatment effect. The first yields a negative 'direct effect' and the second a positive 'direct effect'.

The Problem

Because the conclusions drawn from these analyses are contradictory, which of these should we trust? In Simpson's paradox: Is the treatment harmful — as the difference $E(Y|X=1)-E(Y|X=0)$ suggests? Or is it beneficial as suggested by the differences $E(Y|X=1,Z=z)-E(Y|X=0,Z=z)$, controlling for sex? Which of these comparisons are meaningful for the evaluation of the causal effects of the treatment? Similarly, in the second example: are there treatment effects, overall? Or are the effects nil on average? And, are the conditional effects dependable, or could it be that they would also be reversed if we condition on an additional covariate, such as *age* or *educational status*? As demonstrated in Simpson's paradox, we can neither expect that the difference $E(Y|X=1)-E(Y|X=0)$ is the average of the corresponding differences $E(Y|X=1,Z=z)-E(Y|X=0,Z=z)$, nor can we expect that a difference $E(Y|X=1,Z=z)-E(Y|X=0,Z=z)$ is the average over the corresponding differences if we condition on an additional covariate such as age. Note, these questions are not related to *statistical* inference; they are not raised at the sample level, but on the level of true parameters!

Hence our examples show that the conditional expectation values and their differences, the prima facie effects, can be totally misleading in evaluating the effects of a treatment variable X on an outcome variable Y. This conclusion can also be extended to conditional probabilities, to correlations and to all other parameters describing relationships and dependencies between random variables. They all are like the shadow in the metaphor of the invisible man (see the preface).

If this is true, is the whole idea of *learning from experience* — the core of empirical sciences — wrong? Our answer is 'No'. However, we have to be more explicit in what we mean by terms like 'X affects Y', 'X has an effect on Y', 'X influences Y', 'X leads to Y', etc. used in our theories and hypotheses. How can these terms be translated into a language compatible with statistical analyses of empirical data?

Box 1.1 Glossary of New Concepts

$PFE_{xx'}$ — *Prima facie effect* of treatment x compared to treatment x'. It is defined by

$$PFE_{xx'} := E(Y|X{=}x) - E(Y|X{=}x').$$

$PFE_{xx';Z=z}$ — $(Z{=}z)$-*Conditional prima facie effect* of treatment x compared to treatment x'. It is defined by

$$PFE_{xx';Z=z} := E(Y|X{=}x, Z{=}z) - E(Y|X{=}x', Z{=}z).$$

$E(PFE_{xx';Z})$ — *Expectation of the* $(Z{=}z)$-*conditional prima facie effects* of treatment x compared to treatment x'. It is defined by

$$E(PFE_{xx';Z}) := \sum_z PFE_{xx';Z=z} \cdot P(Z{=}z).$$

How to design a study and how to look at the resulting data if we want to probe our theories empirically and learn about the causal dependencies postulated in these theories and hypotheses?

We know that a reversal of total effects does not occur in the randomized experiment, i. e., in an experiment in which observational units (in the social and behavioral sciences, usually the subjects or individuals) are randomly assigned to one of at least two treatment conditions. In the randomized experiment comparing expectation values *is* informative about total causal treatment effects. But why? What is so special in the randomized experiment? Which are the conditions allowing for causal inference in the randomized experiment? Can we create these conditions also in quasi-experimental studies? How can we estimate causal effects in quasi-experiments? And why does randomization not help if we analyze direct treatment effects? Obviously, conclusive answers to these questions can be hoped for only within a theory of causal effects.

Relevance of the Problem

These questions are of fundamental importance for the methodology of empirical sciences and for the empirical sciences themselves. The answers to these questions have consequences for the design and analysis of experiments, quasi-experiments, and other studies aiming at estimating the effects of *treatments*, *interventions*, or *expositions* on certain outcome variables. No *prevention study* can be meaningfully conducted without knowing the concepts of causal effects and how they can be estimated from empirical data, and the same is true for the *evaluation of institutions* such as schools, universities, or clinics with respect to their effects on the outcomes of their clients. Similarly, without a clear concept

of causal effects we are not able to learn from our data about the effects of a certain (possibly harmful) environment on our health, or about the effects of certain behaviors such as smoking or drug abuse. Again, this is similar to the problem of measuring the invisible man's size via the length of his shadow: only with a clear concept of *size*, some basic knowledge in geometry, and the additional information such as the angle of the sun at the time of measurement, are we able to determine his size from the length of his shadow.

Furthermore, without an explicit theory of causal effects we are not able to study direct and indirect effects, and *this is true even in a perfect randomized experiment*. For example, if we are interested in whether or not the effect of vaccination is completely transmitted through the amount of a certain type of antibodies, then this cannot be done relying only on the benefits of a perfect randomized trial. Instead we have to apply certain adjustment techniques. In terms of our metaphor, the 45° angle (the randomized experiment) does not help in determining the parameters we are looking for (the direct effects).

Research Traditions

Of course, raising these questions and attempting answers is not new. Immense knowledge and wisdom about experiments and quasi-experiments has been collected in the Campbellian tradition of experiments and quasi-experiments (see, e. g., Campbell & Stanley, 1963; Cook & Campbell, 1979; Shadish et al., 2002). In the last decades, a more formal approach has been developed supplementing the Campbellian theory and terminology in important aspects: the theory of causal effects in the Neyman-Rubin tradition (see, e. g., Splawa-Neyman, 1923/1990; Rubin, 1974, 2005). Many papers and books indicate the growing influence of this theory (see, e. g., Greenland, 2000, 2004; Höfler, 2005; Rosenbaum, 2002; Rubin, 2006; Winship & Morgan, 1999; Morgan & Winship, 2007) and formidable efforts have already been made to integrate it into the Campbellian framework (West, Biesanz, & Pitts, 2000). Furthermore, these questions have also been dealt with in the graphical modeling tradition (see, e. g., Pearl, 2009; Spirtes et al., 2000) as well as in biometrics, econometrics, psychometrics, and other fields dealing with the methodology of substantive research fields.

Outlook

In this book, we present the theory of total, direct, and indirect causal effects in terms of classical probability theory. We show that a number of questions that have been debated controversially and inconclusively can now be given a clear-cut answer. What kinds of causal effects can be meaningfully defined? Which design techniques guarantee unbiased estimation of causal effects? How to analyze nonorthogonal ANOVA designs (cf., e. g., Aitkin, 1978; Appelbaum & Cramer, 1974; Gosslee & Lucas, 1965; Maxwell & Delaney, 2004; Overall et al., 1975)? How to analyze non-equivalent control-group designs (cf., e. g., Reichardt, 1979)? Should we compare pre-post differences between treatment groups (cf., e. g.,

Lord, 1967; Senn, 2006; van Breukelen, 2006; Wainer, 1991)? Should we use analysis of covariance to adjust for differences in treatment and control that already existed prior to treatment (cf., e. g., Maxwell & Delaney, 2004; Cohen, Cohen, West, & Aiken, 2003)? Should we use new techniques such as propensity score methods instead of the more traditional procedures mentioned above (cf., e. g., Rosenbaum & Rubin, 1984)? How do we deal with non-compliance to treatment assignment (cf., e. g., Cheng & Small, 2006; Dunn et al., 2003; Jo, 2002a, 2002b, 2002c; Jo, Asparouhov, Muthén, Ialongo, & Brown, 2008; J. Robins & Rotnitzky, 2004; J. M. Robins, 1998)? How to analyze direct and indirect effects? We do not treat the statistical sampling models with their distributional assumptions, their implications for parameter estimation, and the evaluation (or tests) of hypotheses about these parameters. However, in chapter 13 we discuss the virtues and problems of general strategies of data analysis such as the analysis of difference scores, analysis of covariance, its generalizations, analysis based on propensity scores, and instrumental variables.

1.5 Exercises

▷ **Exercise 1-1** Why do we need the concept of a causal treatment effect?

▷ **Exercise 1-2** What is the relationship between the unconditional prima facie effect PFE_{10} and the expectations $E(Y|X=0)$ and $E(Y|X=1)$ of the outcome variable Y in the two treatment conditions?

▷ **Exercise 1-3** Verify that Table 1.1 (p. 2) is in fact obtained by collapsing the two corresponding tables for males and females (see Table 1.2, p. 4).

▷ **Exercise 1-4** Which are the three kinds of prima facie effects treated in this chapter?

▷ **Exercise 1-5** What is the difference between statistical inference and causal inference?

▷ **Exercise 1-6** Why are the conditional expectation values $E(Y|X=x)$ in treatment conditions x also probabilities for $Y=1$ in the first example treated in this chapter?

▷ **Exercise 1-7** Compute the conditional probability $P(Y=1 \mid X=1, Z=0)$ from Table 1.2 (p. 4).

▷ **Exercise 1-8** Compute the probability $P(Y=1|X=0)$ of success in the control condition.

▷ **Exercise 1-9** What are the unconditional prima facie effects of the treatments, i. e., the prima facie effects $E(Y|X=1)-E(Y|X=0)$ and $E(Y|X=2)-E(Y|X=0)$ in the second example of this chapter?

▷ **Exercise 1-10** What are the conditional prima facie effects of the treatments, i. e., the prima facie effects $E(Y|X=1,Z=z)-E(Y|X=0,Z=z)$ and $E(Y|X=2,Z=z)-E(Y|X=0,Z=z)$ in the second example of this chapter?

▷ **Exercise 1-11** What are the averages of the conditional prima facie effects

$$E(Y|X=1,Z=z) - E(Y|X=0,Z=z) \qquad \text{and} \qquad E(Y|X=2,Z=z) - E(Y|X=0,Z=z)$$

in the second example of this chapter?

▷ **Exercise 1-12** Compute the coefficients of the equation for the conditional expectation $E(Y|X,M)$ presented in Equation (1.3).

▷ **Exercise 1-13** Compute the coefficients of the equation for the conditional expectation $E(Y|X,M,Z,W)$ presented in Equation (1.4).

▷ **Exercise 1-14** Download *table.1.4.10000.sav* from *www.causal-effects.de*. This data set has been generated from Table 1.4 (p. 8) for a sample of size $N = 10.000$.

(a) Estimate the cell means and the relative frequencies of observations in each of the nine cells of the 3×3 table.

(b) Use each of the procedures offered by your statistical program package to analyze the data including a test of the main effects of the treatment factor (most programs offer Typ I, II and III sums of squares for such an analysis).

(c) Compare the results of these analyses to the parameters presented in Table 1.4 (p. 8).

▷ **Exercise 1-15** Download *table.1.6.10000.sav* from *www.causal-effects.de*. This data set has been generated from Table 1.6 (p. 12) for a sample of size $N = 10.000$.

(a) Estimate the conditional expectation of Y given X and M.

(b) Estimate the conditional expectation of Y given X, M, Z and W.

(c) Compare the estimated regression coefficients to the parameters presented in Equations (1.3) and (1.4), respectively.

Solutions

▷ **Solution 1-1** We need the concept of a causal treatment effect, because Simpson's paradox shows that differences between expectations are meaningless for the evaluation of the effects of a treatment, unless we can show how the differences between expectations are related to the causal effects. Without a definition of causal treatment effects, this would not be possible. Estimating causal treatment effects is crucial for answering questions such as 'Does the treatment help our patients with respect to the outcome variable considered?'

▷ **Solution 1-2** The unconditional prima facie effect PFE_{10} is defined as the difference between the two expectations $E(Y|X=1)$ and $E(Y|X=0)$.

▷ **Solution 1-3** This can easily be verified by adding the probabilities for the observations of the pairs (x,z) of X and Z over males and females. This yields $.144 + .096 = .240$, $.004 + .228 = .232$, $.336 + .024 = .360$ and $.016 + .152 = .168$.

▷ **Solution 1-4** The three kinds of prima facie effects treated in this chapter are: the *unconditional prima facie effect*, the *conditional prima facie effect* given the value z of a covariate Z, and the *average of the* $(Z=z)$*-conditional prima facie effects*. The unconditional prima facie effect of treatment 1 compared to treatment 0 is the difference $PFE_{10} := E(Y|X=1) - E(Y|X=0)$ between the expectations of an outcome variable Y in the two treatment conditions. The $(Z=z)$-conditional prima facie effect is the difference $PFE_{10;Z=z} := E(Y|X=1,Z=z) - E(Y|X=0,Z=z)$ between the $(Z=z)$-conditional expectations of the outcome variable Y in the two treatment conditions. The average prima facie effect is the expectation of the conditional prima facie effects [see Eq. (1.2)].

▷ **Solution 1-5** In *statistical* inference we estimate and test hypotheses about parameters characterizing the distribution of a random variable from sample data. In *causal* inference we interpret some of these parameters as causal effects.

▷ **Solution 1-6** $E(Y|X{=}x) = P(Y{=}1|X{=}x)$, because, in this example, Y is dichotomous with values 0 and 1. In this case, $E(Y|X{=}x) := \sum_y y \cdot P(Y{=}y|X{=}x)$ [see Steyer & Nagel, in press-a, Eq. (9.19)] yields $E(Y|X{=}x) = 0 \cdot P(Y{=}0|X{=}x) + 1 \cdot P(Y{=}1|X{=}x) = P(Y{=}1|X{=}x)$.

▷ **Solution 1-7** According to Table 1.2 (p. 4) ,

$$P(Y{=}1|X{=}1,Z{=}0) = \frac{P(X{=}1,Y{=}1,Z{=}0)}{P(X{=}1,Z{=}0)} = \frac{.016}{.016+.004} = .80.$$

▷ **Solution 1-8** First of all, note that the theorem of total probability, can also be applied to conditional probabilities, in this exercise, the $(X{=}0)$-conditional probabilities. Hence, according to this theorem,

$$P(Y{=}1|X{=}0) = P(Y{=}1|X{=}0,Z{=}0)\cdot P(Z{=}0|X{=}0) + P(Y{=}1|X{=}0,Z{=}1)\cdot P(Z{=}1|X{=}0).$$

The probabilities $P(Y{=}1|X{=}0,Z{=}0) = .70$ and $P(Y{=}1|X{=}0,Z{=}1) = .20$ are computed analogously to Exercise 1-7 and the other two probabilities occurring in this formula are $P(Z{=}0|X{=}0) = .48/.60$ and $P(Z{=}1|X{=}0) = .12/.60$ (see Table 1.2, p. 4). Hence,

$$P(Y{=}1|X{=}0) = \frac{.70\cdot.48}{.60} + \frac{.20\cdot.12}{.60} = .60.$$

▷ **Solution 1-9** The prima facie effects $E(Y|X{=}1) - E(Y|X{=}0)$ and $E(Y|X{=}2) - E(Y|X{=}0)$ can be computed from Table 1.3 (p. 7). They are as follows:

$$PFE_{10} = E(Y|X{=}1) - E(Y|X{=}0) = 100.00 - 111.25 = -11.25$$

and

$$PFE_{20} = E(Y|X{=}2) - E(Y|X{=}0) = 114.25 - 111.25 = 3.00.$$

▷ **Solution 1-10** The conditional prima facie effects $E(Y|X{=}1,Z{=}z) - E(Y|X{=}0,Z{=}z)$ and $E(Y|X{=}2,Z{=}z) - E(Y|X{=}0,Z{=}z)$ can be computed from Table 1.4 (p. 8). For *low neediness* $(Z{=}0)$, they are:

$$PFE_{10;Z=0} = E(Y|X{=}1,Z{=}0) - E(Y|X{=}0,Z{=}0) = 100 - 120 = -20$$

$$PFE_{20;Z=0} = E(Y|X{=}2,Z{=}0) - E(Y|X{=}0,Z{=}0) = 80 - 120 = -40.$$

For *medium neediness* $(Z{=}1)$, they are:

$$PFE_{10;Z=1} = E(Y|X{=}1,Z{=}1) - E(Y|X{=}0,Z{=}1) = 100 - 110 = -10$$

$$PFE_{20;Z=1} = E(Y|X{=}2,Z{=}1) - E(Y|X{=}0,Z{=}1) = 90 - 110 = -20.$$

Finally, for *high neediness* $(Z{=}2)$, the conditional prima facie effects are:

$$PFE_{10;Z=2} = E(Y|X{=}1,Z{=}2) - E(Y|X{=}0,Z{=}2) = 100 - 60 = 40$$

$$PFE_{20;Z=2} = E(Y|X{=}2,Z{=}2) - E(Y|X{=}0,Z{=}2) = 140 - 60 = 80.$$

▷ **Solution 1-11** Using the results of the last exercise, the average of the $(Z{=}z)$-conditional prima facie effects can be computed from the conditional effects as follows:

$$\begin{aligned} E(PFE_{10;Z}) &= PFE_{10;Z=0}\cdot P(Z{=}0) + PFE_{10;Z=1}\cdot P(Z{=}1) + PFE_{10;Z=2}\cdot P(Z{=}2) \\ &= -20\cdot\frac{1}{4} - 10\cdot\frac{1}{2} + 40\cdot\frac{1}{4} = 0. \end{aligned}$$

$$\begin{aligned} E(PFE_{20;Z}) &= PFE_{20;Z=0}\cdot P(Z{=}0) + PFE_{20;Z=1}\cdot P(Z{=}1) + PFE_{20;Z=2}\cdot P(Z{=}2) \\ &= -40\cdot\frac{1}{4} - 20\cdot\frac{1}{2} + 80\cdot\frac{1}{4} = 0. \end{aligned}$$

▷ **Solution 1-12** The two coefficients $\beta_1 \approx -3.7528$ and $\beta_2 \approx 1.1876$ are obtained by

$$\begin{aligned} \boldsymbol{\beta} = \boldsymbol{\Sigma}_{VV}^{-1}\boldsymbol{\Sigma}_{Vy} &= \begin{pmatrix}\beta_1\\ \beta_2\end{pmatrix} \approx \begin{pmatrix}0.25 & 5.00\\ 5.00 & 189\end{pmatrix}^{-1}\begin{pmatrix}5.00\\ 205.70\end{pmatrix} \\ &\approx \begin{pmatrix}8.4944 & -0.2247\\ -0.2247 & 0.0112\end{pmatrix}\begin{pmatrix}5.00\\ 205.70\end{pmatrix} \approx \begin{pmatrix}-3.7528\\ 1.1876\end{pmatrix} \end{aligned}$$

[see Steyer & Nagel, in press-a, Eq. (12.54)]. The appropriate statements in R are:

```
a=matrix(c(.25,5,5,189),byrow=T,nrow=2,ncol=2)
b=matrix(c(5,205.7),byrow=T,nrow=2,ncol=1)
round(solve(a,b),4)
```

In this equation, $\boldsymbol{\Sigma}_{VV}^{-1}$ denotes the inverse of the covariance matrix of $V := (X, M)$ and $\boldsymbol{\Sigma}_{Vy}$ the covariance vector of $V = (X, M)$ and Y. The intercept $\beta_0 \approx 34.989$ is obtained by

$$\begin{aligned} \beta_0 &\approx E(Y) - \beta_1\cdot E(X) + \beta_2\cdot E(M) \\ &\approx E(Y) + 3.7528\cdot E(X) - 1.1876\cdot E(M) \\ &\approx 140 + 3.7528\cdot 0.50 - 1.1876\cdot 90 \approx 34.9924 \end{aligned}$$

[see Steyer & Nagel, in press-a, Eq. (12.53)].

▷ **Solution 1-13** The coefficients γ_1 to γ_4 of

$$E(Y|X, M, Z, W) = \gamma_0 + \gamma_1\cdot X + \gamma_2\cdot M + \gamma_3\cdot Z + \gamma_4\cdot W$$

are obtained by

$$\begin{aligned} \boldsymbol{\gamma} = \boldsymbol{\Sigma}_{RR}^{-1}\boldsymbol{\Sigma}_{Ry} &= \begin{pmatrix}\gamma_1\\ \gamma_2\\ \gamma_3\\ \gamma_4\end{pmatrix} = \begin{pmatrix}0.25 & 5.00 & 0.00 & 0.00\\ 5.00 & 189 & 80 & 68\\ 0.00 & 80 & 100 & 85\\ 0.00 & 68 & 85 & 100\end{pmatrix}^{-1}\begin{pmatrix}5.00\\ 205.70\\ 116.50\\ 124.00\end{pmatrix} \\ &= \begin{pmatrix}20.0000 & -0.8000 & 0.6400 & 0.0000\\ -0.8000 & 0.0400 & -0.0320 & 0.0000\\ 0.6400 & -0.0320 & 0.0616 & -0.0306\\ 0.0000 & 0.0000 & -0.0306 & 0.0360\end{pmatrix}\begin{pmatrix}5.00\\ 205.70\\ 116.50\\ 124.00\end{pmatrix} = \begin{pmatrix}10.00\\ 0.50\\ 0.00\\ 0.90\end{pmatrix} \end{aligned}$$

[see again Steyer & Nagel, in press-a, Eq. (12.54)]. In this equation, $\boldsymbol{\Sigma}_{RR}^{-1}$ denotes the inverse of the covariance matrix of $R := (X, M, Z, W)$ and $\boldsymbol{\Sigma}_{Ry}$ the covariance vector of $R = (X, M, Z, W)$ and Y. The appropriate statements in R are:

```
a=matrix(c(.25,5,0,0,5,189,80,68,0,80,100,85,0,68,85,100),
         byrow=T,nrow=4,ncol=4)
b=matrix(c(5,205.7,116.5,124),byrow=T,nrow=4,ncol=1)
round(solve(a,b),4).
```

The intercept $\gamma_0 = 0.00$ is obtained by

$$\begin{aligned}\gamma_0 &= E(Y) - [\gamma_1 \cdot E(X) + \gamma_2 \cdot E(M) + \gamma_3 \cdot E(Z) + \gamma_4 \cdot E(W)] \\ &= E(Y) - 10 \cdot E(X) - 0.50 \cdot E(M) - 0.00 \cdot E(Z) - .90 \cdot E(W) \\ &= 140 - 10 \cdot 0.50 - 0.50 \cdot 90 - 0.00 \cdot 100 - .90 \cdot 100 \;=\; 0.00.\end{aligned}$$

[see again Steyer & Nagel, in press-a, Eq. (12.53)].

▷ **Solution 1-14** No solution provided. Just compare your results to the parameters presented in Table 1.4 (p. 8).

▷ **Solution 1-15** No solution provided. Just compare your estimated parameters to the true parameters presented in Equations (1.3) and (1.4).

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