Thinking Clearly About Correlations and Causation: Graphical Causal Models for Observational Data

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Abstract

Correlation does not imply causation; but often, observational data are the only option, even though the research question at hand involves causality. This article introduces readers to causal inference based on observational data with the help of graphical causal models, a powerful tool to think more clearly about the interrelations between variables. It expounds on the rationale behind the statistical control of third variables, common procedures for statistical control, and what can go wrong during their implementation. Certain types of third variables—colliders and mediators—should not be controlled for, because in these cases, statistical control can actually move the estimated association farther away from the underlying causal effect. More subtle variations of such "harmful control" include the use of unrepresentative samples that can undermine the validity of causal conclusions, and conceptual problems associated with mediation analysis. Drawing valid causal inferences on the basis of observational data is not a mechanistic procedure but rather always depends on assumptions that require domain knowledge and that can be more or less plausible. However, this caveat holds not only for research based on observational data, but for all empirical research endeavors.

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Psychologists in many fields face a dilemma. Whereas most researchers are aware that randomized experiments are considered the "gold standard" for causal inference, manipulation of the independent variable of interest will often be unfeasible, unethical, or simply impossible. One can hardly assign couples to stay married or get a divorce; nonetheless, one might be interested in the causal effect of divorce on well-being. One cannot randomly resettle individuals into different strata of society, but one might be concerned about the causal effects of social class on behavior. One cannot randomize children to different levels of adversity, yet one might care about the potential negative consequences of childhood adversity on adult health. This article will provide very general guidelines for researchers who are interested in any of the many research questions that require causal inferences to be made on the basis of observational data.

Researchers from different areas of psychology have chosen different strategies to cope with the weaknesses of observational data. To circumvent the issue altogether, some researchers have implemented "surrogate interventions": If the real-life cause of interest cannot be manipulated, there might be a proxy that can be randomized in the lab. For example, an influential study on the effects of social class on prosocial behavior included an experimental manipulation of *perceived* social class. Participants were asked to compare themselves with either the top or the bottom of the "social ladder," temporarily changing their subjective assessment of their social class (Piff, Kraus, Côté, Cheng, & Keltner, 2010). Such surrogates can result in valuable insights, but they are not a panacea as they come with a well-known trade-off (e.g., Cook & Campbell, 1979): Whereas they substantially improve confidence in the *internal* validity of a study (i.e., clear causal relationships can be established with only minimal additional assumptions), they might substantially decrease the *external*

validity, that is, it becomes uncertain whether the finding says much about other situations, other operationalizations of the independent variable, or the world outside the lab in general. For example, how is the effect of being instructed to compare yourself with the bottom of the ladder related to the effect of being born with a silver spoon in your mouth? How is the effect of comparing yourself with the top of the ladder related to the effect of constantly having to worry about how to pay your bills? These questions are non-trivial research projects on their own.

Researchers who instead decide to rely on observational data often attempt to deal with its weaknesses by cautiously avoiding causal language: They use terms such as "associations," "relationships," or tentative "links" between variables instead of clear cause—effect relationships, and they usually add a general disclaimer ("Of course, as the data were only observational, future experiments are needed…"). But again, in many instances, this is not a satisfactory solution. Most substantive questions are concerned with *causal* effects, and, "[a]s humans, we cannot avoid thinking in terms of causality" (Asendorpf, 2012). Carefully crafted language will not prevent readers—let alone the public—from jumping to causal conclusions, and many studies that are based on observational data will probably get published only because they *suggest* that they are able to provide information about meaningful causal effects.

Last but not least, many researchers have tried to bridge the gap between observational data and (more or less explicit) causal conclusions by statistically controlling for third variables. Alas, such attempts often lack proper justification: The choice of control variables is determined by norms in the domain and by the variables available in the data set. Often, the analysis follows the rationale that "more control" is always better than less. Models resulting from such an approach have been labeled "garbage-can regression" (Achen, 2005) because the idea that the inclusion of a multitude of control variables will *necessarily* improve (and

will not worsen) causal inference is a methodological urban legend at best (Spector & Brannick, 2011). In addition, even if the right variables are statistically included in the models, other issues (e.g., neglecting measurement error) can result in the wrong conclusions (Westfall & Yarkoni, 2016). These issues will be discussed throughout this article.

The purpose of this article is to provide psychologists with a primer to a more principled approach to making causal inferences on the basis of observational data. Such coherent frameworks (see, e.g., Morgan & Winship, 2014, for a comprehensive yet accessible introduction) are more common in social science domains that rely heavily on observational data (e.g., economics and sociology). Because of the nature of the research questions pursued in these fields, randomized experiments are often not an option—thus, a systematic approach to make sense of observational data is needed.

This article makes use of *Directed Acyclic Graphs* (DAGs). DAGs provide visual representations of causal assumptions. They were mainly developed by the computer scientist Judea Pearl (e.g., Pearl, 1995; see Pearl, Glymour, & Jewell, 2016, for an introduction), and share many features with *Structural Equation Models* (SEMs).[1] Importantly, DAGs offer an intuitive approach for how to think about causal structures. Even if one does not wish to completely adopt a comprehensive formal framework for causal inference, some basic knowledge of DAGs can be helpful for addressing a number of questions that are of interest to psychologists who work with observational data. What third variables need to be controlled for? Which third variables can be ignored? And in which situations will statistical control *worsen* causal inference?

[1] Knowledge of SEMs will be helpful but not necessary to follow this article.

The answers to these questions necessarily depend on assumptions about the causal web underlying the variables of interest. It is *impossible* to infer causation from correlation without background knowledge about the domain (e.g., Robins & Wasserman, 1999). However, the need to make certain assumptions should not be a reason to abandon observational research. In fact, experimental studies require assumptions as well—for example, experiments might take place in restricted laboratory settings, and to generalize results from such studies to everyday life will require assumptions as well. The critical point is thus not *whether* a research design hinges on additional assumptions, but *which* assumptions need to be made. Regardless of the research design, *awareness* and *transparent communication* of these assumptions allows critical assessments of causal claims to be made and thus lays the foundation for productive scientific debates.

A Brief Introduction to Directed Acyclic Graphs

Assume that we are interested in the causal effect of educational attainment on income. To keep it simple, let's assume that educational attainment has only two levels: college degree versus no college degree by age 30. To establish temporal order, we measure income at age 40. We observe that individuals with a college degree have an average income of $1,500 per week, whereas those without a degree make about $700. From this observation, we cannot conclude that getting a college degree *causes* income to increase by $800. It is very likely that individuals who have a college degree differ from people who do not on many other variables, and these variables might also affect income. Potentially, these variables might even fully account for any difference in income between the two groups, rendering the effect of a college degree to be zero.

Such a situation is depicted in Figure 1: The relationship between educational attainment and income is confounded by a common cause: intelligence. To keep it simple,

let's assume that intelligence is a stable trait that does not change from childhood to adulthood, although a later example in this article considers a more complex scenario.

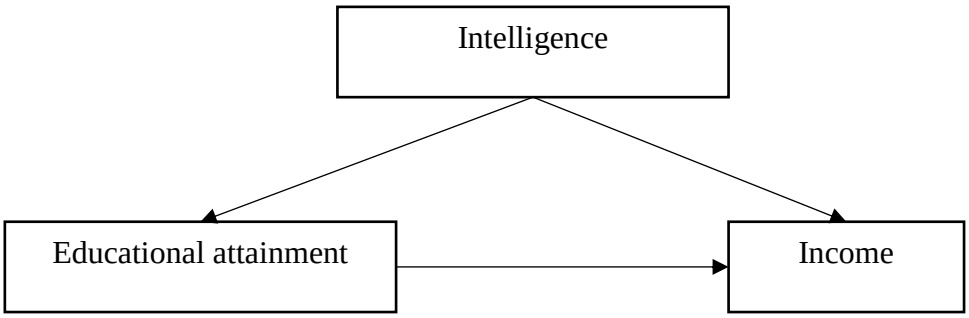

Figure 1. A simple Directed Acyclic Graph (DAG) depicting a causal model in which intelligence has a causal effect on educational attainment and income. In addition, educational attainment also has an effect on income.

Figure 1 encodes causal assumptions. Intelligence, one variable[2] in the model, has a causal effect on educational attainment and a causal effect on income, as denoted by the arrows pointing away from it to the other nodes. Furthermore, an arrow points from educational attainment to income, capturing the idea that educational attainment has a causal effect on income. This figure depicts a Directed Acyclic Graph (DAG) in its most minimalistic version. DAGs consist of nodes (variables) and arrows (also called directed edges) between these nodes, reflecting causal relationships: The assumption is that an experimental manipulation of a variable at which an arrow begins (educational attainment) would change the variable at the end of the arrow (income) if it could be manipulated directly (i.e., in this case, *without* changing intelligence, holding intelligence constant).

[2] Notice that whether or not one assumes that the psychological constructs represented by the variables actually *exist* might have consequences for the interpretation of causal models; however, this topic is outside the scope of this article. Readers interested in the ontological status of psychological constructs—a topic intrinsically related to questions of causality—are referred to, for example, Borsboom, Mellenbergh and van Heerden (2003).

One popular way to think about DAGs is to interpret them as nonparametric SEMs (Elwert, 2013), a comparison that highlights a central difference between DAGs and SEM. Whereas SEMs encode assumptions regarding the form of the relationship between the variables (i.e., per default, arrows in SEMs indicate linear, additive relationships, unless indicated otherwise), an arrow in a DAG might reflect a relationship following *any* functional form (polynomial, exponential, sinusoidal, step function, etc.). The two arrows pointing to the income node in Figure 1 indicate that income can be expressed as an arbitrary function of intelligence and educational attainment, including interactions between the two causes. In this sense, a DAG is *qualitative*: $A \rightarrow B$ means only that *A* causally affects *B* in some way.

Furthermore, in contrast to SEMs, DAGs only allow for single-headed arrows, which is why they are called *directed* graphs. Sometimes, there might be a need to indicate that two variables are non-causally associated because of some unspecified common cause *U*. A double-headed arrow *could* be used to indicate such an association (i.e., $A \leftarrow\rightarrow B$), but this would just be an abbreviation of $A \leftarrow U \rightarrow B$, which again contains only single-headed arrows.

Paths and Elementary Causal Structures

From these two simple building blocks—nodes and arrows—one can visualize more complex situations and trace so-called paths. To make this a bit more interesting, the example from Figure 1 is extended to Figure 2 by adding a new node, school grades, which are affected by intelligence and which affect educational attainment.

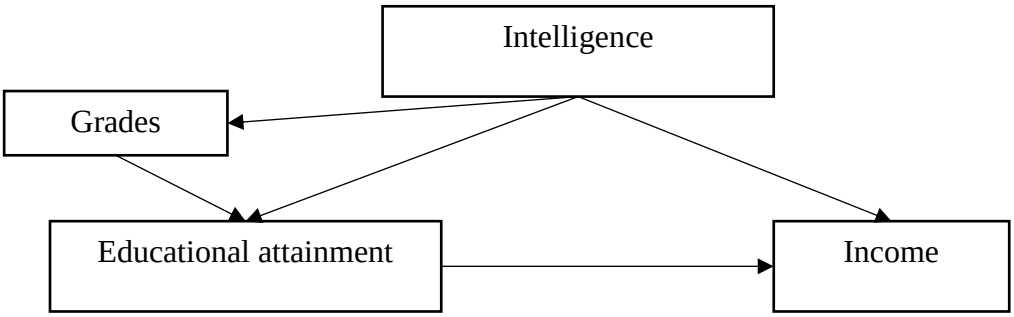

Figure 2. Intelligence affects grades in school which in turn affect educational attainment.

From this DAG, various paths can be discerned by moving from node to node traveling along arrows. In the simplest case, a path leads just from one node to the next one: *Intelligence→Income*. Paths can also include multiple nodes. For example, *Intelligence* and *Income* are additionally connected by the paths *Intelligence→Educational attainment→Income* and *Intelligence→Grades→Educational attainment→Income*. A path can also travel against the direction indicated by the arrows, as for example this path connecting educational attainment and income: *Educational attainment←Grades←Intelligence→Income*. While such paths can become arbitrarily long and complex, they can be broken down into three elementary causal structures: Chains, forks, and inverted forks (see also, Elwert, 2013).

Chains have the structure *A→B→C*, for example, *Intelligence→Educational attainment→Income*. Chains can transmit an association between the node at the beginning and the node at the end: If intelligence causally affects educational attainment, and educational attainment causally affects income, then intelligence and income can be correlated. Importantly, this association reflects a genuine causal effect: In this chain, intelligence causally influences income via educational attainment.

Forks have the structure *A←B→C*, for example, *Educational attainment←Intelligence→Income*. Such a fork can transmit an association, but it is not

causal. If one just considers this fork in isolation, *Educational attainment* and *Income* may be correlated because they share a common cause: *Intelligence*. Forks are the causal structure most relevant for the phenomenon of confounding.

Inverted forks have the structure *A*→*B*←*C*, for example, *Educational attainment*→*Income*←*Intelligence*. Such an inverted fork does not transmit an association: Just because educational attainment and intelligence both affect income does not imply they are in any way correlated. Inverted forks will become relevant later in this article in the context of so-called collider bias.

These three elementary causal structures determine the features of longer paths. If a path only consists of chains, such as *Intelligence*→*Grades*→*Educational attainment*→*Income*, it can transmit a causal association. Along such a chain, variables that are directly or indirectly causally affected by a certain variable are called its *descendants*; conversely, all variables that directly or indirectly affect a certain variable are considered its *ancestors*. For example, *Intelligence* is an ancestor of *Grades*, *Educational attainment*, and *Income*; *Income* is a descendant of *Grades*.

If a path additionally contains forks, such as *Educational attainment*←*Grades*←*Intelligence*→*Income*, it still transmits an association—but it is no longer a causal association because of the confounding variable, *Intelligence*. And whenever a path contains an inverted fork, it is blocked: No association is transmitted. For example, the path *Educational attainment*→*Income*←*Intelligence*→*Grades* does not transmit a correlation between educational attainment and grades.

No Way Back: Acyclicity

DAGs are *acyclic* because they do not allow for cyclic paths in which variables become their own ancestors/descendants. A variable cannot causally affect itself; for example,

in Figure 1, the direction of the path between intelligence and income cannot simply be reversed because this would result in a cyclic path (*Intelligence→Educational attainment→Income→Intelligence*). This may seem counterintuitive: Psychological systems often contain feedback loops, such as reciprocal relationships in which intelligence influences education but education also influences intelligence. Such a feedback loop can be modeled in a DAG (to some extent) by taking the temporal order into account and adding nodes for repeated measures. For example, a DAG could be drawn to show that early childhood intelligence causally influences educational attainment, which in turn influences adult intelligence. Temporal resolution could be "magnified" even further and increased to annual, monthly, or even daily assessments of multiple variables, resulting in more and more nodes in the DAG.[3]

Confounding: The Bane of Observational Data

Having covered the central terminology and rules of DAGs, we are now equipped to approach observational data in a more systematic manner. The central problem of observational data is *confounding*, that is, a common cause that lurks behind the potential cause of interest (the independent variable, in experimental settings often considered the treatment) and the outcome of interest (the dependent variable). Such a confounding influence can introduce what is often called a spurious correlation, which ought not to be confused with

[3] If such a causal process continuously unfolds over time, one would end up drawing an infinite number of nodes capturing each moment in time. In most cases, it will of course not be possible to continuously measure variables; but the (often arbitrary) spacing between time points can have a considerable influence on estimates, making causal inference even more complicated. Continuous time modeling, which lies outside of the scope of this article, relates variables measured at *discrete* time points to an underlying *continuous* model (see, e.g., Voelkle, Oud, Davidov, & Schmidt, 2012, for an SEM-based approach).

a causal effect.[4] Our aim is now to use a DAG to figure out how to remove all these non-causal association so that only the true causal effect remains.

To do that, one must make sure that the DAG includes everything that is relevant to the causal effect of interest. In theory, one can extend the simple DAG in Figure 2 in a large number of different ways. For example, intelligence is certainly not the only cause of educational attainment, and one might want to include additional variables that point to this or other nodes or add generic residuals to indicate that there are other unrelated causal influences as well as measurement error. But not all of these possible extensions of the model are of interest if one plans to investigate the causal relationship between educational attainment and income. A variable that affects educational attainment but has no causal effect on *any* of the other variables in the DAG—neither directly nor indirectly (mediated by other variables)—would not need to be included. Such idiosyncratic factors, including uncorrelated measurement error, are usually not displayed as they do not help in identifying the causal effect (Elwert, 2013). If we want to derive a valid causal conclusion, we need to build a so-called *causal* DAG that is complete if it includes all common causes of any pair of variables that are already included in the DAG (Spirtes, Glymour, & Scheines, 2000). Any additional variable that causally affects at least two variables already included in the DAG—again, either directly or indirectly mediated by other variables that are not yet included—should be included.

After building such a DAG, so-called back-door paths can be discerned. Back-door paths are all paths that start with an arrow pointing to the independent variable and end with an arrow pointing to the dependent variable. In other words, back-door paths indicate that

[4] The extraordinary role of randomized experiments in the testing of causal inferences stems from the simple fact that if the independent variable is randomly assigned, for example by the flip of a coin, it cannot share a common cause with the outcome *by design*.

there might be a common factor affecting both the "treatment", the independent variable, and the "outcome", the dependent variable of interest. In Figure 2, two such back-door paths exist between educational attainment and income: *Educational attainment*←*Grades*←*Intelligence*→*Income* and *Educational attainment*←*Intelligence*→*Income*. Such back-door paths are problematic whenever they transmit an association. In this case, both back-door paths consist of only chains and forks *without* any inverted forks (which would block any transmitted association). Thus, these two back-door paths are open, they can transmit a spurious association. The zero-order correlation between educational attainment and income is a mix of the true causal effect (*Educational attainment*→*Income*) of interest, plus any non-causal association transmitted by the two back-door paths. To remove the undesirable non-causal association, the two back-door paths must be blocked.

Statistical Control: Blocking Back-Door Paths

The purpose of third-variable control is to block these open back-door paths. *If* all back-door paths between the independent and dependent variables can be blocked, *then* the causal effect connecting the independent and dependent variables can be identified, *even if* the data are purely observational. Such a causal effect would be considered *identifiable*, always under the assumption that the DAG captures the true underlying causal web. This is the so-called back-door criterion described by Pearl (1993).[5] Notice that the assumption that one has correctly captured the causal web and successfully blocked all back-door paths is in most cases a *very* strong one, because it posits that no relevant variables have been omitted from

[5] Accessible introductions to the underlying concept of d-separation, which determines whether paths transmit association or not, can be found in Hayduk et al. (2003) or, in a very brief form, in Thoemmes' (2015) Appendix A.

the causal graph. Whether this is plausible or not needs to be evaluated on a case-by-case basis.

Back-door paths can be blocked by "cutting" the transmission of association at any point in the path by statistical control of a node. Take, for example, the non-causal path *Educational attainment*←*Grades*←*Intelligence*→*Income*. One could, for example, control for grades. This would effectively block this back-door path, and it would no longer be able to transmit a non-causal association. However, one could also control for intelligence. This again cuts the transmission of this specific back-door path, but at the same time, it also blocks the transmission of the second back-door path, *Educational attainment*←*Intelligence*→*Income*. In Figure 1 and Figure 2, assuming that the DAG correctly captures the underlying causal web, controlling for intelligence would be sufficient to identify the causal effect of educational attainment on income because it blocks all back-door paths.

Various practices allow to control for nodes in a DAG and thus for the blocking of back-door paths. Although these procedures might appear quite different from each other (i.e., they require different statistical procedures to be run), they serve the same purpose. In any case, if one wants to control for a certain variable, one must have measured it.

Even if the DAG correctly captures the underlying causal model, if the back-door paths that should be blocked are correctly determined, and if all the variables necessary to block all back-doors are measured, a lot can still go wrong during the actual estimation of the effect. *Qualitative* causal identification and the subsequent *quantitative* (usually parametric) estimation of the desired effects are two distinct problems (Elwert, 2013): The right variables can be controlled for, but this can be done in the wrong way, as I will discuss below.

How to Control for a Variable

Stratified analysis. In some cases, it might be possible to fully stratify the sample to control for confounders. For example, if the variable to be controlled for is a categorical variable (e.g., biological sex), the sample can be split into sex-homogenous groups, analyses can be run within these groups (i.e., conditional on sex), and the estimates from the subgroups can be combined into an overall estimate. These steps guarantee that sex effects cannot provide an alternative explanation for the findings because, for example, women are compared only with other women. This analytic approach might be appealing because it is highly transparent. However, stratification becomes unfeasible if the third-variable has many levels, if it is continuous, or if multiple third-variables variables and their interactions need to be taken into account simultaneously. Thus, in this case, other options for statistical control might need to be considered.

Including third variables in regression models. A widespread approach in the social sciences is statistical control in multiple regression models.[6] The dependent variable can be regressed on both the independent variable and the covariate to "control for" the effects of the covariate and thus to potentially block back-door paths.

In the standard case, psychologists will run models in which linear relationships are assumed without explicit justification. However, this is not guaranteed to adequately adjust for the covariate. For example, if the effects of the covariate on the dependent and independent variables both follow a quadratic trend, linear control might leave residual confounding between the independent and dependent variables. Both the covariate *and* the covariate raised to the second power would need to be controlled for to properly remove the influence of the covariate in such a scenario. This also applies to the widespread practice of

[6] However, notice that all of the following considerations also hold for other common statistical approaches such as ANOVA and ANCOVA, as most models used by psychologists are simply special cases of generalized linear models.

"controlling for age": Simply including age in a linear regression model will adequately adjust for age only *if* the age trends that are being controlled for are approximately linear; in other cases, the statistical models might need to be refined (e.g., by including higher order polynomials). Similarly, if covariates have interactive effects, these interactions must be considered in the model as well.

Matching. In many cases, there might be a need to control for not only a single third variable but multiple background characteristics. Furthermore, one might want to control for covariates in a fully non-parametric fashion, that it, without assuming specific functional forms for the effects of the covariates. Matching is one way to approach such a situation. Different matching methods exist, but propensity score matching has shown to be particularly popular in the social sciences. The usage of propensity scores for matching is controversial, and critics have remarked that other procedures might be preferable (King & Nielsen, 2016). Nonetheless, because of the popularity of propensity score matching, and because the fundamental rationale of matching approaches is independent of the specific method used, the following example is a study that used propensity score matching.

Jackson, Thoemmes, Jonkmann, Lüdtke, and Trautwein (2012) were interested in the effects of military training (in comparison to civilian community service) on personality. Young men who choose to enter the military are most likely different with respect to personality even before they enter the military and will also differ on a number of other background variables. Including all of these variables in a regression model can lead to estimation issues and can result in unwieldy models. Furthermore, such a practice does not provide an actual model of who chooses military training, which might be of interest in itself. In their study, Jackson et al. (2012) thus used propensity-score matching.

In this procedure, first, the probability of entering the military is predicted from the covariates. This results in a single number, a propensity score, which indicates how "typical"

a respondent is of somebody joining the military. There are some individuals with high propensity scores who do not join the military as well as some individuals with very low propensity scores who join the military nonetheless. Subsequently, matched groups can be created: For every individual with a certain propensity score who joined the military, one individual with the same (or a similar) propensity score who instead chose civilian community service is included in a control group. Under idealized conditions, this procedure guarantees that the two resulting groups (i.e., military vs. civilian service) are balanced with respect to all control variables that were used to generate the propensity scores. Thus, these variables can no longer be the cause of any differences between the two groups that are being compared, and a large number of potentially confounding back-door paths have been blocked.

Such matching procedures serve the same purpose as the more common approach of including control variables. Whereas propensity scores might, depending on the circumstances, have certain advantages with respect to the *estimation* of the effect, they do not change anything about the specifics of causal *identification*: If an important confounder is omitted, or if variables that should not be included are included (Sjölander, 2009), propensity scores fail to properly identify the causal effect, just like other methods of statistical adjustment. In addition, whether the model properly captures the effects of the covariates must again be considered. In this case, the researcher must make sure that the model underlying the propensity scores properly captures the relationships between background characteristics and the propensity to join the military.

Measurement Error in Confounding Variables

Measurement error can affect all methods of statistical control. In the previous example regarding *Educational attainment* and *Income*, the confounding variable *Intelligence* will not be measured perfectly (Figure 1 and 2). Thus, the statistical adjustment for intelligence will most likely not be able to completely remove its confounding influence, and

the effect of educational attainment on income might be mistakenly assumed to be stronger than it actually is, due to residual confounding. The same problem holds for propensity score matching if the scores have been based on variables that are affected by measurement error.

Westfall and Yarkoni (2016) assessed what the imperfect measurement of covariates means for the false positive rate regarding the associations of interest. It is worrisome that the false positive rate can reach very high levels, approaching almost 100%. In a worst-case scenario, applied to our example, the conclusions would almost *always* be that there is a significant effect of educational attainment on income after intelligence is controlled for, even if the association between the two variables could actually be completely attributed to the confounder intelligence. Somewhat counterintuitively, this problem becomes worse in situations in which sample sizes are large. A latent variable approach in which the measurement error is explicitly represented in an SEM can be used to address this problem and reduce the rate of false positives; however, under realistic conditions, it might require hundreds to thousands of participants to achieve an acceptable level of statistical power (see Westfall & Yarkoni, 2016, for details).

Excursion: Genetic Confounding and Control by Design

One source of potentially spurious associations that might have gone underappreciated in psychology is genetic confounding (e.g., between parents and their offspring). Assume that children who were rarely held and cuddled by their mothers are observed to grow up to be depressed adults.[7] Before the conclusion can be drawn that being raised by a cold, distant mother causes depression, it is important to consider potential back-door paths, as depicted in Figure 3. Mothers who have a certain genetic predisposition might be prone to depressiveness,

[7] This example was adapted from Turkheimer, 2000.

which could, in turn, affect the way they interact with their child. A child will be genetically similar to his or her mother and thus, the child might have inherited this predisposition, which could result in depression later in life.

The knowledge that all traits are to some extent heritable has consequences for the ability to draw causal inferences, as summarized by Turkheimer (2000): "It is no longer possible to interpret correlations among biologically related family members as prima facie evidence for sociocultural causal mechanisms" (p. 162). To figure out whether maternal displays of affection causally influence children's depressiveness, the back-door paths connecting maternal behavior to the child's depressiveness via genetic predispositions must be blocked.

The genetic back-door path could be blocked in different ways. For example, assuming that Figure 3 reflects the causal DAG, measuring and controlling for the mother's depressiveness would remove any spurious association.

However, in this case, an alternative to statistical adjustment is available: control *by design*. For example, the path between maternal and offspring genes could be blocked by analyzing only adopted children, in which case there would be no link between the genetic dispositions of mothers and offspring.[8] Another potentially powerful solution makes use of individuals who are matched on a wide range of variables: twins.

[8] This holds only under "idealized" conditions in which there is no selectivity in adoptions.

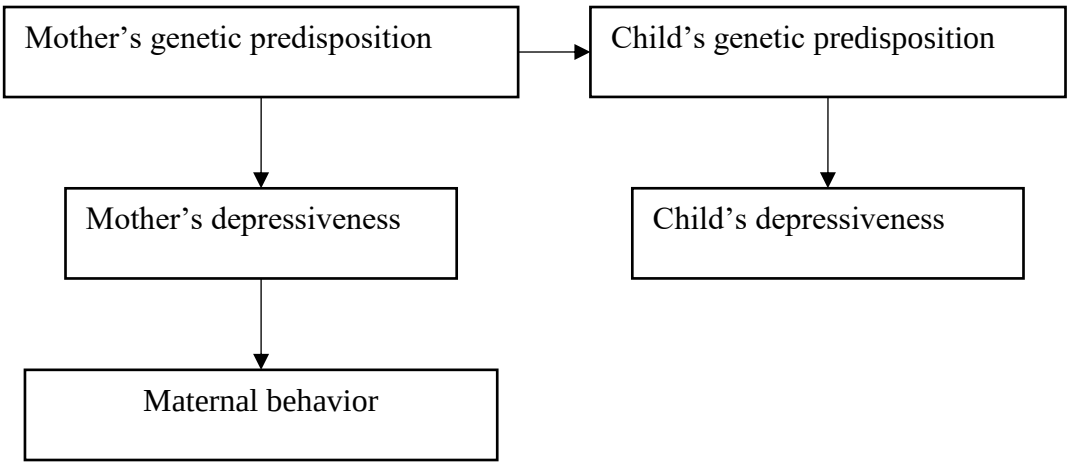

Figure 3. A mother who is genetically prone to depression might pass this genetic vulnerability on to her child, who in turn might suffer from similar issues later in life. In this graph, there is no causal effect of maternal behavior on the child's depressiveness, any observed association can be attributed to genetic confounding.

Monozygotic twins are of special interest for causal inference, even if a researcher is not interested in genetics at all. They are matched with respect to both their genetic predispositions and a wide range of shared family background characteristics. Thus, they provide an attractive way to test causal claims: If a certain association is found *within* monozygotic twin pairs, these cannot be attributed to confounding by genes or shared family background characteristics because all these covariates are controlled for *by design*.

For example, Turkheimer and Harden (2014) investigated whether religiosity has a causal effect on delinquency. They found a negative correlation between religiosity and delinquency when they simply correlated the variables across the whole sample. Although a causal effect might seem plausible—many religions try to encourage ethical behavior and are embedded in supportive social communities—confounders such as family background characteristics could provide an alternative explanation. Turkheimer and Harden thus

analyzed the association between religiosity and delinquency *within* monozygotic pairs of twins and found that the association disappeared: The more religious twin was not more (or less) likely to become delinquent than his or her twin. This finding challenges the assumption that there might be a causal effect: If religiosity actually affected delinquency, there should be an association even after family background and genes are controlled for.

Not only the birth of twins but other "lucky accidents" or specific variable situations can also result in research designs that control for a wide range of potential confounders. Under ideal conditions, such designs can render additional post hoc statistical control unnecessary. They are called natural experiments and constitute an interesting intermediate case between ordinary observational studies and randomized experiments. Such design-based approaches to causal inference have become more popular in economics because they often require substantially fewer assumptions, and they might be one reason why empirical microeconomics have experienced a "credibility revolution" (Angrist & Pischke, 2010). Dunning (2012) offered an excellent introduction to natural experiments ranging from "standard" natural experiments to regression-discontinuity designs to instrumental variables, including an extensive discussion of potential trade-offs in comparison with other research designs.

Learning to Let Go: When Statistical Control Hurts

In certain fields, it has become common practice to include as many covariates as possible—to the point where authors claim that they have additional confidence in their findings because their study "uses more control variables than previous studies" (e.g., p. 85 in Tiefenbach & Kohlbacher, 2015, and implied in many other articles). Whereas, in many cases, a failure to control for important confounders will indeed undermine the conclusions, the idea that simply adding more covariates will improve the estimate of a causal effect is not true. There are two types of variables that researchers should not control for without taking

into account potential negative side effects: colliders and mediators. Both have in common that, instead of causally affecting the independent variable (as confounders do), they are causally *affected by* the independent variable of interest. As a solid rule of thumb, researchers should not control for such posttreatment variables (Rosenbaum, 1984; Rubin, 1974). The following sections explain *why*.

Conditioning on a Collider Can Introduce Spurious Associations

A collider variable for a certain pair of variables is any variable that is causally influenced by both of them. Controlling for such a variable (or any of its descendants), that is, *conditioning on a collider*, can introduce a spurious (i.e., noncausal) association between its causes. Returning to DAG terminology, a collider is the variable in the middle of an inverted fork, variable *B* in $A \rightarrow B \leftarrow C$. The collider variable normally blocks the path, but when one controls for it, a spurious association between A and C can arise. This might open up a non-causal path between the independent variable and the dependent variable of interest. In recent years, this potential source of bias has been pointed out in a variety of research fields such as epidemiology (Greenland, 2003), personality psychology (Lee, 2012), and genetic research (Munafò, 2017).

To visualize why conditioning on a collider can have this effect, imagine that we are interested in whether the methodological rigor of a scientific study impacts its innovativeness, as depicted in Figure 4. Such an association could go either way: Methodological rigor might "tie" the hands of a researcher, leading to less original research designs; but methodological rigor might also require researchers to come up with creative solutions for addressing methodological problems. For this thought experiment, assume that there is *no* causal effect of methodological rigor on innovativeness.

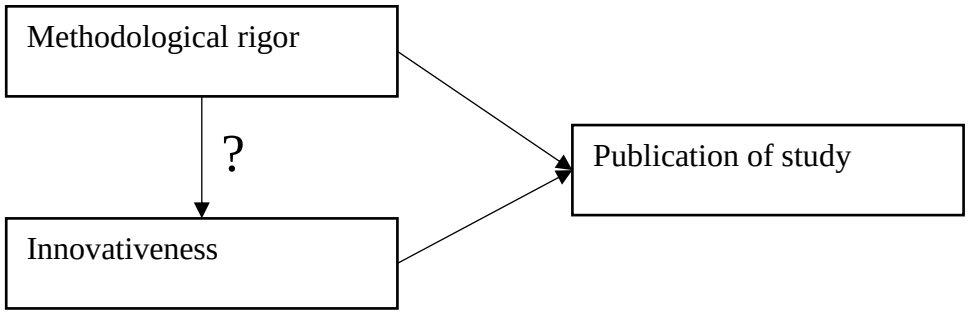

Figure 4. Does methodological rigor influence the innovativeness of research? Both methodological rigor and innovativeness affect whether a study will be published or not. Thus, controlling for publication status (e.g., by looking only at published studies) will bias the estimate of the relationship between methodological rigor and innovativeness.

To investigate the association between methodological rigor and innovativeness, we consider all psychological studies that have been published. Say we notice that among these studies, there is a sizeable negative association: Studies higher in methodological rigor are less innovative and vice versa. Next, we notice that publication bias might be an issue here, so we decide to additionally assess all psychological studies that have not been published.[9] In a follow-up study, we investigate the association between methodological rigor and innovativeness among unpublished studies and again find a sizeable negative association.

By assessing published and unpublished studies separately, we have stratified our analyses by publication status, in other words, we have conditioned on publication. However, both methodological rigor and innovativeness are likely to causally affect publication status. In the simplest case, both have a positive, non-interactive effect: With increasing rigor, the

[9] Notice that this is a thought experiment and is thus unconstrained by any considerations of practicality or feasibility.

likelihood of publication increases; with increasing innovativeness, the likelihood of publication increases. Thus, publication status is a collider. Controlling for this collider variable will bias the estimate of the effect of methodological rigor on innovativeness, and in this thought experiment, it introduces a negative association where no causal effect exists.

Collider bias seems less intuitive than spurious associations caused by confounders. However, consider what the body of published studies will look like: There will be studies that are both rigorous and innovative, but there will also be studies that met the publication threshold thanks to high methodological rigor (despite being less innovative) and studies that met the publication threshold thanks to high innovativeness (despite being less rigorous). Studies that are low in both rigor and innovativeness will not end up in this analysis: They simply never got published. Thus, looking at all published studies will give the impression of a certain trade-off: Studies tend to be either rigorous *or* innovative, and the resulting association, *conditional on publication*, is negative. Similarly, among the unpublished studies, we will observe studies that are low on both dimensions as well as studies that are low on only one of the dimensions, but there will not be many studies that are high on both dimensions because these ended up getting published more frequently. Again, this might result in a negative association between methodological rigor and innovativeness, *conditional on nonpublication*.

However, in this thought experiment, there is no association between methodological rigor and innovativeness if all studies—published and unpublished—are considered simultaneously. The spurious negative correlation emerges only if the joint outcome of the two variables of interest is controlled for. This observation generalizes to similar situations in which selections are based on multiple dimensions, exaggerating the trade-offs between desirable features. For example, people might notice that there is a negative correlation between attractiveness and intelligence among their former romantic partners. However,

dating somebody is a collider of multiple causes of attraction, and thus, it would be invalid to assume that all potential romantic partners are *either* attractive *or* intelligent. The issue is simply that a person is less likely to date somebody who is low on both dimensions, whereas potential partners who are high on both dimensions are simply rarer. The spurious correlation results from the selection procedure.

Returning to the thought experiment displayed in Figure 4, the solution to the collider problem seems straightforward: If we realize that publication is a collider, we can decide to run the analysis *without* controlling for this variable. By extension, we should also not control for descendants of the collider variable. For example, the publication of a study might have a causal effect on whether or not popular media report about the findings. If we only look at studies which have been covered by popular media—i.e., if we condition on the descendant of the collider—we might observe the same spurious negative association between methodological rigor and innovativeness.

Avoiding collider bias requires two steps: First, one must be *aware* of the collider variable; and second, one must be able to run analyses that are not conditional on the collider (i.e., we must have observed studies that have not been published). Outside of thought experiments, one might often be unaware of collider variables or collect data in such a way that collider bias is "built in."

Variations on the Same Theme: Endogenous Selection Bias

Endogenous selection bias is collider bias that results from the sampling procedure (i.e., spurious associations that are introduced when data are collected). Elwert and Winship (2014) provided a succinct summary of the many ways in which endogenous selection bias can arise. In the following, I briefly illustrate scenarios with examples that might be relevant to psychologists.

Nonresponse bias. Nonresponse bias occurs if, for example, a researcher analyzes only completed questionnaires, and the variables of interest are associated with questionnaire completion. Assume that we are interested in the association between grit and intelligence, and say our assessment ends up being very burdensome. Both grit and intelligence make it easier for respondents to push through and complete the assessment. *Questionnaire completion* is thus a collider between grit and intelligence. For example, although there might be no association between grit and intelligence in the population, we might find a spurious negative association if we analyze only completed questionnaires. Completers low on intelligence may have "compensated" with their high levels of grit, completers low on grit may have "compensated" with their high levels of intelligence, and those who were low on both variables were less likely to finish the assessment and are thus underrepresented in the analyzed sample.

Attrition bias. Assume that we are conducting a longitudinal study and are interested in the effects of health problems on work satisfaction. We make sure that we assess work satisfaction at a later point in time, which supposedly gives us greater confidence in the direction of the causal flow. However, over time, respondents will inevitably drop out of the study (e.g., they move away, cannot be found, are no longer willing to participate), and this attrition will likely be selective. Some respondents might leave the study because of health problems; others might drop out because their workplace is too stressful. Now, assume that we analyze only the respondents who remain in our sample.

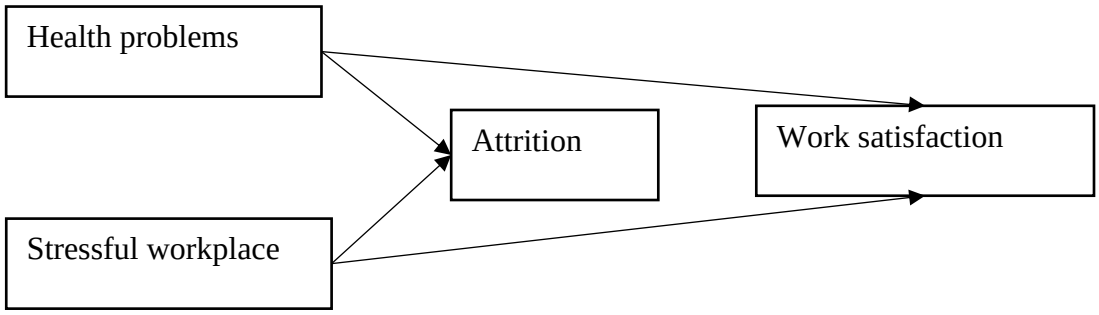

Figure 5. Attrition—leaving the sample—is causally affected by both health problems and having a stressful workplace. If analyses are computed on only the respondents who remain in the sample, we condition on a collider and potentially induce a spurious association between health problems and a stressful workplace. This opens a back-door path that introduces a non-causal association between health problems and work satisfaction.

If only the respondents remaining in the panel are analyzed, spurious associations between all causes of attrition are introduced, and this noncausal association then might open up a back-door path between the variables of interest. In this example, attrition will introduce a spurious association between health problems and a stressful workplace: Both health problems and a stressful workplace will likely lead to attrition; respondents with health problems might remain in the study if they have low-stress jobs; respondents with a stressful workplace might remain in the study if they are in particularly robust health. Assuming that there is a negative effect of health problems on work satisfaction, the strength of this association would be underestimated if only the remaining sample is analyzed because the respondents high in health problems are more likely to work in low-stress workplaces that are generally more likely to leave individuals satisfied.

Related Issues: Missingness and Representativity

Thoughts about endogenous selection bias quite naturally lead to considerations of problems that normally are not framed as issues of causal inference. The first problem is missing data: Nonresponse and attrition bias lead to missing data, and these missing data must be handled properly if the goal is to draw valid causal conclusions on the basis of observational data. Schafer and Graham (2002) provide an introduction to the management of missing data for psychologists. Thoemmes and Mohan (2015) use DAGs to provide visual representations of missing data problems on the basis of formalizations developed by Mohan, Pearl, and Tian (2013).

The second problem is the representativeness of samples: Does the sample accurately reflect the underlying population about which the researchers want to make statements? For example, if a researcher investigates only college students, endogenous selection bias is introduced between all variables that causally affect whether or not somebody becomes a college student, including socioeconomic status, cognitive abilities, attitudes, parents' characteristics, and so on.

Controlling for Mediators: Removing the Association of Interest

Overcontrol bias is another phenomenon in which statistical control can hurt instead of help: If mediating variables are controlled for, the very processes of interest are "controlled away." Returning to the very first example from Figure 1, let's additionally assume that educational attainment has an influence on adult intelligence (see Figure 6). Although this might still seem like a grossly oversimplified model of reality, it results in considerably more complex considerations. In addition, let's incorporate a variable labeled *U*. Although it is possible to come up with plausible ideas about what *U* stands for (i.e., some variable that affects both adult intelligence and income, potentially something unobserved), let's simply leave it unspecified here, as conceptual considerations derived from DAGs do not depend on the concrete variables but only on the underlying abstract causal web.

Again, childhood intelligence is a confounder that needs to be controlled for. However, a new node has been added: adult intelligence. Should one—or should one not—control for adult intelligence?

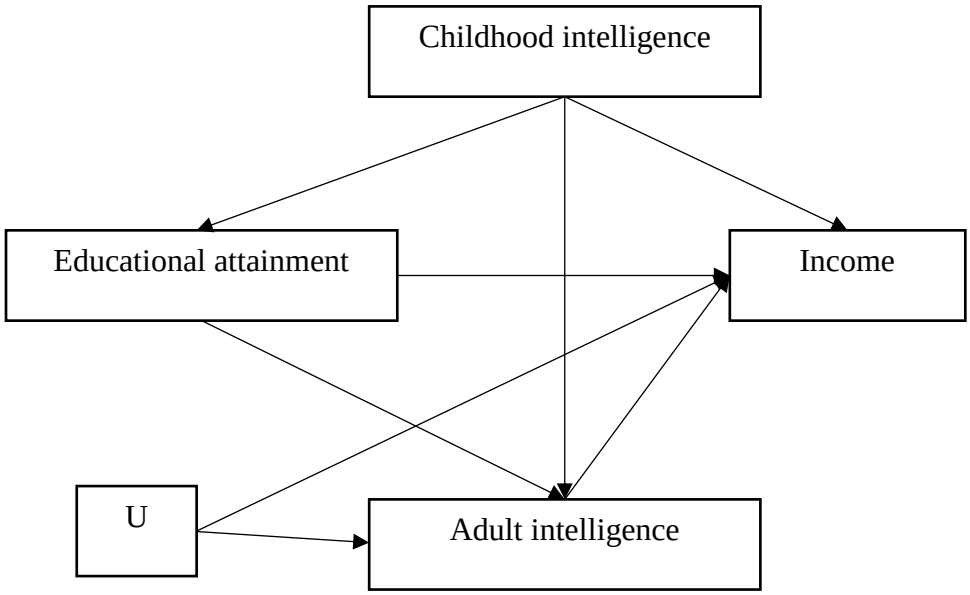

Figure 6. In this scenario, childhood intelligence confounds the association between educational attainment and income, but at the same time, adult intelligence is a mediator of the effect of educational attainment on income. In addition, adult intelligence and income are confounded by the unknown variable U.

Adult intelligence is a mediator of the effects of educational attainment on income; it is a node on a *causal* pathway from educational attainment to income. If one were able to randomize whether or not participants had a college degree, this manipulation would also have an indirect effect on income mediated by intelligence. Controlling for adult intelligence would block this genuinely causal pathway, and one would likely underestimate the positive payoff of a college degree. If one is interested in the magnitude of a causal effect, one should

not control for mediating variables (i.e., the mechanisms driving the effect). By extension, the same applies for any descendant of a mediating variable. Consider, for example, adult chess performance as an outcome of adult intelligence: Chess performance is a noisy proxy for intelligence, and controlling for it will remove some of the variation in adult intelligence that is caused by the independent variable of interest (i.e., educational attainment). Thus, this descendant of the mediating mechanism should also not be controlled for.

Mediation analysis can induce endogenous selection bias. In some cases, researchers might actually be interested in the effect of an independent variable on a dependent variable after accounting for the effect of a mediating variable, i.e. the effect net of a certain mediated path. This is a common goal of mediation analysis. Both old and newer common approaches to estimating the remaining ("direct") effect after accounting for a mediator (e.g., Baron & Kenny, 1986; Hayes, 2009) rely on the statistical control of the mediating variable, but such approaches can introduce endogenous selection bias (Elwert & Winship, 2014).

In Figure 6 adult intelligence is a collider with respect to educational attainment and the unspecified other variable *U*. As long as *Adult intelligence* is not controlled for, *U* is unproblematic: It affects the outcome variable (i.e., *Income*), but it does *not* causally affect the independent variable (i.e., *Educational attainment*); thus, *U* is not a confounder of the effect of interest and can simply be ignored. However, if *Adult intelligence* is controlled for, a non-causal association between its two causes, *U* and *Educational attainment*, is introduced, which could be represented as *Educational attainment* ←→*U*. Now, a back-door path has been opened: *Educational attainment* ←→*U*→*Income*, which potentially introduces a non-causal association. If the goal is to correctly estimate the "direct" effect of a college degree on income, all back-door paths that were opened by conditioning on the mediating variable must be blocked.

Maybe somewhat surprisingly, this problem of mediation analysis also applies to experimental studies *unless* the mediating variable itself was randomly assigned. Randomized assignment of the independent variable rules out back-door paths between the independent variable and dependent variable, but back-door paths between the mediator and the outcome remain unaffected. In such a study, estimating the direct effect by controlling for the mediating variable will lead to biased estimates. Bullock, Green, and Ha (2010) offer recommendations for experimental research programs but also highlight that in any case, uncovering a mediating mechanism might be much harder than most social scientists are aware of.

Conclusion: Making Causal Inferences on the Basis of Correlational Data is Very Hard

To summarize, the practice of making causal inferences on the basis of observational data crucially depends on the awareness of potential confounders and meaningful statistical control (or noncontrol), that is, taking into account estimation issues such as nonlinear confounding and measurement error. Back-door paths must be considered *before* data are collected to make sure that all relevant variables are measured. In addition, variables that should not be controlled for (i.e., colliders and mediators) need to be considered. This might require careful planning *before* data collection begins because researchers must consider how sample recruitment might result in endogenous selection bias, which threatens the validity of any conclusions drawn.

In reality, researchers might often end up with data that do not contain reliable measures of central confounders—because a back-door path was not considered before the data were analyzed (or before comments were made by peer reviewers), because somebody else collected the data (e.g., if data that came from nationally representative panel or survey studies were analyzed), or because the confounder might be some unobservable factor that could not be measured with the available methods. In such a situation, thorough consideration

of the causal web underlying the variables can result in the conclusion that the data do not warrant causal claims.

In addition, in a messy psychological reality, causal graphs quickly become substantially more complex than the previous illustrations. For example, in certain variable constellations, controlling for a variable might *reduce* one type of bias (because the variable is a confounder) but at the same time *increase* another type of bias (because the variable is a collider on a different path that transmits a spurious association when the collider is controlled for). Such a constellation, called butterfly bias, can be easily visualized in a DAG. Quite pragmatically, one can gauge which of the two biases is more problematic and then settle for the lesser evil (Ding & Miratrix, 2015).

Apart from complex variable situations, sometimes it might be genuinely unclear whether a given variable is a confounder, collider, or a mediator. Stronger theories that posit clear directional links between variables might solve such problems, but in some cases, theory might simply tell us that different data are needed. For example, if educational attainment, intelligence, and income are measured at only one point in time, it becomes unclear whether intelligence should be controlled for or not—part of the variable certainly captures confounding influences, but at the same time, it will also capture parts of the "treatment" of education. Reciprocal effects seem plausible for many psychological variables, and to disentangle causes and effects in such a situation, there is a need for data with a higher temporal resolution. Again, thoughtful consideration of the underlying causal web might lead to the conclusion that the data at hand are not sufficient and that different sampling designs are needed, such as intensive time series of repeated measures (Borsboom et al., 2012).

Causal inferences based on observational data require researchers to make very strong assumptions. Research who attempt to answer a causal research question with observational data should not only be aware that such an endeavor is challenging, but also understand the

assumptions implied by their models and communicate them transparently. In addition, instead of reporting a single model and championing it as "the truth," researchers should consider multiple potentially plausible sets of assumptions and see how assuming any of these scenarios would affect their (causal) conclusions. This practice of robustness-checking is already common in parts of economics and could also improve inference in psychological research (see, e.g., Duncan et al., 2014, for recommendations for developmental research). As a positive side effect, performing and reporting multiple analyses, i.e. conducting a "multiverse analysis" (Steegen, Tuerlinckx, Gelman, & Vanpaemel, 2016), can greatly improve transparency and thus boost productive and open debates.

One could argue that—given the complex nature of human behavior—causal modeling of observational data might not be worth the hassle, as it requires a great deal of effort, with respect to both theoretical reasoning and data collection, and nonetheless results in claims that can be often easily challenged. However, this should not be a reason to give up the endeavor altogether.

Observational Data are an Indispensable Part of Empirical Science

Psychologists are interested in a *broad* range of questions about human behavior and cognition, and whereas (properly implemented) randomized experiments leave researchers with great confidence in internal validity, "their meaning and significance for the target phenomenon are often questionable" (Rozin, 2001, p. 12). That is, whereas randomized experiments allow researchers to be confident about a cause-effect relationship with only very few additional assumptions, a lot more assumptions might be needed to convincingly argue that this cause-effect relationship is *actually* the one of interest. Which method is suited for drawing causal inference for a specific research question—randomized experiment, natural experiment, or observational study—must be decided on a case-by-case basis (see also arguments that *there is no gold standard* in Cartwright, 2007).

It is instructive to consider cases in which most people readily accept causal claims in the absence of randomized experiments. Nowadays, few doubt the effects of tobacco smoking on lung cancer. But in the 1950s, tobacco lobbyists embraced the idea that a genetic disposition caused both a disposition for smoking and lung cancer (Mukherjee, 2010, pp. 253). In other words, they claimed that there was an unblocked back-door path. This idea was not dispelled by randomized, controlled experiments in humans, but by highly consistent evidence from observational studies using various controls and different sampling designs, experimental evidence *from rodent studies*, and a plausible *mechanism*. Inhaled carcinogens correlate with visible malignant changes in the lung which in turn correlate with lung cancer (see Mukherjee, 2010, for a summary of the history of cancer research).

A plausible mechanism is also what greatly increases our confidence in the causal effect of human activity on the climate: Human activity, such as industrial processes, increases the atmospheric concentrations of greenhouse gases. Atmospheric greenhouse gasses in turn warm the Earth's surface through an uncontroversial mechanism: the greenhouse effect (see Silver, 2012, pp. 374, for this line of argument). And a plausible mechanism is also the reason why we do not need randomized controlled trials to conclude that parachute use during free fall reduces mortality (but cf. Smith & Pell, 2003).

Thus, causal inference based on observational data is not a lost cause *per se*—indeed, in combination with additional knowledge from the domain, highly convincing causal arguments can be made. Further research into psychological mechanisms and processes, which will frequently involve experimental studies including well-designed "surrogate interventions," can strengthen the potential of observational data. Likewise, observational data can increase the external validity of constrained experimental settings and also hint towards new phenomena that potentially warrant further research. Different research designs are neither mutually interchangeable nor rivals, but can contribute unique information to help

answer common research questions. The most convincing causal conclusions will always be supported by *multiple* designs (Angrist & Pischke, 2010, p. 25): "In the empirical universe, evidence accumulates across settings and study designs, ultimately producing some kind of consensus."

Glossary

Ancestor. A variable causally affecting a given variable; influences the variable either directly (*Ancestor→X*) or indirectly (*Ancestor→Mediator(s)→X*). Direct ancestors are also called parents.

Arrow. A directed edge; indicates a direct causal effect between two variables.

Back-door path. A non-causal path that connects the independent variable of interest with the dependent variable of interest.

Blocked/unblocked path. A path that contains (1) a collider that has not been conditioned on, or (2) a non-collider (confounder or mediator) that has been conditioned on is considered blocked and does not transmit an association between variables. A path that is not blocked is unblocked/open and can transmit an association.

Causal/non-causal path. A causal path consists only of chains and can transmit a causal association if unblocked. Non-causal paths contain at least one forks or inverted forks and can transmit a non-causal association if unblocked.

Chain. An elementary causal structure of the form *A→B→C* (or, in short, A→C) which transmits a causal effect of A on C. The variable in the middle, B, mediates the effect of A on C.

Collider. A variable in the middle of an inverted fork (A→Collider←C). A collider blocks a path unless it (or one of its descendants) is conditioned on.

Conditioning on a variable. In the most abstract sense, conditioning introduces information about a variable into an analysis (Elwert & Winship, 2014). This may happen through various means of statistical control or through sample selection.

Confounder. A variable in the middle of a fork (A←*Confounder*→C).

Directed Acyclic Graph (DAG). A graph is an abstract structure that connects nodes with edges (lines). In a DAG, every edge is an arrow (hence *directed*). Directed cycles are not allowed (hence *acyclic*). Thus, in a DAG, a variable cannot causally affect itself.

Descendant. A variable causally affected by a given variable; influenced either directly (X→*Descendant*) or indirectly (X→Mediator(s)→*Descendant*). Direct descendants are also called children.

Fork. An elementary causal structure of the form A←B→C. The variable in the middle, B, is called a confounder and can transmit a non-causal association between A and C. Conditioning on B blocks this non-causal path.

Inverted Fork. An elementary causal structure of the form A→B←C. The variable in the middle, B, is called a collider and blocks the path. Conditioning on the collider opens the path, which may then transmit a non-causal association.

Mediator. A variable in the middle of a chain, *A*→*Mediator*→*C*.

Node. A variable in a DAG.

Path. A sequence of edges which connect a sequence of nodes. In a DAG for observational data, a path is a sequence of *arrows* connecting *variables*. The arrows of a path need not point into the same direction.

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Conflicts of Interest

The author declares that she has no conflicts of interest with respect to the authorship or the publication of this article.

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