


## Definition and Identification of Causal Ratio Effects


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# Abstract

In non-linear regression models, the effect of a treatment or intervention is often expressed as a ratio (e.g., risk ratio, odds ratio). There is discussion about when ratio-based effect measures can be interpreted in a causal way. For example, it is well-known that ratio-based effect measures suffer from non-collapsibility, that is, the average over individual ratio effects is not equivalent to the ratio of group means in a randomized experiment. Even more, different ratio-based effect measures (e.g., simple ratio, odds ratio) can point into different directions regarding the effectiveness of the treatment making it difficult to decide which one is the causal effect of interest. While causality theories do in principle allow for ratio-based effect measures, the literature lacks a comprehensive derivation and definition of ratio-based effect measures and their possible identification from a causal perspective (including, but not restricted to randomized experiments). In this paper, we show how both simple ratios and odds ratios can be defined based on the stochastic theory of causal effects. Then, we examine if and how expectations of these effect measures can be identified under four causality conditions. Especially, we will show that ratio-based effect measures are collapsible under certain conditions. Finally, we discuss an alternative computation of ratio-based effect measures as ratios of causally unbiased expectations instead of expectations of individual ratios, which is identifiable under all causality conditions and consistent with difference-based effect measures.

*Keywords:* causality

### Definition and Identification of Causal Ratio Effects

The effect of a treatment or intervention on an outcome-of-interest is often expressed as a ratio (e.g., risk ratio, odds ratio), especially if the outcome variable is a count variable or dichotomous variable. In these cases, non-linear regression models (e.g., Poisson regression, logistic regression) are applied for estimating treatment effects accounting for covariates. The (transformed) parameters of these models can often be interpreted on a multiplicative ratio scale. For example, the exponentiated regression parameters of a logistic regression model can be interpreted as odds ratios.

There is an ongoing debate – predominantly in the fields of biostatistics and epidemiology – on the use of ratios as measure of treatment effectiveness. This debate considers various aspects of ratio-based effect measures, such as the comparability across samples and covariates (Mood, 2010), collapsibility (Greenland, 1996, 2021; Greenland et al., 1999), and (causal) interpretation (Niu, 2020). Collapsibility means that an effect measure based on group means resembles an average over corresponding conditional or individual measures, when the effect measure varies across individuals. This is the case for difference-based effect measures, but, for instance, not necessarily for risk ratios and odds ratios in a randomized controlled trial – under this condition, ratio-based effect measures are called *non-collapsible*. While the debate on (non-)collapsibility is not new in biostatistics (see, e.g., a contribution four decades ago by Miettinen & Cook, 1981), the phenomenon seems less known in psychology and the social sciences (with prominent exceptions, e.g., Hanmer and Ozan Kalkan, 2013; Mood, 2010).

We believe that the definition of (average) ratio-based effects measures as well as their statistical and causal properties (e.g., collapsibility) deserve greater attention in the literature. We identified three open research questions in the current literature on ratio-based effect measures and (non-)collapsibility on which we want to focus: First, there is no discussion of different types of definitions of (average) ratio effects from the perspective of causality theories and comprehensive derivations of their properties are

lacking. Many papers on ratio-based effect measures and their collapsibility only consider the interpretation of regression coefficients (e.g., Gail et al., 1984; Mood, 2010). Others state that “the ratio or any such comparison could be used to define [...] causal effects” (p. 323, Rubin, 2005; further examples are Greenland et al., 1999; Imbens and Rubin, 2015), but we are not aware of any contribution in the causal inference literature that actually examined the properties and implications of such a definition.

Second, non-collapsibility is usually examined for randomized experiments (Gail et al., 1984; Greenland et al., 1999) and, thus, is described as a phenomenon that occurs even in absence of confounding. For observational studies, Daniel et al. (2021) state that one should be careful in interpretation of ratio-based effect measures as it would be difficult to disentangle non-collapsibility from confounding. Newman (2001) provides conditions for *strict collapsibility* of both risk and odds ratios, but these conditions do not necessarily allow for a causal interpretation. Thus, we are not aware of any contribution considering whether ratio-based effect measures are (non-)collapsible under causality conditions that allow causal inferences in observational studies.

Third, ratios of group averages as alternatives allowing treatment evaluation on a multiplicative scale, which are also collapsible under regular causality conditions and also provide consistent interpretation comparable to difference-based effect definitions, are seldom discussed. It is usually recommended to avoid ratio-based effect measures, and stick to difference-based effects (e.g., Hanmer & Ozan Kalkan, 2013), but we believe that being able to express treatment effects in terms of a multiplicative scale can be beneficial and informative in its own right.

In this paper, we aim at addressing all three aforementioned research questions by (a) providing definitions of individual and average ratio-based effect measures based on the stochastic theory of causal effects, (b) examine the identification of average ratio effects under four causality conditions commonly used in both experimental and observational studies, and (c) provide an alternative way to compute *average* ratio effects, which also

have a clear causal interpretation and are identifiable under the four causality conditions.

The rest of the paper is structured as follows: First, we lay out the basic concepts and definitions of the stochastic theory of causal effects. Second, we provide definitions of both ratio- and difference-based effect measures on an individual level and give an intuition and comparison of their substantive interpretation. Third, we consider how average causal effects can be computed as aggregates of the effect measures on the individual level and discuss an example, where the difference-based effect, the simple ratio-based effect, and the odds ratio-based effect result in three different indications for the treatment. Fourth, we examine the identification of the average ratio effects. That is, we examine whether the effects can be computed under four different causality conditions that are commonly applied in experimental and observational studies. Fifth, we discuss an alternative way to compute average ratio effects as ratios of averages rather than average of ratios, which also has a clear causal interpretation, but is also collapsible under all four causality conditions. Finally, implications and limitations will be discussed.

### Definitions and Terminology of Causal Effects

The concept of causal inference, definitions and terminology of causal effects, as well as methods of estimating such effects have a long tradition. In the biostatistics and epidemiology literature on ratio-based effect measures and collapsibility, one can often find the graphical modeling approach to causal inference by Pearl (2009). Another well-known tradition approaches causal inference with the potential outcome framework, sometimes called Rubin’s causal model (Rubin, 2005). In this paper, we will make use of the stochastic theory of causal effects (Mayer, 2019; Steyer et al., 2014). This theory is grounded in probability theory which has advantages for the definition and identification of ratio-based effect measures. For example, if we are considering potential outcomes of a binary outcome (i.e., 0 or 1), definition of a simple ratio is challenging as division by 0 is not properly defined. In the stochastic theory of causal effects, these quantities are replaced by conditional probabilities of the outcome (i.e., *expected outcomes*), which range

between 0 and 1 and facilitate definition of ratio-based effect measures.

In the following, we introduce some key concepts of the stochastic theory of causal effects. Our notation and the conceptualization is oriented on Mayer (2019) and Mayer et al. (2014). However, the probabilistic foundation of the stochastic theory of causal effects is presented in a less formal notation. For the formal notation and corresponding definitions and theorems, we refer to Steyer and Nagel (2017) and Steyer et al. (2014). We are not looking at variables mediating between treatment and the outcome variable and, thus, do not differentiate between direct and indirect effects of the treatment effects, but only consider total effects.

### Random Variables and Their Temporal Ordering

Let us consider an outcome variable  $Y$  with real values  $y$ , a dichotomous treatment variable  $X$  with  $P(X = x) > 0, \forall x = 0, 1$  (where we will refer to  $X = 1$  as treatment group and  $X = 0$  as *control group* or reference group), and a (multi-dimensional) covariate  $Z$  with real values  $z$ . Then, we consider the following random experiment (sometimes called single-unit trial):

1. A person  $u$  is sampled out of a set of persons  $\Omega_U$ .
2. The value  $z$  of covariate  $Z$  of that person is observed.
3. The person  $u$  is assigned or assigns itself to level  $x$  of treatment  $X$  (i.e., either randomized assignment or self-selection into treatment).
4. The person's value  $y$  of the outcome variable  $Y$  is observed.

This is a random experiment describing the temporal order of occurrence of the random variables  $U, Z, X, Y$ . In more formal presentations, the four steps of the random experiment are considered as landmarks of an underlying stochastic process (Mayer, 2019; Steyer et al., 2014). That is, the experiment starts at a time point  $t_0$  and the corresponding person variable  $U_{t_0}$  reflects a person and its history at beginning of the

experiment. But persons are allowed to evolve throughout the experiment as well. For instance, experiences made between assessment of the covariates and the actual start of the treatment at time point  $t_X$  may also influence the effect of the treatment. The actual start of the treatment at  $t_X$  is crucial for defining causal effects and the person right before this time point is denoted as  $U_{t_X}$  (Mayer, 2019). It is important to adhere to the temporal order to ensure a causal interpretation of the following quantities. For example, variables observed after time point  $t_X$  are considered as mediators and not covariates in the stochastic theory of causal effects.

### True Outcome Variables

Based on the random variables and the random experiment defined above, the *true outcome variable*  $\tau_x$  of treatment condition  $X = x$  can be defined as

$$\tau_x := E(Y|X = x, U_{t_X})$$

where  $U_{t_X}$  is the person variable just before the treatment takes place and includes all previous experiences up to this point. Thus, the person variable  $U_{t_X}$  comprises all covariates influencing either the choice of the treatment variable or the outcome variable. Consequently, the true outcome variable  $\tau_x$  reflects the expected outcome for a person  $u$ , if given to treatment condition  $x$  and controlling for potential confounders that are functions of  $U_{t_X}$ .

### Definition of Individual Causal Effects

Most theories of causal effects start with defining causal effects on an individual level and typically as a difference between two causal quantities (e.g., potential outcomes, expected outcomes) (Imbens & Rubin, 2015; Mayer, 2019; Pearl, 2009; Rubin, 2005; Steyer et al., 2014). In addition, it has often been stated that “the ratio or any such comparison could be used to define [...] causal effects” (p. 323, Rubin, 2005; further examples are Greenland et al., 1999; Imbens and Rubin, 2015), but we are not aware of any contribution

in the causal inference literature that actually examined the properties and implications of such a definition. Thus, we will provide a definition of individual causal effects as simple ratios (sometimes also called risk ratios or rate ratios) or odds ratios and show how they relate to the difference-based treatment effect. In addition, we will discuss benefits of ratio-based effect measures compared to a difference-based effect measure and provide an illustration how their substantive interpretation differs from each other.

### Definition of Difference Effect

A common way to express treatment effects, is by using a difference between the true outcome under treatment  $X = 1$  and under control  $X = 0$ :

$$\delta_{10} = \tau_1 - \tau_0 \tag{1}$$

which is called the individual causal effect variable. It can be directly interpreted on the scale of the outcome variable and reflects additive changes due to the treatment. Let us consider, for example, the effect of an alcohol abuse intervention on Joe. If the outcome variable is the amount of alcoholic drinks consumed on a regular Friday evening, we could expect Joe to consume  $\tau_0(U_{t_X} = \text{Joe}) = 8$  drinks without the intervention and  $\tau_1(U_{t_X} = \text{Joe}) = 4$  with the intervention. The individual causal effect would be  $\delta_{10}(U_{t_X} = \text{Joe}) = -4$  drinks for Joe, that is, four drinks less if he was treated. Having an effect measure on the scale of the outcome and an additive interpretation might be two reasons, why treatment effects are often defined as difference effects.

### Definition of Simple Ratio and Odds Ratio Effect

The first ratio effect that we are going to consider is simply a ratio of the two true outcomes under treatment  $\tau_1$  and under control  $\tau_0$ . Hence, it will be called *simple ratio* effect and it is defined by:

$$\rho_{10} := \frac{\tau_1}{\tau_0}$$

It is required that the expected outcome must not be zero in the control group (i.e.,  $\tau_0(U_{t_x} = u) \neq 0, \forall u$ ). Simple ratios are often used if non-negative outcome variables (e.g., counts) are examined. That is, the outcome variable itself can take the value of zero, but the conditional expectation must be  $\tau_0(U_{t_x} = u) > 0, \forall u$  in these cases. As a consequence of the limitation to non-negative outcomes, the simple ratio itself is bounded between zero and infinity, meaning that values between 0 and 1 reflect higher values in the control group and values above 1 reflect higher values in the treatment group. Returning to our alcohol abuse intervention example, the simple ratio effect for Joe would be  $\rho_{10} = 0.5$ , meaning that he is expected to consume half the drinks under control than he would be expected to consume under treatment. We see that the simple ratio effect is no longer directly interpretable on the scale of the outcome and it provides relation (rather than additive) information on the treatment effectiveness. We will discuss and compare these differences between effect measures in more detail below.

For defining an odds ratio with the true outcomes, we need both  $\tau_0(U_{t_x} = u) \neq 0$  and  $\tau_1(U_{t_x} = u) \neq 1$  to hold for all  $u$ . That is, the true outcome must not be zero in the control group and not be one in the treatment group. These conditions are typically met if the true outcomes are considered as probabilities (i.e.,  $\tau_x(U_{t_x} = u) \in ]0, 1[, \forall u$ ) of a dichotomous or categorical outcome variable. Note that this would – strictly speaking – not be possible in Rubin’s potential outcome framework, as the potential outcomes reflect values of the outcome variable and not their probabilities. However, the odds ratio based on true outcome variables is defined as:

$$o_{10} := \frac{\frac{\tau_1}{1 - \tau_1}}{\frac{\tau_0}{1 - \tau_0}}$$

The denominator and the numerator consist of an odd each, that is, the ratio of success to failure for both the treatment and the control group. Similar as the simple ratio, the odds ratio is bounded between 0 and infinity.

**Table 1**

*Joe, Ann, and Sue With Randomized Assignment: Homogeneous Difference Effects, but Heterogeneous Simple Ratios and Odds Ratios*

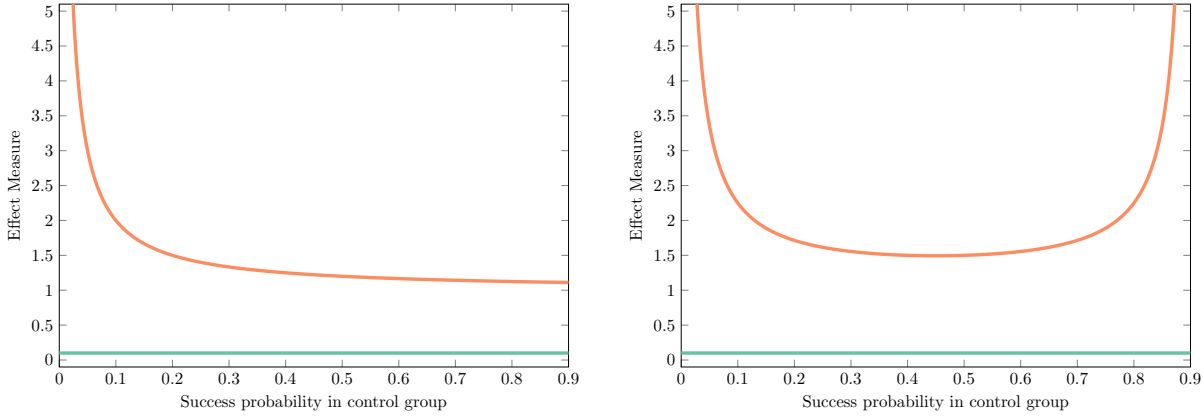
$u$	$E(Y X=0, U=u)$	$E(Y X=1, U=u)$	$\delta_{10}$	$\rho_{10}$	$o_{10}$	$P(U=u)$	$P(X=x U=u)$
Joe	0.85	0.95	0.100	1.118	3.353	.333	.5
Ann	0.05	0.15	0.100	3.000	3.353	.333	.5
Sue	0.40	0.50	0.100	1.250	1.500	.333	.5

*Note.* Conditional expectations for three different values of  $u$ , with corresponding individual effects: difference-based effect  $\delta_{10}$ , simple ratio-based effect  $\rho_{10}$ , and odds ratio-based effect  $o_{10}$

### Comparison of Difference- and Ratio-Based Causal Effect Measures

So far we have seen, that ratio-based effect measures are defined on a more limited domain of the true outcomes  $\tau_x$  as their difference-based counterpart from Equation (1) and that they provide an asymmetric effect scale (i.e., bounded between 0 and infinity). Thus, the question may arise which benefits are associated with ratio-based effect measures. In contrast to difference-based effect, which are absolute effect measures, the ratio-based effects are relative effect measures. That is, the treatment effect is expressed in relation to a baseline and, thus, incorporates additional information.

Table 1 provides a minimal example of the benefit of ratio-based effects over the difference-based effect. We consider a random experiment where Joe, Ann, and Sue are randomly assigned to the treatment or control group and the expected outcome is the probability of success (e.g., cure from an illness). As we can see, Joe's success probability is already quite high even if he is not treated (i.e., 85%) and his chances are increasing to 95% if he is treated. This is very different for Ann. Her success probability without treatment is about 5%, but with treatment it increases to 15%. And we have yet different success probabilities for Sue (i.e., 50% with treatment, 40% without treatment). On the



**Figure 1**

Illustration of the relationship between a homogeneous difference-based treatment effect ( $\delta_{10} = 0.1$ ; green line) and a simple ratio effect (left panel, orange line) or a odds ratio effect (right panel, orange line) for different levels of success probability in the control group (i.e.,  $\tau_0$ ).

difference-based effect measure, the treatment works equally well for all three patients (i.e.,  $\delta_{10}(U = u) = 0.1$  for all  $u \in \{\text{Joe}, \text{Ann}, \text{Sue}\}$ ). However, both the simple ratio and the odds ratio would lead to differential indications.

The simple ratio effect indicates that the treatment is most effective for Ann, as her success probability is three times higher under treatment than under control. The effect measures takes into account that her success probability is very low if she was not treated. In contrast, for Joe the simple ratio is smaller as he already has a high success probability even if he was not treated. Hence, the simple ratio gives an indication for whom the treatment is especially beneficial with regard to what would happen without treatment. If the absolute difference effect  $\delta_{10}$  is held constant, the simple ratio will be higher for persons with low success probability under control. This is illustrated in the left panel of Figure 1.

The odds ratio offers an effect measure, which is yet different from the simple ratio. While the simple ratio is very different for Joe and Ann in Table 1, the odds ratio is the same for both of them. The reason is, that the odds ratio reflects how much change is going on in relation to the proximity to the bounds of the success probability (i.e., 0 and 1). Thus, the closer the effect of  $\delta_{10}$  is shifted to either the lower or the upper bound, the

larger the corresponding odds ratio gets – and Joe and Ann are equally close to their respective bounds. In contrast, the closer the effect comes to a success probability of 50% (as is the case for Sue, in this example), the smaller the odds ratio gets. This is illustrated in the right panel of Figure 1.

### Average Causal Effects

In applied scenarios, we cannot observe either the true outcome variable nor the individual treatment effect variable, unless very strong assumptions hold. However, we might be able to estimate aggregates of these variables depending on the conditions of our study. One noteworthy aggregate of individual treatment effects is the average treatment effect  $ATE_{10}$  of treatment  $X = 1$  compared to the control group  $X = 0$ :

$$ATE_{10} = E(\delta_{10}) \tag{2}$$

The ATE can be identified and estimated if certain causality conditions hold, for example, as a difference in group means in a randomized controlled trial. Using the (multidimensional) covariate  $Z$ , we can compute conditional treatment effects using the effect function

$$g_1(Z) := E(Y|X = 1, Z) - E(Y|X = 0, Z)$$

Note that conditional effects of  $Z$  are not necessarily aggregates of  $\delta_{t0}$ . That is, we have to ensure conditions under which

$$g_1(Z) = E(\delta_{10}|Z)$$

holds. In this case, the effect function is called causally unbiased and we will introduce and check some of the (empirically testable) causality conditions below.

### Average Ratio Effects

As for the difference-based individual causal effect, we cannot empirically observe simple or odds ratios on an individual level, unless very strong assumptions hold. However, we can compute (and estimate) aggregates of the individual ratio-based effects, either unconditional or up to the levels of a covariate  $Z$ . In the following, we will provide the definition of an average simple ratio and an average odds ratio effect. Then, we will briefly discuss how these average ratio effects and their interpretation relate to the ATE from Equation (2).

We define an average simple ratio (ASR) of the treatment  $X = 1$  compared to control group  $X = 0$  as the unconditional expectation of the individual simple ratio effects  $\rho_{10}$ :

$$\text{ASR}_{10} := E\left(\frac{\tau_1}{\tau_0}\right) = E(\rho_{10})$$

Similarly, the average odds ratio (AOR) of the treatment  $X = t$  compared to control group  $X = 0$  is defined as the unconditional expectation of the atomic odds ratio effects  $o_{t0}$ :

$$\text{AOR}_{10} := E\left(\frac{\frac{\tau_1}{1-\tau_1}}{\frac{\tau_0}{1-\tau_0}}\right) = E(o_{t0})$$

In the previous section, we explained that difference- and ratio-based effect measures are associated with varying interpretations. Thus, it may come as no surprise, that averaging over these effect measures, can result in very different results and interpretations for one and the same random experiment. In the following, we provide a minimal example where all three average effect measures (i.e., ATE, ASR, AOR) yield different indications regarding the treatment. The overview of the example is given in Table 2.

For this example, we chose a dichotomous outcome again, as it is possible to compute all three effect measures for this kind of outcome. We consider treatment effects for Joe and Ann, where the success probability for Joe is 90% without treatment and 80% with treatment (i.e., treatment is harmful for Joe) and is 40% without treatment and 50%

**Table 2***Joe and Ann With Randomized Assignment: Three Different Average Effects*

$u$	$E(Y X=0, U=u)$	$E(Y X=1, U=u)$	$\delta_{10}$	$\rho_{10}$	$o_{10}$	$P(U=u)$	$P(X=x U=u)$
Joe	.90	.80	−0.100	0.889	0.444	.5	.5
Ann	.40	.50	0.100	1.250	1.500	.5	.5

*Note.* Conditional expectations for three different values of  $u$ , with corresponding individual effects: difference-based effect  $\delta_{10}$ , simple ratio-based effect  $\rho_{10}$ , and odds ratio-based effect  $o_{10}$

with treatment for Ann (i.e., Ann benefits from treatment). The three average effect measures for this example are as follows:

$$\text{ATE}_{10} = 0; \quad \text{ASR}_{10} = 1.069; \quad \text{AOR}_{10} = 0.972 \quad (3)$$

The difference-based ATE suggests that there is no treatment effect on average. This is because, Joe’s success probability decreases under treatment (i.e., −10%) as much as Ann’s is gaining (i.e., 10%) and, consequently, the ATE is zero in this case. The simple ratio-based ASR indicates a positive effect of the treatment on average. That is, the positive effect for Ann is stronger than the negative effect for Joe, because Ann’s success probability under control is lower than Joe’s. Yet, the odds ratio-based AOR indicates a negative effect of the treatment on average. Here, the negative effect for Joe is stronger than the positive effect for Ann, because his success probabilities under both conditions are closer to one of the bounds of the probabilities (i.e., the upper bound of 1).

While this is a minimal example, it illustrates the intricacies of averaging over non-linear effect measures, such as ratio-based effects. While the interpretation of these effect measures is fairly comprehensible on an individual level, averaging over individual-level odds ratios and interpreting these averages might be a major challenge.

**Table 3***Identifiability of Average Effects*

	Condition			
	CC1	CC2	CC3	CC4
$ATE_{t0}$	✓	✓	✓	✓
$ASR_{t0}$	–	–	✓	✓
$AOR_{t0}$	–	–	✓	✓

*Note.* CC1: Independent Cause Condition; CC2: Conditional Independent Cause Condition; CC3: Regressively Independent Outcome Condition; CC4: Conditional Regressively Independent Outcome Condition

Thus, applied researchers in psychology and the social sciences should be aware of these interpretative challenges of ratio-based effect measures.

### Identification of Average Ratio Effects

It is crucial for causal inference that the quantities that we can compute and estimate in an applied setting (e.g., group means, treatment effects conditional on a covariate) are actually aggregates of the underlying individual treatment effects. Otherwise, these quantities are called *causally biased*. While it is not possible to empirically test causal unbiasedness of a (un-)conditional expectation given  $X$  and/or  $Z$ , there exists a set of causality conditions, which imply unbiasedness of the conditional expectations  $E(Y|X=x)$  and  $E(Y|X=x, Z)$  and some of these causality conditions are empirically testable.

In this paper, we will examine under which causality conditions the average ratio effects (i.e., ASR and AOR) can be identified. That is, under which conditions is it possible to compute an average simple ratio or an average odds ratio? In the following, we focus on four causality conditions that are also examined for total, direct, and indirect difference-based effects by Mayer et al. (2014). Note that these four conditions are not exhaustive (for an overview of further conditions, see, e.g., Steyer et al., 2000), but they are representative for common strategies for causal inference in both randomized and observational studies and all four can be tested in an empirical setting. An overview of our findings is provided in Table 3.

### Unbiasedness of Conditional Expectations

We start with briefly defining causal unbiasedness of a conditional expectation, which means that a conditional expectation is actually an aggregate of the true outcome variable. Causal unbiasedness of the expected outcome given a treatment group (e.g., the group mean of  $Y$ ) is defined as

$$E(Y|X = x) = E(\tau_x) \quad (4)$$

Similarly, causal unbiasedness of the expected outcome given a treatment group and conditional on the covariate  $Z$  is defined as

$$E(Y|X = x, Z) = E(\tau_x|Z) \quad (5)$$

Each of the four causality conditions considered in the following is implying at least one of these two causal unbiasedness definitions.

#### CC1 – Independent Cause Condition

The first causality condition (CC1) to imply causal unbiasedness of the expectation of the outcome given treatment  $X = x$  (e.g., a group mean) – as defined in Equation (4) – is the independent cause condition. That means, that the treatment variable  $X$  is independent from the person variable  $U_{t_X}$ .

$$P(X = x) = P(X = x|U_{t_X}) \implies E(Y|X = x) = E(\tau_x) \quad (6)$$

In applied scenarios, this can be achieved through randomization, that is, individuals are randomly assigned to one of the treatment conditions. Randomization is often referred to as the gold standard for causal inference. It is also possible to empirically test the

independent cause condition, because Equation (6) implies that

$$P(X = x|Z) = P(X = x) \quad \text{for all } Z \text{ that are functions of } U_{t_x},$$

that is, if CC1 holds there is no covariate  $Z$  predicting the treatment condition, which can in principle be falsified<sup>1</sup>.

It can be shown that CC1 does neither suffice for identification of the ASR nor of the AOR, that is:

$$\begin{aligned} P(X = x) = P(X = x|U_{t_x}) &\not\Rightarrow \frac{E(Y|X=t)}{E(Y|X=0)} = \text{ASR}_{10} \\ P(X = x) = P(X = x|U_{t_x}) &\not\Rightarrow \frac{\frac{E(Y|X=t)}{1 - E(Y|X=t)}}{\frac{E(Y|X=0)}{1 - E(Y|X=0)}} = \text{AOR}_{10} \end{aligned}$$

We can prove these propositions by using counterexamples. More specifically, we can use the minimal Joe-Ann example from Table 2 where the independent cause condition holds. The respective expectations in the example are  $E(Y|X=1) = 0.675$  and  $E(Y|X=0) = 0.675$  and, consequently, the ratios of these expectations neither equal  $\text{ASR}_{10}$  nor the  $\text{AOR}_{10}$ :

$$\text{ASR}_{10} = 1.069 \neq \frac{0.675}{0.675} ; \quad \text{AOR}_{10} = 0.972 \neq \frac{\frac{0.675}{1 - 0.675}}{\frac{0.675}{1 - 0.675}}$$

This phenomenon is well-known as *non-collapsibility* and has often been shown for randomized experiments (Greenland, 1996; Greenland et al., 1999). Greenland (2021, p, 264) describes:

“[...] group odds are not simple averages of individual odds when the odds vary

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<sup>1</sup> Note that condition CC1 implies that there is no true predictor of the treatment condition. Nevertheless, this does not prevent that one might a false positive significant predictor due to chance in applied settings.

across individuals. Let us now move on to an even more odd phenomenon: The odds ratio measuring a treatment effect on a group need not equal any average of its subgroup odds ratios, even if there is no confounding.”

Importantly, *no confounding* refers to the independence of treatment from potential confounders (i.e., CC1) in this quote.

## CC2 – Conditional Independent Cause Condition

In applied settings, randomization is sometimes carried out conditional on a covariate. This can be useful to ensure a balance of treatment and control group within subgroups of the sample. But the condition can also be achieved using propensity score or matching techniques.

This procedure can also be described in terms of a causality condition, the conditional independent cause condition, where we consider  $Z$ -conditional independence of the treatment variable  $X$  from the person variable  $U_{t_X}$ , which implies unbiasedness of the conditional expectation as defined in Equation (5):

$$P(X = x|Z) = P(X = x|U_{t_X}) \implies E(Y|X = x, Z) = E(\tau_x|Z)$$

which means that the conditional expectations are causally unbiased as long as we control for the covariate  $Z$ . Similarly as condition CC1, this causality condition can be empirically falsified, if a covariate  $W$  can be identified for which

$$P(X = x|Z) \neq P(X = x|Z, W)$$

does not hold. Note that this causality condition is also implied if CC1 holds.

Similarly as for CC1, it can be shown that neither the ASR nor the AOR can

**Table 4***Joe, Ann, Jim, and Sue With Conditional Randomized Assignment*

$u$	$Z$	$E(Y X=0, U=u)$	$E(Y X=1, U=u)$	$\rho_{10}$	$o_{10}$	$P(U=u)$	$P(X=x U=u)$
Joe	m	.90	.80	0.889	0.444	.25	.4
Ann	f	.50	.40	0.800	0.667	.25	.7
Jim	m	.70	.80	1.143	1.714	.25	.4
Sue	f	.40	.50	1.250	1.500	.25	.7

*Note.* Conditional expectations for three different values of  $u$ , with corresponding individual effects: difference-based effect  $\delta_{10}$ , simple ratio-based effect  $\rho_{10}$ , and odds ratio-based effect  $o_{10}$

generally be identified under this condition, that is:

$$P(X=x|Z) = P(X=x|U_{t_x}) \not\Rightarrow E \left[ \frac{E(Y|X=t, Z)}{E(Y|X=0, Z)} \right] = \text{ASR}_{10}$$

$$P(X=x|Z) = P(X=x|U_{t_x}) \not\Rightarrow E \left[ \frac{\frac{E(Y|X=t, Z)}{1 - E(Y|X=t, Z)}}{\frac{E(Y|X=0, Z)}{1 - E(Y|X=0, Z)}} \right] = \text{AOR}_{10}$$

Again, we prove these propositions by using counterexamples. Here, we use the Joe-Ann example from Table 4 where the conditional independent cause condition holds as treatment probabilities are constant conditional on the covariate  $Z$ . The respective  $Z$ -conditional expectations in the example are

$$\begin{aligned} E(Y|X=1, Z=m) &= 0.800 ; & E(Y|X=1, Z=f) &= 0.450 ; \\ E(Y|X=0, Z=m) &= 0.800 ; & E(Y|X=0, Z=f) &= 0.450 \end{aligned}$$

and the expectation of these ratios of  $Z$ -conditional expectations do neither equal  $\text{ASR}_{10}$

nor the  $\text{AOR}_{10}$ :

$$\begin{aligned}\text{ASR}_{10} &= 1.021 \neq 0.5 \cdot \frac{0.800}{0.800} + 0.5 \cdot \frac{0.450}{0.450} = 1 ; \\ \text{AOR}_{10} &= 1.081 \neq 0.5 \cdot \frac{\frac{0.800}{1-0.800}}{\frac{0.800}{1-0.800}} + 0.5 \cdot \frac{\frac{0.450}{1-0.450}}{\frac{0.450}{1-0.450}} = 1\end{aligned}$$

While we are not aware of any study demonstrating the phenomenon of non-collapsibility in this setting, these results are unsurprising as they are closely related to the setting of unconditional randomization (as in CC1). In the next step, we will examine the identification of average ratio effects under causality conditions that do not require (conditional) independence of treatment and potential confounders.

### CC3 - Regressively Independent Outcome Condition

Causal inferences cannot only be drawn if (conditional) independence of the treatment variable and potential confounders holds. It can also be achieved by creating regressive independence of the outcome variable  $Y$  from the potential confounders for person  $U_{t_x}$ . In a very general version, this is achieved when

$$\text{E}(Y|X=x) = \text{E}(Y|X=x, U_{t_x}) \implies \text{E}(Y|X=x) = \text{E}(\tau_x).$$

holds. This is a scenario, where the expected outcome of  $Y$  only depends on the assigned treatment condition. Note that this scenario is very unrealistic in applied settings, as, for example, not even the pretest variable is assumed to be predictive of the outcome. Thus, this causality condition is presented for the sake of completeness. Its conditional version (i.e., CC4), on the other hand, is very common and often applied in observational research. While CC3 cannot be created by randomization or covariate selection techniques, it can be

empirically falsified by finding a covariate  $Z$  for which

$$E(Y|X = x) = E(Y|X = x, Z)$$

does not hold.

It can be shown that under CC3, both the ASR and the AOR can be identified, that is:

$$\begin{aligned} E(Y|X = x) = E(Y|X = x, U_{t_x}) &\implies \frac{E(Y|X = t)}{E(Y|X = 0)} = \text{ASR}_{10} \\ E(Y|X = x) = E(Y|X = x, U_{t_x}) &\implies \frac{\frac{E(Y|X = t)}{1 - E(Y|X = t)}}{\frac{E(Y|X = 0)}{1 - E(Y|X = 0)}} = \text{AOR}_{10} \end{aligned}$$

Proofs for this propositions can be found in Appendix A. While CC3 describes a rather hypothetical situation, it already illustrates that ratio-based effect measures can be collapsible. This finding is a special case of the *strict collapsibility* shown by Newman (2001, p. 50), which requires the  $\rho_{10}$  and  $\sigma_{10}$  to be homogeneous (i.e., they do not vary as a function of the covariate). Under CC3 this is achieved as the group-specific conditional expectations of  $Y$  given  $Z$  are constant.

#### CC4 - Conditional Regressively Independent Outcome Condition

Related to CC3, but more interesting from an applied point of view, is the conditional regressive independence of the outcome  $Y$  from the person variable  $U_{t_x}$  given the treatment variable  $X$  and the covariate  $Z$ :

$$E(Y|X = x, Z) = E(Y|X = x, U_{t_x}) \Rightarrow E(Y|X = x, Z) = E[\tau_x|Z].$$

In this case, the person variable  $U_{t_x}$  does not carry any additional information beyond the covariate  $Z$ . In applied settings, this can be achieved through careful covariate selection

and this strategy is commonly applied for causal inference in observational studies. The condition can also be empirically falsified by an additional covariate  $W$  for which

$$E(Y|X = x, Z) = E(Y|X = x, Z, W)$$

does not hold.

It can be shown that under CC4, both the ASR and the AOR can be identified, that is:

$$E(Y|X = x, Z) = E(Y|X = x, U_{t_x}) \implies E \left[ \frac{E(Y|X = t, Z)}{E(Y|X = 0, Z)} \right] = \text{ASR}_{10}$$

$$E(Y|X = x, Z) = E(Y|X = x, U_{t_x}) \implies E \left[ \frac{\frac{E(Y|X = t, Z)}{1 - E(Y|X = t, Z)}}{\frac{E(Y|X = 0, Z)}{1 - E(Y|X = 0, Z)}} \right] = \text{AOR}_{10}$$

Proofs for this propositions can be found in Appendix A. This is a new and important finding, as both average ratio-based effect measures can be shown to be collapsible under weaker conditions than required for Newman's strict collapsibility. Both the ASR and the AOR can be accurately computed based on covariate selection and adjustment, which is often used for causal inference in observational studies.

In sum, we identified two common challenges with (average) ratio-based effect measures: First, the substantive interpretation of both the ASR and the AOR can be tricky, as the aggregation of individual ratio-based effect measures happens on a different effect scale. Second, both the ASR and the AOR cannot generally be identified in randomized experiments, unless additional covariates are accounted for. As randomized experiments are commonly considered the gold standard for causal inference, the non-collapsibility is a unfavorable property in these cases. This is partly related to the phenomenon of non-collapsibility. However, we have also shown that for the regressively independent outcome conditions it is actually possible to identify the average ratio-based

effect measures and, hence, these effects are collapsible under these conditions.

### **Ratios of Causally Unbiased Expectations**

Despite their aforementioned downsides, there are still scenarios where ratio-based effect measures are desirable. For example, (Newman, 2001, p. 36) provides a number of examples, where difference- and ratio-based effect measures are of interest from different substantive perspectives. Thus, we present a solution for average ratio-based effect measures which are not aggregates over individual effects, but consistent with magnitude and direction of each other and the average difference-based effect measure (i.e., the ATE), and are identifiable under all four considered causality conditions.

These average ratio-based effect measures are defined as ratios of causally unbiased conditional expectations instead of as expectations of individual ratio effects.

Consequently, we define a simple ratio of averages (SRA) as

$$\text{SRA}_{10} := \frac{E(\tau_1)}{E(\tau_0)}$$

and a odds ratio of averages (ORA) as

$$\text{ORA}_{10} := \frac{\frac{E(\tau_1)}{1 - E(\tau_1)}}{\frac{E(\tau_0)}{1 - E(\tau_0)}}$$

The advantage of these definitions is that they are based on quantities that are causally unbiased under the causality conditions CC1 to CC4. Thus, both the SRA and the ORA can be identified under these conditions, especially in randomized experiments – in contrast to their counterparts ASR and AOR. We provide proofs for the identification both the SRA and the ORA under the four causality conditions in Appendix B

It is also noteworthy, that the inconsistent treatment indications demonstrated in

Equation (3), vanish for the SRA and ORA. Indications from these effects are always consistent with each other and the ATE. Remember, that in Equation (3) we provided an example where ATE, ASR and AOR pointed into different directions regarding the average effectiveness of the treatment (i.e., no average effect, positive average effect, and negative average effect). However, with the effects defined as ratios of averages, all three effects (i.e., ATE, SRA, ORA) always provide the same indication, that is:

$$ATE_{10} = 0 \iff SRA_{10} = 1 \iff ORA_{10} = 1$$

$$ATE_{10} > 0 \iff SRA_{10} > 1 \iff ORA_{10} > 1$$

$$ATE_{10} < 0 \iff SRA_{10} < 1 \iff ORA_{10} < 1$$

That means, for the example provided in Table 2, the ATE, the SRA, and the ORA indicate that there is no average treatment effect.

Thus, SRA and ORA allow researchers to express their treatment effects on a ratio-based scale (i.e., simple ratio, odds ratio), while still maintaining identification under randomized experiments and keeping interpretations consistent among different effect measures.

### Comparison of Causal Effect Quantities

Under the previously outlined causality conditions, the SRA and ORA accurately reflect the ATE when CC1 is satisfied, unlike the ASR and AOR. As displayed in Table 2, the SRA and ORA suggest no ratio effect, aligning with the ATE, while the ASR and AOR imply a ratio effect with conflicting directions.

$$SRA_{10} = 1 \neq ASR_{10} = 1.069$$

$$ORA_{10} = 1 \neq AOR_{10} = 0.972$$

When causality condition CC1 is satisfied, the SRA and ORA are calculated from a ratio of causally unbiased expectations. Their definitions inherently allow them to circumvent the issue of non-collapsibility because the ratio is computed last. This sets them apart from the ASR and AOR, which remain vulnerable to this issue. Under CC2, the discrepancy persists between the SRA and ORA versus the ASR and AOR. The reasoning behind this is analogous to CC1.

As previously discussed, under CC3, both the ASR and AOR are collapsible. Consequently, they yield the same results as the SRA and ORA. This equality emerges because CC3 assumes a scenario where the outcome variable depends solely on the treatment variable, irrespective of potential confounding variables. Therefore, both group-specific and the overall ratio effects are uniform, causing the ASR and AOR to be collapsible.

However, under CC4, a more realistic scenario in empirical settings, the correspondence between the effects as ratios of expectations and expectations of individual ratios does not hold. Although the ASR and AOR are collapsible under CC4, their equivalence to SRA and ORA is not guaranteed. This is because, unlike under CC3, the individual ratio effects under CC4 might vary with different levels of the covariates, thereby causing a discrepancy between the ASR and AOR and SRA and ORA. This is an important finding, as both effect definitions have a causal meaning, but can have different implications in practical settings, as we will show in our illustrative example below. A proof illustrating why the ratio effects coincide under CC3 but not under CC4 is provided in Appendix C.

The choice of effect measure significantly influences the interpretation of the treatment effect due to their varying properties and behaviors under different causality conditions. The newly introduced measures, SRA and ORA, consistently align with the average treatment effect across all causality conditions. However, these measures can yield results and interpretations that differ significantly from traditional measures such as the

**Table 5***Summary of comparison of ratio effect definitions*

	Causality Condition			
	CC1	CC2	CC3	CC4
SRA = ASR	×	×	✓	×
ORA = AOR	×	×	✓	×

*Note.* CC1: independent cause condition; CC2: conditional independent cause condition; CC3: regressively independent outcome condition; CC4: conditional regressively independent outcome condition; SRA: simple ratio of averages; ASR: average of simple ratios; ORA: odds ratio of averages; AOR: average of odds ratios

ASR and AOR, particularly under more realistic causality conditions like CC1, CC2, and CC4. Under CC3, all measures do align. Table 5 provides a summary of these distinctions.

### Illustrative Example

We provide a small simulated data example to illustrate the theoretical findings and show how the effects can be computed based on data. We generated a random sample of  $N = 600$  observations where each person was randomly assigned to either treatment ( $X = 1$ ) or control ( $X = 0$ ) with equal probability of  $P(X = 1|U = u) = 0.5$  (i.e., randomized assignment). The outcome variable  $Y$  was generated as a binary variable, with the probability of success being determined by a Bernoulli distribution. The success probabilities varied based on whether the individual had a certain precondition  $Z = 1$  (or not,  $Z = 0$ ), and also depended on their respective treatment status. The resulting dataset contains a precondition variable  $Z$  with values 0 or 1, the treatment variable, and the binary outcome. Based on a logistic regression model of  $E(Y = 1|X, Z)$ , we can compute all five of the above defined effect measures. The R script for both the data generation and the data analysis of this example as well as the dataset are provided in the Supplementary Materials.

### Group-Level Effects

As we generated the dataset with randomized assignment to the treatment groups, the causality condition CC1 (i.e., independent cause condition) holds in this example. Thus, we can judge the average effectiveness of our treatment by estimating the

**Table 6***Contingency table for the whole sample of the illustrative example.*

	$Y = 0$	$Y = 1$	
$X = 0$	133	159	292
$X = 1$	152	156	308
	285	315	600

*Note.* The numbers reflect absolute frequencies of observations within the combinations of the treatment variable and the outcome variable.

difference-based effect measure  $ATE_{10}$ , the simple ratio-based effect measure  $SRA_{10}$ , and the odds ratio-based effect measure  $ORA_{10}$ . However, the ratio-based effect measures which average over individual ratio effects (i.e.,  $ASR_{10}$  and  $AOR_{10}$ ) are not identified under this condition. A contingency table of the absolute frequencies of success (i.e.,  $Y = 1$ ) and failure (i.e.,  $Y = 0$ ) divided by treatment groups is given in Table 6.

An overview of the estimated effect measures and their 95% confidence intervals (CIs) in this illustrative example is given in Table 8. The estimated  $\widehat{ATE}_{10} = -0.038$  is quite small and the 95% CI includes zero, suggesting an insubstantial overall impact of the treatment on average. Furthermore, the  $\widehat{SRA}_{10} = 0.930$  and  $\widehat{ORA}_{10} = 0.858$  are both close to unity with CIs including the 1, indicating that the newly defined ratios of expectations reflect an average effect of the treatment similar to the ATE.

### Average Effects with Covariate Adjustment

Not entirely coincidental, we generated the data in a way that the causality condition CC4 (i.e., conditional regressively independent outcome) also holds, if one accounts for the precondition covariate  $Z$ . Under this condition, all five of the above defined average effect measures can be identified. A contingency table distinguishing the absolute frequencies of success (i.e.,  $Y = 1$ ) and failure (i.e.,  $Y = 0$ ) by treatment groups and the precondition covariate is given in Table 7.

It can be seen that the covariate  $Z$  has a strong moderating effect on the treatment-outcome relationship. While the treatment works very well for persons without the precondition (i.e.,  $Z = 0$ ), it has a detrimental effect for persons with the precondition

**Table 7***Contingency table for two subgroups of the illustrative example.*

	$Z = 0$			$Z = 1$			
	$Y = 0$	$Y = 1$		$Y = 0$	$Y = 1$		
$X = 0$	56	94	150	$X = 0$	77	65	142
$X = 1$	7	148	155	$X = 1$	145	8	153
	63	242	305		222	73	295

*Note.* The numbers reflect absolute frequencies of observations within the combinations of the treatment variable and the outcome variable depending on the precondition covariate.

(i.e.,  $Z = 1$ ). The three effect measures which we already computed on the group level should be invariant to this additional information, as they were already identified through CC1. Indeed, the  $\widehat{ATE}_{10} = -0.033$ ,  $\widehat{SRA}_{10} = 0.940$ , and  $\widehat{ORA}_{10} = 0.878$  accounting for the covariate  $Z$  are still close to our estimates from the group level (see also Table 8). One notable effect of the covariate-adjustment, however, is the shrinkage of the corresponding CIs as the inclusion of covariate information would be expected to increase the statistical power for these estimates. Thus, the example illustrates that these three effect measures are collapsible.

In contrast, the  $\widehat{ASR}_{10} = 0.831$  shows a strong negative effect of the treatment and its 95% CI = [0.724, 0.973] does not include the 1, suggesting that this is a significant effect. The  $\widehat{AOR}_{10} = 6.435$ , on the other hand, reflects an overall positive effect of the treatment, for which the 95% CI = [1.139, 11.731] does also not include the 1, suggesting that this is also a significant effect. It is crucial to understand, that both these effects have a clear causal interpretation, even though their implications are in stark contrast to the ATE, SRA, and ORA. While the SRA and the ORA reflect average effectiveness based on causally unbiased expectations on a group level, the ASR and the AOR reflect average effectiveness as causally unbiased expectations of individual simple ratios or odds ratios, respectively. Only the ATE can be interpreted as both a difference between causally unbiased expectations on a group level as well as an expectation over individual difference effects.

Note that the moderating effect of the covariate  $Z$  is extraordinarily strong in this

**Table 8***Estimated Effect Measures based on Simulated Data Example*

Effect	Group-level		Average over $Z$	
	Estimate	95% CI	Estimate	95% CI
$\widehat{ATE}_{10}$	-0.038	[-0.118, 0.042]	-0.033	[-0.023, 0.024]
$\widehat{SRA}_{10}$	0.930	[0.789, 1.072]	0.940	[0.833, 1.047]
$\widehat{ORA}_{10}$	0.858	[0.583, 1.134]	0.878	[0.661, 1.094]
$\widehat{ASR}_{10}$	—	—	0.831	[0.724, 0.973]
$\widehat{AOR}_{10}$	—	—	6.435	[1.139, 11.731]

*Note.*

case and designed to result in a significant and positive AOR as well as a significant and negative ASR. We would not expect such strong effects in applied settings. Moreover, large sample sizes are required to detect significant and contradictory estimates of the ASR and AOR. However, situations where at least one of the ATE, ASR, and AOR estimates significantly points into another direction than the remaining two seem to be realistic in applied settings.

### Discussion

In this paper, we have provided definitions of simple ratios and odds ratios on an individual and average level from a causal inference perspective. We examined whether or not these effects are identifiable under common causality conditions and found that this is not always the case. Thus, we discussed an alternative way to define average ratio-based effect measures based on causally unbiased conditional expectations, which are always identifiable under the common causality conditions.

We want to highlight the two main contributions: First, we have shown that ratio-based effect measures can be collapsible under conditions that are relevant in practice of causal inference research. While it is well-known that (conditional) independence of treatment and potential confounders is not sufficient for collapsible ratio-based effect measures (e.g., Greenland et al., 1999), we have shown that regressive independence of the

outcome and potential confounders is a sufficient condition. These causality conditions are especially common in observational studies where randomization is not feasible and, hence, covariate selection techniques are employed to infer causal effects. Our findings show, that ratio-based effect measures are collapsible in these cases.

Second, we recommend the use of ratios of causally unbiased expectations as alternative average ratio-based effect measures. This is in contrast to previous literature, which suggests to refrain from ratio-based effect measures and to choose difference-based effect measures instead (e.g., Hanmer & Ozan Kalkan, 2013; Mood, 2010). We argue that ratio-based effect measures can be useful in some scenarios and, hence, our proposed alternative offers a way to get collapsible, consistent, and causally unbiased ratio-based effects.

## Implications

It might be asked which of the five effect measures should be preferred in an applied scenario. While this depends on the concrete research questions, we try to give some broad recommendations based on our theoretical derivations and the illustrative examples.

In randomized controlled trials, where the main focus lies on the evaluation of average effectiveness of the treatment, we recommend the use of ATE, SRA, or ORA. All three of these effects are identified in this scenario through the (conditional) independent cause condition (i.e., CC1 or CC2) and, thus, provide consistent results, even when accounting for covariates. Covariate adjustment is predominantly done to increase the efficiency and precision of the average effect estimation (Hernández et al., 2004; Negi & Wooldridge, 2021) and the SRA and ORA offer an alternative to pursue this goal without running into the issue of non-collapsibility. We illustrated this point in our illustrative example.

From a causal perspective, the phenomenon of non-collapsibility seems to stem from the attempt to estimate a non-identified effect – and this does not only hold true for ratio effects. For example, Steyer et al. (2000) provide an example, where only the conditional

regressively independent outcome condition (i.e., CC4) holds. They call the (non-identified) effect on the group level as *prima facie effect*, which differs substantially in magnitude and direction from the (identified) ATE. This phenomenon is sometimes also referred to as Simpson's paradox.

### Limitations

In this paper, we treated the issue of ratio-based effect measures mostly from the perspective of the stochastic theory of causal effects and, therefore, on a level of probability theory and not quality of statistical inferences (with exception of our illustrative example). When it comes to applied research and real data, researchers will need to come up with a (statistical) model or technique for estimating the conditional expectations. Depending on the outcome variable and the nature of the relationship among outcome, treatment, and covariates this might be addressed using, for example, linear and non-linear regression techniques (e.g. OLS regression, logistic regression, Poisson regression) or matching techniques. It is important to note that the correctness of the model (or method) also plays a role whether the underlying causal effects can be estimated without bias. Nevertheless, we think it is beneficial to examine the properties of effect measures based on a random experiment to be able to disentangle properties and inconsistencies that are purely to the effect measure itself (e.g., non-collapsibility) from properties and (perhaps) biases that are due to statistical modeling of these effect measures.

In the same vein, it should be stated that the stochastic theory of causal effects – just as Rubin's potential outcome framework and Pearl's graphical approach – often assume an idealized world, where, for instance, randomized treatment assignment is imagined as a perfectly random flip of a coin. In practice, however, randomized assignment is done with real persons, who might or might not accept the assigned treatment and might choose not to comply. Such treatment non-compliance can also compromise the causal interpretation of effect measures (Meier, 1991; Sheiner & Rubin, 1995). For a broader overview of causal inference in applied settings, see West et al. (2014) or Shadish et al. (2001).

## References

- Daniel, R., Zhang, J., & Farewell, D. (2021). Making apples from oranges: Comparing noncollapsible effect estimators and their standard errors after adjustment for different covariate sets. *Biometrical Journal*, 63(3), 528–557.  
<https://doi.org/10.1002/bimj.201900297>
- Gail, M. H., Wieand, S., & Piantadosi, S. (1984). Biased estimates of treatment effect in randomized experiments with nonlinear regressions and omitted covariates. *Biometrika*, 71(3), 431–444. <https://doi.org/10.1093/biomet/71.3.431>
- Greenland, S. (1996). Absence of confounding does not correspond to collapsibility of the rate ratio or rate difference. *Epidemiology*, 7(5), 498–501.  
<https://doi.org/10.1097/00001648-199609000-00008>
- Greenland, S. (2021). Noncollapsibility, confounding, and sparse-data bias. Part 2: What should researchers make of persistent controversies about the odds ratio? *Journal of Clinical Epidemiology*, 139, 264–268. <https://doi.org/10.1016/j.jclinepi.2021.06.004>
- Greenland, S., Pearl, J., & Robins, J. M. (1999). Confounding and collapsibility in causal inference. *Statistical Science*, 14(1). <https://doi.org/10.1214/ss/1009211805>
- Hanmer, M. J., & Ozan Kalkan, K. (2013). Behind the curve: Clarifying the best approach to calculating predicted probabilities and marginal effects from limited dependent variable models. *American Journal of Political Science*, 57(1), 263–277.  
<https://doi.org/10.1111/j.1540-5907.2012.00602.x>
- Hernández, A. V., Steyerberg, E. W., & Habbema, J. F. (2004). Covariate adjustment in randomized controlled trials with dichotomous outcomes increases statistical power and reduces sample size requirements. *Journal of Clinical Epidemiology*, 57(5), 454–460. <https://doi.org/10.1016/j.jclinepi.2003.09.014>
- Imbens, G., & Rubin, D. B. (2015). *Causal inference for statistics, social, and biomedical sciences: An introduction*. Cambridge University Press.

- Mayer, A. (2019). Causal effects based on latent variable models. *Methodology*, 15, 15–28.  
<https://doi.org/10.1027/1614-2241/a000174>
- Mayer, A., Thoemmes, F., Rose, N., Steyer, R., & West, S. G. (2014). Theory and Analysis of Total, Direct, and Indirect Causal Effects. *Multivariate Behavioral Research*, 49(5), 425–442. <https://doi.org/10.1080/00273171.2014.931797>
- Meier, P. (1991). Compliance as an Explanatory Variable in Clinical Trials: Comment. *Journal of the American Statistical Association*, 86(413), 19.  
<https://doi.org/10.2307/2289709>
- Miettinen, O. S., & Cook, E. F. (1981). Confounding: Essence and detection. *American Journal of Epidemiology*, 114(4), 593–603.  
<https://doi.org/10.1093/oxfordjournals.aje.a113225>
- Mood, C. (2010). Logistic regression: Why we cannot do what we think we can do, and what we can do about it. *European Sociological Review*, 26(1), 67–82.  
<https://doi.org/10.1093/esr/jcp006>
- Negi, A., & Wooldridge, J. M. (2021). Revisiting regression adjustment in experiments with heterogeneous treatment effects. *Econometric Reviews*, 40(5), 504–534.  
<https://doi.org/10.1080/07474938.2020.1824732>
- Newman, S. C. (2001). *Biostatistical methods in epidemiology*. John Wiley & Sons.
- Niu, L. (2020). A review of the application of logistic regression in educational research: Common issues, implications, and suggestions. *Educational Review*, 72(1), 41–67.  
<https://doi.org/10.1080/00131911.2018.1483892>
- Pearl, J. (2009, September 14). *Causality: Models, Reasoning, and Inference* (2nd ed.). Cambridge University Press. <https://doi.org/10.1017/CBO9780511803161>
- Rubin, D. B. (2005). Causal inference using potential outcomes: Design, modeling, decisions. *Journal of the American Statistical Association*, 100(469), 322–331.  
<https://doi.org/10.1198/016214504000001880>

- Shadish, W. R., Cook, T. D., & Campbell, D. T. (2001). *Experimental and quasi-experimental designs for generalized causal inference*. Houghton Mifflin.
- Sheiner, L. B., & Rubin, D. B. (1995). Intention-to-treat analysis and the goals of clinical trials\*. *Clinical Pharmacology & Therapeutics*, 57(1), 6–15.  
[https://doi.org/10.1016/0009-9236\(95\)90260-0](https://doi.org/10.1016/0009-9236(95)90260-0)
- Steyer, R., Gabler, S., von Davier, A. A., & Nachtigall, C. (2000). Causal Regression Models II: Unconfoundedness and Causal Unbiasedness. 5(3).
- Steyer, R., Mayer, A., & Fiege, C. (2014). Causal Inference on total, direct, and indirect Effects. In A. C. Michalos (Ed.), *Encyclopedia of Quality of Life and Well-Being Research* (pp. 606–630). Springer Netherlands.  
[https://doi.org/10.1007/978-94-007-0753-5\\_295](https://doi.org/10.1007/978-94-007-0753-5_295)
- Steyer, R., & Nagel, W. (2017). *Probability and conditional expectation: Fundamentals for the empirical sciences*. Wiley.
- West, S. G., Cham, H., & Liu, Y. (2014, February 24). Causal Inference and Generalization in Field Settings: Experimental and Quasi-Experimental Designs. In H. T. Reis & C. M. Judd (Eds.), *Handbook of Research Methods in Social and Personality Psychology* (2nd ed., pp. 49–80). Cambridge University Press.  
<https://doi.org/10.1017/CBO9780511996481.007>

## Appendix A

### Proofs for CC3 and CC4

Proof for CC3 and ASR

$$\text{ASR}_{t0} = E(\rho_{t0}) = E \left[ \frac{E(Y|X=t, C_X)}{E(Y|X=0, C_X)} \right] \stackrel{\text{CC3}}{=} E \left[ \frac{E(Y|X=t)}{E(Y|X=0)} \right] = \frac{E(Y|X=t)}{E(Y|X=0)}$$

Analog for AOR.

Proof for CC4 and ASR

$$\text{ASR}_{t0} = E(\rho_{t0}) = E \left[ \frac{E(Y|X=t, C_X)}{E(Y|X=0, C_X)} \right] \stackrel{\text{CC4}}{=} E \left[ \frac{E(Y|X=t, Z)}{E(Y|X=0, Z)} \right]$$

Analog for AOR.

## Appendix B

### Proofs for SRA and ORA

Proof for CC1 and SRA

$$\text{SRA}_{t0} = \frac{E(\tau_t)}{E(\tau_0)} \stackrel{\text{CC1}}{=} \frac{E(Y|X=t)}{E(Y|X=0)}$$

Analog for ORA.

Proof for CC2 and SRA

$$\text{SRA}_{t0} = \frac{E(\tau_t)}{E(\tau_0)} = \frac{E[E(\tau_t|Z)]}{E[E(\tau_0|Z)]} \stackrel{\text{CC2}}{=} \frac{E[E(Y|X=t, Z)]}{E[E(Y|X=0, Z)]}$$

Analog for ORA.

Proof for CC3 and SRA

$$\text{SRA}_{t0} = \frac{E(\tau_t)}{E(\tau_0)} \stackrel{\text{CC3}}{=} \frac{E(Y|X=t)}{E(Y|X=0)}$$

Analog for ORA.

Proof for CC4 and SRA

$$\text{SRA}_{t0} = \frac{E(\tau_t)}{E(\tau_0)} \stackrel{\text{CC4}}{=} \frac{E[E(Y|X=t, Z)]}{E[E(Y|X=0, Z)]}$$

Analog for ORA.

## Appendix C

### Proof for Comparison of SRA and ASR under CC3 and CC4

$$\begin{aligned}
\text{ASR}_{t0} &= \text{E} \left[ \frac{\text{E}(\tau_t|Z)}{\text{E}(\tau_0|Z)} \right] \\
&= \text{E} \left[ \frac{\text{E}(Y|X = t, Z)}{\text{E}(Y|X = 0, Z)} \right] && \text{(CC4)} \\
&\neq \frac{\text{E}(Y|X = t, Z)}{\text{E}(Y|X = 0, Z)} && \text{(Only true if the ratio is constant for all } Z \text{ (CC3))} \\
&= \frac{\text{E}[\text{E}(Y|X = t, Z)]}{\text{E}[\text{E}(Y|X = 0, Z)]} && \text{(Law of Iterated Expectations)} \\
&= \frac{\text{E}[\text{E}(\tau_t|Z)]}{\text{E}[\text{E}(\tau_0|Z)]} && \text{(CC4)} \\
&= \text{SRA}_{t0}
\end{aligned}$$

Analog for AOR and ORA.