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Causal models accounted for research participation effects when estimating effects in a behavioral intervention trial

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Abstract

Objective: Participants in intervention studies are asked to take part in activities linked to the conduct of research, including signing consent forms and being assessed. If participants are affected by such activities through mechanisms by which the intervention is intended to work, then there is confounding. We examine how to account for research participation effects analytically.

Study design and setting: Data from a trial of a brief alcohol intervention among Swedish university students is used to show how a proposed causal model can account for assessment effects.

Results: The proposed model can account for research participation effects as long as researchers are willing to use existing data to make assumptions about causal influences, for instance on the magnitude of assessment effects. The model can incorporate several research processes which may introduce bias.

Conclusions: As our knowledge grows about research participation effects, we may move away from asking *if* participants are affected by study design, toward rather asking by how *much* they are affected, by which activities and in which circumstances. The analytic perspective adopted here avoids assuming there are no research participation effects.

Keywords: Research participation effects; Behavioral interventions; Randomized controlled trials; Causal models; Bias; Statistical analysis

1. Introduction

The research participation effects (RPE) construct has been proposed in order to facilitate the identification of separate sources and mechanisms of participation effects which have traditionally been bundled into a single concept known as the Hawthorne effect [1–4]. RPEs include generally acknowledged sources of bias in trials, such as lack of blinding, contamination, and compensatory behavior. They also give attention to overlooked prerandomization activities such as collecting informed consent and screening [5]. In the context of randomized controlled trials (RCTs), RPEs may introduce bias in effect estimates which may shift findings both away from and toward the null [6], and may thus in turn affect policy decisions based on intervention study findings.

The CONSORT Statement [7] recommends that sources of potential bias be discussed as study limitations in trials, and while such discussion is important to aid the interpretation of findings, it is rare that analyses and effect estimates account for them. This may be for several reasons, including a belief that any such biases are small enough to be ignored, and this may often be the case [8,9], or that apart from measurement reactivity [10], it is simply hard to make any informed adjustment for them. The reasoning goes that all participants are involved in such activities in RCTs, and those differences among participants will be equally divided between treatment groups due to randomization. Also, the assumption is generally that any influence that prerandomization activities may have on the intended outcome will be additive, and thus separate from the effects of the intervention under

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study, leaving group level measurements at follow-up equivalently shifted and contrasts unchanged [5,11].

Evidence of this conventional view can be found in almost all reports of RCTs, where analyses typically consist of regression models which aid the contrast of two or more groups of individuals receiving different treatments. These regression models may include covariates for baseline variables in order to make effect estimates more precise, however they do not account for any interaction between prerandomization activities and allocated treatment. Omitting such causal substructures may be of particular importance in trials of behavioral interventions, as it has for instance long been known that measuring behavior may both bias subsequent measurements and change behavior [12,13]. Although less prominent in the literature, participants are also unlikely to be completely neutral or inert in respect of other research activities such as signing consent forms and facing the uncertainty of randomization [14,15]. When the mechanism of effects of research activities at least partially overlaps those of the novel treatment under study [16], then conventional contrasts between groups may not account appropriately for bias.

Awareness of RPEs may lead to decisions to mitigate their effects, or to include nested studies within the trial to measure their effects and account for them as biases in the analyses [17]. Sometimes, however, it is not possible to mitigate or measure, and in such cases one may instead use external data to study the consequences of making particular assumptions about RPEs in a trial. This paper explores this approach, proposing causal models which aid accounting for RPEs when estimating the effects of interventions.

1.1. Studies of research participation effects

While RPEs are usually considered as potential sources of bias in studies of other phenomena, they have also been studied directly. One experiment compared engagement with alcohol educational material and alcohol consumption outcomes among those who were told that they had been allocated to the intervention arm vs. those who were told that they had been allocated to the control arm [18]. Despite both groups receiving the same material, those who were told that they were in the intervention arm were much more likely to access the material (78% vs. 57%) and spent more time on average viewing it (35 seconds vs. 8 seconds). While the experiment found no clear effect on 1-month alcohol outcomes, the evidence suggests that being informed about allocation may change individuals' view of what is expected of them and their actions, despite other activities being equal. A similar experiment found evidence indicating that some individuals (those more ready to change) who were told that they were placed on a waiting list were less likely to change their alcohol consumption despite being given the same material as those told they would have it immediately [19]. Thus, the information given to participants about the study may affect their behavior in unintended ways, with implications for estimates of engagement and effect. Other experiments of RPEs include those estimating the effects of social desirability considerations in reporting on one's own behavior [20], and other studies of the effects of obtaining informed consent [21]. Most studies, however, concern the effects of assessment or measurement, for which systematic review evidence indicates there are small effects across multiple topics [10].

Assessment is an activity that may play a role at different stages of a trial, including at screening, baseline, and follow-ups. In the form of observation, this was the activity that gave rise to the eponymous effect in the Hawthorne factory [22]. Trials of behavioral interventions where it is thought that some of the mechanisms by which the intervention is intended to work are triggered by assessment alone are particularly vulnerable to RPEs. We shall therefore in the following section consider a trial of a brief alcohol intervention which consists of a single session of assessment and feedback [23,24]. In this case, assessment by asking questions about alcohol consumption obviously overlaps with the intervention itself.

1.2. Alcohol email assessment and feedback study dismantling effectiveness for university studentS

The Alcohol eMail Assessment & feedback study Dismantling Effectiveness for University Students (AMADEUS-1) study was a unique trial in which the effects of an alcohol assessment and feedback intervention was estimated in a highly naturalistic setting, but which also included a substudy of RPEs [23–25]. Briefly, 14,910 email addresses of first, third and fifth semester students at two universities in Sweden were in 2011 randomized (1:1:1) to three groups (Group 1, Group 2 and Group 3). Group 1 and Group 2 were sent an email with a link to an alcohol assessment instrument (10 items). Group 1 were advised that they were to receive feedback on their response, which they subsequently did, whereas Group 2 were thanked for their participation and offered a link to a commonly used alcohol website without content understood to be effective in assisting behavior change. Group 3 were not contacted at all at this stage.

Three months postrandomization, all three groups were sent an email with an invitation to participate in an online lifestyle survey (15 items), with no particular emphasis on alcohol. Trial outcomes were derived from the three AUDIT-C items [26] embedded in the survey.

The key design element of the AMADEUS-1 study relevant to the study of RPEs was that none of the participants were aware that they were part of an intervention trial, nor that they had been randomized. The initial invitation was common screening practice among university students, and the follow-up was masked as a seemingly unrelated survey of lifestyle. By doing so, it was possible to isolate the RPE of assessment alone (Group 2 vs. Group 3) without bias

arising from other research awareness sources. The study was prospectively registered (ISRCTN28328154) and received ethical approval by the Regional Ethical Committee in Linköping, Sweden (No. 2010/291-31). The ethical issues were discussed in the bioethics literature [27], and it is not recommended that deception be widely practiced in research.

In what follows, we will first revisit the original analysis of the AMADEUS-1 study by contrasting groups without any adjustment for potential RPEs. These analyses have been described and reported previously [24,28,29]. We will then use this as a basis to develop a causal model to underpin the proposed analytic approach to RPEs in the subsequent section.

We begin by contrasting Group 1 (assessment and feedback) and Group 2 (assessment only). Differences between groups were estimated using multivariable normal linear regression with log-transformed AUDIT-C scores as outcome using Bayesian inference [30]. As can be seen in the distribution in Fig. 1a, the ratio is centered around 1, suggesting that we should not expect any marked difference between groups in terms of their AUDIT-C scores. The mean of the distribution in Fig. 1a suggests that the geometric mean of AUDIT-C scores among Group 1 was 0.4% higher than in Group 2. Given only this evidence, we may have concluded there was an absence of evidence of intervention effect. Some awareness of assessment effects may have played a role in our thinking, but may also have been considered likely to be small enough to ignore.

However, in AMADEUS-1 we also had a group which received neither assessment nor feedback (Group 3). This allowed us to estimate the assessment effect by contrasting Group 2 and Group 3. In Fig. 1b, we now see that the distribution is shifted away from 1, suggesting the presence of a noteworthy assessment effect. The mean of the distribution in Fig. 1b suggests that the geometric mean of AUDIT-C scores among Group 2 was 96.5% of that of Group 3, that is, a relative difference of 3.5%. This evidence suggests that assessment alone may have an impact on alcohol outcomes. Although small, it is not negligible, especially in light of the Group 1 Group 2 contrast.

Finally, the study design also allowed for estimation of the effects of combining assessment and feedback by contrasting Group 1 and Group 3. The mean of the distribution in Fig. 1c indicates that the geometric mean of AUDIT-C scores among Group 1 was 96.9% of that of Group 3, that is, a relative difference of 3.1%. This evidence suggests that feedback may not have had any additional effect above assessment.

The standard view is that baseline assessment is almost always necessary for trials to be analyzed appropriately, conferring precision in effect estimation. Thus, we choose to tolerate the presence of assessment effects in our trials. We can, however, use causal models to account for assessment effects when we analyze data from trials, even when there is not a no-contact/no-assessment group included in the trial. If we are willing to make assumptions about the ways in which assessment effects behavior, and to what degree they may do so, we can account for them when analyzing data from trials in which all participants have been assessed. Such judgments may be informed by existing data.

In the following section, we will propose a model for accounting for RPEs in trials where they are believed to be present yet have not been explicitly measured. Since we will use causal graph and causal mediation notation, we offer a primer on these in Appendix A.

Deception should not be used widely to counter RPEs. Instead we need to find a way to account for them in our analyses. To illustrate how this may be done, we now imagine that the AMADEUS-1 trial did not include Group 3. We previously showed that comparing Group 1 and Group 2 directly suggested no difference between groups, however, now we will account for assessment effects using the causal model in Fig. 2.

1.3. Causal model and assumptions

The causal model in Fig. 2 contains a variable representing baseline assessment (A), feedback (T), and follow-up AUDIT-C scores (Y). These could more generally be thought of as assessment, treatment and outcome. The model posits that assessment may have an effect on AUDIT-C scores through some unmeasured mechanism M. The model further suggests that the effects of feedback may also be mediated through the same mechanism M. For instance, M may represent a factor which is triggered by reflection on one's drinking or other behavior. We are aware this is possible for assessment, and expect that feedback is normally intended to do exactly this. There is, therefore, commonality between how assessment affects the outcome and the way the treatment is designed to operate[1].

Since M is unmeasured, there is no way of estimating its direct effect on Y, however, we can estimate the effects of A and T which are mediated through M by making assumptions about their influence on M. By doing so, we can still reason about total, direct and indirect effects involving A and T on Y. We make the following assumptions:

- **Assumption 1:** We *assume* that the effect that assessment (A) has on the log of AUDIT-C scores (Y) follows a normal distribution with mean -0.036 and standard deviation 0.019. Note again that we have no access to Group 3 data, thus this assumption would have to be inferred from previous studies.
- **Assumption 2:** Being assessed (A) increases the mediator (M) by 1 unit. Since there are no other paths

[1] Note the difference from Appendix A, where f_M now takes an additional parameter representing whether or not an individual has been assessed at baseline, and the shorthand notation is changed to $M_{t,a}$.

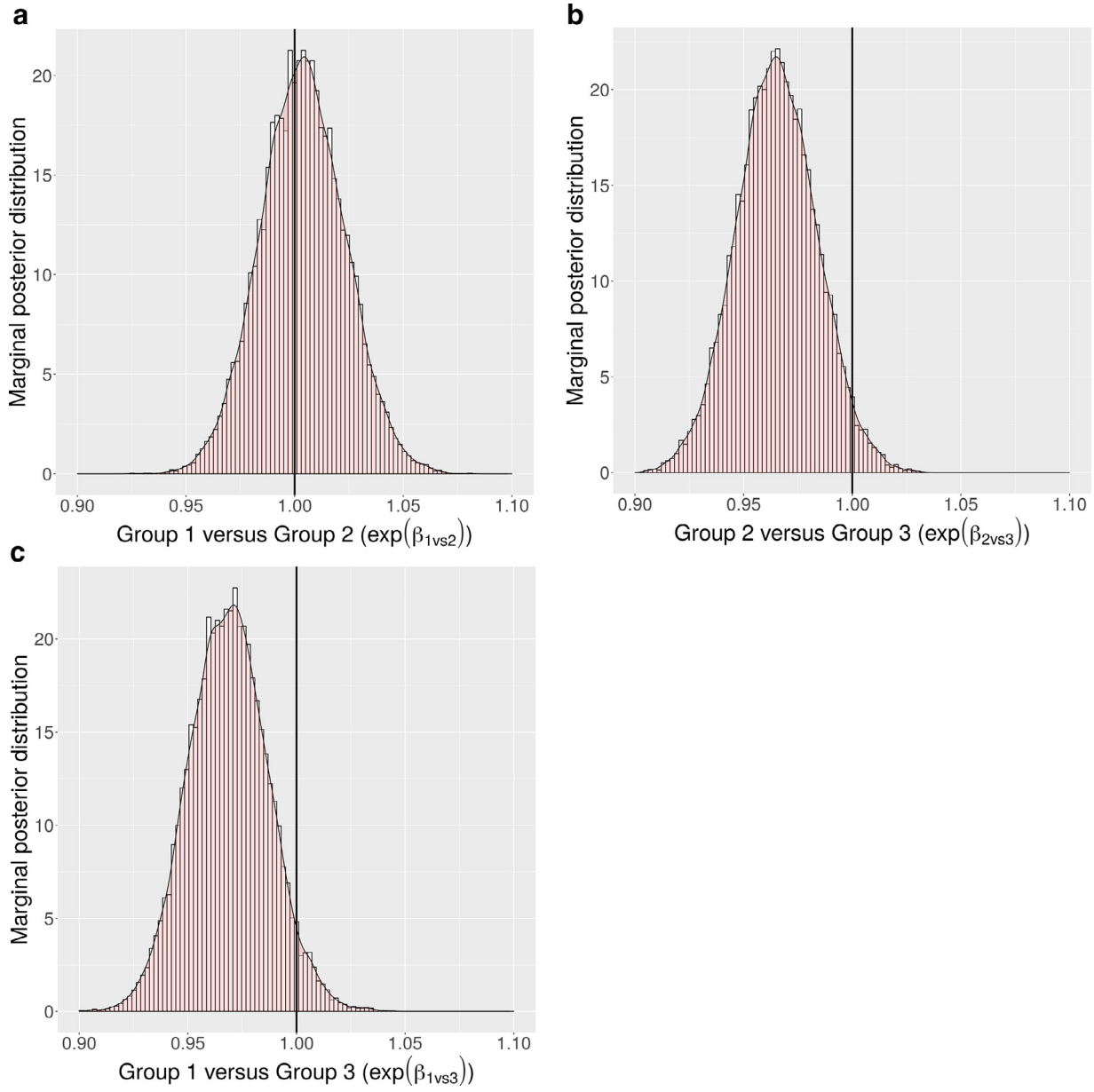

Fig. 1. Compatibility of different geometric mean ratios when contrasting (a) Group 1 and Group 2; (b) Group 2 and Group 3; and (c) Group 1 and Group 3 from the AMADEUS-1 trial (normal linear regression with log transformed AUDIT-C as dependent).

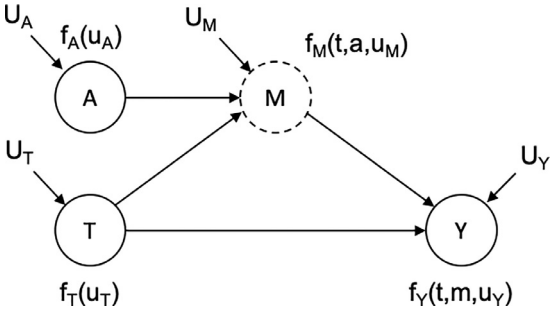

Fig. 2. Causal model describing how assessment (A) and feedback (T) affects AUDIT-C scores (Y) through a mediating factor (M).

from A to Y, the distribution of the effect of a 1 unit increase of M on the log of Y is the same as that of A on Y (i.e., a normal distribution with mean -0.036 and standard deviation 0.019, as per Assumption 1).

- **Assumption 3:** Feedback (T) potentially also increases the mediator (M) by a number of units above the effect of assessment (A), but we are uncertain by how much. We assume that the influence of T on M is positive and follows a half-normal distribution with mean 0 and standard deviation 1.

In summary, we wish to estimate the effects of being allocated to assessment and feedback on AUDIT-C scores, but since all participants have been offered assessment,

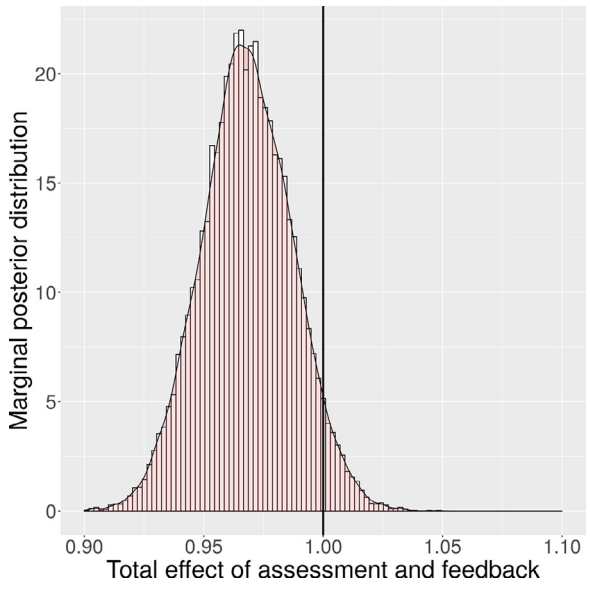

Fig. 3. Total effect of assessment and feedback versus no assessment and no feedback (geometric mean ratio). The effect has been estimated using the model in Figure 2, assuming that we have some external knowledge about the effects of assessment.

we need to use external data to account for this. Using the notation introduced in Appendix A, the effect which we wish to estimate is the total effect without the control group not being assessed: $Y_{1,M_{1,1}} - Y_{0,M_{0,0}}$. This represents the difference when going from the control group (0) to the intervention group (1), while allowing the mediator to track this change in treatment but removing assessment from the control group.

1.4. Estimation and results

The relationship between T and Y is modeled using a multivariable normal linear regression model with log AUDIT-C as the outcome. This regression model is adjusted for M, which we have assumed follows a normal distribution (see Assumption 1 and 2), and thus does not need to be estimated from data. Using Bayesian inference [30], and in particular Markov Chain Monte Carlo methods, we can then estimate the entire model and the effect of particular interest ($Y_{1,M_{1,1}} - Y_{0,M_{0,0}}$). It should be noted that these estimation techniques are flexible, thus the same techniques can be used in cases where outcomes are for instance multinomial or binary. In Appendix B, we have described the model specification in more detail, and included the code in the Stan modeling language [31] used to estimate the model.

In Fig. 3, we depict the distribution of the effect of receiving assessment and feedback versus no assessment and no feedback ($Y_{1,M_{1,1}} - Y_{0,M_{0,0}}$). As the distribution is shifted away from 1, there is some evidence of a marked effect. The mean of the distribution is located at 0.968, suggesting a relative difference in the geometric mean of AUDIT-C of 3.2%. It should be noted that this is essentially identical to our previous direct comparison between Group 1 and Group 3 of the AMADEUS-1 trial, yet Group 3 data were not available to us in this particular analysis.

An examination of the consequences of Assumptions 1 through 3, which make the estimation presented here possible, is warranted. We will defer this examination to the next section, and there incorporate it within a more general discussion about the model itself, implications, and future work.

2. Discussion

In this paper, we have proposed a causal model which can be used to account for research participation effects when analyzing findings from trials. Using data from the AMADEUS-1 study [24,28], we showed that the model can be used to account for unintended effects arising from prerandomization assessment, resulting in a less biased estimate of the effect of the intervention. In this example, we found that the observed overall effect was largely driven by assessment, rather than by feedback.

It is not always possible to mitigate sources of RPEs, nor may it be possible to measure them. The concrete model presented herein is a simple one, but which still allows us to get an estimate of the total effect of assessment and feedback, despite all participants included in the analyses having been assessed. This was possible as we were willing to make assumptions about the assessment effect, yet it will be rare to have access to such an appropriate estimate of the effects of assessment as we had in the exemplar (Assumption 1). However, Assumption 1 could be conservatively attenuated to assume that the assessment effects follow a distribution closer and narrower around the null, which would still help to get an unbiased measure of the total effect in a trial in which effects of assessment were not measured.

We have focused on assessment effects in our exemplar, however, causal models are in general not restricted, thus a number of pre- and postrandomization research processes could be incorporated into the model. The basic RPE construct can be further operationalized by mapping all research activities from initial contact through follow-up, and think through the potential effects of these activities. To aid this identification process, it may prove helpful to consider the issues from the perspective of the participant [1], including consideration of intentions in enrolling in the trial, expectations of the treatment and reactions to not receiving it, and decisions made throughout the trial period [32,33]. As an example of this, our model has been expanded in Fig. 4 with a variable to account for the reading and signing of consent forms (S), affecting the outcome by means of a mediator (D), which could for instance represent the mechanism by which a declaration of commitment may affect behavior [34], as may also be promoted by the intervention, so T has been modeled to affect Y through

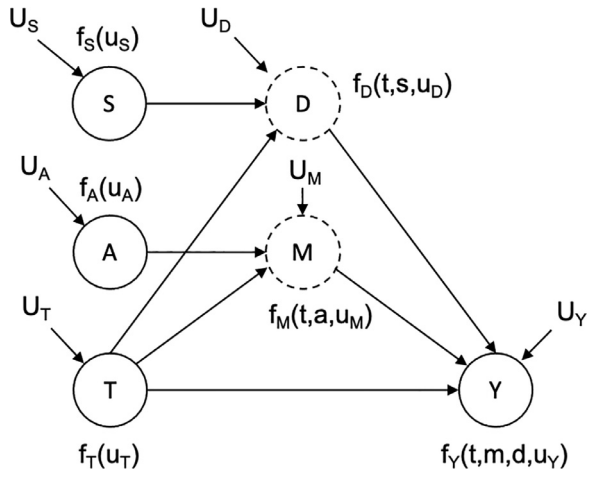

Fig. 4. Causal model describing how assessment (A) and treatment (T) affects outcome (Y) through a mediating factor (M), and reading and signing consent forms (S) and treatment (T) affects outcome (Y) through a mediating factor (D).

D. This now opens up the possibility of further interactions between pre-randomization activities and allocation, which also necessarily requires making more assumptions about the effects' magnitude in order to correct for these potential biases.

In our exemplar, we also made an assumption about the degree to which T influences M (Assumption 3). As long as the focus of inquiry is on the total effect of assessment and feedback on the outcome, then variations of Assumption 3 have little bearing on estimates (as long as the assumption does not in some way violate Assumption 1 or 2). We could use both a wider and narrower distribution to describe how much T influences M, and the total effect estimate would be principally unchanged. This would not be the case if our focus was instead on direct and indirect effects, for example, the natural direct effect of treatment on the outcome, which would then depend on how much T influences M. This should be taken into consideration in studies of mediators, and we will leave for the future work how this could be modeled.

Our final remark on the model presented herein is that our example focused on the total effect of both assessment and feedback. The focus would be different in studies where assessment was not intended to be any part of treatment. Take the case of the use of accelerometers for assessment purposes in a trial of a mobile phone app promoting physical activity. Assuming that the app was intended to be disseminated without the use of an accelerometer, the effect estimate which more accurately represents the effectiveness of the app would be the one where neither the control nor the intervention group have been assessed (i.e., $Y_{1,M_{1,0}} - Y_{0,M_{0,0}}$). This could also be readily estimated using the model proposed.

2.1. Concluding remarks

Given current evidence of RPEs, although component effects may be small, it is unwise to ignore them, particularly in trials of behavioral interventions. It is not so much a question of whether RPEs exist, but rather who they affect, what size they are, and how they vary in different research contexts. Therefore, while assumptions will be necessary to estimate the models proposed herein, it should be noted that the traditional approach (a causal model including only T and Y), implies an assumption that RPEs do not exist. That is a strong assumption.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jclinepi.2021.03.008.

CRediT authorship contribution statement

Marcus Bendtsen: Conceptualization, Methodology, Software, Investigation, Formal analysis, Writing - original draft. **Jim McCambridge:** Conceptualization, Methodology, Investigation, Writing - review & editing.

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