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Clinical Hypothesis

Resilience selection: a grave potential bias in clinical trials

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ABSTRACT

Physiological and psychological resilience has important implications for health, disease and treatment. Resilience is shown to boost treatment compliance as well as response and thereby reduce mortality. We consider the possibility that individuals having lower resilience are more likely to discontinue treatment in response to side effects of a drug. In randomized control trials (RCT) if a considerable proportion of individuals discontinue from the treatment group because of side effects, the average resilience in the remaining treatment group would be greater. As a result, the frequency or severity of adverse outcomes in the treatment group will be smaller than the control even when the drug has no effect. This bias is more likely to be serious for drugs with more frequent and/or serious side effects, but following intention to treat (ITT) protocols with some additional precautions can help in avoiding it. We suggest testable predictions of the resilience selection bias hypothesis along with ways to quantify and correct for the bias in RCTs. Attempts to detect, measure and correct for the resilience selection bias should be considered necessary for realistic evaluation of drug action in a clinical trial. Retrospective studies are more sensitive to RS bias than RCTs and need to be interpreted carefully.

Keywords: Resilience, Randomised control trials, Adverse events, ITT, Correction for resilience, GLP-1RA drugs

INTRODUCTION

Clinical trials are designed with the intention of minimizing potential biases and confounding factors and reflect the effect of treatment in comparison with appropriate control group(s). Randomization, blinding and placebo control are the common practices used to minimize certain kinds of biases. Nevertheless, there are other possible biases that these practices are insufficient to arrest. It is necessary to identify them and design appropriate measures to minimize, if not eliminate, them. It is possible that with appropriate data the biases can be corrected. Further even when biases cannot be eliminated or corrected, the possibility of biases, the contexts in which they arise and their possible misleading effects on the inference need to be made explicit. We will describe one such bias that remains underappreciated so far and has the potential to mislead the inference significantly.

In a typical clinical trial, there is a randomized group under the drug being examined along with a placebo control group. For chronic diseases the treatment duration as well as follow up periods are typically long during which some attrition in the number in both the groups is inevitable. The attrition can be because of reasons unrelated to the treatment. Such drop outs can be considered random and therefore unlikely to introduce a systematic bias in the results. However, the drug is also likely to have some side effects of varying severity and some individuals may quit because they cannot tolerate them. On the other hand, some participants in the placebo group are likely to discontinue because they do not experience any of the expected effects. Such discontinuations are non-random and if sufficient in numbers they can defeat the purpose and effect of randomization used in making the groups. We propose here that a type of potential bias introduced by this non-random element is resilience selection which is not being

adequately considered in designing clinical trials and interpreting results. This needs focussed research towards developing, testing and validating means to avoid, estimate and correct for the bias. We suggest here possible ways to achieve this goal.

THE RESILIENCE SELECTION BIAS HYPOTHESIS

Resilience is an identified factor in patient care and treatment. Although the definition of resilience varies in a context specific manner, a generalized definition is the capacity to achieve positive outcomes despite exposure to significant challenges. Resilience is a significant factor affecting coping with side effects and treatment adherence.[1] Individuals with lower level of resilience are more likely to withdraw from the treatment wing of the clinical trial because of the side effects of the drug. If not complete withdrawal, they are less likely to be adherent to the treatment and are thereby short of fulfilling the treatment target.[2-4] This is less likely to happen in the control group. This creates a potential bias.

In life style interventions, coping with side effects may not be a concern, but the necessary self-control and determination to adhere to lifestyle changes needs resilience. Furthermore, it is also likely that the ones who continue and comply with the intervention boost their resilience further. As a result, although initially the groups might be randomized, after attrition, the treatment group has a greater probability of retaining more resilient individuals than the control group. In comparisons without randomization, if there is prior knowledge about side effects and patients have the freedom to choose the treatment, only more resilient individuals are likely to be in the group with the drug having serious side effects. Any one or more of these factors systematically create a difference in mean resilience of the two groups. Resilience is positively correlated with better treatment response and effectiveness for a number of disorders examined.[5-9] Resilience helps in de-addiction.[10,11] More resilient individuals also have a lower cause specific as well as all-cause mortality.[12,13] Because of resilience selection, even when the treatment has no effect, the treatment group may report smaller frequency and/or severity of the symptoms/adverse events. The inability to appreciate this bias may lead to a misleading belief in the efficacy of the drug (while there may not be any), or lead to overestimation.

TESTABLE PREDICTIONS OF THE RESILIENCE SELECTION BIAS HYPOTHESIS

Having identified a potential source of bias, it is important to consider ways to detect the presence of such bias. How can we examine whether such a bias is present in a given clinical trial and whether it is serious enough to mislead the inference?

A provision for detection and correction of RS bias needs to be there in the design of the clinical trial itself. Indices to quantify resilience have been used in specific contexts.[14-16] If an appropriate context specific index is available, resilience should be estimated for every individual in the treatment as well control groups. After attrition, it is possible to see whether there is systematic difference in the distribution of resilience scores. It is also possible to see whether resilience is significantly related to the incidence or severity of adverse events in both the groups. If it is seen to be significant, an attempt to correct the bias can be made. The limitation of this approach is that currently there is heterogeneity in resilience measurements, and more research is needed to standardize and choose the right index in right context.[17]

If follow up data are collected on the group that discontinued the treatment then qualitatively the presence of RS bias can be detected as well as quantitatively a crude estimate of the possible impact of RS bias on the results can be obtained by the following model. Table 1 assumes that intolerance related discontinuation happens in the treatment group only and there is no non-random discontinuation from the placebo group. Random discontinuations will not affect the model.

Table 1: Variables used in the model.

Numbers recruited	Placebo control arm		Treatment arm	
	Np		Nt	
	Number of individuals	Event rate	Number of individuals	Event rate
Continued	Npc	Rpc	Ntc	Rtc
Discontinued	Npd	Rpd	Ntd	Rtd

We assume that in both the arms some individuals continue with the treatment (denoted by the suffix c) and some discontinue (d) in the placebo (p) and treatment (t) arms. If some proportion of individuals discontinue because of the treatment side effects, we expect Npd/Np<Ntd/Nt.

Assuming that discontinuation from the placebo group is for reasons independent of the treatment, we expect the rate of the adverse event in both to be similar, i.e. Rpc=Rpd. If resilience bias exists then it is expected that Rtd >Rpc. This inequality can be used as a testable prediction of resilience bias as this difference will be independent of the treatment effect. If Rtd is not significantly different from Rpc, the absolute risk reduction (ARR) will be given by Rpc-Rtc. But if significant bias is detected, a corrected ARR can be calculated as

Rpc-(Rtc.Ntc + Rtd.Ntd)/Nt.

This approach has a limitation. A possible reason for discontinuation in the placebo group is not experiencing the expected beneficial effect. This is also a non-random cause of discontinuation whose effects can be different than those of resilience selection. Therefore, even when the proportion of discontinuation from the treatment and placebo groups is not different a difference in the outcomes attributable to RS bias is possible.

An alternative solution is that all results are expressed on the ITT basis, i.e. ignoring whether the treatment was continued or discontinued, compliance or non-compliance, treatment target achieved or unachieved, the group intended to be treated should be considered as treated and compared with the intended control group. Unless attrition is not too large, the difference will remain significant if the treatment is really effective. Nevertheless, it is necessary to report the frequency of adverse events in the discontinued group which has important implications. If the outcome in the discontinued subgroup is not different from the continued treatment group, i.e. Rtd=Rtc the necessity of continuation of treatment can be questioned. If Rtd>Rtc but Rtd=Rpc continued use of drug is justified. But if Rtd>Rpc it indicates resilience selection but bias is avoided when the total incidence is used for statistical analysis.

Even when resilience scores are not maintained and follow up on the drop outs is not available, some attempt to suspect RS bias is possible. Since resilience is a more generalized phenomenon, related to a wide diversity of conditions and treatment effects, one should find lower frequencies in the treatment group for multiple, even unrelated outcomes.[9] It should be easy to monitor this in the groups even at a later stage for trials in which a provision for resilience data is not made from the beginning. Finding favourable effects of the treatment on multiple (but not necessarily all) unintended outcomes is a strong indicator, though not a proof, of RS bias.

If the absolute risk reduction is greater than the absolute attrition difference i.e. Rpc-Rtc>(Ntd/Nt-Npd/Np), the treatment can be safely concluded to be effective. For example, if ARR is 10 % but the attrition difference is only 5%, resilience selection cannot account for this difference and the treatment must be effective. The reverse is not necessarily true, if the attrition difference is greater than ARR, it is not sufficient to conclude that ARR is only a result of resilience selection.

The RS bias hypothesis expects that in the long run, meta-analysis of several drug trials will show a positive correlation between severity of side effects or proportion of treatment drop outs and absolute risk reduction.

Using one or more of the testable predictions it should be possible to estimate how common and how serious the RS bias is across different clinical trials. Also, in future clinical trials it should be possible to maintain the data necessary to detect and even correct RS bias, if any. It is critical that the follow up data of the continued and discontinued groups is separately maintained and made public. Meta-analysis of such data from multiple trials is likely to throw light on the occurrence, frequency and strength of the RS bias.

RE-EXAMINING SOME OF THE RECENT CLINICAL TRIALS FOR THE POSSIBILITY OF RS BIAS

In one recently published clinical trial the data are indicative of RS bias. This trial is not of a drug but of a diet regime that led to significant weight loss as compared to the control group.[18] The trial has certain parallels but also certain differences with the context that we described above. Not involving any drug, there is no question of side effects. However, the diet regime was very strict and we expect only individuals with high resilience to comply with it and reach the weight loss and other targets. The paper gives data on the mortality and diabetic complications among the target achieved and unachieved groups. If inability to achieve the target is at least partly because of low resilience we can use some of the testable predictions above. The main success claimed by Lean et al was that the group that achieved the weight loss target at one year by the treatment had an incidence of major adverse diabetic events (MADE) of 12.8% and the group not achieving the target had 23% (see table S9 in supplementary appendix).[18] This difference was significant and the authors take it as evidence for the success of the treatment. But if we look at the corresponding control group the incidences were 15.9% for target achieved (presumably spontaneously) and 16.1 for non-achieved group and the difference was non-significant. Here we see that the condition Rtd>Rpc is satisfied. This inequality was significant by chi square test (chi sq=8.35, $p<0.05$), but Rtc was not significantly different than the control group (chi sq=1.35, NS). We tested that going by ITT, no difference is seen between the control and intervention. The same pattern was seen for other targets such as HbA1c or remission at 1 year. Wherever Lean et al claim that the target achieved intervention group had significantly reduced incidence, it is seen to be accompanied by increased incidence in the target unachieved group.[18] This is very likely to be a case of resilience selection bias. Such a post hoc analysis was possible because Lean et al separately report the incidence of adverse events in the target achieved and unachieved groups.[18] If such reporting is made mandatory for all clinical trials, detecting, estimating and correcting for RS bias will be possible and clinical trials will become more sound and robust.

The RS bias is also likely to be potentially relevant to the GLP-1 RA drugs that have made a sensational entry as promising anti-obesity drugs. Interestingly apart from weight loss, glucose normalization and diabetic complications, GLP 1RAs are also being claimed to be

effective in preventing a wide variety of conditions.[19-21] Retrospective studies or trial unintended outcome data typically claim significant clinical benefit in preventing conditions including 10 different types of cancers, chronic kidney disease, alcohol and other drug abuse disorders, Alzheimer's disease, cardiovascular outcomes, fertility, seizure and epilepsi, sleep apnea, steatohepatitis, inflammatory bowel disease, type 1 diabetes, and all cause mortality.[20,22-33] The apparent effectiveness of the drugs against widely different end points in retrospective studies is the first suggestion (but not a proof) that RS bias may be at work.

In many of the GLP1 RA trials over 2-to-5-fold greater proportion of participants discontinued in the treatment group as compared to the control.[34-36] Outside RCTs treatment adherence is reported to be less than 50%.[37] The resilience bias is therefore expected to be very strong for GLP 1RA drugs. Comparisons without randomization are most likely to have a strong bias.[21] Many of the clinical trials follow ITT, which should be sufficient to avoid RS bias, but unfortunately these trials have certain other problems in statistical rigor, and broader scope meta-analysis remains inconclusive.[38-40] Also, the data in the public domain does not give us the incidence of adverse events specific to the discontinuation group. Potentially it should be still possible to test the possibility of RS bias and estimate its strength if these data are made public. In the absence of such efforts the results of GLP 1RA clinical trials with respect to arresting cardiac, renal and other complications should be considered inconclusive. The RS bias principle needs to be applied to many other clinical trials for long term treatment aimed at preventing complications in chronic conditions. In the long run it should be possible to include the necessary corrections in clinical trial design itself and methods to detect, estimate and correct for the bias designed, standardized and validated.

Limitations of the concept

As of today, the definition of resilience, the possible psychological and physiological components, mechanisms and pathways are not clearly known.[41,42] But so is the case of placebo. The mechanisms of placebo effect are also not clearly known, but still the effects are demonstrable and accepted in designing the trial protocols as a routine. Resilience selection bias needs to be incorporated as another necessary routine in clinical trials to make the inferences more robust.

The second limitation is that the indices for resilience measurement also need to be refined and validated for context of the underlying types of disorders, treatment effect and side effects. Some refinement of statistical methods and appropriate validation is also needed on application of the RS bias correction. All this needs research inputs but such developments are certainly possible in near future and crucial for increasing the reliability of clinical trials.

CONCLUSION

If side effects or difficulties in continuing the treatment is the cause of discontinuation from the trial then the mean resilience of individuals in the treatment group is likely to increase. Since resilience can affect treatment response and mortality, selection for higher resilience can result into lower incidence and severity of adverse events in the treatment arm even when the treatment has no effect. This bias needs to be recognized, detected and corrected if possible. If the follow up data on the continued and discontinued individuals in both control and treated arms is separately reported, it is possible to detect and correct the bias. Clinical trial designs hence forth need to incorporate this principle for more sound and robust inferences.

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