In the pursuit of truth, we should not prioritize correlational over causal evidence

Article in Psychotherapy Research · January 2017
DOI: 10.1080/10503307.2016.1249434

CITATIONS
0

READS
406

2 authors:

Ioana Alina Cristea
Babeş-Bolyai University
58 PUBLICATIONS 221 CITATIONS
SEE PROFILE

Pim Cuijpers
VU University Amsterdam
732 PUBLICATIONS 23,656 CITATIONS
SEE PROFILE

Some of the authors of this publication are also working on these related projects:

Which patients benefit specifically from short-term psychoanalytic psychotherapy for depression? An individual patient data meta-analysis. View project

NESDA : The Netherlands Study of Depression and Anxiety View project

All content following this page was uploaded by Ioana Alina Cristea on 27 November 2016.

The user has requested enhancement of the downloaded file. All in-text references underlined in blue are added to the original document and are linked to publications on ResearchGate, letting you access and read them immediately.
In the pursuit of truth, we should not prioritize correlational over causal evidence

Ioana A. Cristea & Pim Cuijpers

To cite this article: Ioana A. Cristea & Pim Cuijpers (2017) In the pursuit of truth, we should not prioritize correlational over causal evidence, Psychotherapy Research, 27:1, 36-37, DOI: 10.1080/10503307.2016.1249434

To link to this article: http://dx.doi.org/10.1080/10503307.2016.1249434

Published online: 24 Nov 2016.

Submit your article to this journal

Article views: 23

View related articles

View Crossmark data
COMMENTARY

In the pursuit of truth, we should not prioritize correlational over causal evidence

IOANA A. CRISTEA & PIM CUIJPERS

1Department of Clinical Psychology and Psychotherapy, Babes-Bolyai University, Cluj-Napoca, Romania; 2Department of General Psychology, University of Padova, Padova, Italy; 3Department of Clinical Psychology, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands & 4EMGO Institute for Health and Care Research, Amsterdam, The Netherlands

(Received 29 July 2016; revised 15 August 2016; accepted 28 September 2016)

Wampold et al. (2017) offer a compelling critique of three recent meta-analyses maintaining superior effects of cognitive behavioral therapy (CBT) over other psychotherapies, for psychopathology in general and for social phobia. The paper illustrates how easy it is to make basic errors in meta-analyses, and that the results of such meta-analyses can, like any other type of research, be interpreted in different ways. It is also in line with a series of papers and other publications led by the author advocating the notion that all psychotherapies have comparable effects and work mainly through non-specific mechanisms that all therapies have in common (Wampold, 2001). However, while the ideal of identifying causal ingredients is in itself of value, there are several problems with its implementation. For one, the notion that these general ingredients exist in the first place and that they are the driving force behind change in clinically relevant outcomes still needs to be empirically demonstrated. This is a hypothesis in itself and it needs to be falsified on its own. In other words, the fact that evidence is scarce for specific differences between psychotherapies, both in terms of efficacy and in terms of purported mechanisms of change (Cristea et al., 2015), simply means that the null hypothesis “there are no specific differences” cannot be rejected. However, stating that all therapies work by some common ingredients or mechanisms is another, new, hypothesis, which needs its own empirical substantiation. This brings us to the next point, regarding the kind of evidence that would be necessary to falsify this claim. As we have argued elsewhere (Cuijpers, 2013), most of the evidence that has been offered in support of this assertion is simply correlational and cannot form the basis for causal inferences. But we have also hinted to an even deeper problem: in practical terms, studying treatment mechanisms with designs that are as sound in terms of clinical validity as that of the randomized controlled trial is simply not feasible at the moment.

In their article, Wampold et al. recommend that “the field needs to identify the ingredients of psychotherapy responsible for change.” While the authors restrain from giving examples of such ingredients, it can be assumed that they have common factors in mind, a research area the lead author himself championed and expanded (Wampold, 2001). However, while the ideal of identifying causal ingredients is in itself of value, there are several problems with its implementation. For one, the notion that these general ingredients exist in the first place and that they are the driving force behind change in clinically relevant outcomes still needs to be empirically demonstrated. This is a hypothesis in itself and it needs to be falsified on its own. In other words, the fact that evidence is scarce for specific differences between psychotherapies, both in terms of efficacy and in terms of purported mechanisms of change (Cristea et al., 2015), simply means that the null hypothesis “there are no specific differences” cannot be rejected. However, stating that all therapies work by some common ingredients or mechanisms is another, new, hypothesis, which needs its own empirical substantiation. This brings us to the next point, regarding the kind of evidence that would be necessary to falsify this claim. As we have argued elsewhere (Cuijpers, 2013), most of the evidence that has been offered in support of this assertion is simply correlational and cannot form the basis for causal inferences. But we have also hinted to an even deeper problem: in practical terms, studying treatment mechanisms with designs that are as sound in terms of clinical validity as that of the randomized controlled trial is simply not feasible at the moment.

For example, we recently showed that more than one hundred comparative outcome trials, directly comparing two or more psychotherapies for adult depression have been published (Cuijpers, 2016). However, all trials are heavily underpowered and do not even come close to having sufficient power for detecting clinically relevant differential effect sizes. Moreover, the quality of the majority of these was subpar, so even meta-analyses of these trials cannot
say with any degree of certainty whether there really are differences between psychotherapies or not. We also showed in another systematic review that dismantling and other component studies in depression are even more severely underpowered (Cuijpers, Cristea, Karyotaki, Reijnders, & Hollon, 2010). Of the 19 identified component studies of psychotherapies for depression, only one had sufficient power to detect a relevant differential effect between the treatment with the added component (emotional regulation) and the standard therapy without the component (Berking, Ebert, Cuijpers, & Hofmann, 2013). Incidentally, this study also found a significant benefit of the added component over the standard therapy. None of the other trials had enough power to detect an effect size smaller than $g = 0.49$, and more than half of the trials only had sufficient power to detect an effect size smaller than $g = 1.0$.

These reviews demonstrate that the empirical evidence for the contention that all therapies are equally effective is not that strong at all and may be related to the low quality and lack of statistical power of most studies in this field. As we stated, the present state of the field is such that the only thing we can affirm with some degree of certainty is that the null hypothesis “there are no specific differences” cannot be rejected. However, this does not imply that this hypothesis can be considered proven. It also does not mean that an alternative specific hypothesis about common mechanisms or ingredients is most likely true.

In conclusion, we commend Wampold and colleagues for their painstaking critical analysis. Many of their arguments are valuable food for thought and will undoubtedly be much discussed. It was also disquieting to read about refusals of data or protocol sharing, particularly from lead CBT researchers. It is our hope that these claims will be further explored and answered. We stand in full agreement with the authors in affirming that the current balance of evidence indicates that it is unlikely that CBT is more effective than other psychotherapies and that this notion probably holds for psychopathology in general and for many specific disorders. In fact, it needs to be said that the meta-analyses critically discussed by Wampold et al. (2017) add to an array of others that did not find added benefits of CBT over other psychotherapies (Barth et al., 2013; Cuijpers et al., 2013). Nevertheless, the converse contention that hence all therapies must work through universal mechanisms or common ingredients has yet to be supported with internally valid empirical evidence.

Funding

Ioana A. Cristea was supported for this work by a grant of the Unitatea Executiva pentru Finantarea Invatamantului Superior, a Cercetarii, Dezvoltarii si Inovarii (UEFISCDI), Romanian National Authority for Scientific Research and Innovation, CNCS – UEFISCDI, project number PN-II-TE-2014-4-1316 awarded to Ioana A. Cristea.

ORCID

Ioana A. Cristea © http://orcid.org/0000-0002-9854-7076
Pin Cuijpers © http://orcid.org/0000-0001-5497-2743

References


