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Annual Review of Clinical Psychology

The Role of Common Factors in Psychotherapy Outcomes

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psychotherapy, outcome research, mechanisms of change, common factors, meta-analysis

Abstract

Psychotherapies may work through techniques that are specific to each therapy or through factors that all therapies have in common. Proponents of the common factors model often point to meta-analyses of comparative outcome studies that show all therapies have comparable effects. However, not all meta-analyses support the common factors model; the included studies often have several methodological problems; and there are alternative explanations for finding comparable outcomes. To date, research on the working mechanisms and mediators of therapies has always been correlational, and in order to establish that a mediator is indeed a causal factor in the recovery process of a patient, studies must show a temporal relationship between the mediator and an outcome, a dose–response association, evidence that no third variable causes changes in the mediator and the outcome, supportive experimental research, and have a strong theoretical framework. Currently, no common or specific factor meets these criteria and can be considered an empirically validated working mechanism. Therefore, it is still unknown whether therapies work through common or specific factors, or both.

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Contents

INTRODUCTION	208
COMMON FACTORS: DEFINITION AND MODELS	209
DO ALL THERAPIES HAVE COMPARABLE EFFECTS?	211
What Can We Learn from Meta-Analyses of Comparative Outcome Studies?	211
How Can Differences Between Therapies Be Explained by the Common Factors Model?	214
Can Comparable Effects Be Seen as Evidence for the Common Factors Model?	215
Alternative Explanations for Finding Comparable Outcomes	216
COMPONENT STUDIES AND THE COMMON FACTORS MODEL	217
HOW CAN SPECIFIC AND NONSPECIFIC FACTORS BE EXAMINED?	218
A Model for the Process of Clinical Change	218
Research Methods for Examining the Process of Clinical Change	219
Research on Specific and Nonspecific Components of Therapies	220
EVIDENCE FOR SELECTED COMMON FACTORS	221
The Therapeutic Alliance	221
Other Common Factors	222
Specific Factors	222
RECOMMENDATIONS FOR FUTURE RESEARCH	224
CONCLUSIONS	225

INTRODUCTION

It is well established that psychotherapies can effectively treat mental disorders, including depression (Barth et al. 2013, Mohr et al. 2014), anxiety disorders (Cuijpers et al. 2014a, 2016a; Mayo-Wilson et al. 2014; Pompoli et al. 2018), posttraumatic stress disorder (PTSD; Cusack et al. 2016, Gerger et al. 2014), obsessive-compulsive disorder (Olatunji et al. 2013), psychotic disorders (Velthorst et al. 2015), eating disorders (Godfrey et al. 2015), bipolar disorder (Chatterton et al. 2017, Oud et al. 2016), and personality disorders (Cristea et al. 2017). The effects of these therapies have been demonstrated in randomized controlled trials (RCTs). From a scientific perspective, these trials have a strong design, and when positive effects from a therapy are found, this can be considered strong evidence that a therapy is indeed effective. However, showing that a therapy is effective in treating a mental disorder does not provide evidence about how a therapy works (Kazdin 2007). As we see later in this review, showing how a therapy works is much more complicated, requires a whole series of studies and experiments, and will never have the strength of evidence of RCTs showing that a therapy works.

The consequence of this complexity of showing how psychotherapies work is that little is known about the mechanisms of change of these therapies. Although there are many different kinds of therapy, and most therapies were developed with a clear theoretical explanation of how the therapy is supposed to bring about change in the patient, the actual scientific knowledge about these mechanisms is limited. This lack of knowledge has resulted in different views and models of how therapies work—the so-called mechanisms of change or action—but none of these models has sufficient empirical support. The majority of these models explain the effectiveness of therapies through specific effects. Specific effects are those that are realized through the approach of the therapy. For example, cognitive therapies are assumed to realize their effects through changing maladaptive cognitions in patients, and behavioral therapies realize their effects through changing

maladaptive behaviors, both of which are in keeping with the theoretical models these therapies are based on.

However, there is an alternative model that says that the effects of therapies are not or not only realized by these specific effects, but instead are realized predominantly through common factors. These common factors—also often called nonspecific or universal factors—are those factors that all therapies have in common, such as the alliance between the patient and the therapist, expectations, and a rationale that helps patients understand why they have problems and what can be done about them.

In this review, we first describe the most important theoretical models of common factors and how they are assumed to work. Then we focus on comparative outcome research and component studies because these types of studies and the findings that all therapies seem to have comparable effects were the starting point for the common factors model. We discuss the research that is needed to find out how psychotherapy works. Then we discuss the most important common factors, including the therapeutic alliance, empathy, and expectations, as well as the evidence supporting these factors as the active ingredients of therapies. We end by discussing the most important findings and future directions for examining common factors and other potential mechanisms of change.

COMMON FACTORS: DEFINITION AND MODELS

The notion that all therapies work through common factors was first introduced by Saul Rosenzweig (1907–2004) in 1936 (Rosenzweig 1936). He observed that all therapies resulted in comparable outcomes, and based on this observation, he suggested that they probably worked through factors that were common to them all. He also used an analogy to the Caucus-race in Lewis Carroll's *Alice's Adventures in Wonderland*, in which the Dodo suddenly announces the end of a race and states that, “*Everybody* has won, and all must have prizes” (Carroll [1865] 1998, p. 34). This Dodo Bird Verdict, indicating that all therapies have comparable effects and do not significantly differ from one another, has become a much-discussed issue and the subject of heated debate in the psychotherapy literature (Budd & Hughes 2009, Cuijpers 1998, Luborsky et al. 2002, Marcus et al. 2014).

Since Rosenzweig's statement, several models of common factors have been developed. The most influential early model was developed by Jerome Frank, who wrote a book in the early 1960s which became the basis of the modern views on common factors. In his book *Persuasion and Healing: A Comparative Study of Psychotherapy* (Frank 1961), he described four nonspecific factors that were common to all therapies and were assumed to be the working elements of these therapies: a functioning relationship between patient and therapist, a rationale that provides credibility to the treatment being delivered, certain procedures or rituals that are provided in a structured manner, and a healing context or setting.

The best developed and most modern common factors model, however, is the contextual model (Wampold 2001, 2015; Wampold & Imel 2015). This model is intended to be an alternative to the medical model in which therapies are supposed to work through specific ingredients that are “purportedly beneficial for particular disorders due to remediation of an identifiable deficit” (Wampold 2015, p. 270). In the contextual model, a patient and a therapist first have to create a basic bond to work together. After the establishment of this initial bond, therapy is hypothesized to work through three pathways. The first pathway is the real relationship, defined as “the personal relationship between therapist and patient marked by the extent to which each is genuine with the other and perceives/experiences the other in ways that benefit the other” (Gelso 2014, p. 119). This relationship is assumed to provide the patient with a connection to a caring and empathic person.

Table 1 Overview of common factors in psychotherapy outcomes^a

Common factors		
Support	Learning	Action
Catharsis	Advice	Behavioral regulation
Identification with therapist	Affective experience	Cognitive mastery
Mitigation of isolation	Assimilating problematic experiences	Encouragement to face fears
Positive relationship	Cognitive learning	Taking risks
Reassurance	Corrective emotional experience	Mastery efforts
Release of tension	Feedback	Modeling
Structure	Insight	Practice
Therapeutic alliance	Rationale	Reality testing
Active participation of both therapist and client	Exploration of internal frame of reference	Experiencing success
Therapist expertise	Changing expectations of personal effectiveness	Working through
Therapist warmth, respect, empathy, acceptance, genuineness		
Trust		

^aTable adapted with permission from Lambert & Ogles (2004).

This connection is assumed to be healing in itself, especially for patients with suboptimal social relationships. The second pathway is through a patient's expectations or hope. Therapies provide an explanation or rationale for how the patient developed the mental disorder and what can be done about it. The therapy provides patients with the means to cope with their problems. Therapies give patients hope that they are capable of doing the tasks necessary to complete therapy, and when these tasks are completed, the problems will be able to be coped with. The third pathway consists of the specific ingredients of therapies. These specific ingredients create expectations in patients (thereby activating pathway two) and also produce some salubrious actions (Wampold 2015). These actions differ across therapies; for example, they may improve a patient's social relationships (interpersonal psychotherapy), aid the patient in adopting helpful ways of thinking (cognitive behavioral therapy), or help a patient become more accepting toward him- or herself (acceptance and commitment therapy). It is not assumed that these specific ingredients exert a direct effect through the medical model by repairing an apparent deficit, but rather that, in general, they stimulate healthy actions that are beneficial to patients.

The term common factors can be conceptualized in different ways. Lambert & Ogles (2004) gave an overview of the different common factors that can be derived from the literature (Table 1). As can be seen in Table 1, the factors are grouped into categories of support, learning, and action. These categories are based on the sequential order of change that is hypothesized to happen during psychotherapy. For example, a strong therapeutic alliance can lead to a corrective emotional experience, which then leads to patients facing their fears or dealing with their problems (Huibers & Cuijpers 2015). However, this sequential process of change is a theoretical assumption that has yet to be confirmed by empirical research. It is beyond the scope of this review to describe all common factors in detail, but in the section Evidence for Selected Common Factors, we discuss the research on the most important common factors, including the therapeutic alliance between a therapist and a patient. We also describe how complicated it is to empirically prove that these factors are indeed directly causing change in patients.

In 1992, a pie chart was published that displayed how much of the change in patients receiving psychotherapy could be attributed to common factors, expectations, or placebo effects, and to specific factors, as well as to factors outside the therapy (Lambert 1992). This chart indicated that 30% of the change can be attributed to common factors and 15% to specific factors (and 40% to extratherapy factors and 15% to the placebo effect). Although this pie chart appeals to many clinicians due to its simplicity (echoing the sentiment that “it’s all about the relationship”), and is often referred to as if these were empirical estimates, the numbers in the chart are in fact no more than rough estimates of correlates based on impressions from the literature. There is no empirical evidence to support the percentages given in the chart. There is one meta-analysis of nondirective supportive counseling in which an attempt was made to estimate improvement rates under control conditions (change through extratherapy and placebo factors), additional improvement in counseling (the common factors), and the superior effects of specific therapies above counseling (the specific effects) (Cuijpers et al. 2012). The estimates found in this study did not differ considerably from the estimates of Lambert (1992), with extratherapeutic factors being responsible for about one-third of the improvement (33.3%), nonspecific factors responsible for about half (49.6%), and specific factors responsible for the remaining sixth and smallest share (17.1%). However, these findings should be considered with great caution because this meta-analysis also found that the difference between counseling and specific therapies was heavily influenced by a researcher’s allegiance, and the risk of bias in the majority of the included studies was considerable (Cuijpers et al. 2012). This means that true estimates of the contribution of specific and nonspecific factors cannot be based on this meta-analysis either or on any other meta-analysis, as a matter of fact.

DO ALL THERAPIES HAVE COMPARABLE EFFECTS?

The discussion about whether therapies work through specific or nonspecific factors started with the Dodo Bird Verdict, the observation that all (or most) therapies have comparable effects. In this section, we summarize the knowledge about whether all therapies do indeed have comparable effects by reviewing meta-analyses of comparative outcome trials.

The best way to examine whether two types of therapy have comparable effects is to conduct trials in which patients are randomized to one of the two therapies and then examine whether the improvement in participants is comparable between the two therapies. Many such comparative outcome trials have been conducted during the past decades, and dozens of meta-analyses have pooled the results of these trials. For proponents of the common factors model, the comparable effectiveness of therapies is one of the cornerstones that is supposed to prove their claim that all therapies work through common factors. They would predict that meta-analyses of these comparative trials indicate no significant differences between therapies. However, proponents of specific effects assume that such differential effects are entirely possible but not necessary in all cases. So who is right?

In this section, we first give an overview of the results of meta-analyses that have been conducted during the past 10 years that compared two types of therapy. As we show, some of these meta-analyses indeed point toward comparable effect sizes for the examined therapies, but others do not. Therefore, we describe how these results can be interpreted, why proponents of the common factors model argue that the significant differences found in meta-analyses are only artifacts, and whether these results can be seen as evidence for the common factors model.

What Can We Learn from Meta-Analyses of Comparative Outcome Studies?

According to the common factors model, meta-analyses of comparative studies should first find no, or only small and nonsignificant, differences because all therapies are assumed to have comparable

effects. However, in the common factors model it is also expected that there is no statistical heterogeneity. Heterogeneity is the variability in effect sizes that is found in a meta-analysis. It is present when the observed effect sizes are more different from each other than what would be expected due to chance (random error) alone (Cuijpers 2016). The common factors model assumes that all therapies have comparable effects, so heterogeneity should be low because there is no variability in effect sizes that can be caused by differences between therapies. Heterogeneity is usually measured in meta-analyses through I^2 , which is an indication of heterogeneity in percentages (Higgins et al. 2003). An I^2 of 25% indicates low heterogeneity, 50% indicates moderate, and 75% indicates high.

In **Table 2**, we summarize the results of meta-analyses of comparative outcome studies. We have focused on meta-analyses of broad categories of mental disorders (depression, anxiety disorders, PTSD, obsessive-compulsive disorder, psychosis, and bipolar, eating, and borderline personality disorders) that reported standardized mean differences (SMD, also called Cohen's d or Hedges's g), were conducted during the past 10 years, and included at least 10 comparisons between two therapies.

As can be seen from **Table 2**, several meta-analyses do indeed find nonsignificant differential effects between therapies with low heterogeneity. For example, cognitive behavioral therapy (CBT) versus supportive therapy for depression has a small, nonsignificant effect size (SMD = 0.10) and low heterogeneity ($I^2 = 27%$) (Cuijpers et al. 2013). Other meta-analyses, however, find a significant differential effect and higher levels of heterogeneity. For example, eye movement desensitization and reprocessing (EMDR) has been found to be significantly more effective than CBT, with a differential effect size of SMD = -0.43 and with high heterogeneity ($I^2 = 75%$) (Chen et al. 2015).

Recently, a number of network meta-analyses of the effects of psychotherapies have been published (Barth et al. 2013, Gerger et al. 2014, Kriston et al. 2014, Linde et al. 2015, Mayo-Wilson et al. 2014, Pompoli et al. 2018, Skapinakis et al. 2016, Slade et al. 2018). Network meta-analyses include multiple comparisons in one analysis, and they include both direct evidence of the effects of therapies (e.g., from studies directly comparing therapy A with therapy B) and indirect evidence (e.g., from studies comparing therapy A with control group C, and studies comparing therapy B with the same control group C). If both A and B have comparable effect sizes compared with control condition C, then that strengthens the evidence that A and B are indeed comparable. Therefore, network meta-analyses make optimal use of all available evidence and are superior to conventional meta-analyses from that perspective. It is beyond the scope of this article to comprehensively review these studies, but overall they show the same pattern as conventional meta-analyses. For some comparisons, significant differential effects for therapies are found, and nonsignificant differences are found for others. For example, Mayo-Wilson and associates (2014) found significantly better effects for CBT compared with psychodynamic therapies for treating social anxiety disorders [$d = -0.56$; 95% confidence interval (CI), -1.03 to -0.11], but no significant difference between psychodynamic therapy and exposure and social skills training. Barth and associates (2013) did not find a significant difference between CBT and behavioral activation therapy for treating depression, but they did find a significant difference between interpersonal psychotherapy and supportive counseling ($d = -0.16$; 95% CI, -0.33 to 0.22).

One problem with the level of heterogeneity found in meta-analyses is that this estimate has its own level of uncertainty (measurement error); therefore, calculating 95% confidence intervals around I^2 is recommended (Ioannidis et al. 2007). Usually these confidence intervals are wide when the number of studies is small. So in our example of the meta-analysis comparing CBT with supportive therapy, we found low heterogeneity ($I^2 = 27%$), but the confidence interval ranged from 0% to 59%. This means that we actually do not know what the true level of heterogeneity is, and it could range from zero to a moderate or high level. Therefore, the second hypothesis of

Table 2 Meta-analyses of comparative outcome trials of psychotherapies^a

Study	Disorder	Therapy	Control	<i>k</i>	SMD	95% CI	<i>I</i> ²	95% CI ^c
Baardseth et al. 2013	Anxiety disorders	CBT	Other therapies	13	0.14	−0.08 to 0.35	69	
Bisson et al. 2013	PTSD	TFCBT	Other therapies	10	0.48 ^b	0.14 to 0.83	69	29 to 82
Braun et al. 2013	Depression	CBT	Other therapies	41	−0.01	−0.10 to 0.08	5	0 to 68
		Behavior activation	Other therapies	16	−0.08	−0.30 to 0.14	52	17 to 87
		Psychodynamic	Other therapies	11	0.19	−0.01 to 0.40	44	0 to 91
		IPT	Other therapies	10	−0.09	−0.30 to 0.13	39	0 to 88
		Supportive therapies	Other therapies	17	0.14	0.00 to 0.29	22	0 to 71
Chen et al. 2015	PTSD	CBT	EMDR	11	−0.43 ^b	−0.86 to −0.01	75	49 to 85
Cuijpers et al. 2018	Depression	PST	Other therapies	12	0.18 ^b	0.01 to 0.35	27	0 to 63
Cuijpers et al. 2016b	Depression	IPT	Other therapies	14	0.06	−0.14 to 0.26	52	0 to 72
Cuijpers et al. 2013	Depression	CBT	Supportive therapies	16	0.10	−0.06 to 0.25	27	0 to 59
Cuijpers et al. 2012	Depression	NDST	Other therapies	30	−0.20 ^b	−0.32 to −0.08	30	
Driessen et al. 2015	Depression	Psychodynamic	Other therapies	15	−0.25 ^b	−0.49 to −0.02	63	24 to 77
Grenon et al. 2018	Eating disorders	CBT	Other therapies	11	0.31 ^b	0.16 to 0.46	0	
Kivlighan et al. 2015	Anxiety, depression, PDs, bulimia, anorexia, PTSD	Psychodynamic	Other therapies	20	−0.05	−0.18 to 0.07	15	
Leichsenring & Rabung 2011	Chronic depression and anxiety, PDs, eating disorders	Long-term psychodynamic	Other therapies	10	0.54 ^b	0.26 to 0.83	23	0 to 63
Shinohara et al. 2013	Depression	BT	Other therapies	18	−0.03	−0.20 to 0.15	0	0 to 43
Steinert et al. 2017	Depression, anxiety, eating disorders, PDs, substance dependence, PTSD	Psychodynamic	Other therapies	23	−0.15 ^b	−0.23 to −0.08	0	0 to 40
Turner et al. 2014	Psychotic disorders	CBT	Other therapies	22	0.16 ^b	0.04 to 0.28	12	0 to 48
		Befriending	Other therapies	11	−0.37 ^b	−0.60 to −0.13	53	0 to 75
		Cognitive remediation	Other therapies	11	0.13	−0.05 to 0.31	32	0 to 65
		Social skills training	Other therapies	16	0.06	−0.17 to 0.28	67	38 to 79
		Supportive counseling	Other therapies	17	0.00	−0.21 to 0.22	60	23 to 75

Abbreviations: BT, behavioral therapy; CBT, cognitive behavioral therapy; CI, confidence interval; EMDR, eye movement desensitization and reprocessing; IPT, interpersonal psychotherapy; NDST, nondirective supportive therapy; PD: personality disorder; PST, problem-solving therapy; PTSD, posttraumatic stress disorder; SMD, standardized mean difference; TFCBT, trauma-focused cognitive behavioral therapy.

^aWe conducted a systematic search for comparative meta-analyses in PubMed, PsycINFO, and the Cochrane Database of Systematic Reviews in which we combined search terms (key words and text words) for psychotherapy, mental disorders, and words indicating comparative studies. We selected conventional meta-analyses comparing two types of psychotherapy that reported the SMD, 95% CI, and *I*² (heterogeneity) and that had been published since 2010 (search date, June 19, 2018).

^b*p* < 0.05.

^cEmpty cells in this column indicate that the 95% CI of *I*² was not given and could not be reproduced based on the published data.

the common factors model (i.e., there should be low levels of heterogeneity in meta-analyses of comparative trials) is difficult to validate, especially through meta-analyses of small numbers of studies.

Some meta-analyses do not focus on the comparative effects of two therapies but instead focus on any trial that compares any kind of therapy with any other kind of therapy (Marcus et al. 2014,

Wampold et al. 1997). The problem with this kind of meta-analysis is that the direction of the outcome is not clear. Suppose that CBT is compared with psychodynamic therapy in a trial, and psychodynamic therapy is found to be superior. Is that a positive effect size for psychodynamic therapy or a negative effect size for CBT? Both options are equally valid. This is not a problem when, for example, psychodynamic therapy is compared with any other therapy because then one can say that a positive effect size indicates that psychodynamic therapy is superior to other therapies. But if all comparisons for any therapy are included in a meta-analysis, one can choose both directions, positive for psychodynamic or negative for CBT, and there is no way of resolving the issue. In these types of meta-analysis, this problem is solved by giving all effect sizes a positive direction (i.e., it is assumed that there is no difference anyway, so the pooled effect gives only the upper limit of the differential effect) or by randomly assigning the direction of the effect size across the studies. The original study that developed this method of meta-analysis showed no significant difference between therapies (Wampold et al. 1997). However, since then, so many more specific meta-analyses have been conducted that directly compare two therapies with each other that an overall meta-analysis using this methodology would be a huge enterprise and, to the best of our knowledge, this has not been done.

How Can Differences Between Therapies Be Explained by the Common Factors Model?

Proponents of the common factors model say that when a meta-analysis shows a significant differential effect size and has a high level of heterogeneity, this is the result of artifacts of the included comparative trials, and it does not represent true differences between treatments. They give several reasons for this.

One important reason why some comparative outcome studies may find differences between therapies is that some therapies are not bona fide therapies. These therapies are “designed to fail” and have been called “intent-to-fail” treatments (Westen et al. 2004). Researchers working from a specific model of therapy (cognitive, behavioral, psychodynamic, interpersonal, or any other specific model) want to compare their therapy with another therapy to show that their therapy works. But often, this control therapy is not really intended to have a therapeutic effect and only mimics the therapeutic situation, without meeting the criteria for a bona fide therapy. Bona fide therapies can be defined as therapies that are “based on psychological principles, [are] offered to the psychotherapy community as viable treatments (e.g., through professional books or manuals),” and are “delivered by trained therapists” (Wampold et al. 1997, p. 205). Proponents of the common factors model would say that only comparisons of bona fide therapies can give a fair indication of whether therapies do indeed differ significantly from each other. For example, in a meta-analysis of trials comparing CBT for depression with other therapies, it was found that CBT was superior to other therapies (Gloaguen et al. 1998). However, when the other therapies were separated into bona fide and non-bona fide therapies, it was found that CBT was no more effective than other bona fide therapies (Wampold et al. 2002).

In many of the comparisons in **Table 2**, the only therapies compared are those that can be considered bona fide therapies. However, the decision about whether a therapy is bona fide requires a judgment from expert raters, and these judgments have not been made in all meta-analyses. So in meta-analyses comparing a specific type of therapy with the category all other therapies, it is possible that this category includes non-bona fide therapies. This could explain why some meta-analyses find superior effects for a therapy when it is compared with the category other therapies. However, this cannot explain why, for example, EMDR has been found to be more effective than CBT in treating PTSD (Chen et al. 2015) because both are bona fide therapies.

Another important reason why comparative outcome studies may find differences between therapies and still fit into the common factors model is known as researcher allegiance. This can be defined as the “belief in the superiority of a treatment [and] it usually also entails a belief in the superior validity of the theory of change that is associated with the treatment” (Leykin & DeRubeis 2009, p. 55). The assumption is that researchers with an allegiance toward one type of therapy are inclined to design or interpret the results of a comparative study in such a way that their preferred therapy is found to be superior to other therapies. This can be seen as a specific type of intellectual conflict of interest (Cristea & Ioannidis 2018). Indeed, there is evidence that researcher allegiance can have an impact on the outcomes of comparative outcome studies. A systematic review of 29 meta-analyses reporting on researcher allegiance found that the correlation between the outcome of an intervention and allegiance was $r = 0.26$, which can be seen as a moderate effect size (corresponding to $SMD = 0.54$) (Munder et al. 2013).

It is not clear how allegiance works. The assumption is that it favors the preferred therapy over the other therapy, for example in terms of the number of sessions that participants undergo, the training of therapists, and supervision. Another possibility, for which some support exists, is that allegiance works through the risk of bias. Risk of bias refers to the validity of a trial or the weak spots of a trial (Cuijpers & Cristea 2016). It is usually assessed with the Cochrane Risk of Bias assessment tool (Higgins et al. 2011). The tool rates whether the randomization has been done correctly and by an independent person, whether outcome assessors were blinded, whether all randomized patients were included in the analyses, and whether only selected outcomes (the positive ones) are reported. There is some evidence that allegiance is indeed associated with the risk of bias (Munder et al. 2011), and it may be the case that allegiance is a problem only in studies with a high risk of bias and not in those with a low risk of bias.

Can Comparable Effects Be Seen as Evidence for the Common Factors Model?

Some meta-analyses find clear differences between the effects of therapies, suggesting that therapies can have effects that are specific to that therapy. However, proponents of the common factors model say that this may be true, but these differences can always be explained by the inclusion of non-bona fide therapies or those that are intended to fail and by researcher allegiance. This has not been systematically assessed in all meta-analyses, so whether it is true cannot be determined. Thus, it may or may not be true that all therapies have comparable effect sizes. Also, it is not known whether heterogeneity is low in these meta-analyses because the confidence levels around I^2 are typically wide.

Comparative outcome studies also have several other important methodological problems that may affect their results. One substantial problem is statistical power. Differences between therapies can be assumed to be small, and, therefore, comparative trials need large sample sizes. For example, a rough estimate of the threshold for clinical relevance in depression, based on the minimally important difference from the patient’s perspective, has been found to be $d = 0.24$ (Cuijpers et al. 2014b). In order to find such a difference, a trial would need 548 patients (274 in each condition; Cuijpers 2016). Trials with such large sample sizes are hardly ever conducted in psychotherapy research. In a meta-analysis of 46 outcome trials comparing CBT for depression with another therapy, we found that the mean number of patients included was 52, and the range was 13 to 178 (Cuijpers et al. 2013). The largest trial (Dowrick et al. 2000) had sufficient power only to detect a differential effect size of $d = 0.42$, and the average study with 52 patients had enough power only to find a differential effect size of $d = 0.79$.

Another important problem is the low methodological quality of many trials. For example, in a meta-analysis of controlled trials of psychotherapy for depression in adults we found that

only 22% of the 289 studies could be rated as having a low risk of bias (Cuijpers et al. 2019b). For anxiety disorders, we found that only 29% (9/31) of trials assessing treatment for generalized anxiety disorder, 10% (4/42) of those for panic disorder, and 17% (8/48) of those for social anxiety disorder had a low risk of bias (Cuijpers et al. 2016a). It is well known from controlled trials that poor-quality trials considerably overestimate the effects of therapies (Cuijpers et al. 2010, 2019b; Furukawa et al. 2014). It has not been examined whether the poor quality of many trials has affected the outcomes of comparative outcome studies. However, the results of many comparative trials may also be biased and may not accurately represent the true differentials between therapies.

In principle, the power problem of comparative outcome trials can be solved by pooling the results of multiple trials in meta-analyses. However, if we focus only on trials with a low risk of bias, then even meta-analyses do not have enough power to detect smaller differences between therapies (Cuijpers 2016).

Alternative Explanations for Finding Comparable Outcomes

But suppose that all meta-analyses show that therapies have comparable effects and heterogeneity is low. Can that be considered as evidence for the common factors model? In general, there is little empirical evidence about how therapies work (Kazdin 2007), so whether therapy works through common factors is mostly a matter of speculation. There are alternative explanations for why therapies may have comparable effects. One logical explanation is that many roads lead to Rome, and different therapies may lead to comparable outcomes via different mechanistic pathways (DeRubeis et al. 2005). Mental disorders are complex and affect many areas of life. If a therapy—itsself a multifactorial, complex process—focuses on one area of life and successfully changes it, then it may in turn change the other areas that are affected by the disorder. For example, depression is a disorder that affects cognitions, behavior, interpersonal relations, intrapersonal functioning, and other areas. If someone learns how to change his or her cognitions and is successful in doing so, then this change may also result in improvements in other areas. And if someone is treated with a therapy that focuses on intrapersonal issues that may also affect all the other areas. So in these cases it would mean that the outcomes of all therapies are comparable, but how they realize their effects may still differ.

Related to this discussion of complex processes, mental disorders are significantly associated with dozens of psychological, biological, and social factors. Therapies are also highly complicated interactions between a therapist and a patient. So it is very possible, even probable, that there are endless ways in which the therapy is shaped, with some interactions having an impact on specific patients with certain characteristics. These interactions probably differ considerably for each patient and each therapy. However, when these outcomes are taken together in trials and those trials are pooled in a meta-analysis, the highly individual outcomes are blended into one outcome. The lack of precision in measuring mental health problems and the outcomes of therapies makes the therapeutic process seem even more uniform, when in fact, therapy is a highly individualized process, and it is impossible to say whether these outcomes are the result of common or specific factors.

The only conclusion we can draw from meta-analyses of comparative outcome trials is that they partly support the common factors model because some show comparable outcomes for therapies. However, there is insufficient evidence to be certain whether all therapies do indeed have comparable effects. And even if the final conclusion turns out to be that all therapies have comparable effects, this cannot be seen as final proof that all therapies work through common factors, as we have argued.

COMPONENT STUDIES AND THE COMMON FACTORS MODEL

One important type of research that is used as an argument by proponents of specific therapy models is the component study. These studies provide an elegant way to identify the active ingredients of psychotherapies (Borkovec & Costonguay 1998). These studies decompose multicomponent therapies and compare the full therapy with a therapy in which one component is left out (dismantling studies) or in which a component is added to an existing therapy (additive studies; Bell et al. 2013). Because of their design, the strength of evidence resulting from component studies is high. If a component study finds a difference between a therapy with a component and a therapy without that component, this indicates that the particular component is indeed responsible for (at least part of) the effects of the intervention. Therefore, proponents of common factors assume that component studies do not result in significant differences between studies with or without a specific component, as long as both the therapy with the component and the one without are *bona fide*.

There are a few meta-analytical studies of component studies. The first one, which included 27 studies, did not find evidence that specific components were responsible for part of the effects of therapies (Ahn & Wampold 2001). In keeping with the common factors model, no significant differences between therapies with and without specific components were found. A second major review examined a much larger set of component studies ($n = 66$; Bell et al. 2013) and found that the use of dismantling studies did not result in significant differences between the full and the partial treatment (in which one component was left out; Cuijpers et al. 2019a). However, a small but significant effect was found when a component was added to a therapy, and this effect was larger at follow-up. This finding was not in keeping with the common factors model. A reanalysis using longitudinal meta-analytical methods could not replicate the finding that the effects of components were found in the long term (Flückiger et al. 2015, Wampold & Imel 2015). In a third meta-analysis of 16 component studies of treatments for depression, a small but significant difference ($SMD = 0.21$) was found between the full treatments and treatments with one component removed (Cuijpers et al. 2019a).

There are several methodological problems with component studies that make them hard to interpret and make it even harder to decide whether they support the common factors model. Again, the first problem is statistical power. Just as with other comparative trials, most component studies are heavily underpowered. In the meta-analysis by Cuijpers et al. (2019a), none of the studies had more than 19% of the number of participants needed to detect the significant effect of a component, and 16 of the 22 comparisons had less than 10% of the participants needed. The only exception was a study in which emotion regulation skills training was added to CBT for treating depression (Berking et al. 2013), and this study found a superior effect of CBT with emotion regulation skills training compared to CBT without this training. In the same meta-analysis, three studies compared cognitive restructuring plus behavioral activation for depression with cognitive restructuring alone. These studies also found a significant effect of adding behavioral activation to cognitive restructuring. In sum, these findings suggest that component studies may be suited to demonstrating the significant effects of components, but they need sufficient power to be able to do so.

Again, another methodological problem is the risk of bias. As mentioned earlier, this refers to the weak spots of a trial and, for example, to problems with the randomization and the blinding of outcome assessors, and whether all randomized patients are included in the analyses (Cuijpers & Cristea 2016). Of the 16 studies included in the meta-analysis by Cuijpers et al. (2019a), only 1 had a low risk of bias. This suggests that based on the literature, we are not able to state whether specific components of therapies are essential elements or whether the effects result from common factors.

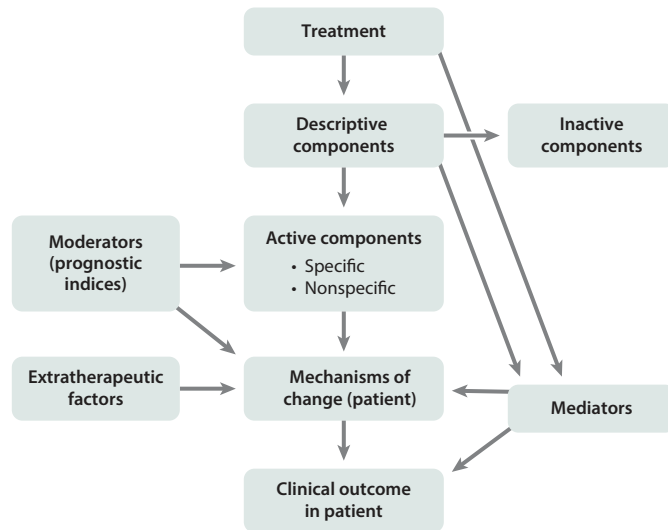


Figure 1

Model of the clinical change process and the components of psychotherapy.

In addition, it can be questioned whether the results of component studies should be pooled in meta-analyses anyway. The components of therapies that are examined in these studies differ from one another, and it is possible that a few components are essential components of therapies while others are not. If the pooled result of all component studies finds no significant effect, then that cannot be considered as evidence that essential components do not exist. Finding nonsignificant effects of pooled component studies is not evidence that these components do not exist and that therapies work through common elements.

HOW CAN SPECIFIC AND NONSPECIFIC FACTORS BE EXAMINED?

At the start of this review, we stated that RCTs result in strong evidence that a therapy works, but it is much more complicated to show how a therapy works, and the trial designs used to examine these mechanisms do not provide the same strength of evidence as results from RCTs. In this section, we will explain this problem in more detail and describe the methods that can be used to examine the mechanisms of therapies. These methods are important because this is the research that can tell us whether it is the specific mechanisms, the nonspecific, or both that are responsible for the change that results from therapies.

A Model for the Process of Clinical Change

In **Figure 1** we present a simplified model of how therapies lead to clinical outcomes in patients. This model is based on earlier work by Hollon and associates (1990) and builds on the definitions of relevant concepts described by others (Borkovec & Sibrava 2005; Kazdin 2007, 2009; Kraemer et al. 2008, 2002).

A therapy consists of several descriptive components, some of which may be inactive (e.g., they do not contribute to change in a patient) and others that are active (e.g., specific therapeutic procedures). The active components are the specific or nonspecific factors, or both, of the therapy. The mechanisms of change are the therapeutic processes or events that are responsible for the

change and the reasons why change occurs or how change comes about (Kazdin 2007, 2009). These are the results of the active components (i.e., therapeutic procedures) of the therapies. Mechanisms of change happen in the patient, while the active components are part of the therapy.

Mechanisms of change can be influenced by events outside of therapy. These extratherapeutic factors may include life events or a lack of competencies in the patient that limit the possibilities of the mechanisms of change. Such effects are not so much a consequence of treatment as an extraneous influence on outcome, unless they interact with treatment to produce the change observed.

Moderators, or prognostic indices, are characteristics that influence the direction or magnitude of the relationship between treatment and outcome (Kazdin 2007, 2009; Kraemer et al. 2008). In our model, it is assumed that moderators are related to active components and mechanisms of change. Moderators that are found to be statistically significant indicate that different active components and mechanisms of change are involved in specific subgroups. For example, if an RCT finds that gender is a significant moderator of outcome, this would imply that the active components or subsequent mechanisms of change, or both, differ for men and women.

Mediators are intervening variables that statistically may account for the relationship between the treatment and the outcome (Kazdin 2007). Mechanisms of change are expected to consist of a mediator or a group of mediators. However, mediators are not necessarily part of a mechanism of change because statistically they may account for only the association between treatment and outcome. It is possible for a variable that is found to be a mediator to be, in fact, a proxy for one or more other variables. A mediator may point to a possible causal mechanism, but it is not necessarily part of the mechanism itself (Kazdin 2007).

Research Methods for Examining the Process of Clinical Change

RCTs showing that a therapy is effective are not sufficient to examine how treatments result in clinical outcomes. This is similar to attempts to show that smoking leads to lung cancer (Kazdin 2007, 2009). Showing that such an association exists says nothing about the exact biological processes in the human body that lead from the smoke being inhaled into the lungs to the development of cancer over time.

We have already shown that component studies are a good option for examining the effective ingredients of therapies because they provide strong evidence that the examined components are indeed responsible for the effects of a treatment. However, component studies do not explain fully how these treatments work. The components activate mechanisms of change in the patient, but component studies do not identify what these mechanisms are or explain how they operate. In order to examine mechanisms of change, it is necessary to examine mediators. Mediators are the intervening variables that account for the relationship between the treatment and the outcome of the treatment (Kazdin 2007). To show statistically that a variable is a mediator, several conditions must be met (Baron & Kenny 1986, Kazdin 2007, Kraemer et al. 2008, MacKinnon et al. 2007).

First, participants in the intervention condition must show significantly greater decreases in measures of symptoms over time than the controls (i.e., the treatment condition predicts the change in the outcome). Second, participants in the intervention condition must show significantly greater decreases on measures of the mediator over time than controls (i.e., the treatment condition predicts changes in the mediator). Third, in the intervention condition, the change in the mediator over time must be significantly correlated with change in the outcome over time. And fourth, the predictive effect of the intervention condition on the change in the outcome—after controlling for change in the mediator—must be significantly reduced (for partial mediation) or eliminated (for complete mediation) relative to when the outcome is regressed

only for the intervention condition. A decade ago, the MacArthur group (Kraemer et al. 2008) proposed a somewhat different analytical approach that basically replicates this logic but adds the notion that an interaction between a treatment and a purported mediator also indicates mediation even in the absence of a documented relation between treatment and outcome.

Studies of mediators are not sufficient to examine mechanisms of change. If a mediator meets the criteria described above, there is still not sufficient evidence of a causal association between the mediator and the outcome. It is possible that a mediator is, in fact, a proxy for one or more other variables. Furthermore, if a variable is indeed a mediator, it must be shown that changes in the mediator come before changes in the outcome, thus demonstrating a temporal relationship. To show that a mediator is indeed a component of a mechanism of change, several other types of research are helpful, including direct experimental manipulation of the proposed mediator, studies showing a dose–response relationship between the mediator and the outcome, and theoretical studies presenting a plausible explanation for why and how the mediator results in change in the patient (Kazdin 2007, 2009). Multiple, converging lines of research are needed to explain precisely how mechanisms of change result in better outcomes for the patient.

To demonstrate how difficult it is to show how mechanisms of change actually work, we return to the example of tobacco smoking. Although many carcinogens have been identified in tobacco, and a considerable body of research has tried to disentangle how these substances cause cancer, “we may never be able to map each detail of the complex process by which cigarette smoking causes lung cancer” (Hecht 1999, p. 1194).

Research on Specific and Nonspecific Components of Therapies

There are two major groups of studies examining how therapies work: component studies and studies directly examining mediators and working mechanisms. We have already showed that a considerable number of component studies have been conducted during the past decades, but their methodological weaknesses do not warrant drawing strong conclusions about whether there are specific effects. The best conclusion is that this body of research is not strong enough to say whether there are specific components.

Hundreds of studies have examined the processes that are active in psychotherapy. However, the majority of these studies have examined only the association or correlation between the outcome of a therapy and the characteristics of patients, the therapist, and the therapy process, and they did not include formal tests of mediators. For example, one large meta-analysis included almost 200 studies examining the correlation between improvement in patients and the therapeutic alliance (Horvath et al. 2011).

Many hundreds of other studies have examined the association between patients’ improvement and expectations, the therapist’s warmth and mastery, and other characteristics (Orlinsky et al. 2004). However, this large body of research lacks the strength of evidence from RCTs and is only correlational, with all of the pitfalls and limitations attached to that. One problem is that the association between many of these factors and outcomes can be examined only within treatments because factors related to the therapist and therapy are irrelevant in control conditions. This means that these variables are completely integrated into the treatment and cannot be examined in controlled trials. Furthermore, in order to plausibly discover whether these variables are indeed mediators, we need to find a temporal association, with a change in the mediator preceding the change in the outcome. Finding a dose–response relationship would strengthen the hypothesis that a variable is indeed a mediator. But then it might still be possible that a third variable causes a change in the outcome as well as a change in the assumed mediator. Even if a large number of such third variables were to be examined (which is almost impossible to achieve because of the

limited time patients can spend completing questionnaires), the possibility can never be excluded that there is a third, unmeasured variable causing the change in the outcome and in the assumed mediator. Only supporting evidence from experimental studies and a clear theoretical framework can strengthen the hypothesis that a particular variable is indeed a mediator.

The complexity of showing whether specific or nonspecific factors are accountable for the effects of psychotherapy is complicated even further by the fact that many studies of psychotherapy suffer from a considerable risk of bias, low statistical power, publication bias, and researcher allegiance. Furthermore, mental health problems and changes in these problems can be measured only by self-report or by clinical interview: There are no objective tests. Together these problems make it almost impossible to examine how therapies work, and it is not surprising that there is little knowledge about this. Examining mechanisms of change requires considerable investment in research, and unfortunately sufficient investment has not been available to disentangle the mechanisms of change in specific therapies. The implication is that we know only a little about whether therapies work through specific or nonspecific factors.

EVIDENCE FOR SELECTED COMMON FACTORS

The Therapeutic Alliance

Of all the common factors that have been proposed, the therapeutic alliance or therapeutic relationship is championed by many as the most important. The alliance has three components: the bond between the therapist and patient, agreement about the goals of therapy, and agreement about the tasks of therapy (Wampold 2015). The association between alliance and outcome in psychotherapy has been extensively examined in treatment studies, and the most recent meta-analysis, based on more than 200 studies, found that stronger alliances are indeed associated with better outcomes, although the magnitude of the association is modest, with an explained variance in outcome of 7.5% (Horvath et al. 2011). The alliance–outcome association is often presented as strong proof in favor of the common factors theory, despite the relatively modest magnitude of the association and the fact that this association is merely correlational in nature. Correlational data cannot be used for causal inferences, and a temporal relationship, a dose–response relationship, the exclusion of other potential mediators, supporting experimental research, and an explanatory theoretical framework are also needed to establish the role of the alliance in realizing change.

However, recent studies have begun to disentangle the relationship between alliance and outcome. The core of this new line of research is the establishment of a time line of repeated assessments so that temporality can be investigated, and this is combined with the use of advanced statistical methods that account for potential confounders. Zilcha-Mano (2017) reviewed these new studies in an elegant overview and concluded that the studies that account for temporality and disentangle the effects of between- and within-patient variability show that changes in the alliance may indeed precede symptom reduction. She proposes a new model for understanding the potential therapeutic role as sufficient to induce change by itself. This model, based on the integration of previous research (Zilcha-Mano 2017), stresses the importance of differentiating between patients' general tendencies to form satisfying relationships with others, which affect also their relationship with the therapist (the trait-like component of alliance with which the patient comes into the therapy), and the process of the development of changes in such tendencies through interaction with the therapist (the state-like component of alliance that changes as a result of therapy). A good trait-like alliance is a prerequisite to engaging in therapy and making it effective, while the state-like changes in the alliance during treatment can predict the subsequent outcome and, thus, might make the alliance therapeutic in itself.

Zilcha-Mano (2017) also argues that disentangling the trait-like and state-like components of the alliance might elucidate differences in the role of the alliance between therapy schools. Indeed, in some therapies that place more emphasis on the alliance (e.g., psychodynamic therapy), the alliance might be more strongly linked to outcome than in therapies that place less emphasis on the alliance (e.g., CBT) (Huibers & Cuijpers 2015).

Despite the sophistication of Zilcha-Mano's (2017) review, it should be emphasized that temporality is only one of many criteria that should be met before speaking of a causal mechanism (Kazdin 2007). The studies reviewed by Zilcha-Mano (2017) are definitely a step in the right direction, but they are observational and cannot be seen as proof of the causal role of the alliance in the process of psychotherapy. For example, it is still entirely possible that a third, unmeasured variable is driving the change in both the alliance and outcome, even if temporality is accounted for.

One more step toward evidence for a causal role of the alliance could come from experimental studies, in which the alliance is isolated as a process and directly manipulated. For example, one can imagine a randomized experiment in which therapists in one condition are instructed to maintain a steady alliance, while therapists in the second condition are instructed to actively strengthen the alliance over the course of therapy (Zilcha-Mano 2017). In the case of the alliance, conducting such experiments is challenging, primarily from an ethical point of view, but it is not entirely impossible (Huibers & Cuijpers 2015).

Other Common Factors

In addition to the therapeutic alliance, a wide array of other common factors have been proposed (Table 1; Lambert & Ogles 2004) and investigated in the context of treatment. Wampold (2015) summarized the evidence in the form of aggregated effect sizes for common factors—such as therapist empathy, patients' expectations of outcome, the cultural adaptation of evidence-based interventions (for an optimal fit with the patient's beliefs about illness and effective therapy), and the therapist's effects—and concluded that they all should be considered therapeutic, although the evidence in favor of specific factors is weak. However, this distinction between common and specific factors is based purely on correlational data, and, therefore, it is not supported by evidence for or against the potentially causal role of common or specific factors. Moreover, the differences in effect sizes between nonspecific and specific factors that Wampold presents are questionable. Factor–outcome correlations or effect sizes based on correlational data cannot be used to determine the relative importance of these factors, let alone to inform us about the causal impact of therapy factors on outcome.

On the basis of several meta-analyses, the American Psychological Association's Interdivisional Task Force on Evidence-Based Therapy Relationships concluded that expressed empathy (Elliott et al. 2011), the therapeutic alliance (Horvath et al. 2011), and collecting structured client feedback (Lambert & Shimokawa 2011) were demonstrably effective elements of the therapeutic relationship across all models of psychotherapy (Norcross & Wampold 2011). The complexities involved in examining how therapies work make this an overly optimistic summary of the literature.

Specific Factors

Apart from the research on common factors, there is also a vast literature on the specific factors that might account for the effects of psychotherapy, mostly organized around specific forms of psychotherapy (mostly CBT) and specific psychological disorders (mostly depression and anxiety). What can be said for the evidence on common factors can, broadly speaking, just as well be said for the evidence on specific factors: Most older studies are correlational, and the number of studies is

small that address temporal changes, dose–response relationships, and experimental manipulation of putative mechanisms. It is beyond the scope of this article to present a complete overview of these studies. However, we discuss a few recent reviews and meta-analyses that give an impression of how this field has developed during the past decades, particularly in regard to CBT, the most extensively researched form of psychotherapy.

Kazantzis et al. (2018) reviewed 30 meta-analyses that synthesized process–outcome relationships in CBT (mostly used to treat depression and anxiety) that were categorized as involving either treatment processes (e.g., cognitive change work) or in-session processes (e.g., the therapeutic alliance). Note that the distinction between therapeutic procedures and therapeutic processes is not made in this review. In the category of treatment processes, they found the strongest support for cognitive and behavioral strategies as processes of change in CBT in depression and anxiety. For in-session processes, the strongest support was found for the therapeutic alliance and homework assignments. At a glance, this comprehensive review seems to highlight the importance of both specific and nonspecific factors. However, it should be kept in mind that the evidence reviewed is merely correlational.

Lemmens et al. (2016) reviewed the mechanisms of change in psychotherapy (CBT and other forms of therapy) for depression. This review included 35 individual studies in which an actual test of statistical mediation [using the methods of Baron & Kenny (1986) or more advanced methods] was performed. In total, 39 different potential mediators (i.e., therapeutic processes) were investigated, mostly related to CBT and CBT processes. The strongest support was found for changes in dysfunctional attitudes, negative automatic thoughts, rumination, worry, and the use of mindfulness skills, but it should also be noted that about half of the studies did not detect a mediational role for the construct under investigation. Moreover, Lemmens and associates (2016) also investigated the quality of these mediation studies based on six methodological criteria (RCT design, inclusion of a control group, sufficient sample size, assessment of multiple mediators, temporality, and experimental manipulation) and found that only 17 out of 35 studies met 4 or more of these criteria. Their conclusion is that the evidence for the mediational role of the various constructs (specific and nonspecific) is largely mixed and that better designed studies are urgently needed to understand the mechanisms of psychotherapy.

Cristea et al. (2015) performed a meta-analysis of the effects of CBT on dysfunctional thinking (the presumed core process in CBT) compared with the effects on dysfunctional thinking in control groups, other forms of psychotherapy and antidepressant medication. They included 26 studies, of a less-than-optimal quality, and found a strong association between the effects of CBT on dysfunctional thinking and the effects on depression. However, the effects on dysfunctional thinking did not differ significantly between CBT and other psychotherapies (except when the comparison was restricted to a specific measure, the Dysfunctional Attitude Scale, or DAS) or antidepressant medication, suggesting that the effect on dysfunctional thinking is not specific or exclusive to CBT. The authors concluded that their findings can be interpreted in opposite directions: as a confirmation of the primacy of cognitive change as a specific factor in symptom change, regardless of how it is attained, or as support for the notion that dysfunctional thinking is merely another symptom of depression that changes during therapy.

Similar to the review by Cristea et al. (2015), Spinhoven et al. (2018) conducted a meta-analysis of 36 studies that investigated the effects of various forms of CBT on repetitive negative thinking (i.e., rumination) compared with control groups and other treatments. They found a medium-size effect of any treatment compared with the control group, but they also found significantly larger effect sizes for CBT treatments such as rumination-focused CBT and original CBT compared with treatments such as antidepressant medication and counseling. The authors concluded that CBT and, in particular, rumination-focused CBT might have specific and pronounced effects

on repetitive thinking, which might be interpreted as establishing a link between a therapeutic procedure (i.e., specific CBT interventions) and a therapeutic process (i.e., rumination).

In sum, the association between specific factors and outcome is well established, especially in the field of CBT, but proof of the causal role of these factors is generally lacking, and there is insufficient evidence that these specific factors are core elements of how CBT works. More than 30 years after the introduction of mediation analysis (Baron & Kenny 1986), we must conclude that we still do not know whether the factors that bring about change in psychotherapy are specific, nonspecific, or both. New lines of research are needed that account for temporality and other criteria that constitute the requirements for establishing a causal link. Moreover, by definition, psychotherapy is a complex process involving multiple elements that most likely interact with one another, and simple causal models probably will not advance our understanding of how it works (Lemmens et al. 2016, Lorenzo-Luaces & DeRubeis 2018).

RECOMMENDATIONS FOR FUTURE RESEARCH

Research on common and specific factors, and understanding how therapies bring about change in patients, is important, not only from a scientific perspective but also from a public health view. For example, the effect sizes of treatments in depression have not increased during the past decades, and many patients do not respond to treatment or relapse soon after recovery. Furthermore, the uptake of therapies is low, even in high-income countries (Chisholm et al. 2016), and one reason may be that therapies are not sufficiently acceptable to patients. Understanding how therapies work may make it possible to develop treatments that focus on the core processes and are, therefore, more effective and efficient, and more acceptable to patients.

Until now, much research has focused on RCTs, with many hundreds of such trials showing that therapies work but not how they work. There is also a lot of research on the processes and potential working mechanisms of therapies. However, this large body of research consists mostly of correlational research, and few studies have focused on the type of research that is needed to show precisely how these mechanisms work, with evaluations of the temporal associations, the dose–response relationship, and the other requirements of such research. This means that we have conducted a large number of studies examining how therapies work but have never managed to focus this research on the issues that are needed to understand the mechanisms of change. It is as if we have been in the pilot phase of research for five decades without being able to dig deeper.

If we want to take a step forward, we need to conduct research that goes beyond examining, on the one hand, simple correlational associations between specific and common factors and, on the other, outcomes. We need research on these factors that includes temporal associations, dose–response relationships, multiple alternative potential mediators, experimental manipulation, and theoretical models. Research to discover how one type of therapy works and whether a specific or a common factor is responsible for change requires major funding, many individual studies, and a deep breath. There are no easy solutions, and such research will require considerable resources. However, we have invested resources in this research for five decades and if we could put only part of these resources toward making a coordinated effort to examine mechanisms of change, it would certainly become feasible.

Within such broad frameworks, it is important to conduct research with carefully planned assessments of both procedures (i.e., the interventions employed by the therapist) and treatment processes (i.e., the changes that occur within the patient as a result of those interventions). Although therapeutic procedures and processes have often been lumped together as mechanisms of change, it is important to differentiate between them and examine them and their interplay in detail.

Accounting for individual differences between patients is vital in this research. Causal pathways of change are likely to differ between patients. Although RCTs focus mainly on groups and mean values for the group, trial data can be used to investigate individual differences. One way to approach the issue of “different strokes for different folks” is to analyze moderated mediation. For example, recent studies have shown that the alliance may indeed be a predictor of outcome but only in certain subgroups of patients (Lorenzo-Luaces et al. 2014, 2015; Zilcha-Mano et al. 2018).

Another important element that studies should address is not only between-person variability (e.g., a good versus bad alliance) but also within-person variance (e.g., change in the alliance over time) to assess how changes within a patient during the course of treatment lead to individual outcomes. It may be helpful to use advanced methods such as experience sampling (see, for example, Hoet et al. 2018) to track the change processes of individual patients before and during treatment so that these can then be linked to outcome.

CONCLUSIONS

Although it has been shown that psychotherapies are effective in treating mental disorders, how they work is not well understood. Examining how therapies work cannot be done with RCTs alone, and the analysis requires multiple, complicated, and very large studies that should be complemented by experimental studies and theoretical work. Psychotherapies may work through techniques that are specific to each therapy or through factors that all therapies have in common, but currently, there is insufficient evidence to enable either common factors or specific factors to explain how therapies work. Proponents of the common factors model often point to other evidence to show that these factors are, in fact, the ones that make therapies work. One important body of knowledge consists of comparative outcome studies, which have suggested that all therapies have comparable effects. However, not all meta-analyses support the common factors model; the studies included in them often have methodological problems; and there are alternative explanations for finding comparable outcomes. Component studies, in which one component is added to or removed from a therapy, are assumed by proponents of the common factors model to result in nonsignificant differences between the full and the partial therapies. However, these studies are typically underpowered, and there are few sufficiently powered component studies, meaning that this body of research also cannot tell us whether therapies work through common or specific factors.

Psychotherapy is a complex, multifactorial process, and it is most likely that both common factors and specific factors play a part in the process that leads to recovery, most likely in complicated ways that cannot be captured by simple causal models. That being said, the only empirical conclusion that can be drawn is that it is not known whether therapies work through common factors, specific factors, or both, and that more and better research is needed to establish this.

SUMMARY POINTS

1. Although hundreds of randomized controlled trials have shown that psychotherapies are effective in treating mental disorders, it is not known how they work.
2. Therapies may work through techniques that are specific to each therapy, through factors that all therapies have in common, or through a combination of the two.

3. The discussion about whether therapies work through common or specific mechanisms has been going on for several decades, but it has not been resolved because it is not known how therapies work.
4. Meta-analyses of comparative outcome studies do not all point to comparable effects for different therapies and because alternative explanations are possible for comparable effects, it is not known whether all therapies do, in fact, have comparable effects.
5. Component studies (in which one component is removed from or added to a therapy and this is compared with the complete therapy) are also inconclusive, regardless of whether specific components are partly responsible for the effects of therapies.
6. There is no straightforward method for examining how therapies work, and most research on specific and common factors has been conducted using correlational studies; there has been little research on temporal associations, dose–response relationships, supportive theoretical frameworks, and laboratory studies.
7. Although hundreds of correlational studies have been conducted during the past decades, little progress has been made in understanding the mechanisms of change of therapies: It is as if we have been in a pilot phase of research for five decades.

FUTURE ISSUES

1. We must realize that examining how a therapy works is completely different from examining that it works.
2. To come to an understanding of how a therapy works requires major funding, many individual studies, and a deep breath.
3. Moderated mediation is a promising focus of future research.
4. Research should focus not only on between–person variability but also on within–person variance, for example, with experience sampling methods.

DISCLOSURE STATEMENT

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Contents

Positive Psychology: A Personal History <i>Martin E.P. Seligman</i>	1
History of Psychopharmacology <i>Joel T. Braslow and Stephen R. Marder</i>	25
Bifactor and Hierarchical Models: Specification, Inference, and Interpretation <i>Kristian E. Markon</i>	51
The Utility of Event-Related Potentials in Clinical Psychology <i>Greg Hajcak, Julia Klawohn, and Alexandria Meyer</i>	71
An Active Inference Approach to Interoceptive Psychopathology <i>Martin P. Paulus, Justin S. Feinstein, and Sabib S. Khalsa</i>	97
Implicit Cognition and Psychopathology: Looking Back and Looking Forward <i>Bethany A. Teachman, Elise M. Clerkin, William A. Cunningham, Sarah Dreyer-Oren, and Alexandra Werntz</i>	123
The MMPI-2-Restructured Form (MMPI-2-RF): Assessment of Personality and Psychopathology in the Twenty-First Century <i>Martin Sellbom</i>	149
Normal Versus Pathological Mood: Implications for Diagnosis <i>Ayelet Meron Ruscio</i>	179
The Role of Common Factors in Psychotherapy Outcomes <i>Pim Cuijpers, Mirjam Reijnders, and Marcus J.H. Huibers</i>	207
One-Session Treatment of Specific Phobias in Children: Recent Developments and a Systematic Review <i>Thompson E. Davis III, Thomas H. Ollendick, and Lars-Göran Öst</i>	233
Augmentation of Extinction and Inhibitory Learning in Anxiety and Trauma-Related Disorders <i>Lauren A.M. Lebois, Antonia V. Seligowski, Jonathan D. Wolff, Sarah B. Hill, and Kerry J. Ressler</i>	257

Mindfulness Meditation and Psychopathology <i>Joseph Wielgosz, Simon B. Goldberg, Tammi R.A. Kral, John D. Dunne, and Richard J. Davidson</i>	285
Prenatal Developmental Origins of Future Psychopathology: Mechanisms and Pathways <i>Catherine Monk, Claudia Lugo-Candelas, and Caroline Trunpff</i>	317
Using a Developmental Ecology Framework to Align Fear Neurobiology Across Species <i>Bridget Callaghan, Heidi Meyer, Maya Opendak, Michelle Van Tieghem, Chelsea Harmon, Anfei Li, Francis S. Lee, Regina M. Sullivan, and Nim Tottenham</i>	345
Man and the Microbiome: A New Theory of Everything? <i>Mary I. Butler, John F. Cryan, and Timothy G. Dinan</i>	371
Estrogen, Stress, and Depression: Cognitive and Biological Interactions <i>Kimberly M. Albert and Paul A. Newhouse</i>	399
Adolescent Suicide as a Failure of Acute Stress-Response Systems <i>Adam Bryant Miller and Mitchell J. Prinstein</i>	425
Abnormal Sleep Spindles, Memory Consolidation, and Schizophrenia <i>Dara S. Manoach and Robert Stickgold</i>	451
The Development of the ICD-11 Classification of Personality Disorders: An Amalgam of Science, Pragmatism, and Politics <i>Peter Tyrer, Roger Mulder, Youl-Ri Kim, and Mike J. Crawford</i>	481
A Reciprocal Model of Pain and Substance Use: Transdiagnostic Considerations, Clinical Implications, and Future Directions <i>Joseph W. Ditre, Emily L. Zale, and Lisa R. LaRowe</i>	503
Anxiety-Linked Attentional Bias: Is It Reliable? <i>Colin MacLeod, Ben Grafton, and Lies Notebaert</i>	529
Biomedical Explanations of Psychopathology and Their Implications for Attitudes and Beliefs About Mental Disorders <i>Matthew S. Lebowitz and Paul S. Appelbaum</i>	555
Psychology's Replication Crisis and Clinical Psychological Science <i>Jennifer L. Tackett, Cassandra M. Brandes, Kevin M. King, and Kristian E. Markon</i>	579

Errata

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TABLE OF CONTENTS FOR VOLUME 1:

Introduction, Susan A. Gelman, Sandra R. Waxman

Eleanor Maccoby: An Abridged Memoir,
Eleanor Maccoby

*Using Developmental Science to Distinguish
Adolescents and Adults Under the Law*,
Laurence Steinberg, Grace Icenogle

*Adolescent–Parent Relationships: Progress, Processes,
and Prospects*, Judith G. Smetana, Wendy M. Rote

The Life Course Consequences of Very Preterm Birth,
Dieter Wolke, Samantha Johnson, Marina Mendonça

*Early Deprivation Revisited: Contemporary Studies
of the Impact on Young Children of Institutional Care*,
Megan R. Gunnar, Brie M. Reid

The Development of Cumulative Cultural Learning,
Cristine H. Legare

*Neighborhood Effects on Children’s Development
in Experimental and Nonexperimental Research*,
Tama Leventhal, Veronique Dupéré

*Cognitive Aging and Dementia: A Life-Span
Perspective*, Elliot M. Tucker-Drob

*Brain Plasticity in Human Lifespan Development:
The Exploration–Selection–Refinement Model*,
Ulman Lindenberger, Martin Lövdén

The Pervasive Role of Pragmatics in Early Language,
Manuel Bohn, Michael C. Frank

*Early Development of Visual Attention:
Change, Stability, and Longitudinal Associations*,
Alexandra Hendry, Mark H. Johnson, Karla Holmboe

*Childhood Adversity and Neural Development:
A Systematic Review*, Katie A. McLaughlin,
David Weissman, Debbie Bitrán

*Social Relations Across the Life Span: Scientific
Advances, Emerging Issues, and Future Challenges*,
Toni C. Antonucci, Kristine J. Ajrouch, Noah J. Webster,
Laura B. Zahodne

*Safety Net Policies, Child Poverty, and Development
Across the Lifespan*, Benard P. Dreyer

The Development of Social Categorization,
Marjorie Rhodes, Andrew Baron

Developmental Effects of Parent–Child Separation,
Anne Bentley Waddoups, Hirokazu Yoshikawa,
Kendra Strouf

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