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Corresponding Author	Family Name	Taylor
	Particle	
	Given Name	**David**
	Suffix	
	Division	Adult Department, Tavistock Clinic
	Organization	Tavistock & Portman NHS Foundation Trust
	Address	London, United Kingdom
	Email	dtaylor@tavi-port.nhs.uk
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Chapter 5
Psychoanalytic and Psychodynamic Therapies for Depression: The Evidence Base*

David Taylor

Keywords Common mental disorders • Depression and depressive disorders • Placebo effect • Psychoanalytic • Psychodynamic • Publication bias • Researcher allegiance • Short-term and long-term therapy outcomes

Introduction

In 1825, the great English essayist William Hazlitt published *The Spirit of the Age* [1]. His accounts of 25 of the leading figures of his time and of some of their debates succeeded in capturing the defining features of his epoch. Among them, the great poets Wordsworth and Coleridge had been responsible for introducing the ideas of German literature and philosophy into the Anglophone world (see [2]). These insights were based upon the way the human imagination continually projects on to, continually colors, the natural world we observe around us. In terms of the history of ideas, these were amongst the antecedents of psychoanalysis. However, personal loss and disappointment with the turn taken by the French Revolution had led one of them, Wordsworth, famously to lose his early hope in revolutionary progress. For him, "Bliss was it in that dawn to be alive but to be young was very heaven" had given way to membership of the Establishment. Hazlitt, a contentious man, fiercely rebuked another of his subjects, Malthus, for his scientific and logical argument that a geometric growth leading to population "surplus" would outpace food supplies, which could only increase arithmetically. Famine, pestilence, or one of the other Apocalyptic horsemen would then cull that excess. In opposition to progressive or humane opinion, social conservatives used this Malthusian prediction to welcome war and to legitimize the withholding of food, warmth, and shelter from the poor and ill-fed.

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D. Taylor, MB, BS, MRCP, FRCPSYCH, FInstPA (✉)
Adult Department, Tavistock Clinic, Tavistock & Portman NHS Foundation Trust,
London, United Kingdom
e-mail: dtaylor@tavi-port.nhs.uk

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Do we live in totally different times today? Radical discontinuities separate then and now, but dimly we discern regularities. An adequate treatment of the possibility of cyclical epochal variations is beyond my competence, and in any case, a history of ideas is not the subject of this chapter. In spite of this, the idea contained in Hazlitt's ringing title is of value to us: the deep-seated and diffusely disseminated attitudes, preferences, and prejudices which go to make up the Spirit of an Age exert powerful and long-lasting effects upon choices and methods in science and social policy, as indeed they do upon all the activities of a human culture.

Thus, when we come to look at the much more restricted and local matter of evidence-based medicine, it is clearly correct to attribute its growing importance to the increasing potency of medicines and other interventions. Their potency means that treatment decisions must be based on explicit appraisals of the evidence. Otherwise, the likelihood of doing serious harm will outweigh that of doing good. It also seems right to adopt the Malthusian-sounding argument that the accelerating cost of medical innovation means that governments and other funders of healthcare such as social or private healthcare insurers across the developed world must turn to evidence-based medicine as a necessary and sensible instrument to ration an otherwise limitless demand.

But is there is also a sense in which both of these statements are platitudes? Among many other things, they do not explain why, for instance, there has been such a strong trend toward managed care. Or for that matter, why the UK's National Institute for Health and Clinical Excellence (NIHCE or NICE) has, as have equivalent bodies in other countries, required its mental health guideline development groups to use a medical-model approach to the evidence pertaining to the various psychological treatments used in specified common mental disorders, when there are so many reasons to think that there are better methods of doing that.

To make use of a traditional, conservative medical model in this area seems to mean associated assumptions about short-term treatments and about the existence of single disease entities—assumptions which the evidence plainly does not support. Most patients with common mental disorders do not suffer from dysfunctions restricted to those of a single diagnosis. Instead, they meet the criteria for several different symptom-based diagnoses, and they also have to cope with many long-term, suboptimal functions of the personality which are not captured by the nosology [3, 4]. Patients also complain of doubt of love, of past and present difficulties in terms of personal achievement and work, as well as of existential and intimate problems of being and identity which are unmeasured and may in fact be unmeasurable. Certain other experimental conditions, usually equally unmet, are necessary if the view that RCT methodology sets the "gold standard" is to be sustained in relation to psychological therapy outcomes. For example, the most significant of depressive disorders are relapsing, recurrent, and chronic, but, for both theoretical and practical reasons, most RCTs of psychological therapy are short term and test brief treatments. The methodology of these studies succeeds in sidestepping many of the most important features of many depressive disorders, whereas mental health workers should not and patients cannot do so.

Of course, the factors which go to determine these methodological choices are exceedingly complex, but they include the Spirit of the Age; and for better or worse, this is something which happens to be relatively resistant to discrepant findings or arguments. Merely to inveigh is insufficient. To an extent, we have to live with it as we find that, with something of the mass of a tectonic plate, the Zeitgeist has shifted toward positivism and instrumentalism. To the serious detriment of its subject, we find that psychology cravenly cleaves to the physical sciences as a model [5]. We also find that a conveniently narrow horizon upon "what works" offers a defense against the real and disturbing nature of mental disorder: we tend to be deeply uncomfortable with those states of human disorder which have to be recognized and understood before they can be modified.

What underlies the mental disorders' capacity to disturb has long been the preoccupation of psychoanalysis and depth psychology. As in Hazlitt's time, when disillusionment and mistrust had set in after the failure of revolutionary hopes and ideals, we encounter the alleged irrelevance of psychoanalysis. Of course, as with every other field of scientific enquiry, new techniques and new

explanations have become possible. Notwithstanding the undoubted significance of these advances, psychoanalysis and psychoanalytic therapies continue to be an active factory and storehouse of experience and understanding, with a rich potential for new hypotheses, and for the common good as well as for the good of the individual.

If in this light, we turn to consider the effectiveness of psychoanalytic and psychodynamic therapies in the treatment of depressive disorders, we find that the restrictive definition of evidence-based medicine in the mental health field which I have just touched on leads to an overly negative view of the empirical evidence available to support their use. When determined by the constriction of Salkovskis' [6] "hour-glass model," namely the availability of sufficient numbers of high quality double-blind random allocation controlled trials and meta-analyses, it may be possible to conclude that the evidence base for the most used treatments for depression is weak across all modalities. However, such a nugatory posture strangulates the growth of knowledge and the development of practice. It is, moreover, misleading because, as I hope to show, the approach to evidence from which this type of verdict derives ineluctably leads to findings possessed of a banal quality, when the clinical issues to which they relate most certainly are not.

First amongst all the confounds is a linguistic or logical fallacy that a statement about a "strong" or a "weak" evidence base actually says something about a treatment rather than about the research method used in respect of that treatment. As we will see, the natural course of depression is often prolonged, relapsing, or recurrent, and, although there always may be exceptions, the difficulties operating in most depressed patients are not of a kind which conceivably could resolve with brief interventions of whatever variety. While patients may be significantly helped by any well-informed and responsive professional intervention, including really good clinical management, the resistant core of any significant psychopathology is a fact of life.

As every serious clinician well knows, a mental health service for patients with a serious predisposition to depression requires a longer-term perspective, with care and support, and specific interventions available when needed, and some of them in long-term packages. The situation is not so different from that which obtains in long-term conditions like juvenile-onset diabetes. The RCT dominated research agenda has so far been unable sufficiently to take these features into account. To compensate, weak research or flawed guidelines have been endowed with a status they do not merit.

Part of the position underlying this situation seems to be that different theoretical approaches such as the cognitive, the pharmacological, the psychiatric, or the psychoanalytic, the clinical, or the scientistic have very different ideas about the principles which should underpin the methodology of outcome research and of evidence-based guidelines. These are serious disagreements about what counts as quality in evidence and fact, and about the place of clinical knowledge in the discourse. These different positions translate into the way questions about efficacy and effectiveness are asked and the way in which they are answered. They also give rise to serious problems of incommensurability. When we ask questions about complex issues, we must of necessity choose one amongst several approaches to frame our questions, and to provide the concepts and terminologies which we think are best suited to define, collect, observe, and interpret what we count as empirical data. Empirical data are, in truth, only empirical up to a point. Beyond that point, it is highly method dependent. We simply cannot properly examine the facts obtained by one method with the concepts belonging to another.

To continue its line of argument, this chapter is organized in three sections. The first is a set of preliminaries necessary to provide a frame of reference for a review of evidence which is based on principles which differ from those conventionally used by the trialists. The trialist's evidence base is revealed to consist largely of an inconclusive and usually contradictory set of findings derived from treatment outcome studies with only a narrow perspective on depression. Often, it seems to more resemble an evidence quagmire than a firm base. Naturally, my account will then focus upon the evidence pertaining to psychoanalytic/psychodynamic approaches. I hope that it will be possible to

see how, when judged by standards other than those subscribing to the primacy of a particular method, the evidence supporting psychodynamic approaches to the treatment of depression is significant and continues to accumulate. There is also an extensive web of supporting evidence drawn from so many different domains that it would be a major task to review it comprehensively. The final section will discuss this evaluation and some of its implications.

A Frame of Reference for a Developmental/Psychoanalytic View of Depressive Disorders

The paragraphs which follow provide the main facts about what is known about the prevalence, course, and nature of depression. It has been found that over the course of a year, about 6% of the adult population will suffer a major depressive disorder and 6% will experience dysthymia, with the lifetime prevalence of the disorder being about 17% [7]. In the community, between 20% and 30% of untreated depressed individuals will still be depressed a year later [8]. Put the other way round, 70% of people with untreated depression will recover without intervention in the course of a year. Generally, the risk of further episodes over the 10–15 years after an index episode is more than 85% [9]. Thirty percent of those who initially respond to medication will relapse within a year, rising to as many as 75% after 5 years [10]. Each patient with major depressive disorder will suffer on average four episodes with each episode lasting an average of 20 weeks. They will also suffer ongoing symptoms and disability during the periods of remission [11, 12]. As these figures suggest, depression can be a circumscribed, self-limiting condition for some people, but for a significant proportion, the condition is characterized by a pattern of remission and relapse and shows a tendency for a deteriorating outlook as time goes by [13]. Yet, as has already been noted, the majority of RCTs of psychological therapy and pharmacotherapy involve short-term treatments of acute phase depression, with follow-ups which are nonexistent or very brief. These studies therefore cannot provide reliable information about efficacy over the medium and longer term, especially in relation to those who suffer from more chronic or severe forms of depression.

To summarize, we have seen how the natural course of depression follows a varying path: for about 50% of people who develop a major depressive disorder in the community, the duration of illness is likely to be in the order of 3 months; thereafter, the probability of full remission diminishes rapidly; 60% of those treated with antidepressant medication after 1 year and 20% after 2 years will still meet the criteria for MDD; up to 90% of all patients will develop new episodes after recovery, and only 20% of patients with depression who are serious enough to be hospitalized depressives remain well over a follow-up period of 15 years (see [14–17]).

If we interrogate the kind of data quoted previously more closely, we find that the data were arrived at using diagnostic schemes based on the idea that depression, generalized anxiety disorder, panic disorder, and obsessive-compulsive disorder are distinct entities. If truly they were, they would be expected to occur independently of each other, to have their own particular etiology, and each would respond to its own specific treatment acting upon pathological processes specific to the disorder. What we find does not match these predictions.

In fact, single disorder research and treatment guidelines for common mental disorders based upon research of this kind cannot deal with a whole range of key disconfirmations. First, the precise rates provided for its diagnoses are artifacts which endow the conventional taxonomy with an air of objectivity and exactitude which is deceptive. The figures vary according to which diagnostic scheme is used while, at the same time, no one of these schemes is more valid than another [18]. It has repeatedly been found that only a minority of patients meet the criteria of only one diagnosis. Patients satisfying the criteria for MDD are nine times more likely to meet the criteria for other conditions than chance would predict [19, 20]. There is ≥57% "comorbidity" between depression and anxiety [21, 22].

Between 50% and 90% patients with Axis I conditions will be "comorbid" with other Axis I or Axis II disorders [3]. Index presentations commonly change over time with the features of depression, anxiety, phobic, or obsessive-compulsive disorders now uppermost, now unobtrusive [23]. Furthermore, the measures of symptoms upon which categorical diagnoses depend only capture a small, selected portion of the problems patients experience. These include significant problems of interpersonal and intrapsychic functioning and of the life the patient experiences.

The case for a composite group of anxiety/depression and common mental disorder is strengthened by the finding that the temperamental factors found amongst the various common mental disorders (neuroticism or negative affect) cannot be distinguished between diagnoses. There are marked similarities in terms of developmental and environmental antecedents, as well as in natural course and response to treatment. Finally, the genes for generalized anxiety and major depressive disorder are the same, and there is substantial overlap with those of fear disorders.

Findings like these are prompting a major unification of the classification of this group of common emotional disorders in the revision of DSM-IV [24, 25]. It is hard to escape the conclusion that most of the meta-analyses and reviews of evidence of recent decades have rested upon scoping criteria which have employed a research diagnostic framework of distinctly questionable validity [26, 27].

The data described earlier indicate a little of how in the field of depression research supposedly empirical observations bear the unmistakable imprint of the presuppositions at work when making them. Basically, claims that a given group of observations or facts are neutral or theory-free are false. Rather than pretending like an ostrich that a theory in the background is a theory that does not exist, spelling out the theory being used allows its assumptions to be tested and the order it is capable of bringing to otherwise unconnected or unexplained findings to be appraised. That a theory can give an account of known facts is also valuable because of the way it points to what is going to be required of treatments and mental health service provisions if they are to be effective.

Scientific explanation as a whole proceeds on the basis of general laws or theories deduced on the basis of the findings gathered by observation and experiment. The explanatory theories which result serve not only to predict but also as a model necessary to guide the direction of future enquiries. It is openly acknowledged that observation and theory are not independent of each other and that this complicates enormously the way that scientific knowledge develops (see [28]).

It is by reason of these considerations that I give an outline of a developmental/psychoanalytic theory of depressive and allied disorders. In this way, we can evaluate what the data look like through this particular lens. My account of a psychoanalytic/developmental model is not the only one possible. For instance, other models, with slightly different emphases, have been put forward by Bleichmar [29] and by Blatt [30].

According to psychoanalytic theory, mourning and depression are seen as two different types of biopsychological reactions to loss. Any loss, or even a threat of loss, is considered to stimulate fear, anxiety, and anger. The emotional systems connected with fight/flight and with nurturance are immediately activated and disturbed. All the efforts of the organism may be mobilized to find ways of managing these potentially powerful emotional reactions. It was Freud [31] who compared the mental processes of mourning with those of depression. The fact that these two major states of mind share a common origin in loss explains why they coexist and interact with each other in significant ways. In relation to the psychological importance of loss, an important distinction is to be drawn between those losses which are accidental, such as the premature death of a love object, and those which are inbuilt. For example, the loss of infantile omnipotence arising from a child's experience of oedipal defeat as a result of observing his or her parents operating together as a couple is inevitable, and both the experience and its acceptance are necessary for normative development to proceed.

Fundamentally, the ability to mourn is an adaptive capacity involving relinquishment. Its psychological processes are seen as crucial to mental growth and lead to the return of a capacity to feel love, but at a new level. Mourning a lost love object or a lost state is one of the ways it is possible to recover from depression. This is what is happening over the course of the self-limiting depressions

which so commonly occur at the turning points of life. In those who are susceptible to less productive forms of depression—dysthymia, long-term "subthreshold" symptoms, recurrent, chronic, or "treatment-resistant" depression—the psychoanalytic model posits the existence of long-term difficulties with tolerating and adapting to the fundamental separateness of love objects.

The diathesis underlying depressive is seen as having been established over the course of infantile, childhood, and adolescent development. This has involved complex interactions between both genetic and suboptimal environmental influences, mainly mediated through early relationships. As a result, such individuals are unable to cope with the conflicting affects of love and of hate which necessarily arise in connection with absence and loss, while at the same time continuing to feel secure to some degree. Acceptance of loss and relinquishment are much more difficult in these circumstances. There is a desperate effort to spare the loved object from aggression which lends intense painfulness to the internal struggles of the depressed person. This "sparing" is a sine qua non of depression and is its ethical dimension.

Whereas in mourning, the person remains consciously preoccupied with whom or with what has been lost, the deeply depressed person often no longer knows why he is depressed. In the mourner, it is the world that is felt to have lost something or someone essential to its meaning. In depression, it is more noticeably the self that is experienced as diminished or bad. The self undergoes this alteration through its becoming identified with the lost object. Identification is seen as a largely unconscious mechanism which involves "becoming the same as." In depression, identification occurs on [AU1] the basis of sympathy with the object, sometimes out of guilt or sometimes as a way of keeping the relationship with the lost person present—within rather than absent—without. Because aggression cannot simply be wished out of existence, the attempt to spare the object inevitably means that the hostility must be turned inward. This turning inward of dissatisfied or critical feelings is how depressed people come to believe that they are bad, disliked, hated, or hateful. Many of the somatic and vegetative symptoms of depression arise from feelings of arousal, hostility, and fear which have been partially inhibited psychologically but which continue to operate at the somatic level as well as within the more familiar symbolic and representational levels of feeling and thought.

This turning of aggression and anxiety against the self is mediated by an internalized structure, the critical agency, known as the super-ego. Because of the dual character of this critical agency, the person shows both a persecutory fear of criticisms or reproaches and a sympathy for the object. In the course of a depressive illness, the severity of the super-ego increases, sometimes reaching delusional levels. The super-ego is colored by the individual's own much increased hostile and envious feelings so that, other things being equal, the more an individual controls his or her aggression toward others who are felt to be absent or frustrating, the more severe his or her superego becomes. Losing the love and approval of the super-ego represents the greatest of dangers to the self. Without that feeling of internal regard, there is much less reason to want to stay alive, as well as a reduced capacity to do so.

To summarize, the basis of the psychoanalytic/developmental theory of depression is the idea that those who are susceptible experience excessive neediness, dependence, and ambivalence in their love relationships and that these features have their origins in infancy and childhood. They predispose to depression after object loss and give rise to a vulnerability which is lifelong. The surface manifestations of this vulnerability might be a tendency to low mood, to excessive anxiety, and to various difficulties in stable love relationships. But the early feeding and affective bond experiences most centrally involved in the development of this vulnerability have an important role in the optimal development of both nurturance and fight/flight systems. The suboptimal functioning of these is also of importance in the later vulnerability to depressive disorders in adult life. The earlier developmental difficulties with object loss are continuously re-evoked in the course of all later relationships of work and love. This means that just as "every finding of an object is a re-finding" [31], every losing revives an earlier losing [32]. A mixture of genetic and environmental factors influences the various levels of severity, chronicity, and refractoriness at which depression exists. Some of these

factors are mediated through the development of an excessively severe or envious super-ego. Environmental (including inter-generational) and constitutional (genetic) factors continue to interact throughout life.

Reasons of space preclude my giving an account of the considerable amount of hard empirical evidence from developmental, attachment, genetic, neuroscientific, and psychoanalytic studies which can be marshaled in support of this model. For reviews of this, the reader is referred to Goldberg [33], Hill [34], Murray [35], Taylor [36], Fonagy [37], and a number of chapters in this volume.

The Outcome Evidence for Psychoanalytic/Psychodynamic Approaches in the Treatment of Depression

The observations made earlier lend support to the view of many researchers and clinicians that the current way of classifying and analyzing data dismembers otherwise instructive relationships that exist between the different types of symptoms found in patients suffering from a broader category of "common mental disorder." However, the idea of distinct, homogeneous mental "diseases" has dominated research and guideline development (including those of NIHCE) in the last 25 years. It has exerted great influence upon the type of study done and the kind of results available. What follows therefore goes against this template by looking more carefully at the results of some meta-analyses and trials that have used the concept of common mental disorders as a way of describing their study populations, and in which often measures of depressive symptoms have been employed.

Short-Term Psychodynamic Psychotherapy (STPP)

In line with general usage, the various shorter-term formats, whether structured or nondirective, will be referred to collectively as short-term psychodynamic psychotherapy (STPP). Most of the RCTs examining the efficacy of psychoanalytic or psychodynamic approaches have studied forms of STPP. Usually, this means no more than 20 sessions. These adaptations of psychodynamic or psychoanalytic psychotherapy usually involve a focus or some relatively structured model of construing the way patients present. Briefer therapies tend to employ psychoanalytic understandings of transference and the nature of the preconscious and unconscious conflicts in depression but not at all to the same extent as the practice in longer-term psychoanalytic treatments (LTPP). In LTPP, the patient is followed wherever the natural flow of their material proceeds. Evidence concerning these longer treatments is considered later in this chapter.

The Generic Efficacy of Psychological Therapy in the Treatment of Generic Disorders

In the mother of all meta-analyses, a meta-analysis of 45 meta-analyses of the results of a wide variety of brief psychological, educational, and behavioral treatment trials, in an equally wide range of nonpsychotic disorders, Lipsey and Wilson [38] came up with an estimate of the efficacy of psychological interventions as a "generic" type. They found an effect size of +0.76 for all forms of psychological therapy when compared to waiting list or minimal treatment controls. This indicates that the post-treatment condition of those who receive any form of brief psychological therapy will be in the

order of three-quarters of the standard deviation (of a very heterogeneous composite data-set) better off than those who do not receive them. This probably represents a worthwhile benefit, but, with the restricted capabilities of brief therapy research, it has been hard to demonstrate a clear order of difference between this size of effect and that associated with placebo or so-called "treatment as usual," especially should the treatment be of a good standard.

The reader will come to see how, in spite of numerous efforts to prove otherwise, a persistent thread of nonspecificity runs through the findings of psychological therapy research trials. This is the so-called "equivalence paradox." It refers to how difficult it has proved for this kind of short-term outcome research to demonstrate specific differences, whether according to degree of effect, condition, or type of psychological therapy. For this reason, experienced psychotherapy researchers such as Lambert [39] summarizes the previous decades of psychotherapy research with conclusions such as this: "…the generic efficacy of psychotherapy compared to no-treatment has been established for a long time…..….. from mildly disturbed persons with specific limited symptoms as well as from severely impaired patients……study after study, meta-analysis after meta-analysis, have empirically validated psychotherapy as a treatment" (p. 97). Yet, the same findings caused Luborsky [40] to coin the phrase the "Dodo bird effect," to describe the way every research camp in psychological therapy would present data which prove that its favorite is the best, while its rival is perfidious. This refers to Lewis Carroll's Alice in Wonderland, where to deal with the bitter squabbles at the end of the race, the Dodo announced, "everybody has won and all must have prizes." However, the Dodo verdict may provide false consolation. It is not the intention of managed care that all should have prizes. There are winners and losers.

The Efficacy of "Generic" Psychological Therapies in the Treatment of Depression

Robinson et al. [41] in their admirably lucid review of 58 controlled studies of the use of psychotherapy with patients with a formal diagnosis of depression found post-treatment (0.73) and follow-up effect sizes (0.68). These are of the same order as those quoted previously for a much wider group of disorders. Their figures indicated that collectively, these different types of psychological therapy offer a "moderate benefit" in the treatment of depression compared with no-treatment. While the authors' initial analysis found the effect sizes of cognitive, behavioral, and cognitive behavioral therapy to be approximately twice those of "general verbal therapies" (a category which included STPP), they found that this was a difference that disappeared when the treatment allegiance of the researcher and the quality of the trial were taken into account. Their ultimate finding was that there was "no reliable difference" between the efficacies of different forms of therapy in the treatment of depression. Each form was making roughly equal contributions to the final overall effect.

Cuijpers et al. [42, 43] have examined more closely the extent of the relationship between the quality of outcome studies and meta-analyses of psychological therapy for adult depression and the effect sizes they report. They found that only 11 of the 115 RCTs in their sample met all the quality criteria. The effect size reported by high-quality studies ($d=0.22$) was significantly smaller than in the other studies ($d=0.74$, $p<0.001$). Another way of putting this is in terms of number needed to treat (NNT) when these effect sizes translate to 8 in high-quality studies and 2 in lower-quality studies. This means that the findings of high quality studies suggest that eight individuals need to be treated in order to get one recovery more than found in the comparison group. These findings strongly suggest that the effectiveness of all forms of psychological therapy for adult depression has been overestimated by virtue of the low methodological quality of many RCTs, and that the effects of brief treatments of all kinds are considerably less than often stated.

The Efficacy of STPP Specifically, Compared to "No or Minimal Treatment" Controls in Patients with the Specific Diagnosis of Depression

The Cochrane review of Abbass et al. [44] found only two RCTs of sufficient quality giving data on the more exact question of the efficacy of STPP vs. "no treatment" or TAU in patients with a formal diagnosis of depression. This was out of a total of 23 RCTs examining STPP in the treatment of common mental disorders. In an earlier review, Leichsenring et al. [45] had found a third study. These three studies were:

1. In the treatment of adults with mild to moderate major depressive disorder (75% having an illness of less than 2 years duration), de Jonghe et al. [46] compared a form of STPP alone (max 16 sessions) with STTP and antidepressant medication. Although there were marked differences in their acceptability to patients, the efficacy of both formats in terms of effect size, imputed on the basis of the difference between baseline measures and measures at 24 weeks in the two groups, was large (i.e. ≥0.8), and more or less equivalent. Note how similar this is to the figure of Lipsey et al. [38]. This study is considered further later in this chapter.
2. In the treatment of major depressive disorder in the elderly, Thompson et al. [47] compared 16–20 sessions of STPP with an equivalent number of CBT and behavior therapy sessions and a waiting list control group. By the end of 6 weeks, patients in the treatment conditions showed improvement of the familiar order, whereas controls did not. The results of this study also bear upon the comparative efficacy of the different forms of psychotherapy that are considered further later on in this chapter.
3. In the treatment of postnatal depression, Cooper et al. [48] investigated the efficacy of three forms of brief interventions, one of which was ten sessions of psychodynamic therapy, compared to routine primary care in postpartum women meeting the criteria for major depressive disorder. At 4½ months, 70% of the psychodynamic cohort was judged to be recovered compared with 40% of the control condition (R.R 1.89). By the 9-month follow-up, however, this difference between treatment and usual care was no longer evident. Moreover, the three forms of therapy did not reduce the incidence of further episodes of postnatal depression. Psychodynamic psychotherapy did, however, seem to accelerate a recovery which would have occurred eventually in the majority of cases but more slowly. This study had a long 5-year follow-up, unlike most RCTs. It, and the benefit it found, is of particular importance because of the role of postnatal maternal depression in the intergenerational transmission of depression [35].
4. Subsequently, a fourth RCT study, not included in the Abbass review, has compared STPP with a waiting list condition and with a brief supportive therapy in patients with primary DSM-IV dysthymic disorder, depressive disorder not otherwise specified, or adjustment disorder with depressed mood [49]. Symptoms, assessed at baseline, the end of treatment, and after a 6-month follow-up showed a significant and large effect for STPP in comparison with untreated controls (SMD −1.09, 95% CI −2.04, −0.13), and a slightly smaller but still significant effect compared to the supportive therapy condition (SMD −0.97; -).03). The study was, however, a small one, total $N = 30$.

[AU2]

The Efficacy of STPP Compared with No or Minimal Treatment Controls in the Treatment of Common Mental Disorders

According to the psychoanalytic theory described previously, depression, anxiety, obsessional features, personality problems, and a variety of more subtle manifestations are all thought to be interconnected in a dynamic way. Like an increasing number of psychiatrists, psychoanalytically informed clinicians and researchers have long had serious reservations about the validity of using the single

diagnosis concept of depression in outcome trials. Many have argued that it makes much more sense to study the effects of treatment in patient samples which include mixed or combined disorders. As noted, the search criteria used in single disorder guidelines like those of NIHCE have excluded these sorts of studies:

1. Leichsenring et al. [45] found seven RCTs of sufficient quality which had examined STPP as against "no treatment" or TAU in a mixture of common mental disorders. STPP was found to be "significantly superior" to these "placebo" conditions with effect sizes ranging from 0.59 to 1.17, depending upon the domain measured (target problem, general psychiatric symptoms, and social functioning) and the measure used. "No treatment" conditions were found to have "before" and "after" effect sizes which ranged from 0.12 to 0.27, while TAU was found to be capable of more substantial benefits ranging from 0.22 to 0.95, depending presumably upon the intensity, adequacy, and quality of the TAU provided.
2. The review of Abbass et al. [44] of 23 RC trials examined the efficacy of STPP relative to no-treatment or TAU in the category of common mental disorders. They pooled patient samples which had originally been selected on the basis of several specific common disorder diagnoses. These included somatoform disorders (irritable bowel syndrome, chronic pain, etc.), anxiety and personality disorders, general psychiatric outpatients, as well as depression. Some of the studies were of chronic, difficult-to-treat patient populations with comorbid features. Only eight overlapped with Leichsenring's et al. [45] review. Combined, the 23 trials amounted to 1,431 patients. Results indicated that STPP (mean number of sessions =15) produced modest to moderate gains across a wide range of symptom categories for a wide variety of patients. Furthermore, these gains were often sustained or had increased at longer-term follow-up.
3. In respect of mixed disorders, the meta-analyses of Svartberg and Stiles [50], Crits-Christoph [51], and Anderson [52] all found STPP to be superior to no treatment or minimal treatment controls at both short- and longer-term follow-ups.

The Efficacy of STPP Compared to No or Minimal Treatment in Relation to the Depressive Symptoms of Patients Suffering from Common Mental Disorders

The composite, common mental disorder group assembled in the reviews of Leichsenring [45] and Abbass [44] included many patients with significant depressive symptomatology across the diagnostic categories. Fourteen of Abbass's 23 studies had employed measures of depression. Relative to controls in terms of relief from depressive symptoms, these showed over the short-term SMD 0.59 (≤3 months, 11 studies), the medium-term SMD 0.41 (3–9 months, five studies), and the "long"-term SMD 0.98 (≥9 months, six studies). These amount to moderate treatment effects, and, in line with this trend, the longer-term follow-up finding is more substantial.

The Efficacy of STPP Compared with Other Forms of Psychological Therapy

The meta-analyses of Crits-Christoph [51] and Anderson [52] similarly found STPP to be equal in efficacy to other forms of psychological therapy, as did the Leichsenring [45] study. Only one meta-analysis, that of Svartberg and Stiles [50], found STPP to be less effective than other short-term treatments such as CBT. Again, when controls for quality were introduced, this putative superiority was found to be much reduced.

The Efficacy of STPP Compared Specifically with Cognitive-Behavioral Therapy and Behavior Therapy in the Treatment of Depression

Churchill's et al. [53] systematic review of controlled trials of brief psychological treatments for depression, part of the UK's NHS Health Technology Assessment program, found six studies (five RCTs and one CCT) comparing STPP with CBT in formats of less than 20 sessions. Pooling dichotomous recovery/nonrecovery data suggested that the odds of recovery for patients receiving CBT were at least *twice* that for those receiving STPP by the end of the treatment.

The reviewers noted the possibility of a bias toward the CBT condition in these trials. Considerably more than half the studies were conducted by researchers with a CBT preference; these trials had used psychodynamic therapy only as a secondary comparison condition. When ratings for the quality of the trial and the quality of treatment delivery were taken into account, the reviewers found that the marked difference between CBT and STPP disappeared. Also, those trials where follow-up data were reported found there to be no difference between CBT and brief psychodynamic treatments. In two of these studies, follow-up was at 3 months, and in the third at 1 year. The rest provided no follow-up data.

Leichsenring [54] came to similar conclusions. He confirmed that STPP was associated with statistically significant reductions in depressive symptoms before and after treatment. Out of a total of 60 possible comparisons between STPP and CBT, he found no difference in 58, while two showed a small effect in favor of CBT. He calculated the mean before and after success rates of STPP and CBT as 46% and 54%, respectively—equivalent to a small effect size in favor of CBT. Wampold et al. [55] came to similar conclusions.

The allegiance of the researcher has consistently been shown to have a marked influence upon the size and direction of the effect reported (the researcher effect!). Cuijpers et al. [42, 43] examined the effect sizes reported in trials comparing psychological treatments with a control condition, including other forms of psychological therapy and medication, in the treatment of depression. In their sample of 117 randomized trials, the reported overall mean effect size was 0.67. However, asymmetry in the funnel plots suggested there were 26 missing studies presumably not reported, and imputing the results of these pointed to a true effect size of 0.49. Eighty nine of 175 comparisons were concerned with the efficacy of CBT, and they showed this same highly significant evidence indicating bias and overestimation of the efficacy of CBT. However, the same indications of bias were also found for other forms of psychotherapy (including STPP). Research on psychotherapy for women with postpartum depression (most importantly the study of Cooper et al. [48], quoted previously) and on interpersonal psychotherapy were honorable exceptions. Neither showed any evidence of publication bias, although the numbers of such studies were small.

The Efficacy of Psychotherapy Compared with Antidepressants

According to Roth and Fonagy [56], when psychotherapy and medication are offered alone, they seem to be of roughly equivalent efficacy. Most, but not all, of this comparison data concern IPT and CBT studies. The earlier review of Robinson et al. [41] found 15 studies examining this question, including a few looking at the effect of combining psychological therapy with antidepressants. Eight of these 15 studies were of CBT, three were of behavioral therapy, while four tested what the authors describe as "general verbal" therapy which includes dynamic approaches. The studies yielded a small effect size in favor of psychological therapy, but this may be artifactual.

In general, the interpretation of these and other similar findings is not straightforward. Most comparative trials do not include drug placebo controls. Obviously, in the case of psychological therapy,

effective concealment or blind conditions are extremely difficult if not impossible to contrive. Also, the more that allegiance effects are taken into account, the more difficult it becomes to demonstrate the existence of reliable, stable patterns of differences between psychological therapy and pharmacotherapy. Furthermore, meta-analyses re-examining the results of trials of antidepressant medication have also provided strong indications of publication bias—the preferential reporting of those trials which show a positive effect—an effect known colloquially as the "file-drawer effect" (see [57, 58]). The file-drawer effect has certainly led to an inflation of the efficacy of antidepressants. However, as was discussed in the preceding section, similar evidence of publication bias and selective reporting has now been found to be operating in respect of psychological therapy trials.

Salminen [59] compared the efficacy of STPP (16 weekly sessions) and fluoxetine in major depressive disorder of mild or moderate severity in a primary care setting. Both treatments were found to be highly effective in reducing the HDRS ($p < 0.0001$) and BDI ($p < 0.0001$) scores, as well as in improving functional ability ($p < 0.0001$). There were no statistically significant differences between the two treatments (SMD 0.03; 95% CI −0.52, 0.58). Forty of the 51 patients completed a 4-month follow-up when it was found that 57% of the STPP group and 68% of those in the fluoxetine group showed remission (=HDRS ≤7). While the results of this trial indicate that both STPP and fluoxetine are effective in reducing symptoms and in improving functional ability of patients with mild or moderate depression, the interpretation of the findings is, as is so often the case, limited by the absence of a placebo condition, the short and incomplete follow-up, and the wide confidence intervals.

To estimate head-to-head the efficacy of antidepressants in comparison to a 16-session short psychodynamic supportive psychotherapy (SPSP) in a somewhat larger sample, de Maat et al. [60] combined the data sets of three earlier RCTs [46, 61]. These studies, which are examined in more detail later in this chapter, had examined the efficacy of this SPSP combined with antidepressants vs. antidepressants alone in patients with mild to moderate major depressive disorder [61], with combined antidepressants and SPSP vs. SPSP alone [46], and finally comparing two intensities of combined therapy (medication plus eight or 16 sessions of SPSP). Hamilton ratings showed no difference between this short psychodynamic supportive psychotherapy (SPSP) and pharmacotherapy, but patients and therapists seemed to rate the psychotherapy better than medication in regard to symptom reduction, but paradoxically not in improving quality of life.

There is some evidence [62] that antidepressants are more efficacious with more severely depressed patients than in those with mild to moderate severity, or in those where complex psychosocial factors of history and personality are significant parts of the presentation.

The Efficacy of Combined Treatments

Augmenting STPP with Antidepressants (Versus STPP Alone)

The study of de Jonghe et al. [46] cited previously examined whether the addition of antidepressants to 16 sessions of STPP would improve on the outcome of major depressive of mild to moderate severity treated with STPP alone. A sample of approximately 200 psychiatric outpatients with major depressive disorder was treated and followed up over a 6-month period.

Both packages produced broadly equivalent positive effects. The success rate in the STPP alone arm was 73% as against 81% in the combined, with pre–post HRSD effect sizes of 1.22 and 1.53, respectively. By 24 weeks, Hamilton ratings had fallen from an initial average of 18 to one of 10. The comparison findings indicated a small benefit in favor of combining antidepressants with STPP, but these only reached significance in respect of the patient's self-report measure (with a medium effect size of 0.49). Because there was no drug placebo control in the trial, this effect cannot with certainty be attributed to the pharmacological agent.

Other important findings concerned the strength of patients' attitudes to the two kinds of treatment. Although 25% of psychotherapy patients broke off their treatment, psychotherapy still seemed overall to be more acceptable than drugs. More patients refused to enter the pharmacotherapy arm in the first place, and by 6 months, a further 35% had withdrawn from it.

Augmenting Antidepressants with STPP (Versus Antidepressants Alone)

As already indicated, de Jonghe et al. [61] investigated the question of combined therapy the other way around: augmenting antidepressant medication with short psychodynamic supportive psychotherapy (SPSP) and comparing the combination with antidepressants alone in patients with mild to moderate major depressive disorder. SPSP plus drugs was found to be more effective than medication alone in terms of both symptom reduction and quality of life.

Burnand et al. [63] investigated the effect of augmenting antidepressant medication time with a brief ten session form of STPP, administered in this case by well-trained nurses, to enhance the outcomes of patients in an acute phase major depressive episode. The patients had been referred to an outpatient psychiatric service and were suffering from moderate to severe degrees of depression; half had suffered previous episodes; half had some form of personality disorder. They were as a whole slightly more ill than the de Jonghe sample. The final sample analyzed consisted of 74 patients randomized to the two conditions—antidepressants (clomipramine, mainly) combined with supportive time with a designated key worker vs. antidepressants plus ten sessions of STPP with a trained nurse therapist.

After the 10-week treatment period, the mean Hamilton scores in both groups had reduced from ≈24 to ≈9 ($p > 0.001$) but with little difference between the comparison conditions on this measure. However, the combination of STPP and antidepressants was better than antidepressants alone on several other important indices: patients who received STPP were significantly less likely to still meet the criteria for MDD after 10 weeks (9%) than those who did not (28%). The combination group needed significantly fewer days off work (46 vs. 57), and they had both a lower rate and fewer days of hospitalization. Cost benefit comparisons showed that the combined STPP/antidepressants had resulted in savings of $2,311 over the 10-week period. For those patients in full employment before they became ill, savings were greater ($3,394).

The Evidence Concerning the Treatment of Complex, Chronic, or "Treatment-Refractory" Depression

Many of the patients in the samples of the studies cited previously were selected to exclude comorbidity [64]. Since it is now understood that comorbidity is better regarded as an intrinsic characteristic, particularly of those disorders which tend to be more chronic, long term, difficult to treat and recurrent, it cannot be assumed that the evidence of effectiveness presented so far applies to many of the patients actually encountered in clinical practice.

Addressing this complexity factor, Kool [65] investigated the possibility of differential effectiveness in using antidepressants with or without STPP (16 sessions) in an RCT sample of 128 depressed outpatients with and without personality disorders. Findings indicated that STPP had more effect in those with both PD and depression than in those without (SMD −1.15 vs. SMD 1.50). At follow-up (40 weeks), patients receiving the combined treatment showed a significant reduction in "comorbid" personality disorder, both in those who had recovered from their depression and in those who had not. In the medication-alone condition, only those who had recovered from their depression showed a reduction in their personality disorder. As might be expected, the finding was most striking for Cluster C psychopathology (anxious inhibited) whereas those with cluster B pathology (dramatic) changed less.

Additionally, and importantly, the reader will recollect that it is well-established that at least 10% of depressed patients (depending upon the reference sample) go on to suffer from a serious long-term relapsing condition. This is sometimes placed under the heading of "treatment-resistant" or "treatment refractory depression." These patients show only partial recovery between episodes, limited response to treatments of whatever kind, and in some cases active treatment may seem to cause them to deteriorate. While there is general agreement that the term is simply a descriptive one and that it denotes a heterogeneous group, the lives of patients with these disorders may be seriously disabled. Most treatments seem to have only limited benefits for them.

Stimpson et al. [66] examined various pharmacological approaches to the treatment of patients with unipolar depression who had not responded to a minimum of 4-week antidepressant treatment and found 17 RCTs. They concluded that there was little in the way of evidence to guide the management of patients who had not responded to a first course of antidepressants. Within their criteria, they found no satisfactory trials of psychological therapy with chronic or treatment resistant depression. By using more inclusive criteria, McPherson et al. [67] did find a few studies of psychological treatments, including single case studies. These mainly employed CBT. Although they mostly showed reduction in symptoms, they all had short follow-ups and very small numbers. Interestingly, and presumably in response to the severity of the psychopathology, the CBT treatments involved seemed to be showing a marked tendency to become longer than the short 10 or 20 sessions which has been a principle selling point of the method. In one study, this reached a total of 39 sessions over 8 months—something like a medium-length psychodynamic treatment!

Abbass [68] reported on a pilot study of a small series of patients [10] diagnosed with "treatment resistant depression" (TRD) treated with Davanloo's [69] intensive short-term dynamic psychotherapy mostly over a 20-week period. In this study, TRD was defined as an inadequate response to adequate durations and dosages of at least two different classes of antidepressants. Eight patients remitted (Hamilton-D >22 → <8). Concomitantly, there were beneficial changes in the patient's ratings on interpersonal problems, medication usage, occupational status, and costs of healthcare and social support. The scores of the other two patients moved from 31 → 12 and 27 → 14, respectively. In all except these two patients (who both had had short courses of therapy—six and eight sessions), changes were maintained over a 6-month follow-up.

Two RCT trials are currently under-way with chronic or TRD patient groups. The Tavistock Adult Depression Study (Publication ID M0001169680 at http://www.nihr.ac.uk) is examining the efficacy of a "medium-term"[1] treatment—60 sessions of weekly psychoanalytic psychotherapy vs. TAU in a patient sample of 127 suffering from treatment-resistant depression—defined as at least two failed treatments, as well as meeting the criteria for MDD. The usual duration of illness in the sample is 10–15 years, and most patients have been found to meet the criteria for at least one DSM axis II disorder. The trial is due to report in 2012/2013. A similar multicenter German RCT combined with a naturalistic arm, and with a CBT comparison group, began in 2007. Die Langzeittherapie bei chronischen Depressionen (LAC) Studie, led by Professor M. Leuzinger-Bohleber of Frankfurt-am-Main's Sigmund Freud Clinic, is examining a longer psychoanalytic psychotherapy treatment. It has an intended sample of 240. (See http://www.sfi-frankfurt.de/forschung/forschungsfeld-2/depressionsstudie.html.)

[1] Mercifully, there is no generally accepted convention about the terminology to be employed for treatment length or intensity. Abbass et al. [44] categorize all treatments of less than 40 weeks as short term (STPP), whereas others reserve this term for treatments of less than 20 sessions. Likewise, there are different usages concerning the actual length of treatments to which the designations "medium" and "long" term should apply and also what is meant by "intensive."

[AU3]

Longer-Term Psychoanalytic Treatments (LTPP)

In Chapter 2, Rabung and Leichsenring examine longer-term psychoanalytic treatments in detail. There have been two important recent reviews of the effectiveness of LTPP in a range of pathologies ([70, 71], and Chap. 2 in this volume). Here, therefore, I will only consider those issues which have some direct bearing upon the value of longer-term psychoanalytic therapies in the treatment of depression and in relation to depressive symptoms. However, in the context of the dull, uniform impression of general equivalence between all forms of treatment given by the outcomes measured in predominantly short-term RCT depression research, it should be noted that the investigation of longer-term treatments does offer at least some possibility of findings about the possibility of more fundamental and deep-seated changes in an individual's susceptibility to depression, and into the nature of the kind of changes which might be capable of an effect which reduces vulnerability. Such changes may or may not be causally specific to a psychoanalytic approach to the treatment of depressive disorder.

However, as might be predicted, there are few completed studies of longer-term psychotherapy or psychoanalysis with patient samples selected on the basis of a single diagnosis, such as major depressive disorder. As already noted, the psychoanalytic concept of mental disorder is fundamentally different from the nosology associated with research diagnostic criteria. Equally, there are very few completed studies using random allocation controlled trial designs which evaluate medium- or longer-term therapies. These are therapies which require the patients' personal choice, mental work, and their deep engagement. Especially over the medium and longer term, these requirements of human relatedness do not sit at all easily with the choiceless quality basic to the principle of randomization. Except in respect of very unusual conditions and circumstances, practically, ethically, and in terms of acceptability, it is not at all feasible to use random allocation for longer-term psychological treatments. In most clinical contexts, more naturalistic, case-controlled cohort studies offer a better prospect of findings that are both solid and knowledge advancing. Vandenbroucke [72] has examined the necessary conditions in which observational studies become substantially more credible than those of randomized trials. At the same time, there will have to be scrupulous attention to detail if such studies are ever to have the capacity to meet the challenges posed by skeptical and critical scrutiny.

The Efficacy of Long-Term Versus Short-Term Psychodynamic/ Psychoanalytic Treatments in Depression

The Helsinki Psychotherapy RCT ([73, 74] and Chap. 4 in this volume), however, did compare the effectiveness of two forms of short-term therapy with a long-term psychodynamic psychotherapy therapy (LTPP). So far, follow-up data over 3 years have been reported. The study also has an arm for full psychoanalysis with further follow-up data. The two short-term treatments were a problem-solving solution (SFT) (≈10 sessions, including family work over 8 months, Lambert [75]) and a form of STPP based upon the Malan [76] model (20 session over 6 months). The long-term treatment was a 2–3 sessions weekly psychodynamic therapy over 3 years (average number of sessions = 232). The large sample of 326 patients were a mild to moderately disturbed group aged 20–45 years, referred and treated as outpatients with a DSM IV mood or anxiety disorder for at least 1 year. Sixty-eight percent met the criteria for MDD, 44% met those for generalized anxiety disorder, 18% met those for personality disorder, and ≈9% had made a suicide attempt.

All three interventions were associated with significant reductions in all symptom measures (depression, anxiety, and general). Over the 3-year period, the average reduction of the BDI was 51% (with effect sizes ranging from 0.87 to 1.52). At entry, the mean BDI was 18; in those getting

the two short treatments, this had come down to ≈10, in those getting the long term to ≈7. These benefits are comparable to those found in studies involving CBT. Patients receiving the short-term treatments typically showed an early response which reached an upper limit by 12 months. These gains proved lasting over the entire 3-year follow-up.

In contrast, the group receiving LTPP initially lagged behind, showing significantly less improvement over the first year. However, the gains in this group showed a pattern of gradual increase so that by the 3-year point, their symptoms were less than those of the short-term treatments on all measures; with respect to anxiety, they were four times more likely to be recovered; with respect to the depression measures, the difference was not of the same degree. About 20% of those who had met personality disorder criteria no longer did so at 7 months. In the STPP group, this proportion had risen to 46% by 12 months. This improvement did not occur in the SFT group.

The Effectiveness of Longer-Term Psychoanalytic Psychotherapy/ Psychoanalysis (LTPP) in the Treatment of Depression

In their review, Leichsenring and Rabung [71] found five studies of LTPP where a majority of the patients could be ascertained to have been suffering from complex, often comorbid depressive disorders. The studies were Høglend et al. [77], Huber et al. [78], Grande et al. [79], and Leichsenring et al. [80]. The condition was chronic in 71% of the total of 274 patients involved in these five studies. [AU4]

Looking at the sample of Leichsenring et al. [80] (N=36), an indication of the severity and clinical representativeness of the patients involved can be seen from the fact that almost nine-tenths of those with a diagnosis of depressive disorder were also suffering from comorbid disorders such as somatoform, anxiety, or obsessive-compulsive states. In terms of overall outcome, general psychiatric symptoms, and social functioning, LTPP was found to have yielded significant and large effect sizes (all>0.80). In terms of depressive symptom, at 1 year, the effect size was 1.38. As in other research in this area, it was found that symptoms are the first to remit (with changes in personality functioning emerging with a slower gradient).

Evidence from Retrospective Cohort Studies of Effectiveness

A carefully designed naturalistic long-term follow-up in Germany of 402 patients (of whom 128 had affective disturbances) who had received psychoanalysis and long-term psychoanalytic psychotherapy [81] showed that a clinically representative group of patients with significant depressive symptomatology had moved into the normal range of scores. At long-term follow-up, the changes in this group of patients were such that they were doing better in terms of days off work than the population norm. Importantly, the study included in-depth qualitative interviews that made it possible to discern distinct and differing patterns of change in the way that various personality types managed their thoughts and feelings. These patterns included the emergence of reflective functioning [82].

Several studies have confirmed the clinical impression that improvement continues after psychodynamic or psychoanalytic psychotherapy ends (the “sleeper effect”). Sandell et al. [83] and Blomberg et al. [84] demonstrated that patients in the psychoanalysis cohort seemed to continue to gain in strength and capacity after treatment had ended. This pattern was also found in the study of Kopta et al. [85].

Blatt and Shahar [86] have addressed the question of the unique nature and effectiveness of psychoanalysis. According to their results, psychoanalysis contributed significantly to the development of adaptive interpersonal capacities and to the reduction of maladaptive interpersonal behavior, especially with more self-reflective patients. Supportive–expressive therapy, by contrast, only yielded a reduction of maladaptive interpersonal behavior and only with dependent, unreflective patients.

Discussion and Conclusions

Do Short-Term Treatments Cure Depression?

So far in this chapter, as in the literature of psychological therapy outcome in general, estimates of efficacy have mostly been reported in terms of effect sizes. An effect size is the ratio of the difference between the mean of one group and that of another, to the standard deviation of the chosen measure in the initial sample. Generally, an effect size of 0.3 is regarded as small, one of 0.5 as medium, and one of ≥0.8 as large. However, as Jacobson et al. [87] have pointed out, what these statistical values mean in terms of change for an individual patient is entirely opaque. To deal with this, a variety of more rigorous tests have been developed to indicate whether a given change has moved someone from outside the dysfunctional range or to within a functional one [88]. Unfortunately, in this field, the data needed to calculate these statistical measures of clinical significance are often unavailable. In addition, we are still depending upon the assumption that the measures employed actually capture the true nature and extent of the patient's disability or disorder.

Robinson et al. [41] looked at this sort of issue by asking whether the clinical effects of short-term psychological and pharmacological treatments amounted to a cure of depression. In their sample of studies, the mean pretreatment BDI was 21.8 (when 0–13 is considered minimal, 14–19 mild, 20–28 moderate, and 29–63 severe). Post-treatment, the score had fallen by 10 points to 11.8. The corresponding figures for untreated controls were 20.7 and 18.1. The mean BDI score of unselected samples of the general population is 7.0. Amongst that fraction of the general population that is contented, this comes down to 4.9. These treatment effects therefore move patients who are moderately outside the normal range to within one standard deviation of the general population mean, and to within 1.5 SDs of the contented group amongst us. These are not trivial effects but, even by the restricted sensitivity of the BDI, they suggest that on a good day, the previously moderately ill but average responder to short-term treatments is still more depressed than most of us on a bad day, and is still further off from the ideal of normative functioning. The consistency of these estimates points again toward there being a ceiling to what can be achieved with short-term treatments. Additionally, there are more variables in depressive disorders than are measured by the BDI.

Does This Evidence Base Need to Remain an Industry of Small Differences?

It is still possible to sum up the current state of evidence with the following blanket conclusion: antidepressant medication and the different varieties of short-term psychological therapies are capable of generating an improvement of about 12–13 points on the BDI over the short term, but there is not much to choose between them, and there is little evidence about the sustainability of these changes. Also, as clearly indicated by the findings of the NIMH study, the "placebo" effect in trials of treatments for depression can be consistently large, amounting to an average of ten points improvement.

Viewed like this, it is understandable that many clinicians throw up their hands at a research base which often seems more like a heap of dust. It does seem that in respect of treatment research into depression (and other common mental disorders), the dodo bird's judgment, at least, lives on. It lives on in an evolutionary backwater maintained by three factors: the stubborn conviction that short-term RCT trials are guardians of truth, the continued use of instruments that are insensitive to the many aspects of patients' dysfunctionality which lie outside symptom frames of reference, and a wish that short psychological treatments can be highly potent that verges on the homeopathic. If these human factors stay as they are, the difficulty of demonstrating informative differences between therapies will continue until one side has overwhelmed the other, probably through sheer weight of numbers.

Within the remit of this chapter, it has only been possible to present the evidence from outcome trials, and that in a summarized, incomplete way. However, when we questioned the inclusion and exclusion criteria of guidelines and the effect of continuing to use specific psychiatric diagnoses as an organizing principle, we found that relevant evidence had been omitted. Moreover, when we allow clinical experience a little more authority over short-term outcome research trials, other points come into view. If we proceed to admit evidence from naturalistic studies, from other kinds of science, a yet more interesting range of conclusions becomes possible. Epidemiology, observations of natural course, developmental psychopathology, studies of psychosocial factors, life events, experimental studies, and psychoanalytic findings themselves show that we know a great deal about the nature and origins of depression. En passant, these different disciplines provide supporting evidence for psychoanalytic theories about depression.

While this more sophisticated conception of the evidence base cannot tell us directly which treatments will prove effective, it does enable us to know the parameters of the disorder and the limits on the plasticity of persons. These are essential guides to the potential of different treatments. They are scales against which to measure some of the wilder claims. Psychiatry could go further and reintroduce other ideas such as "profound" vs. "banal" to counter the narrowness of the sometimes pseudo-statistical significance/nonsignificance formula.

Conclusions

This wider view of evidence is consistent with:

1. STPP effect sizes approximately similar to other therapies and, like them, superior to minimal treatment controls.
2. Perhaps a tendency for the CBT effect to come into play more quickly than that of STPP but with longer follow-ups this latency effect disappears.
3. Longer-term psychodynamic and psychoanalytic therapies, by addressing the dysfunctions of persons, may have the potential to go above an efficacy ceiling which operates in short-term treatments. There are clear indications that patients in longer-term psychoanalytic approaches begin to manifest qualitatively different types of change which can move them further into the normal range.
4. While studies of combined drug/psychological therapy may be mixed, there is some evidence of "added value" from adding STPP to medication. Also, adding medication to STPP, especially where vegetative symptoms are pronounced, seems sensible. There seem to be no trials addressing benefits from sequences of CBT → STPP or STPP → CBT.
5. The reasons patients have for preferring one treatment over another, including one form of psychological therapy rather than another, have been neglected.
6. The value of qualitative in-depth studies of single or small numbers of cases and of more naturalistic designs has been underestimated.

7. A lot more should be thought about the placebo effect and about the disputes over valuable but limited gains. Like positive transference, the psychological mechanisms involved in the placebo response in depression have a role in normal emotional life. The belief in good medicines dispensed by good people who can provide succor is one of those features of normal mental life which fails in depression. It is possible that the restoration of this belief-function may be important for recovery in depression.

All of these considerations are pointers toward optimal or enhanced services for patients suffering from the complex range of disorders that goes under the general rubric of depression.

Declaration of interest: The author is the clinical lead of the Tavistock Adult Depression Study, an RCT evaluating 18 months of weekly psychoanalytic psychotherapy in the treatment of chronic, "treatment-resistant" depression.

References

[AU5]

1. Hazlitt W. The spirit of the age: or contemporary portraits – various publishers/editions; 1825.
2. Ashton R. The German idea. Cambridge: Cambridge University Press; 1980.
3. Westen D, Novotny CM, Thompson-Brenner H. The empirical status of empirically supported psychotherapies: assumptions, findings, and reporting in controlled clinical trials. Psychol Bull. 2004;130:631–63.
4. Luyten P, Blatt SJ, Van Houdenhove B, Corvelyn J. Depression research and treatment: are we skating to where the puck is going to be? Clin Psychol Rev. 2006;26:985–99.
5. Bruner J. Actual minds, possible worlds. Cambridge, MA: Harvard University Press; 1986.
6. Salkovskis PM. Demonstrating specific effects in cognitive and behavioural therapy. In: Aveline M, Shapiro DA, editors. Research foundations for psychotherapy research. Chichester: Wiley; 1995.
7. Robins LN, Regier DA. Psychiatric disorders in America: the epidemiologic catchment area study. New York: The Free Press; 1991.
8. Sargeant JK, Bruce ML, Florio LP, et al. Factors associated with 1-year outcome of major depression in the community. Arch Gen Psychiatry. 1990;47:519–26.
9. Mueller TI, Leon AC, Keller MB, Solomon DA, Endicott J, Coryell W, Warshaw M, Maser JD. Recurrence after recovery from major depressive disorder during 15 years of observational follow-up. Am J Psychiatry. 1999;156(7):1000–6.
10. Hirschfeld RM. Depressive illness: diagnostic issues. Bull Menninger Clin. 1991;55:144–55.
11. Judd LL. The clinical course of unipolar major depressive disorders. Arch Gen Psychiatry. 1997;54:989–91.
12. Judd LL, Akiskal HS, Paulus MP. The role and clinical significance of subsyndromal depressive symptoms (SSD) in unipolar major depressive disorder. J Affect Disord. 1997;45:5–17.
13. Paykel ES. Historical overview of outcome of depression. Br J Psychiatry. 1994;165(suppl. 26):6–8.
14. Spijker J, de Graaf R, Bijl RV, et al. Duration of major depressive episodes in the general population: results from The Netherlands Mental Health Survey and Incidence Study (NEMESIS). Br J Psychiatry. 2002;181:208–13.
15. Goldberg D, Privett M, Ustun B, et al. The effects of detection and treatment on the outcome of major depression in primary care: a naturalistic study in 15 cities. Br J Gen Pract. 1998;48:1840–4.
16. Coryell W, Akiskal H, Leon AC, et al. The time course of untreated major depression: uniformity across episodes and samples. Am J Psychiatry. 1994;51:405–10.
17. Lee AS, Murray RM. The long-term outcome of Maudsley depressives. Br J Psychiatry. 1988;153:741–51.
18. Goldberg D, Goodyer I. The origins and course of common mental disorders. London: Routledge; 2005.
19. Angst J, Dobler-Mikola A. The Zurich study II. The continuum from depressive to pathological mood swings. Eur Arch Psychiatr Neurol Sci. 1984;234:21–9.
20. Angst J, Dobler-Mikola A. The Zurich study VI. A continuum from depression to anxiety disorders? Eur Arch Psychiatr Neurol Sci. 1985;235:179–86.
21. Kessler RC, Bergland P, Demler O, Jin R, Koretz D, Merikangas K, Rush J, Walters E, Wang P. The epidemiology of major depressive disorder: results from the national comorbidity survey replication. JAMA. 2003;289:3095–105.
22. Kessler R, Chiu WT, Demler O, Walters EF. Prevalence, severity and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005;62:617–27.

23. Vollebergh WAM, Iedema J, Bijl RV, de Graaf R, Smit F, Ormel J. The structure and stability of common mental disorders: the NEMESIS study. Arch Gen Psychiatry. 2008;58:597–603.
24. Andrews G, Anderson TM, Slade T, Sunderland M. Classification of anxiety and depressive disorders: problems and solutions. Depress Anxiety. 2008;25:274–81.
25. Goldberg DG. Should our major classifications of mental disorders be revised? Br J Psychiatry. 2010;196:255–6. doi:10.1192/bjp.bp. 109.072405.
26. Blatt SJ, Zuroff DC. Empirical evaluation of the assumptions in identifying evidence based treatments in mental health. Clin Psychol Rev. 2005;25:459–86.
27. Luyten P, Blatt SJ. Looking back towards the future: is it time to change the DSM approach to psychiatric disorders? The case of depression. Psychiatry. 2007;70:85–99.
28. Braithwaite RB. Scientific explanation. Cambridge: Cambridge University Press; 1955.
29. Bleichmar H. Some subtypes of depression and their implications for psychoanalytic treatment. IJPA. 1996;77:935–62.
30. Blatt SJ. Experiences of depression: theoretical, clinical and research perspectives. Washington, DC: American Psychological Association; 2004.
31. Freud S. Mourning and melancholia. Reprinted (1953–1974) in the standard edition of the complete psychological works of Sigmund Freud. London: Hogarth Press; 1917. 14, p. 237.
32. Klein M. Mourning and its relation to manic–depressive states. Reprinted (1975). In: The writings of Melanie Klein. Vol. 1: Love, guilt and reparation and other works 1921–1945. London: Hogarth Press; 1940.
33. Goldberg D. The interplay between biological and psychological factors in determining vulnerability to mental disorders. Psychoanal Psychother. 2009;23(3):236–47.
34. Hill J. Developmental perspectives on adult depression. Psychoanal Psychother. 2009;23(3):200–12.
35. Murray L. The development of children of postnatally depressed mothers: evidence from the Cambridge longitudinal study. Psychoanal Psychother. 2009;23(3):185–99.
36. Taylor D. Consenting to be robbed so as not to be murdered. Psychoanal Psychother. 2009;23(3):263–75.
37. Fonagy P. Postscript. Psychoanal Psychother. 2009;23(3):276–80. doi:10.1080/02668730903227305.
38. Lipsey MW, Wilson DB. The efficacy of psychological, education, and behavioral treatment: confirmation from meta-analysis. Am Psychol. 1993;48:1181–209.
39. Lambert MJ. Psychotherapy outcome research: implications for integrative and eclectic theories. In: Norcross JC, Goldfried MR, editors. Handbook of psychotherapy integration. New York: Basic Books; 1992.
40. Luborsky L, Singer B, Luborsky L. Comparative studies of psychotherapies. Is it true that "everybody has won and all must have prizes"? Arch Gen Psychiatry. 1972;32:995–1008.
41. Robinson LA, Berma JS, Neimeyer RA. Psychotherapy for the treatment of depression: a comprehensive review of controlled outcome research. Psychol Bull. 1990;108:30–49.
42. Cuijpers P, Smit F, Bohlmeijer E, Hollon SD, Andersson G. Efficacy of cognitive-behavioural therapy and other psychological treatments for adult depression: meta-analytic study of publication bias. Brit J Psychiat. 2010;196:173–8.
43. Cuijpers P, van Straten A, Bohlmeijer E, Hollon SD, Andersson G. Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences. 2010;40(2):211–23. [AU6]
44. Abbass AA, Hancock JT, Henderson J, et al. Short-term psychodynamic psychotherapies for common mental disorders. Cochrane Database of Syst Rev. 2006; issue 4, CD004687.
45. Leichsenring F, Rabung S, Leibing E. The efficacy of short-term psychodynamic psychotherapy in specific psychiatric disorders. A meta-analysis. Arch Gen Psychiatry. 2004;61:1208–16.
46. de Jonghe F, Hendriksen M, van Aalst G, et al. Psychotherapy alone and combined with pharmacotherapy in the treatment of depression. Br J Psychiatry. 2004;185:37–45.
47. Thompson LW, Gallagher D, Breckenridge JS. Comparative effectiveness of psychotherapies for depressed elders. J Consult Clin Psychol. 1987;55:385–90.
48. Cooper PJ, Murray L, Wilson A, et al. Controlled trial of the short- and long-term effect of psychological treatment of post-partum depression. 1: Impact on maternal mood. Br J Psychiatry. 2003;182:412–9.
49. Maina G, Forner F, Bogetto F. Randomized controlled trial comparing brief dynamic and supportive therapy with waiting list condition in minor depressive disorders. Psychother Psychosom. 2005;74(1):43–50.
50. Svartberg M, Stiles TC. Comparative effects of short-term psychodynamic psychotherapy: a meta-analysis. J Consult Clin Psychol. 1991;59:704–14.
51. Crits-Christoph P. The efficacy of brief dynamic psychotherapy: a meta-analysis. Am J Psychiatry. 1992;159:325–33.
52. Anderson EM, Lambert MJ. Short-term dynamically oriented psychotherapy: a review and meta-analysis. Clin Psychol Rev. 1995;15:503–14.
53. Churchill R, Hunot V, Corney R, et al. A systematic review of controlled trials of the effectiveness and cost-effectiveness of brief psychological treatments for depression. Health Technol Assess. 2001;5:1–173.

54. Leichsenring F. Comparative effects of short-term psychodynamic psychotherapy and cognitive–behavioural therapy in depression: a meta-analytic approach. Clin Psychol Rev. 2001;21:401–19.
55. Wampold BE, Minami T, Baskin TW, et al. A meta-(re)analysis of the effects of cognitive therapy versus 'other therapies' for depression. J Affect Disord. 2002;68:159–65.
56. Roth A, Fonagy P. What works for whom? A critical review of psychotherapy research. 2nd ed. New York: Guilford; 2004.
57. Turner EH, Matthews AA, Lindardatos E, Tell RA, Rosenthal R. Selective publication of antidepressant trials and its influence upon apparent efficacy. N Engl J Med. 2008;358:252–60.
58. Kirsch I, Deacon BJ, Huendo-Medina TB, Scobaria A, Moore TJ, Johnson BT. Initial severity and antidepressant benefits: a metaanalysis of data submitted to the food and drug administration. PLoS Med. 2008;5:e45. doi:10.1371/journal.pmed.0050045.
59. Salminen JK, Karlsson H, Hietala J, Kajander J, Aalto S, Markkula J, Rasi-Hakala H, Toikka T. Short-term psychodynamic psychotherapy and fluoxetine in major depressive disorder: a randomized comparative study. Psychother Psychosom. 2008;77(6):351–7. doi:10.1159/000151388.
[AU7] 60. de Maat S, Dekker J, Schoevers R, van Aalst G, Gijsbers-van Wijk G, Hendriksen M, Kool SJ, Peen J, Van R, de Jonghe F. Depress Anxiety. 2008;25:565–74.
61. de Jonghe F, Kool S, Aalst G, Dekker J, Peen J. Combining psychotherapy and antidepressants in the treatment of depression. J Affect Disord. 2001;64:217–29.
62. Thase ME, Greenhouse JB, Frank E, et al. Treatment of major depression with psychotherapy or psychotherapy–pharmacotherapy combinations. Arch Gen Psychiatry. 1997;54:1009–15.
63. Burnand Y, Andreoli A, Kolatte E, et al. Psychodynamic psychotherapy and clomipramine in the treatment of major depression. Psychiatr Serv. 2002;53:585–90.
64. Barkham M, Mellor-Clark J. Rigour and relevance: the role of practice-based evidence in the psychological therapies. In: Evidence-based counselling and psychological therapies: research and applications. New York: Routledge; 2000. p. 127–44.
65. Kool S, Dekker J, Duijsens I, de Jonghe F, Puite B. Changes in personality pathology after pharmacotherapy and combined therapy for depressed patients. J Pers Disord. 2003;17(1):60–72.
66. Stimpson N, Agrawal N, Lewis G. Randomised controlled trials investigating pharmacological and psychological interventions for treatment-refractory depression: systematic review. Br J Psychiatry. 2002;181:284–94.
67. McPherson S, Cairns P, Carlyle J, et al. The effectiveness of psychological treatments for treatment resistant depression: a systematic review. Acta Psychiatr Scand. 2005;111:331–40.
68. Abbass AA. Intensive short-term dynamic psychotherapy of treatment resistant depression: a pilot study. Depress Anxiety. 2006;23:449–52.
69. Davanloo H, editor. Basic principles and techniques in short-term dynamic psychotherapy. New York: Spectrum; 1980.
70. de Maat S, de Jongh F, Schoevers R, Dekker J. The effectiveness of long-term psychoanalytic therapy: a systematic review of empirical studies. Harv Rev Psychiatry. 2009;17:1–23.
71. Leichsenring F, Rabung S. The effectiveness of long-term psychodynamic psychotherapy: a meta-analysis. J Am Med Assoc (JAMA). 2008;300:1551–64.
72. Vandenbroucke JP. When are observational studies as credible as randomised trials? Lancet. 2004;363(9422): 1728–31. doi:10.1016/S0140-6736(04), 16261-2.
73. Knekt P, Lindfors O, Harkanaen T, et al. Randomized trial on the effectiveness of long-and short-term psychodynamic psychotherapy and solution-focused therapy on psychiatric symptoms during a 3-year follow-up. Psychol Med. 2008;38:689–703.
74. Knekt P, Lindfors O, Laaksonen M, Raitasalo R, Haaramo P, Järvikoski A. Effectiveness of short-term and long-term psychotherapy on work ability and functional capacity: a randomized clinical trial on depressive and anxiety disorders. J Affect Disord. 2008;107(1–3):95–106.
75. Lambert MJ, Okiishi JC, Finch AR, et al. Outcome assessment: from conceptualization to implementation. Prof Psychol Res Pr. 1998;29:63–70.
76. Malan DH. Toward the validation of dynamic psychotherapy. New York: Plenum Press; 1976.
77. Høglend P, Amlo S, Marble A, et al. Analysis of the patient-therapist relationship in dynamic psychotherapy: an experimental study of transference interpretations. Am J Psychiatry. 2006;163(10):1739–46.
78. Huber D, Klug G, von Rad M. Die Münchner- Prozess-Outcome Studie, 2001: Ein Vergleich zwischen Psychoanalysen und psychodynamischen Psychotherapien unter besonderer Berücksichtigung therapiespezifischerErgebnisse [The Munich processoutcomestudy: a comparison between psychoanalyses and psychotherapy]. In: Stuhr BM, editor. Langzeit- Psychotherapie.Perspektiven für Therapeuten und Wissenschaftler. Stuttgart: Kohlhammer; 2001. p. 260–70.
79. Grande T, Dilg R, Jakobsen T, et al. Differential effects of two forms of psychoanalytic therapy: results of the Heidelberg–Berlin study. Psychother Res. 2006;16(4):470–85.

80. Leichsenring F, Biskup J, Kreische R, Staats H. The effectiveness of psychoanalytic therapy: first results of the Göttingen study of psychoanalytic and psychodynamic therapy. Int J Psychoanal. 2005;86(pt 2):433–55.
81. Leuzinger-Bohleber M, Target M, editors. The outcomes of psychoanalytic treatment. London: Whurr; 2002.
82. Beutel M, Rasting M. Long-term treatments from the perspectives of the former patients. In: Leuzinger-Bohleber M, Target M, editors. The outcomes of psychoanalytic treatment. London: Whurr; 2002.
83. Sandell R, Blomberg J, Lazar A, et al. Varieties of long-term outcome among patients in psychoanalysis and long-term psychotherapy: a review of findings in the Stockholm outcome of psychoanalysis project (STOPP). Int J Psychoanal. 2000;81:921–43.
84. Blomberg J, Lazar A, Sandell R. Outcome of patients in long-term psychoanalytical treatments. First findings of the Stockholm outcome of psychotherapy and psychoanalysis (STOPP) study. Psychother Res. 2001;11:361–82.
85. Kopta S, Howard K, Lowry J, et al. Patterns of symptomatic recovery in psychotherapy. J Consult Clin Psychol. 1994;62:1009–16.
86. Blatt S, Shahar G. Psychoanalysis–with whom, for what, and how? Comparisons with psychotherapy. J Am Psychoanal Assoc. 2004;52:393–447.
87. Jacobson N, Follette WC, Revenstorf D, Baucom DH, Hahlweg K, Margolin G. Psychotherapy outcome research: methods for reporting variability and evaluating clinical significance. Behav Ther. 1984;17:308–11.
88. Jacobson N, Truax P. Clinical significance: a statistical approach to defining meaningful change in psychotherapy research. J Consul Clin Psychol. 1991;59:12–9.

Author Queries

Chapter No.: 5 0001331385

Queries	Details Required	Author's Response
AU1	Please check if the sentence starting "In depression…" can be edited as "In depression … with the lost person present within, rather than absent without."	
AU2	Please check the data (SMD –0.97; -).03) in the sentence starting "Symptoms, assessed at baseline…"	
AU3	Please check if edit to the section heading "Longer-Term Psychoanalytic Treatments (LTPP)" is okay.	
AU4	In the sentence starting "The studies were…" only four studies have been mentioned, whereas the previous sentence mentions five studies. Please check.	
AU5	Please provide publisher details for Ref. [1].	
AU6	Please provide article title and journal abbreviation for Ref. [43].	
AU7	Please provide article title for Ref. [60].	