Three Problems with the Theory of Cognitive Therapy

S. GIAC GIACOMANTONIO, Ph.D.

Three problems with the theory of cognitive therapy are presented. Each is argued as a problem for the model of change in cognitive therapy, not for their impact (if any) on treatment. They are (a) the unpredictability of cure and relapse, (b) the epistemologically irreconcilable differences between the model of pathology and the model of change, and (c) the inability to conceive of ambivalence. Each problem is discussed, and some counterarguments are offered.

KEYWORDS: cognitive therapy; theory; model of change; CBT; philosophy; epistemology

INTRODUCTION

This paper outlines three problems with the theory of cognitive therapy (CT).[1] These are not problems that will necessarily affect the therapeutic results of cognitive treatments, because treatments are rarely conducted in perfect accordance with the theory. Rather, they are problems with the theory that informs and explains the treatment. They are shortcomings of a model of therapeutic change, and their implications are chiefly for research into therapeutic change. The thesis does not concern empirical evidence, or therapeutic technique, or outcome. This is a paper about theory.

I will begin with a brief review of the empirical status of CT in the extant literature, before addressing these issues of theory in detail.

COGNITIVE THERAPY

Cognitive therapy is a form of psychotherapy that has attained a singular position as the treatment of choice amongst many psychothera-

School of Psychology, Australian Catholic University, Brisbane. ***Mailing address:*** P.O Box 456, Virginia, QLD 4014, Australia. e-mail: giac.giacomantonio@acu.edu.au

[1] I have chosen Beck (1976) and Beck, Shaw, Rush, & Emery (1979) because the former was cited by Beck himself as containing the essence of his model (Beck, 2006) and the latter is often cited in academic psychology literature as the principal reference for classical CT (having had well over 8000 citations to-date in the social sciences literature).

pists and patients (e.g., Cook, Biyanova, Elhai, Schnurr, & Coyne, 2010; Forsyth & Matthews, 2010). In some countries (e.g., England and Australia) accrediting bodies and government funding organisations nominate or mandate it as a therapeutic approach for training and therapy-insurance rebates (e.g., House & Loewenthal, 2008). It is among the most researched of all forms of psychotherapy (Grant, Young, & DeRubeis, 2005; Freeman, & Scott, 2010). But research to date has been more successful in demonstrating psychotherapeutic efficacy than in evincing theoretical validity.

Much modern psychotherapy research concerns itself with the phenomenon of therapeutic change. Each specific theory of the different psychotherapies predicts unique superiority of outcome for its own corresponding treatment. But much outcomes research has failed to find any such differential effect (e.g., Smith & Glass, 1977; Horvath & Symonds, 1991; Martin, Garske, & Davis, 2000; Wampold, 2001; Duncan, Miller, Wampold, & Hubble, 2010). Instead, rigorous studies and comprehensive meta-analyses often confirm the so-called "Dodo Bird finding" that all therapies are roughly equivalent at reducing symptoms of psychopathology. The finding of equal outcomes has (understandably) engendered some skepticism of any specific factor theory of therapeutic change (see, for example, Wampold's contextual model of therapeutic change, 2001). And the theory of CT is another such specific factor model.

Alongside much evidence for equal outcomes across different psychotherapies, some authors maintain that CT produces superior outcomes (e.g., De Rubeis, Brotman, & Gibbons, 2005), suggesting instead that methodological problems have obscured these specific effects in previous studies. Such a case suggests a possible resurrection of the theory of change in CT. Whether superior or equal, CT's efficacy seems doubted by none, but its theory of the mechanisms of that change enjoys no such unanimous faith.

Beyond efficacy alone, remains theory. Many studies looking for evidence of the theory of CT have tried to substantiate its descriptions and formulations of psychopathology. This might include, for example, looking for evidence of the negative triad in depressed patients (see reviews by Haaga, Dyck, & Ernst, 1991; or more recently Beck, 2005). On balance, the predictions about patient characteristics explicated in the theory have been well evidenced. The theory describes pathology.

Such studies are to be distinguished from those that test empirically the mechanisms of change outlined in the theory. Many studies find empirical evidence (e.g., Hofmann, Meuret, Rosenfield, Suvak, Barlow, Gorman,

Shear, & Woods, 2007, & Rapee, Gaston, & Abbott, 2009) lending support to the thesis that CT works for the reasons it claims. While some of these acknowledge other equally-effective non-cognitive mechanisms at play, (e.g., Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010) still other studies have found the reverse, viz., that CT works for reasons other than its hypothesized mechanisms (e.g., Castonguay, et al., 1996; Jacobson, Dobson, Traux, Addis, Koerner, Gollan, Gortner, & Price, 1996). One recent review (Longmore & Worrel, 2007) examined nine studies and concluded that no therapeutic benefit comes from the specifically cognitive interventions in CT (such as challenging cognitions). As with much in clinical psychology, consensus does not yet obtain. Some of these studies are component analyses, where a reduced version of the treatment is compared with full treatment to measure the relative effectiveness of that component (e.g., Rapee et al., 2009). It is typically reasoned from such component analyses that if a component-intervention offers no extra therapeutic gain, then that component-intervention has no therapeutic impact. There are of course alternative interpretations for such findings, e.g., that the benefit of some components cannot be enhanced by the addition of others; in other words, each component may be sufficient to activate a (single) mechanism of therapeutic change. Still other interpretations are possible.

Empirical support for theoretical predictions is, however, not the only avenue of research that should concern those interested in understanding therapeutic change. The topic of this paper, the question of the evaluation of a theory per se by theoretical (not just empirical) means, remains an open one—one that is barely touched-upon in the literature. I will discuss briefly some of the reasons for the possible contribution to psychotherapy research of a purely theoretical focus in the evaluation of mechanisms of change.

The Question of Theoretical Research

In an applied endeavor, like psychotherapy, it might be argued that all theoretical considerations are logically subordinate to pragmatic ones. Our pragmatic task of ameliorating suffering is ethically primary, and this primacy makes us more tolerant of a theory that may be "wrong" in its assertions, if it has sufficient heuristic value to orchestrate a successful treatment (as suggested by studies like Oei, Bullbeck, & Campbell, 2006; or Castonguay et al., 1996; and others cited above). Nevertheless, there are arguments for the central importance of theoretical considerations in

psychotherapy research, each a logical consequence of the role of theory in (any) scientific research. I will stop at three:

First, there are no data without theories. Our conceptual frameworks have an indispensible epistemological role in science, and we cannot hope to set them aside to gather our data. With the ultimate collapse of Bacon's (1620) notion of an unbiased "blind" empiricist, theory as preconcept, as Heidegger's *Vormeinung* (1962, p. 192), has, for decades, been seen as the very means by which data are perceived or are perceivable in principle (see, for example, Hesse, 1980, or even Kuhn, 1970, on the related issue of theory as paradigms that determine the scientific questions to be answered). It is not that we fail to shrug off these conceptual yokes, thus polluting our data gathering, but rather that data gathering is simply not possible without them (Hesse, 1980). Data demand bias. The point here is that persistent empirical exploration without due attention to theory is not merely inadvisable, it is actually impossible. Given that we cannot act without theory, it behooves us to articulate our theoretical positions and to consider them rigorously.

Second, theory is required for establishing coherent lines of empirical research. The theory and especially its auxiliary hypotheses (as Lakatos called them) are required to make coherent steps from one empirical study to the next. The theory must be ahead of the collected data if a research endeavor is not to stagnate (Lakatos, cited in Losee, 2005). And if empirical investigation could ever be truly devoid of theory, it would be incomprehensibly manifold. There is too much that might be measured. Theory tells empirical scientists both what to ignore *and* what to do next—to repeat—no matter how latent that theory may be.

Third, the previous two points feed directly back to our ability to improve psychotherapy, even in the most pragmatic sense. Empirical lines of research boast material assistance to clinician and patient. But if it is impossible for them to step off the foundations of theories, they surely limp without sufficiently well-developed theories. Even the empirical data of psychotherapy failures or shortcomings require some theory if they are to be identified and understood, to generate (rational) alternative practices to be tested empirically. Without theory, the task of improving treatment outcomes becomes an entirely hit-and-miss exercise.

Thus, the earlier distinction between the following theoretical problems and the clinical success of CT relies on the fact that the theory guides, defines, and, therefore, limits treatment, including our very definition of a successful treatment: It implies some theory to describe the improved patient. Else how should the therapist recognize him?

Three Problems

Each of the problems outlined below can be linked conceptually to the exclusion of motivation from the theory of CT. The emphasis on pragmatism in the treatment model, and the corresponding avoidance of the problems of motivation or psychogenesis (see below), have been seen as a credit to the therapy and a feature of decided advantage over other forms of psychotherapy. But this advantage is obtained only after translation of theory into treatment; the problems at the level of theory persist nonetheless, and it is to these that I now turn.

For the sake of simplicity the principal works of A.T. Beck will serve as the major referents for the theory of CT. It may be objected that such an emphasis on the works of Beck presents us a fractionated and possibly archaic rendering of the theoretical edifice of CT—at best too classical, at worst misrepresentative. But I would answer that the three theoretical issues cited here are so central to the cognitive model that if any subsequent development did not continue to subscribe to them, it could scarcely still warrant the appellation "cognitive therapy". In other words, while citing Beck, I believe these issue to apply also to CT beyond Beck. I aim to deal with the essential.

In summary, the cognitive model of A.T. Beck (e.g., 1976), in contradistinction from other models of psychopathology, places emphasis on the conscious mentation of the patient as the source and maintenance of pathology, as well as the route to healing. The external world and internal stimuli are processed with bias (Beck, 2005). Dysfunctional beliefs serve as schemata that structure the patient's experiences in a consistently distorted manner (Beck, 1976, & 2005). Treatment involves clarifying the patient's attribution of meaning and misperceptions of reality, and psychopathology is ameliorated by the changes that ensue in both (e.g., Beck, 1976). Different classes of technique enable the patient to alter the feedback cycle of the perception of reality or the degree of rationality in the lines of internal reasoning, judgment, and attribution. The changes are liberating. Treatment maintains a focus on teaching the *techniques* of cognition alteration and meaning attribution, which the patient can continue to use after termination (Beck et al., 1979). Thus the goal of therapy is not "to cure"[2] the patient during treatment, but rather to establish a therapeutic internal process of which the period of treatment forms only one part—however significant or essential (Beck et al., 1979). The elegance of this

[2] Let the noun "cure" be, from now on, a shorthand for successful therapeutic outcome and the verb "cure" a shorthand for "treat and engender successful therapeutic outcome".

model was likely a contribution to the popularity of the model with patients and therapists alike (Beck et al., 1979), and it has served usefully as a basis for many specific formulations, including neurobiological models of pathology and treatment (e.g., Clark & Beck, 2010).

1. PSYCHOGENESIS AND THE UNPREDICTABILITY OF PATHOLOGY

I have used the words motivation and psychogenesis as though they were synonyms. Some clarification is warranted. Motivation is a broad and almost problematically complex concept to psychology. But to speak of psychogenesis in a psychological theory is inevitably to touch on motivation, especially in the case where psychogenesis is omitted. When the conditions or precursors of a given experience (psychogenesis in the broadest sense) are ignored, the absence of *what* gave rise to something necessarily obscures *why* the rise was given (motivation). If we do not care to know what came before a given behavior, we are prevented from mounting a case for why the behavior was emitted.[3] It is only outside the sciences (e.g., in religion) that *why* may be comprehensible without knowing *what* or *how*; we might accept the Ascension of the Virgin Mary without being able to explain the specific mechanisms at play. Thus wherever I equate psychogenesis with motivation in discussing CT, it is precisely because to occlude the former is necessarily to occlude the latter.

It should go without saying that the motivation to which I refer in this paper is a theoretical construct (as is the tendency toward self-realization in some other theories)—what motivates people in general including patients in particular. It should not be mistaken as motivation *for treatment*, such as the motivation to engage in the therapy and to work collaboratively with the therapist.

Cognitive Therapy rests comfortably on the premise that one need not know the developmental origins of a problem to be able to correct it (e.g., Beck, 1976, p. 3; Beck et al., 1979, p. 7). By dispensing with the burden of uncovering a causal link over time (i.e., between past and present), this premise lends great expediency to treatment, because the task of under-

[3] "Came before" might be argued as inapplicable in the case of behaviorism's reinforcers. But even here, a reinforcer is considered to have been at play in the establishment of the behavior in the patient's repertoire: The behavior must have been emitted earlier before it could ever be reinforced. Thus the verb phrase "came before" means to include previous instances of a given behavior and not only events immediately prior to the behavior. One possible exception in the behavioristic theories might be when a behavior is emitted for the very first time, as in the case of behavioral shaping. But I would reply that this specific instance is again subject to the ideas outlined above.

standing the psychogenesis of pathology might make other treatments lengthy.

Weighing against these benefits, are questions about the lasting value of treatment. Critics might impute that if the genesis of a symptom (say negative self-talk) were not addressed, we could not hope to prevent the re-emergence of that symptom (or its substitute) in the future. In other words, psychologists might disagree over the therapeutic impact of understanding psychogenesis. This disagreement might be easily laid to rest if it were understood *only* as an empirical question—i.e., Do patients of CT need to return to treatment or are the cures more or less permanent? That would be easily settled empirically. (And CT should not have to shoulder the burden of demonstrating permanent cures more than any other therapy, given that no such treatments of universally enduring cure have yet been found.) But there is an entirely separate *theoretical* issue at stake that is central to this paper: Even if empirical investigation were to show cognitive cures to be relatively permanent, without the need for booster sessions,[4] the theory would remain unable to explain *how* lasting change was achieved, because the model has all but neglected the questions of genesis. We cannot talk about what gave rise or activation to the cognitive set. Thus the problem of explaining genesis remains a theoretical shortcoming. In other words, if we imagine for a minute that CT were tomorrow shown to end depression permanently after 12 sessions, then, however we may attribute merit to the theory, it would remain insufficient insofar as it lacks the capacity *to explain* its own lasting treatments: It could not explain how the therapy ended permanently the emergence of pathology, because it does not address the causes of pathology.

Without a theory of psychogenesis, there is a corresponding absence of a functional reason for the pathology.[5] Treatment is left to focus exclusively on the *content* of the pathology, as though to take its very emergence for granted. Eminently pragmatic. But the apparent benefit for any psychotherapy is debatable. We can contrast it with (for example) psycho-

[4] Empirical investigation has, of course, not found that cognitive treatments lead to lasting change without the *expectable* need for follow-up doses (e.g., Ball, Mitchell, Corry, Skillecorn, Smith, & Malhi, 2006) but rather found that its long term effects are no better (e.g., van Oppen, van Balkom, de Haan, & van Dyck, 2005) or worse (e.g., Schedler, 2010), than those of other treatments.

[5] Beliefs are posited in the cognitive model to give rise to cognitions and may therefore be argued as psychogenetic elements of the theory (e.g., Beck et al., 1979, chapter 12). But here the distinction between sequence and genesis is paramount. To say that beliefs lead to cognitions does offer a kind of cause and effect relationship, but it simply displaces the same theoretical problem by one step in the causal chain (beliefs instead of cognitions). It still evades the questions of *why* they do and do not arise when they do (and do not; discussed further below).

analysis, in which the treatment regards a reason for the symptom as often more important than the symptom's contents and details. The very interventions of psychoanalysis are statements of formulation; explaining the emergence more than the content of the symptom is essential. Some cognitive theoretical propositions for psychogenesis are offered (e.g., Beck et al., 1979, p. 245), but these may be considered academic in the sense that they are not required to inform interventions and have little relationship to other constructs in the theory. True: Many successful cognitive treatments involve a sensitive consideration of the patient's past as it sheds light on the development of the present-day beliefs and cognitions. But many equally cognitive treatments do not. Discussing the past is simply not indispensible for CT. This technical point belies the theoretical problem that while some successful cognitive treatments will include the patient's recall of the past and other (equally) successful ones may not, the therapeutic intervention remains a cognitive one in *both* cases. The intervention itself does not *need* the past. The empirical fact of therapeutic success following significant consideration of the past is not to be mistaken for an actual theory/explanation of psychogenesis. The explanation is still lacking.

Following on from the theory, the therapy of CT may teach the patient to change his cognitive distortions, but it cannot supply predictors or heralds of the re-emergence of these distortions. The expectation of relapse (as of permanent cure discussed above) becomes a matter of pure speculation devoid of rational prediction. There are no causal mechanisms to check. Indeed, after termination the patient is *expected* to need to use the techniques learned in the "training period" called treatment (e.g., Beck, 1976, p. 317). So long as the theory says we can ignore the "first mover" of the cognitive phenomena, it must designate the relapse equally as likely as the permanent cure. Relapse in this sense refers to the re-emergence of the symptoms, ignoring whether or not the patient is skilled enough to retreat herself at the point of their re-emergence. The patient is left (according to the theory) knowing that she is capable of altering distorted thinking when it comes, but she remains at the mercy of an ever-present threat; once the onset of the distortion is relegated to a position outside the theory of treatment, it is likewise relegated to the class of the incomprehensible: It must (rationally) be deemed unpredictable.

Against this argument for unpredictability, one might reply that the cognitive distortions are self-perpetuating (owing to their corresponding filter on perception; Beck, 1976), that negativity begets negativity through a feedback cycle, and that the undistorted thinking which makes for more-positive emotions is likewise self-reinforcing. Self-perpetuation

promises to supply an answer of sorts to the question of predictability. But there are several problems with this counter-argument. Despite my use of the word "reinforcing" we cannot properly invoke the model of conditioned behavior to explain this (leaving aside for the moment the thorny problem of classing cognitive phenomena as behaviors). By definition, the negative quality of the associated emotions precludes us from labelling the cognitive phenomena as being "reinforced," from labeling the emotions as "reinforcers." Operant conditioning would predict precisely the opposite because the negative emotional results must be considered "punishing." A case might yet be made for negative reinforcement, but that would require an argument that the negative thinking saves the patient from more (perceived) suffering than it causes, in which case the very problem of cognitive distortions begins to lose its original definition in the model as the crux of the emotional suffering. In other words, this last defense of the CT model argues against the original need for treatment. Thus, either with or without reference to associative learning, the model of CT fails to explain the *emergence* of either the negative- or the positive-feedback loop of cognition/perception.

In summary, the theory of CT, without a well-developed model of psychogenesis, must fail to account for (a) why the patient's psychopathology emerges when it does rather than sooner or later, (b) why the treatment *can* work without addressing whatever genesis there may be—in other words, if we know it is technically unnecessary to address genetic factors, how can we explain this pragmatic fact, when uncovering cause and effect relationships is so basic a scientific activity—and, (c) why the patient does not relapse (for unidentified reasons), or equally, why the psychopathology did not disappear before treatment, (for unidentified reasons). If the genesis of psychopathology requires it to be explained, we are logically constrained in predicting or explaining both the relapse and the enduring nature of the lasting cure. Treatment would ape the hit-and-miss exercise of "purely" empirical research.

2. TWO PRINCIPLES OF MENTAL FUNCTIONING

A further problem lies in the philosophical incompatibility of CT's model of pathology and its model of treatment. The model of pathology is one of indirect perception: We cannot deal with the world directly but rather with our imperfect perception of it. We can never know reality "out there." In the normal course of treatment, this hypothesized gap between perception and reality provides an entry point for the patient to challenge his appraisals and perceptions, thus bringing into question the correspond-

ing emotional reactions *to those perceptions*. In jolting philosophical contrast, the model of treatment is based on a "collaborative empiricism" which seeks (at least in part) to help the patient to bring a realistic (i.e., more correct) apprehension of the world into contact with her cognitions and beliefs. The opportunity for and the direction of both challenging and altering one's beliefs comes from better contact with reality; only the beliefs that tally with reality shall stand. It is philosophically noteworthy that the theory assumes the patient and therapist *capable* of perceiving a reality by which they can examine and verify the patient's (distorted) perceptions.[6] The incompatibility is both nagging and egregious: The epistemology implicit in the theory of *treatment* demands a direct perception that the epistemology of the theory of *pathology* excludes. In other words, when explaining psychopathology, CT relies on a philosophical world-view that makes its own description of treatment impossible. If humans do not perceive the world directly (as many philosophers believe), how then could we ever know any reality ("out there") with which to compare our perceptions? Rationally, these epistemologies cannot be reconciled.

To dispute this criticism, we might argue that the therapy centers not on comparing perception with reality, but that it first deconstructs the patient's perceptions into component-sense data and proceeds to re-assemble or re-construct a differentially interpretable edifice. We might argue that reality is irrelevant. With this counter-argument, the clash of indirect with direct perception is circumvented. But I would reply that the requirement of some direct perception seems indispensible to at least one of the two major aspects of cognitive pathology, viz. (a) the distortions of reality and (b) the illogical thinking or reasoning processes applied to perceptions (e.g., Beck, 1976, pp. 218-219). One may wish to dismiss the former, yet to do so is to undermine the very role of distortion in the cognitive theory (because "distortion" can be defined only with reference to an undistorted reality. We must agree on the original condition of something before we could claim that it has been distorted). Further, dismissing the former would in turn make the collaborative empiricism, by definition, irrelevant. Again, I would argue that this is too central a tenet to be surrendered without implying a different (non-cognitive) model of treatment.

[6] NB. This sentence is a comment on an epistemological standpoint of a theory—that reality can be perceived accurately by human beings—and should not be mistaken for a comment on prognosis of a given patient's capacity to engage in a particular treatment.

If we accept this criticism of incompatible epistemologies (i.e., if we accept my assertion that the theories of pathology and treatment demand mutually exclusive epistemologies), then we are faced with the following conundrum. If the patient were indeed able to perceive the world more or less accurately/directly, how then does the pathology described by the cognitive model ever develop? And conversely, if he were *unable* to perceive reality more or less accurately, how then is he able to participate in the collaborative empiricism? The establishment of either brings the mechanisms of the other into question. One possible counter-argument is, of course, that the patient is able to operate *both* in the manner described as indirect perception *and* in the manner described by naïve empiricism. This counter-argument essentially offers that indirect perception is simply *part* of the pathology, and so it naturally stands along-side other healthy sectors (which both reflect and are in touch with reality). But this counter explanation itself raises its own problem. Because it effectively aligns the two epistemologies with the two conditions of pathology and health, it demands a logical shift in the central issue of treatment. To correct the patient's dysfunctional thinking is now a matter of shifting the patient from one epistemological "mode" to another. And explaining the emergence of pathology is tantamount to explaining when (and why) the patient goes from one epistemological mode to the other. Why should the patient operate in the mode of indirect perception in the area of her pathology when the option remains for her to operate in the "naïve empiricist" manner of direct perception? Again the problem can be seen as a function of the lack of a theory of motivation. Treatment clearly works to direct the patient back to rational and reality-based thinking, but the theory offers no explanation for what "flicked the switch" to the indirect perception of cognitive pathology (either before, during, or after treatment). There is nothing in the theory to say how a patient changes his very system of apprehending sense data, nor anything to explain how the therapist and the treatment are not simply perceived by and made part of the distorted world of the indirect perception. Again, this extends to the problem whereby nothing in the theory can predict whether the patient will revert to indirect perception because nothing is offered as causal *of that mode of functioning*. To restate the problem differently: no part of the theory rests outside these two modes to address what leads to a shift from either to the other. Actual patients obviously do seem to shift, but that is different from the question of theoretical explanation.

This counter-argument of multiple epistemologies has merit. That patients can often generate their "counter-arguments" to their own nega-

tivity demonstrates that both rational and irrational thinking processes are available to them. (Daily life surely demonstrates that we all employ both on a regular basis. In fact, both are required for participation in a cognitive treatment: Without some irrational thinking there is no need for treatment; without at least the capacity for rational thinking there is no participation in the treatment.) But these moments of self-generated counter-argument evidence both kinds of thinking in the one patient with respect to the one issue, and this suggests in turn that pathology may be an issue of the *activation* of the option for distortion, more than distortion per se. In the absence of a theory of motivation, asking *why* the patient thinks irrationally only in the areas of his pathology is far more difficult than to describe *how* he does so. Yet, if treatment is really thought to be helping the patient "to shift epistemologies," this shift seems logically to become the issue of greatest moment for the theory.

3. THE PROBLEM OF AMBIVALENCE

I assert here that ambivalence as a subjective phenomenon cannot be accounted for in the theoretical model of CT. Ambivalence refers, in this sense, to the condition of incompatible sets of thoughts or perceptions, when neither can lay claim to dominance (except temporarily). Such ambivalence has been clinically described as the result of the disavowal (["disowning"] Basch, 1983) of affect (Goldberg, 2001). Some disavowal must be present for an incompatible sector to be activated. I see two reasons why the theory of CT cannot model these phenomena: One is on account of the split between irreconcilable experiences; the other is on account of the disavowal of affect.

First, when such a patient presents, it is invariably because one of these set of thoughts (and any corresponding behavior) is identified as "the problem." Incompatible sets of thoughts and beliefs will be analyzed for their relative consonance with reality and their rational coherence. On this basis one will become seen as the rational side (with which the therapist makes an alliance) while the other is fated the irrational one, "the problem." Take for example the compulsive adulterer who begs the therapist to help him stop committing adultery because he loves and wants to be faithful to his wife. The identified problem is obviously irreconcilable with his professed wish for fidelity, yet, the inconsonant behavior will be examined for its rationality. The ambivalence will elude a cognitive analysis because ambivalence is what we might call a *structural* consideration that stands apart from the *content* of either of the two incompatible sides. In other words, an examination of the thoughts and beliefs of each side of

such a split will not yield an examination of the ambivalence that characterizes the whole. Instead, the treatment is left to juxtapose mental contents on a single plane, which must lead, by definition, to the eventual assertion of one over the other because of their logical incompatibility. We find evidence of this in Beck's formulation of ambivalence as a problem of *relative conviction* only (1976, pp. 222-225). In other words, the condition of ambivalence per se cannot be taken as a unique datum for investigation. The theory directs us to the verbal contents of the two sides of ambivalence in turn.

Second, it is a chief axiom of the model that thoughts lead to feelings; thoughts shape and determine feelings (cf. Beck, 1995, cited in Grant, Young, & DeRubeis, 2005, p. 15). Therefore, a postulated condition in which thoughts and feelings appear *not* to go together (i.e., the very definition of disavowal) leaves literally nothing theoretically to inform treatment except to try to establish greater conviction in the patient. That thoughts lead to feelings is an *assumption* of the model, which (like all assumptions) must be taken for granted, and cannot be manipulated by choice. Cognitive Therapy cannot *make* the patient's thoughts influence his feelings (just as behavior therapy cannot *make* a reinforcer reinforce behavior), whatever those thoughts might be. That is the nature of assumptions. For CT the impact of thoughts on feelings is a process required (by the theory) to be automatic and synthetic.

SUMMARY

Cognitive therapy works. All psychotherapy as an applied endeavor can afford the luxury of pursuing treatments with relatively opaque underlying mechanisms (Kazdin, 2009) provided that there is sufficient evidence to convince scientist-practitioners of therapeutic benefits. Here, the practitioner-aspect serves the patient well. But the scientist-aspect must accept the burden of demonstrating not merely that the treatments work but that we can explain them with sound, well-developed, and specifically psychological theories of human functioning. This imperative urges us to establish criteria by which *theories* themselves can be evaluated, independent of the simple question of the efficacy of their corresponding treatments, or the empirical validation of their predictions. Theory is something more. With this imperative as a point of departure, the preceding paper identified a number of problems with the theory of CT: threats to the integrity of the model of change as a theoretical edifice.

Most of the problems with the theory of this successful psychotherapy can be linked conceptually to its lack of a model of motivation. From this

single issue spring the problems of (a) a weakness of psychogenetic theory and the corresponding unpredictability of pathology or cure, (b) the epistemological irreconcilability of the model of pathology and the model of change, and (c) the inability to model ambivalence.

The de-emphasis on psychogenesis makes treatment pragmatic and brief. But it renders the theory unable to explain (well) how the cure obtains permanence, or how the relapse occurs. Without identifying (much less understanding) any causes of cognitive distortions of dysfunctional beliefs, all areas usually assigned to the precursory role of motivation are occluded: The theory, therefore, hides any mechanisms that may be keeping therapeutic change in place or allowing it to be undermined. There are no identified causal mechanisms or agents to be measured before and after interventions. The source is both obscure and ignored. (This should not be mistaken as an argument for consensus on first causes [in the Wittgensteinian sense]).

It is not a complaint that beliefs are insufficiently primary in a potentially infinite regression of "first movers." No: It is the identification of the absence of psychological motivation, which could enter any model at any point along a potentially infinite regression of first causes, to introduce the wishes, needs, etc., of the person. For CT it never enters. For CT there is no articulation in the model for how the patient might generate or favor certain belief-schemata beyond the utilitarian evaluation in treatment of the identified culprit: symptom. The beliefs are almost incidental, if not accidental, and eminently replaceable on pragmatic grounds, much like changing computer software. (The computer doesn't mind.)

The irreconcilability of the theory of pathology and the theory of change rests on the former's basis in indirect perception and the latter's basis in naïve empiricism. The model of pathology establishes that the patient cannot perceive the world directly and uses this premise to explain why the emotional reactions are essentially needless and senseless suffering. Yet, the model of change requires that the patient-therapist team compare the indirect perceptions of the patient with reality to identify distortions, misperceptions, and conclusions for which poor evidence exists. This is the most purely theoretical of the three problems identified. Some counter-arguments supplied above serve only to leave us in need of explaining why a patient should ever employ the indirect perception (demonstrated by his pathological sector) when the option of direct perception (yoked by the collaborative empiricism) is available.

The inability to conceive of ambivalence is a direct result of an underlying assumption of the theory: It is axiomatic for CT that thoughts

lead to feelings.[7] Any data that might be seen (from the perspective of other models) as a breach of this assumption (e.g., disavowal) are forced through the lens of the theory into a question of conviction. When feelings do not follow-on from cognitions, a model that rests on the synthetic functions of thoughts-determining-feelings cannot conceive of the phenomenon (except within the bounds of the model): It is left to be understood (cf. Beck, 1976, pp. 222-225) as the patient being incompletely convinced of the more realistic and logical thinking.

I have described these problems to be primarily relevant to psychotherapy research, but relevant secondarily (or indirectly) to psychotherapy itself. The benefit to treatment of addressing these theoretical shortcomings may yet prove unexpectedly positive. Only future research will tell.

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[7] Some authors have questioned the oversimplicity of this causal link, suggesting more complex roles for emotions than the classical one outlined above (e.g., Safran, Eubanks-Carter, &, Muran, 2010). But again, we must identify a point where the most central and most "cognitive" aspects of the theory cannot be undone without creating de facto an entirely different model. Can we conceive of CT without the (centrality of the) thoughts-to-feelings axiom? Is this not the problem of the proverbial woodcutter's axe?

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