Competing Visions of the Implications of Neuroscience for Psychotherapy

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COMPETING VISIONS OF THE IMPLICATIONS OF NEUROSCIENCE FOR PSYCHOTHERAPY

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In this third and final article of a series on the confluence of neurobiology and psychotherapy, we consider three current, influential interpretations of the implications of neuroscience for psychotherapy: pharmacological treatment, reparative attachment therapy, and the cognitive regulation of emotion and behavior. We critically examine these clinical strategies, reviewing efficacy data, neuroscientific research, and the model of symptom production by coherent implicit memory as articulated in coherence psychology. We argue that according to current knowledge, (a) each of the three clinical interpretations of neuroscience implements only part of the brain's known capabilities for change; (b) those capabilities are more fully utilized and can yield greater clinical effectiveness for the majority of psychotherapy clients through a therapeutic strategy of selective depotentiation of implicit memory, as exemplified by coherence therapy; and (c) the strategy of counteracting an implicit memory, whether cognitively or psychopharmacologically, is only moderately effective, is inherently susceptible to relapse, and entails a range of undesirable collateral effects.

The field of psychotherapy has wrestled since its inception with the challenging task of integrating an understanding of subjective mental experience with an understanding of how the brain and neural networks function. Freud drafted an attempt to formulate a neurologically based psychology (Freud, 1895/1950) only three years after the discovery of the synapse was published

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(Ramon y Cajal, 1892). Since then, the sought-for synthesis of the physiological and the phenomenological has proven elusive and has more often than not degenerated into a polarized debate over mind versus brain, holism versus reductionism, top-down versus bottom-up.

We are not reductionists, yet we fully value and welcome what the neural viewpoint contributes to an accurate understanding of the functioning of the brain-mind-body system. The latter is so complex and rich as to allow for a wide range of useful approaches for facilitating change and well-being. The almost dizzying diversity of psychotherapies created in the second half of the twentieth century has been a positive development in many ways. Nevertheless, "complex" does not mean "arbitrary." The brain-mind-body system consists of certain structures, processes, forms, laws, and rules, and not others. Having an accurate knowledge of these will contribute to our understanding of the effects and limitations of different psychotheraputic methods, and to this end we believe neuroscience has much to contribute. The psychotherapy field already owes much to authors such as Schore (2001a, 2001b) and Siegel (1999) for calling attention so effectively to the value of understanding the neurodynamic substrates of symptoms that therapists work with every day.

In this article we attempt to sort out the implications for psychotherapy of current knowledge in neuroscience. In doing so, we know we are adding only a few notes to a more than 100-year-long, ongoing conversation.

There are three major current trends in neurodynamically informed mental health treatment: pharmacological treatment, reparative attachment therapy, and the cognitive regulation of emotion and behavior. As we review and critically examine these approaches, it will emerge that each captures only a part of the implications of neuroscience research for psychotherapy and leaves out important areas that have significant clinical relevance. We will conclude that the native, neurodynamic capabilities for therapeutic change are more fully utilized by a therapy of implicit memory depotentiation, as described in the second of this set of three articles (Ecker & Toomey, 2008). As a useful context for our discussion, we begin with a short review of the implicit memory perspective afforded by coherence therapy.

THE VIEW THAT FINDINGS IN NEUROSCIENCE IMPLY A THERAPY OF IMPLICIT MEMORY DEPOTENTIATION

In the first two articles of this series (Ecker & Toomey, 2008; Toomey & Ecker, 2007) we offered a detailed, neural and phenomenological model of a psychotherapy—coherence therapy—that putatively ends symptom production by selectively depotentiating symptom-requiring implicit memory. We now raise the question: How general is this form of symptom causation? What clinical symptoms and problems in living are caused by sub-cortical implicit memory? How generally applicable is a therapy of implicit memory depotentiation?

We argue below that the great majority of clinical symptoms are caused by implicit memory. If this proves to be true, the selective depotentiation of implicit memory would be the therapeutic strategy having broadest applicability and efficacy.

Implicit memory differs qualitatively from the vernacular connotation of the word *memory*, which is the subjective recall of either past personal experiences (episodic, autobiographical memory) or facts (semantic memory), both of which are stored cortically. In contrast, an activated implicit emotional memory in the limbic system is experienced as an immersion in a particular emotional state, such as strong anxiety or anger, with or without thematic or narrative meaning (such as a nonverbal expectation of imminent abandonment or a construal of being used), as well as a compulsion to carry out (or not carry out) a particular behavior. There is no conscious thought as to why the feeling and/or the behavior are occurring, no conscious recall of the experiences and learnings in the past that created this response, and no sense that one is experiencing a memory at all. Yet, as the case examples in our two prior articles have illustrated, full conscious retrieval of the coherent material generating this response usually elicits well-defined, specific knowledge structures as well as explicit, episodic memory of the original, concrete scenes and experiences in which these implicit knowings were formed.

The result of successful implicit memory depotentiation is the dissolution of the implicit knowings driving symptom production. Neurally, the synaptic circuits encoding those knowings then are no longer operational. Phenomenologically, the constituent knowings and constructs then no longer have an emotionally

compelling, subjective realness (although they remain in factual and autobiographical memory, having been translated into explicit memory in the course of therapy). For example, in the main case example used throughout this series, it no longer seemed real to Carol that for her to enjoy marital sex would result in repeating her mother's egregious sexual behavior, and to Tina in our second article (Ecker & Toomey, 2008), it no longer seemed real that she is powerless to keep her mother from taking possession of her interests and pursuits. When the symptom-requiring constructs are depotentiated, symptom production ceases permanently because its very basis and cause are eliminated permanently.

A therapy of implicit memory depotentiation consists, by definition, of locating, accessing, and depotentiating the specific, unconscious personal constructs that require production of the presenting symptom. Coherence therapy (Ecker, 2008; Ecker & Hulley, 1996, 2000a, 2004) is a particular methodology designed for efficiently carrying out these processes.

At the time of this article's publication—15 years since coherence therapy was first presented to psychotherapists—its practitioners have, collectively, several hundred years of experience in observing the effects of its methodology. Anecdotal evidence is a notoriously unreliable basis for assessing the effectiveness of a psychotherapy and the veracity of its model of change, yet a number of positive indications regarding coherence therapy, taken together, appear promising and warrant systematic, rigorous study:

1. Many experienced practitioners of other widely used therapies—such as cognitive-behavioral therapy, psychodynamic psychotherapy, and existential-humanistic therapies—have described a significant enhancement in both their frequency of therapeutic successes and the degree and quality of those successes, across a wide range of symptoms of mood, thought, and behavior.[1]

2. In agency settings, supervisors' recognition of heightened effectiveness appears to be a regular occurrence. For example, a coherence therapy practitioner who works in a large outpatient clinic has been promoted to core staff largely because the average number of sessions of his clients is dramatically lower than that of other clinicians. Another practitioner who was an

intern in an urban community mental health center was asked repeatedly by his supervisor to teach her his methods and subsequently, while still an intern, was offered a postinternship supervisory position, the first such offer to an intern in the long history of this agency.

3. A partial verification of coherence therapy's model of change is built into the methodology. The process with each client generates strong evidence that an implicit memory has actually been depotentiated: The previously vivid, emotionally laden content of the memory (the prosymptom position) fails to be reactivated by potent reminders and triggers, as described in our previous article (Ecker & Toomey, 2008).

4. The methodology embodies the following logic, which potentially also lends support for the model (Ecker, 2006): In the course of coherence therapy properly executed, nothing whatsoever is done to counteract, avoid, or prevent the symptom or the implicit, prosymptom constructs maintaining it. If a symptom then ceases to occur solely as a result of depotentiating a specific implicit memory, the causation of the symptom by that implicit memory is indicated. The clearest case occurs when the juxtaposition experience that depotentiates a prosymptom position is deliberately, overtly orchestrated by the therapist, as in the case of Tina in our prior article. Until that point, the work has amply (and verifiably) incorporated all of the nonspecific common factors widely regarded as the basis of clinical effectiveness (see, e.g., Hubble, Duncan, Miller and Hubble, 1999), but the symptom persists undiminished. It is only after an additional, well-defined event—the juxtaposition experience—that symptom cessation immediately occurs. (In order to conclude reliably that this verifies symptom causation by implicit memory [prosymptom positions], qualitative analysis is needed to establish that nothing counteractive has occurred and that the usual common factors were indeed present but did not dispel the symptom apart from the juxtaposition process.)

The foregoing four points constitute the current basis for the plausibility of coherence therapy. Of particular note, coherence therapy is regularly effective for achieving permanent cessation of longstanding mood problems widely regarded as requiring

neuromodulatory drugs for successful relief. (For detailed case studies of coherence therapy for depression, see Ecker & Hulley [1996, pp. 63–90; 2002a], and for case studies of anxiety and panic see Ecker [2003] and Ecker & Hulley [2000b, 2002b].) We take those results to indicate that the cause of the mood problem was in those cases psychological, not biochemical. This means that the neural and biochemical substrates were driven by the person's phenomenological and emotional material, not the other way around.

Finally, we suggested in the previous article that coherence therapy's methodology for implicit memory depotentiation fulfills plausible criteria for the optimal use of neuroplasticity in psychotherapy: (a) recruitment of the types of synaptic change known to be most potent (b) in the brain regions causing symptom production (c) as rapidly as these neuroplasticity mechanisms can work.

From this perspective, next we offer critiques of the currently widespread views of the implications of neuroscience for clinical treatment.

THE VIEW THAT FINDINGS IN NEUROSCIENCE IMPLY PHARMACOLOGICAL TREATMENT

As knowledge of the neural and molecular correlates of mental life grows, inevitably many new strategies will arise for chemical, bottom-up treatment of mental, emotional, and behavioral symptoms and sufferings. In this section, we examine the notion that pharmaceutical treatment is implied and justified by knowledge of the neurobiological mechanisms involved in mental life. We first consider what the drug effectiveness research shows and then examine the role and relevance of psychiatric medications in treating symptoms that are caused by implicit memory. Our discussion primarily addresses the most widely used medications—the SSRIs (selective serotonin reuptake inhibitors).

Letting the Drug Research Speak for Itself

Meta-analyses of clinical trials that were funded by drug companies have consistently shown that SSRIs are not more effective than placebos to a clinically significant degree. In 27 of

47 studies used to approve them, Prozac, Paxil, Zoloft, Effexor, Serzone, and Celexa failed to significantly outperform placebos (Kirsch & Sapirstein, 1998). Reviewing these data Kirsch, Moore, Scoboria and Nicholls (2002) summarized:

> Approximately 80% of the response to medication was duplicated in placebo control groups, and the mean difference between drug and placebo was approximately 2 points on the 17-item (50-point) and 21-item (62-point) Hamilton Depression Scale. Improvement at the highest doses of medication was not different from improvement at the lowest doses. . . . If drug and placebo effects are additive, the pharmacological effects of antidepressants are clinically negligible. (p. 1)

Fisher and Greenberg (1997) concluded from their review of the SSRI research that in studies showing a slight superiority of SS-RIs over placebos, the difference can more plausibly be ascribed to questionable experimental methodology then to true drug effect.

Likewise, in a more recent meta-analysis, Moncrieff and Kirsch (2005) concluded, "recent meta-analyses show selective serotonin reuptake inhibitors have no clinically meaningful advantage over placebo" and "methodological artifacts may account for the small degree of superiority shown over placebo" (p. 157).

In our experience, our psychotherapy colleagues meet these clear facts regarding the drug companies' own data with great skepticism, and they describe clients whose depression diminished significantly after using these drugs. In this regard, an account by journalist Gary Greenberg (2003) of a participant in a clinical trial at UCLA for Effexor (venlafaxine) is instructive.

Janet Schonfeld had suffered serious depression for over two decades when she read about a trial for antidepressant medication. She felt hopeful and excited about the possibility of a cure, and within a few weeks of enrolling in the study she was largely relieved of feelings of worthlessness and suicidal ideation, which she viewed as a dramatic improvement. She also experienced nausea, one of the drug's known side effects, leading her and her nurse to assume that she was receiving the active drug, not the placebo. However, at the completion of her 6-month participation she was alarmed to learn that she had been

taking an inert placebo and that her improvement could not be attributed to pharmacological action.

For what were no doubt both personal and social reasons, she had been keen to accept the pharmacological narrative stressing biochemical deficiency, corrected imbalances, and exogenous cures. Amazingly, her doctors then told her that despite her great improvement without taking Effexor, she would likely be better off by taking it. She agreed and took the actual drug for an additional two and a half years. Apparently she was unable to accept that she had been able to shed her symptoms entirely psychologically, on the basis of perceptions and expectations.

In drug tests, drug companies often identify and remove from their data pools subjects who demonstrate a strong and early response to placebos and other nonmedical treatments. In a procedure known as the placebo washout strategy, all subjects are placed on a placebo for the first week or two of a drug trial, and those who show a large recovery response are removed from the data pool before randomization, which significantly biases results in favor of drug efficacy. As many as 20% of participitants can be "washed out" in this way (Antonuccio, Danton, DeNelsky, Greenberg & Gordon, 1999; Kirsch, Moore, Scoboria & Nicholls, 2002).

Additionally, many doctors and patients *correctly* infer from the absence or presence of side effects whether they are in the control or treatment group, thus penetrating the double blind and compromising experimental design. Flawed placebo design also is indicated by the observation that when drugs believed not to have antidepressant properties (such as methylphenidate, benzodiazepines, and antipsychotics) are used as "active" placebos in order to mimic side effects, the already clinically insignificant superiority of the SSRI shrinks further or disappears (Moncrieff & Kirsch, 2005).

Despite the widespread clinical notion that SSRIs are more effective in more acute cases, Moncrieff and Kirsch (2005) concluded in their recent meta-analysis, "claims that antidepressants are more effective in more severe conditions have little evidence to support them" (p. 157).

The inability of researchers to clinically differentiate SSRIs from placebos (except with respect to negative side effects) seriously undermines the theory that these drugs reduce symptoms of

depression through a targeted readjustment of neurotransmitter levels.

Drug makers maintain that SSRIs are effective at reducing depressive symptomology because they correct an imbalance of neurotransmitters. This occurs, it is maintained, by blocking the reuptake of serotonin back into the cell after a neuron has fired, causing serotonin to remain longer in the synaptic junction between neurons. This results in an increase in overall serotonin levels, which, it is claimed, in turn alleviates depression. There is some evidence that depressed subjects do show differences in serotonin metabolism and utilization (Meltzer, 1989), and the partial blocking of serotonin reuptake by the drugs appears scientifically confirmed (Mann, 1999).

Nonetheless, while these two facts can be interpreted to support manufacturers' arguments, the placebo equivalence suggests that the decrease in depression experienced by those taking SSRIs is largely due to brain changes that occur naturally in response to expectancy of healing—the placebo effect—rather than to a true drug effect. (For reviews of placebo research, see Benedetti et al., 2005; Vallance, 2006). Indeed, recent evidence suggests the placebo responses to treatment for depression are mediated through changes in serotonerigic functioning (de la Fuente-Fernandez & Stoessl, 2002). The fact that dosage and blood plasma levels of SSRIs show minimal or no causal relation to treatment outcome (Amsterdam et al., 1997; Preskorn, 1997) raises further questions about the drug effects of SSRIs. Truly effective drugs are expected to show a distinct dose-response relationship.

Hyman and Nestler (1996) stressed that drugs in general and psychiatric drugs in particular do not "correct imbalances," but rather create them. They described the process as follows:

> Chronic administration of psychotropic drugs creates perturbations in neurotransmitter function that likely exceed the strength or time course of almost any natural stimulus. . . . The result of these types of repeated perturbations or initiating events is to usurp normal homeostatic mechanisms within neurons, thereby producing adaptations that lead to substantial and long-lasting alterations in neural function. (Hyman & Nestler, 1996, p. 154)

They noted that in the case of drugs of abuse, the cascade of compensatory adjustments initiate the well-studied negative biochemical feedback loops and dependencies that define physical addiction. They observed the same initial pattern in the major classes of prescription psychiatric drugs, which likewise initiate an imbalance or "chronic perturbation," and they commented, "it is less clear how compensatory adaptations to a drug would result in a therapeutic response."

Adding weight to the addiction hypothesis, an SSRI "discontinuation syndrome" has been identified and is under study. Black, Shea, Dursun and Kutcher (2000) reported 53 symptoms that can accompany or follow tapered discontinuation and "are not due to a . . . recurrence of a mental disorder" (p. 255). These symptoms cease within 72 hours of restarting the SSRI. In a review, Tamam and Ozpoyraz (2002) emphasized that (a) many "somatic and psychological symptoms" of discontinuation were "not noted during short-term efficacy studies," and (b) these symptoms "cannot be explained as a remanifestation of the original disorder" (p. 17). The implication is that SSRIs produce a biochemical dependency with withdrawal symptoms, in addition to failing to demonstrate clinically significant antidepressant drug properties.

Further evidence suggests that serotonin reuptake inhibition does not remedy a depression-producing, serotonergic imbalance: (a) A serotonin reuptake *accelerator*, tianeptine, with precisely the *opposite* effect on serotonin reuptake than the SSRIs have, was found to be as effective an antidepressant as Prozac (Lôo, et al., 2001). (b) A blocker of the neurotransmitter glutamate, ketamine, produced far stronger and far faster antidepressant effects than do any of the SSRIs, without affecting serotonin levels (Zarate et al., 2006).

In our view, the totality of the data we have reviewed above indicates that the therapeutic effects of SSRIs are due almost entirely to the endogenous placebo effect, possibly enhanced to a small, clinically insignificant degree by a complex cascade of neural responses constituting an abnormal brain state (as distinct from a corrected imbalance) that has many effects, coincidentally including a reduction of mood symptoms along with a large number of other, unwanted (side) effects. (The array of effects of SSRIs has even been shown recently to include enhanced production of new neurons in the hippocampus [Encinas, Vaahtokari, &

Enikolopov, 2006; Santarelli et al., 2003], bringing the hypothesis that this, rather than the increase of synaptic serotonin levels, could be an actual antidepressant mechanism.)

Psychiatrist Pedro Delgado (2000) summarized three decades of research on the neurotransmitter imbalance/deficiency model of depression with the following frank admission: "Intensive investigation has failed to find convincing evidence of a primary dysfunction of a specific monoamine[2] system in patients with major depressive disorders" (p. 7). He further summarized that while experimentally induced serotonin depletion causes a drop in mood for clients currently taking SSRIs, it has *no effect* on the mood of unmedicated depressives and nondepressed controlled subjects. This indicates from yet another angle that the serotonin depletion model of depression is fallacious.

Thus, there are two major findings that go against the view of depression as caused by a serotonin imbalance warranting pharmacological treatment: first, depressives show no "primary dysfunction" in their neurotransmitter function, and second, the biochemical effects of SSRIs do not produce clinically significant antidepressant drug effects.

The research, including the drug companies' own data, forces us to conclude that SSRI effects are primarily, and perhaps entirely, due to nonpharmacological effects. When inert placebos, other drugs not claimed to have antidepressant effects, and both the inhibition and the acceleration of serotonin reuptake have all proven roughly equivalent across dozens of well-controlled trials, there is a serious problem with the argument that the neuroscientific data imply that depression is caused by a neurotransmitter imbalance that should be corrected exogenously by psychiatric drugs.

In contrast to these impressively unimpressive data, we can consider a drug with a well-documented drug effect. Lipitor (atorvastatin), for example, regulates blood cholesterol through a well-understood biochemical process—the inhibition of a specific enzyme in the liver that metabolizes cholesterol—and was found effective for achieving target cholesterol levels for 85% of subjects. This rate is over five times the 16% efficacy of placebos ($p \leq 0.001$; Harris, Wheeler, & Chong, 2002).[3]

A meta-analysis by Walsh, Seidman, Sysko and Gould (2002) showed that the rate of response to both placebos and

medications grew steadily between 1981 and 2000 at the rate of about 7% per decade. As it is absurd to argue that sugar pills had steadily improved in quality, we believe that a constructivist, meaning-based account of the increase is indicated. Lacasse and Leo (2005) presented a side-by-side comparison showing a striking disparity between the pharmaceutical companies' advertising rhetoric, on the one hand, and the weakness of the scientific evidence for their claim that depression is caused by a serotonin deficit, on the other. Finally, Turner, Matthews, Linardatos, Tell and Rosenthal (2008) documented the Food and Drug Administration's extensive, systematic suppression (nonpublication) of antidepressant drug trials that yielded negative results. Among 74 FDA-registered studies, those "viewed by the FDA as having negative or questionable results were, with 3 exceptions, either not published (22 studies) or published in a way that, in our opinion, conveyed a positive outcome (11 studies). ... A total of 37 studies viewed by the FDA as having positive results were published; 1 study viewed as positive was not published. According to the published literature, it appeared that 94% of the trials conducted were positive. By contrast, the FDA analysis showed that 51% were positive." The elimination of negative results increased the all-important effect size by from 11% to 69% for individual drugs and 32% overall.

Psychiatric Medications for Symptoms Caused by Implicit Memory?

Throughout this three-article series we have argued that subcortical implicit memory is the cause of most of the symptoms and problems presented by most people receiving psychotherapy. This includes depression and anxiety,[4] the symptoms that draw the heaviest use of medications, including SSRIs. If indeed the cause of these symptoms lies in implicit memory, how does the argument that neuroscience implies pharmacological treatment fare?

If a mood symptom is generated by an implicit memory, the cause is a functional, not structural, impairment. As noted earlier, an implicit memory operates not as an autobiographical, episodic memory-of-the-past, but as a model of a particular aspect of the world and a knowing of what to feel and how to act in that context (although only the feeling and the action are conscious, not the knowingness). An implicit memory is a modifiable, functional

schema that is created, held, and applied by the normal operation of the mental system.

In a neural network with an encoded implicit memory that maintains an unwanted mood, the synapses and neurons are *not* damaged or malfunctioning. For instance, Tina, whose therapy for depression we studied in our previous article (Ecker & Toomey, 2008), had, in addition to the prosymptom position (implicit knowing) described above, another one that consisted of expecting to be sharply attacked verbally and emotionally by a family member if she expressed any knowing or feeling of her own, so keeping her energy and feelings deadened (depressed) was urgent in order to be safe. In the subcortical neural circuits encoding that coherent schema, all of the neurons and synapses were working properly. It was the *implicit memory as a whole* that, in a top-down manner, maintained the dulled state required for safety and controlled the synaptic release of neurotransmitters involved in expressing that mood physiologically. (The fact that the behavior of synapses can be governed by top-down, experience-driven processes was demonstrated strikingly in a recent study by Chen et al. [2008]. When rats voluntarily self-administered cocaine by pressing a lever, they formed ultra-long-lasting, extinction-resistant synapses maintaining their memory of and addiction to cocaine. However, when cocaine was instead injected, eliminating any experience of desire, motivation, or choice, no such synapse formation was detected at all. The study indicated that experiential factors are in some situations more important than chemical conditions in governing the behavior of synapses.)

This understanding of the normal functioning of implicit memory can be applied to the finding that the brain region known as the subgenual cingulate, or Brodmann area 25, is chronically highly active in depressed persons (Mayberg et al., 1999), and that applying ongoing electrical stimulation ("deep brain stimulation") to this region largely eliminates depressive symptoms in two-thirds of tested subjects (Mayberg et al., 2005). Area 25 is in the paralimbic cortex, an older part of the cortex that is in close contact with the limbic system and is an important pathway of cortico-limbic communication and coordination. Based on our central tenet that depression, like many other symptoms, is generated by coherent personal constructs in implicit memory, we conjecture that what keeps area 25 highly activated is not a

neurological malfunction, as neuroscientists hypothesize, but a chronic activation of implicit personal constructs. This implies that brain scans of depressed clients in coherence therapy should show a quieting of area 25 as soon as their depressogenic implicit constructs have been revised or depotentiated. In contrast, neither deep brain stimulation nor antidepressant drugs can remove the implicit constructs that may be driving the hyperactivation of area 25, although they might temporarily reduce the depressive effects of those constructs.

Perhaps ultimately most important for understanding the brain's response to SSRIs is a systemic view of the relationship between biochemistry, implicit memory, and psychology. Consider the situation of most people who take an SSRI to counteract depression. When a person's depression is generated by an implicit memory, the depressed state is emotionally necessary according to the content of that subcortical implicit memory (prosymptom position). SSRIs were not developed to block the complex circuits by which an implicit memory in the limbic system implements a necessary state of depression. As a rule, each neural, synaptic, and molecular process in the brain has several independent, alternate pathways, making the brain a supremely flexible and adaptive system (Panksepp, 2004). Subcortical systems that "know" they must create a state of depression in order to avoid a feared attack have many options for doing so, such as reducing the total number or sensitivity of serotonin receptors, creating changes in any of the molecular pathways of many other types of neurotransmitters, altering the strengths and numbers of synapses, or adjusting any of the mood-affecting endocrine hormones in any number of ways.

The same argument can be applied to other classes of antidepressants, which include monoamine oxidase inhibitors, dopamine reuptake inhibitors, norepinephrine reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, tricyclics, and tetracyclics. All of these drugs target a small number of specific elements within a dizzyingly complex system involving hundreds of genes, proteins, synapses, receptors, transmitters, enzymes, and functionally encoded neural networks.

Looking specifically at the complexity of the serotonin system, Carrasco and Sandner (2005) described the remarkable diversity of biological mechanisms recruited across the functioning of different SSRIs, noting that, in addition to modulating

serotonin reuptake, "transsynaptic effects such as modulation of signaling cascades, gene expression processes, and neuroplasticity are also important in the mechanism of action of antidepressants" (p. 1428). Synapses have over a dozen distinct serotonin receptor subtypes, which respond differently to structurally distinct SSRIs such as paroxetine (Paxil) and fluoxetine (Prozac; Olivier, Van Oorschot, & Waldinger, 1998). Likewise, on synapse surfaces there are several different enzymes (proteins) that carry out serotonin reuptake, and these are also subject to different levels of inhibition by different SSRIs (Preskorn, 1997). Again, given this complexity, it appears that in any attempt to biochemically blockade a symptom there will be holes through which a prosymptom position could find a way to express itself. To assume that a pharmaceutical intervention can trump the operation of coherent implicit memory may be wishful, naïve thinking that greatly underestimates the neuro-ecology of the brain.

Proponents of SSRIs would argue, presumably, that the drugs subdue the brain systems that implicit memory activates. Studies have detected a suppression of sympathetic nervous system activity in response to SSRI use (Shores, Pascually, Lewis, Flatness & Veith, 2001). However, such suppression is nonselective and likely causes the troubling and often unacceptable emotional flattening and loss of libido described by many SSRI users. Conceivably, for some users the drugs may induce an actual drug effect in dulling emotional responsiveness broadly. However, suppression of the central nervous system by no means necessarily translates into an effective treatment for depression. Many drugs, including alcohol and barbiturates, also suppress central nervous system activity.

The possible role of nutritional deficiency may be a noteworthy exception to our arguments against the view that psychological symptoms are produced by biological defects or imbalances. For example, there is evidence that depressed persons have decreased blood plasma levels of omega-3 fatty acids and that the reduced levels "are associated with the severity of depression" (Edwards, Peet, Shay & Horrobin, 1998, p. 149.) Omega-3 fatty acids, like certain other micro- and macronutrients, are an essential building block needed by the brain, and it is biochemically plausible that a depletion could generate a structural neural deficiency with deleterious psychological effects, such as depressed mood. Treatment in the form of orthomolecular supplementation and dietary

adjustments would ideally occur in tandem with coherence-focused depotentation of any implicit memory that is further amplifying the mood symptom.

Genes, Drugs, and Serotonin

In contrast to the functional causation of symptoms by implicit memory, neuroscientists have recently begun to identify true structural diseases of the synapse, such as a genetically caused, synaptic molecular deficiency that manifests as fragile X, the most common form of hereditary mental retardation (Huber, Gallagher, Stephen, Warren & Bear, 2002). Medications created for such conditions could be designed to truly remedy their causes, rather than merely suppress symptoms.

The distinction between functional and structural causation of symptoms can also shed light on recent research into the powerful role of neural genes in our emotional life. Genes have been identified that synthesize proteins that play a critical role in the creation of specific emotional states, such as fear or depression, by controlling synapse strength in particular subcortical circuits. For example, mice genetically altered to lack the gene *stathmin* show no fear in situations that reliably engender innate fear responses in normal mice, and also exhibit decreased memory for conditioned fears (Shumyatsky et al., 2005). Mice genetically altered to lack the gene GRP showed, in contrast, a marked intensification of learned fear, but no change of instinctive, innate fear (Shumyatsky et al., 2002). Deletion of the gene TREK-1 yields mice that show clear signs of being resistant to depression and stress (Heurteaux et al., 2006).

What are the implications for psychotherapy of these and similar discoveries of how gene expression influences emotional states? Eric Kandel, coauthor of the *stathmin* and GRP studies just cited, commented that the results indicate potential drug targets for instinctive fear and learned anxiety (HHMI, 2005). He stated also,

> Since GRP acts to dampen fear, it might be possible in principle to develop drugs that activate the [protein that GRP produces], representing a completely new approach to treating anxiety. ... [I]n studies of fear learning we could well have an excellent beginning for animal models of a severe mental illness. (HHMI, 2002)

Similar comments have been offered by authors of the TREK-1 study.

Only if the cause of a person's anxiety or depression happened to be a structural impairment of *stathmin*, GRP, TREK-1, or some other gene involved in the neural circuitry controlling the particular mood-state would the effect of a gene-targeting drug be a true corrective to the actual cause of the mood symptom. We contend that this would prove to be the case for at most a very small percentage of therapy clients.

If, on the other hand, the cause of the individual's anxious, fearful, or depressed mood is an implicit memory, a gene-targeting drug treatment could only serve to suppress the symptom and its actual cause. Such a switching-off of mood circuitry would be global, possibly eliminating appropriate, adaptive fear responses that maintain personal safety. Likewise, switching off the depression circuitry might eliminate the capacity to feel appropriate sadness, grief, and sorrow, which are integral to the human experience of love and meaning.

Moreover, there may be other important functions served by these genes and the proteins they synthesize. Even if the drug reduces anxiety or depression, the implicit memory responsible for the troubled mood would continue to exist, leaving the person fully susceptible to relapsing upon discontinued use of the drug, or to symptom expression via alternate neurophysiological pathways.

By analogy, if the temperature in one's home is chronically too high or too low, the appropriate response depends on whether the cause is a defective central furnace (corresponding to a genetic impairment) or the setting on the thermostat (corresponding to an implicit memory producing, for example, anxiety or depression). One should not tamper with the furnace if the thermostat is causing the problem.

It is well established that as a rule, the activity of genes is highly regulated by both the external environment and the internal, physiological/psychological environment. Genes alone do not determine many manifested traits. Genes can be silenced, regulated, and flexibly switched on and off through interactions with the environment and the organism itself (Cirelli, 2005; Lewontin, 1980, 2001; Meaney, 2004). As the following example shows, research is already finding that certain genetic

vulnerabilities are expressed as psychological symptoms only if the neural system is driven by an implicit memory involving expectations that entail anxiety or depression. In such cases, the psychotherapeutic clearing of those implicit memories should be the first strategy of treatment.

Consider the much-discussed short allele version of 5-HTT, a gene implicated in impaired utilization of serotonin. In human studies, Hariri et al. (2002) found that subjects with this gene demonstrated increased amygdalar activation in response to fearful stimuli, implying that the gene enhances threat appraisal and, we would add, may act as an amplifier on any fear-based implicit memories. Caspi et al. (2003) then found that this gene in humans correlates with an increased susceptibility to depression in subjects with major life stresses or trauma. However, subjects with the short allele, depression-prone gene who had not suffered major stressors had the *same probability* of developing depression as subjects with the normal, long allele version of 5-HTT. The implication of the study is that only in tandem with negative implicit memories does the short allele form of 5-HTT cause expression of psychological symptoms.

Likewise, Bennett et al. (2002) found that the same short allele form of 5-HTT did not have deleterious effects in monkeys who had normal amounts of nurturing from their mothers; but monkeys with deficient rearing showed a wide range of symptoms, including deficiencies in early neurobehaviorial development, central nervous system integrity, serotonin metabolism, increased antisocial aggression, and propensity for alcohol consumption. These researchers concluded that the gene's regulatory region interacts with early experience to affect central 5-HTT functioning. These findings indicate an environment-dependent effect of the 5-HTTL genotype (Bennett et al., 2002, p. 119).

These observations suggest a model in which constructions, meanings, and protective strategies that are formed in response to stressors interact with the 5-HTT gene in a way that increases its depression-inducing activity. In such emotional-stress-related cases, the resultant depression is neither irreversible nor entirely genetically determined. Transformation of the implicit schema(s) affecting this gene could be expected to prevent depression despite the presence of the gene.

Similarly, symptom-producing imbalances of hormones (such as cortisol or adrenaline) could be maintained by a chronic reactivation of implicit memory. For instance, an implicit memory that evaluates some aspect of a client's usual environment as threatening could maintain chronically high levels of adrenaline.

The recognition of bottom-up causation of mood states—the knowledge that ingestion of a substance can strongly influence subjective experience—is prehistoric. However, the fact that a symptom such as a mood state has measurable chemical and/or neural correlates does not necessarily imply bottom-up causation of that symptom. We contend that top-down processes launched by subcortical implicit memory play a primary role in the creation of most psychological symptoms. In support of that contention is our observation that the methodology of coherence therapy, which specifically targets causation by implicit memory, shows consistency of clinical effectiveness across a wide range of clients and symptoms. Controlled studies are of course needed to verify this inference.

THE VIEW THAT FINDINGS IN NEUROSCIENCE IMPLY A THERAPY OF REPARATIVE ATTACHMENT

Neuroscience has shown that new experience can significantly shape the brain's neural wiring throughout the lifespan. During infancy and childhood, neuropsychological development of the brain is powerfully affected by interpersonal interactions and attachment patterns with primary caregivers. It is in response to these interactions that the developing infant organizes and regulates its own subjective states and, to a significant degree, the wiring of its brain. Nonattuned, nonnurturing, and traumatic interaction patterns can have deleterious, symptom-generating effects on brain development, including the nonintegration and discoordination of various brain systems; the formation of behavior- and mood-destabilizing implicit memories, with no corresponding explicit memories or coherent autobiographical narrative; and reduced hippocampal volume (Bremner et al., 1997; Schore, 2001a, 2001b; Siegel, 1999).

Therefore, as indicated in the following quotes, a central goal of psychotherapy in such cases should be to bring about the neural integration and the concomitant, coherent narrative that were

disrupted by disturbed attachment patterns and trauma early in childhood.

> Coherent narratives can thus be proposed to be a product of the integration of left and right hemisphere processes: the drive to explain cause and effect relationships (left) and to understand the minds of others and of the self within autonoetic consciousness (right). In this manner, we can propose that coherent narratives reflect the mind's ability to integrate its processes across time and across the representational processes of both hemispheres. (Siegel, 2001, p. 87)

> The co-construction of a coherent narrative of the trauma may emerge in a relational [therapeutic] context which promotes a callosal transfer of affective information from the right to left orbitofrontal regions. This structural advance allows for left hemispheric retrieval and explicit semantic processing of right hemispheric emotional states encoded in implicit memory. (Schore, 2001b, p. 245)

A sizable fraction of working therapists have interpreted these conclusions from neuroscience to mean that because relational experiences were the cause of a person's neurodevelopmental disruptions, they need to be the cure: Effective psychotherapy should centrally consist of the use of the client–therapist relationship to reverse the effects of the client's original, negative experiences of relationship by reliably producing positive experiences of reparative attachment in the form of the therapist's caring attention and attuned, empathic understanding (see, for example, Lewis, Amini, & Lannon, 2000). The aim is for these experiences to bring about a reorganization of neural linkages, building neural integration and manifesting as emotional and behavioral stabilization.

According to this widespread view, neuroscience implies a therapy of reparative attachment, and the essence of therapy should be a highly skillful reparenting, providing the client with consistent experiences of the reliable, accurate empathy and understanding that were lacking earlier in development. The client's felt experience of receiving the therapist's empathy and understanding is seen as the crucial transformative ingredient in this therapy. The work therefore entails a central, explicit focus on the client's experience of interactions with the therapist.

A therapy of reparative attachment is optimal, undoubtedly, for a certain population of therapy clients. But how generally applicable is this approach? What determines whether reparative

attachment is a suitable form of therapy for dispelling a given symptom presented by a given client? We propose that the situation is defined by a decision tree consisting of two questions:

1. Is the presenting symptom based in a troubled attachment pattern?
2. If an attachment pattern or schema *is* the basis of the presenting symptom, is that attachment pattern changeable through new experiences of interaction with the therapist?

We suggest that only when the answer to both of these questions is *yes* can a therapy of reparative attachment be appropriate. If the answer to either question is no, reparative attachment work is inappropriate because it cannot dispel the client's presenting symptom. The fact that the answer to either question *can* be no and often *is* no is illustrated by case examples below.

Answering these two questions theoretically is a complex and subtle matter that would go well beyond our scope here. However, they can be answered phenomenologically with each therapy client through methods employed in coherence therapy, without relying on interpretations or theorizing. We will consider each of the two questions in turn.

Is the Presenting Symptom Based in a Troubled Attachment Pattern?

A therapy client's attachment pattern is an implicit schema made up of specific procedural constructs, or knowings, of the forms of available connection with others and the specific rules, behaviors, and conditions that constitute connection and relatedness with others. Coherence psychology refers to this material as the *terms of attachment* that caregivers imposed or that the individual has construed in response to interpersonal experiences.

As described in our previous article (Ecker & Toomey, 2008) and recapitulated earlier, the great majority of clinical symptoms are caused by specific personal constructs in implicit memory. Question 1, then, is equivalent to asking: Does the client's presenting symptom, such as insomnia or reactive anger, arise from implicit constructs that define terms of attachment?

The therapist cannot answer that question until the discovery work has revealed the prosymptom constructs. One person's

reactive anger *will* prove to be arising from an attachment-defining pro-symptom position, whereas the prosymptom position producing the anger of another client will have nothing to do with terms of attachment.

For example, a man began therapy for his hair-trigger, excessive anger whenever he perceived anyone as departing even slightly from what he thought had been agreed. He became aware in therapy that he was "stuck" in anger over being betrayed by a business partner who had robbed him of a small fortune five years earlier. He further discovered that he remains "stuck" in this anger for several well-defined purposes: in order not to feel powerless; in order not to feel intense grief; and in order to sustain a fantasy of demanding and getting accountability from the betrayer and the universe, preserving the moral nature of the world. The emotional truth of his anger, in other words, is that he needs it to protect himself from collapsing into feeling powerless, grief-stricken, and in despair and disillusionment over a world that has no moral order.

Guided experientially to see if any of these themes and purposes had a resonant feeling of similarity or connection to what he experienced in his family of origin—his terms of attachment—he found that they did not. These were *existential* issues that he was solving with anger, not attachment issues. For him, the answer to Question 1 was *no*—his symptom of anger was not based in a troubled attachment pattern. Because his symptom-requiring constructs did not define or pertain to terms of attachment, the therapist's attuned empathy could not, in itself, create an experience that would disconfirm and transform his symptom-requiring constructs. Therefore, a therapy of reparative attachment was not appropriate for dispelling his anger.

If an Attachment Pattern Is *the Basis of the Presenting Symptom, Is the Symptom Changeable Through New Experiences of Interaction with the Therapist?*

Consider, for example, Carol in our earlier case example. Her presenting symptom was her aversion to sexuality in her marriage. Her discovered prosymptom position consisted of traumatic memory of her mother's noxious, sexualized relationship with her and her implicit expectation that her own sexuality would have the

same terrible effects on her own daughter (which in turn necessitated her symptom of suppressed sexuality). This is a case, then, in which an attachment pattern (the client's construction of terms of attachment with her mother) *was* the basis of the presenting symptom.

However, the answer to Question 2 is *no* because this was not an attachment pattern changeable through new relational experiences with the therapist. Carol's relationship with her therapist had no subjective relevance to what she expected herself to enact in her relationship with her daughter. Carol's positive experience of her therapist—no matter how empathic, safe, and trustworthy—could never specifically disconfirm her expectation of replicating her mother's sexualized relational role. Therefore reparative attachment methods were not suitable. The therapist's accurate empathy could not, in itself, transform the implicit personal constructs maintaining the symptom. The knowings that would successfully disconfirm Carol's expectations were not to be found in experiences of the client–therapist relationship.

As the foregoing two examples suggest, reparative attachment therapy is not suitable for a large fraction of clients and therefore cannot be the general form of psychotherapy implied by neuroscience.

Of course, with some clients the client–therapist relationship *is* subjectively relevant to an attachment schema generating a presenting symptom. In that case, that attachment schema *is* amenable to being changed by the client's experience of the therapist.[5] Then the answer is yes to both questions, and reparative attachment methods are a valid option—among various other alternatives, as described below.

Reparative Attachment Work Within Coherence Therapy

From the perspective of coherence psychology, the implicit, subcortical personal constructs that make up an attachment schema are not fundamentally different from the implicit constructs that define and govern other areas of personal experience and behavior. The same meta-methodology of change applies: the creation of experiences that discover, integrate, and then transform the symptom-requiring constructions, or prosymptom positions.

Within coherence therapy, a client's attachment schemas can be transformed by various types of experiences. The use of experiences of the therapist's empathic understanding (reparative attachment work) is one option among many. When the therapist opts to use reparative attachment (a valid choice when both questions defined above are answered in the affirmative), coherence therapy's focused methodology tends to enhance the effectiveness of that approach. According to coherence therapy's principles of how constructs are transformed (see Ecker & Toomey, 2008; Toomey & Ecker, 2007), positive experiences of the therapist's empathy and sensitive understanding do not automatically revise a client's troubled, original terms of attachment held in implicit memory. The transformative effect occurs only as a result of a deep disconfirmation produced by a conscious juxtaposition of the new relational experience and the longstanding attachment schema. That is, the two mutually contradicting emotional realities have to be experienced explicitly and simultaneously. By actively working to create this disconfirming juxtaposition experience, a coherence therapist ensures that new relational experiences have a transformative (as distinct from merely counteractive) effect.

In addition, the coherence-guided discovery process of coherence therapy (also described in Ecker & Toomey, 2008) typically yields a swift elicitation of the client's specific attachment schemas, usually in one or two sessions, bringing them from the implicit background to the explicit foreground of awareness. (This is the "left hemispheric retrieval and explicit semantic processing of right hemispheric emotional states encoded in implicit memory" described by Schore.) The high level of explicit verbalization sought in coherence therapy (in tandem with vivid subjective experiencing) enhances the accuracy and reliability of the transformation process because the therapist knows exactly what constructs need to be juxtaposed and disconfirmed.

For example, a 42-year-old man was in therapy because none of the marriages or other couple relationships in his life had ever developed an emotional closeness, and he had finally realized, "It must be my fault." He was the middle of five siblings, with authoritarian parents whose range of behaviors did not include supplying children with personal understanding or emotional attunement. In a guided visualization he was back in that family at 6 years of

age. When he was absorbed in the scene and describing details, the therapist, a woman, asked him, "What's it like for you, there in your family, when you're scared or hurting over something?" Persisting with this focus for 5 minutes resulted in his first awareness of a lifelong emotional truth, verbalized as, "This is not a world where anyone will pay attention to what I feel or give me understanding for how I'm hurting. I don't matter, and I'm all on my own."

This brought quiet tears. The therapist said, "There you are, a little boy of 6, and sometimes you're really hurting or really scared, and you need a grown-up to understand that and take care of you, but it never, ever happens, so you feel very alone, like you're all on your own in this world. Is that right?" His tears increased as he nodded assent. The therapist allowed a short silence and then created an experience of disconfirming juxtaposition by softly saying, "And how is it for you right now to be so in touch with that—your certainty that you will never get caring understanding for what hurts—and at the same time, to recognize that you are actually getting that kind of emotional understanding from me, right now?"

At this he burst into deep crying, fully feeling both the intensity of his long-suppressed anguish and a kind of amazed relief that the impossible was suddenly happening. When his crying subsided, the therapist repeated the same question, again guiding him into the juxtaposition experience. The man thought for a moment and said, "If it's possible for that to happen, then why did I get parents who couldn't do it?" Here he again cried quietly, the beginning of a grieving process that typically accompanies the recognition and revision of troubled attachment schemas.

In subsequent sessions during the next two months, the therapist continued to find opportunities to create disconfirmation experiences. For example, when the client gave a dry, matter-of-fact account of being spoken to demeaningly by his boss at work, the therapist said, "I notice that you mention nothing at all about *how you feel* from being talked to in that way by him. And I don't know if you're assuming I'll be like your parents and won't care about what you're feeling, or if you're assuming that I'm different from them and *will* pay attention and understand what you're going through. Could you tell me about both sides of that?"

In that way the therapist was deliberately guiding him to again hold both emotional realities, side by side, juxtaposed. Because attachment schemas are such central, life-organizing constructs with far-reaching ramifications, a series of disconfirmation experiences across a range of contexts tends to be necessary in order to transform them fully enough for relapses to cease.

Unlike therapies oriented mainly toward achieving reparative attachment, in coherence therapy the therapist's empathy is not regarded as being curative in itself, but only as serving to disconfirm troubled attachment schemas via juxtaposition with them. What is curative is the client's internal act of retrieving unconscious constructs into explicit subjective awareness and subjecting them to experiential disconfirmation. If the client's attachment schemas do not come into experiential juxtaposition with the therapist's empathy and understanding, those attachment schemas will not be altered, no matter how much empathy is delivered by the therapist and enjoyed by the client.

Similarly, original, troubled terms of attachment that have become conscious and integrated, successfully creating a coherent autobiographical narrative, do not automatically transform as a result, because this much can occur without the original attachment schema being subjected to a transforming disconfirmation. The attachment schema continues to feel real, even though fully explicit and conscious, and even though the client might now recognize it cognitively as being true in "the past," not in "the present." Reparative attachment work that relies centrally on creating a past–present distinction is bound to falter because, while the client's neocortex finds the past–present distinction factually meaningful, terms of attachment are held as unconscious knowings in implicit memory, which is timeless and immune to that distinction. The past–present distinction is therefore only a counteractive, nontransformational measure that pits the neocortex against subcortical systems of implicit memory. An attachment schema that has not been experientially disconfirmed continues to feel potently real even when fully conscious and even if the neocortex regards it as being from the past. The neocortex's notion of past versus present can be truly effective only as an adjunct to a disconfirming juxtaposition.

Transformation of Attachment Schemas in Coherence Therapy

Even when use of the client–therapist relationship for reparative attachment work would be a suitable option according to the criteria described above, it is not the therapist's only way to create experiences that transform a client's troubled attachment schema. That is, reparative attachment work is not essential for effective transformation of attachment patterns. Coherence therapy provides for the creation of a wide range of other kinds of experience that can discover, integrate, and transform the personal constructs, or prosymptom positions, maintaining negative attachment patterns.

Imaginal techniques provide myriad ways of creating potent experiences of old and new attachment schemas in relation to any significant attachment figure, past and present. Such techniques become indispensable when reparative attachment work via the client–therapist relationship is *not* suitable. The therapist tailors imaginal experiences in which the client interacts with a selected attachment figure. This is effective because, as described in our previous article (Ecker & Toomey, 2008), personal constructs in implicit memory activate in response to both real and imagined percepts.

For example, an integration experience could be created for the discovered attachment schema of the man described above by guiding him to imagine being 6, looking at his parents, and privately thinking explicitly, "If *they* don't want to pay any attention to me and how I'm feeling, I'm sure this means nobody ever will, so I'd better not try to tell anyone what I'm feeling, because they won't care." This could be followed by visualizing all of his former wives and girlfriends and saying to all of them an overt statement of his emotional truth: "My parents didn't care about me feeling hurt or troubled, so I'm sure you won't either, and I'd only get hurt worse if I tried to tell you what I'm feeling. I wish we were closer, but I know better than to trust you or anybody to care about what I feel." Explicit, highly personal, utterly candid verbalizations of that kind are effective for achieving thorough, stable retrieval of implicit emotional schemas.

In an imaginal version of the *symptom deprivation* technique (Ecker & Hulley, 2000a, 2004), this man would be guided into a preview experience of "how it will be when you're no longer

viewing all others as not caring about you, and instead you know that some people *will* care, pay attention, and understand." The purpose of this exercise is for the client to encounter any *un*welcome consequences that he expects implicitly, making it more important to maintain the symptom than live without it, even with the suffering it brings. Every ecological consequence of transformation that is daunting is a theme of unconscious necessity for maintaining the symptom. In coherence therapy, as these daunting consequences are made conscious, addressed as legitimate challenges, and rendered workable, the resistance fades and transformation proceeds.[6]

These are just a few examples of a wide range of possibilities for experiential work on attachment schemas, other than through relying on the client–therapist relationship to provide reparative attachment experiences.

Coherence and Attachment

Main, Kaplan, and Cassidy (1985) determined across multiple studies that the security of attachment of a child is predicted to better than 80% accuracy by how coherent (well-knit) the parents' conscious narratives (sense-making) of their own childhoods are (for a review, see Hesse, 1999). This finding is widely understood as indicating the great importance of forming a coherent, conscious narrative for the sufferings and ordeals one passed through in childhood. Parents whose narratives lacked coherence had troubled attachment in their own childhoods.

Coherence therapy brings benefits that relate directly to these findings. As understood within the framework of coherence psychology, even when an adult lacks a coherent, conscious narrative of childhood experiences, the person's implicit, *un*conscious knowledge of his or her childhood ordeals nevertheless is already coherent and, if retrieved into conscious experience, is the therapeutically optimal guide for making sense of what was suffered and for forming the needed conscious narrative. Not *any* coherent narrative, not *any* way of making sense of what happened in childhood is emotionally and neurologically healing and symptom-dispelling. The most therapeutic narrative is formed as the honest facing, verbalizing, and declaring of what the subcortical brain already knows in implicit memory. Furthermore, this verbaliza-

tion is an important part of integrating that subcortical memory and setting it up for transformation, as described in our previous article.

Coherence therapy appears to offer a methodology and conceptual framework that is well-suited to the therapeutic agenda described in Siegel's (1999) call for an "understanding [of] the factors and mechanisms the mind can use to achieve a coherent integration . . . in the face of a suboptimal attachment history" (p. 90). The narrative and neurological integration that Schore, Siegel, and Main have emphasized is a built-in, central result of coherence therapy methodology.

How the Therapist's Empathy Fosters Change

As noted above, in coherence therapy the therapist's empathy can be used to create a disconfirmation of troubled attachment schemas, but this is a special case, not a defining feature of the methodology. In addition, the therapist's empathy has other, catalytic effects that always support the core methodology and with some clients are critical to its success. Ecker and Hulley (2004, p. 59) provide the following account of these effects of *coherence empathy,* a phrase that denotes empathy focused on the content of a prosymptom position (as distinct from empathy toward the antisymptom position's wish to be rid of the symptom).

1. *Calming accompaniment.* For some clients, even to approach their prosymptom material on their own would bring feelings of aloneness or fear that are unbearable. Feeling closely accompanied by the therapist greatly reduces these daunting feelings, making it workable to go into the material.

2. *Fostering alignment of neocortical and subcortical knowledges.* Going into unconscious emotional material alone (without the therapist present) would, for some clients, result in merely merging into the troubled material and helplessly resuffering it in its original intensity, a plunge into earlier, developmental states without retaining the neocortical, adult, observing self. In contrast, being in empathic connection with the therapist while going into the troubled, prosymptom material helps retain the participation of the client's adult, observing self. This has two

crucial effects that promote neural integration: (a) As experi-
enced by the client's adult self, the unresolved emotional ma-
terial is now tolerable to experience (whereas it is intolerable
if merely merged into), and the client can open to it and allow
integration of it. (b) Retaining to some degree the perspective
of the adult, neocortical state while immersing in the subcorti-
cal material allows the client to apprehend, verbalize, and in-
tegrate the symptom-generating meanings (prosymptom con-
structs) she or he originally formed, which sets the stage for
transforming them.

3. *Creating the state of neuro-affective well-being.* Most therapy clients
   initially regard their presenting symptom as a form of irra-
   tionality or defectiveness, but upon fully integrating a prosymp-
   tom position, the underlying deep sense and emotional truth
   of the symptom is directly experienced. This inherently in-
   cludes experience of:

- a deep connection-with-self;
- the actual coherence of self;
- a profound authenticity of self; and
- being seen, heard, intimately understood, honored, and af-
  firmed at this same, vulnerable level of depth by another hu-
  man being (the therapist).

In this state of both connection-with-self and connection-
with-other, the client's limbic system and right brain have recov-
ered their native state of integrated well-being. This is the same
state sought by therapists who define therapy in terms of repara-
tive attachment and the use of the client–therapist relationship.
Achieving this state is built into coherence therapy by virtue of
centering the work not on attachment or the dyadic relationship
but on the coherence of the underlying emotional truths generat-
ing the client's symptoms. This state comes about also for clients
for whom no attachment issues arise.

THE VIEW THAT FINDINGS IN NEUROSCIENCE IMPLY A THERAPY OF COGNITIVE REGULATION

Interconnections between brain systems allow for a wide range
of mutual influences between their various functions. For ex-
ample, recruiting the cognitive talents of the cortex to exert a

counteractive, regulating influence on symptoms driven by sub-cortical emotional brain centers is the strategy of cognitive-behavioral therapy (CBT), various forms of which are in widespread, predominant use today. Cognitive regulation is the essence of many counteractive methods designed to stop, fix, prevent, get away from, override, or correct a symptom and replace it with a desired state. Examples include positive thinking, self-soothing, narrative reframing, rational refuting of irrational beliefs, and the standard protocols of anger management. The strategy of cognitive regulation is regarded in some quarters as the primary implication of neuroscience for psychotherapy.

The interest of neuroscientists in cognitive regulation is based on the long hegemony of the view that long-term, emotionally intense implicit memory in the amygdala is indelible, so that conditioned responses of mood and behavior that are rooted in amygdalar memory cannot be eliminated. With the assumption of indelibility regarding the brain's primary emotional center, the only strategy for remediation of amygdala-generated symptoms has seemed to be the enlisting of other brain regions—particularly the cortex—to compete against, moderate, and manage the unwanted responses of amygdalar and other implicit memory systems.

However, as described in our previous article (Ecker & Toomey, 2008), neurodynamic studies since 1997 have shown that amygdalar implicit memory is not indelible after all and *can* be radically depotentiated, that is, erased and eliminated, not just suppressed. This occurs through an experience-driven neurological process termed *reconsolidation*, which unlocks and renders disruptable the synapses of an implicit memory circuit, rather than through pitting a cognitive brain system against that memory circuit.

It is not yet apparent to neuroscientists how the disruption of synapses allowed by the process of reconsolidation can be used therapeutically in humans. Therefore the counteractive, cortical regulation of emotional symptoms arising subcortically continues to be the predominant therapeutic strategy studied by researchers and used by clinicians. For example:

> A characteristic difficulty in many psychological disorders, such as depression or anxiety, is the maladaptive cognitive interpretation of situations or

events. A component of treatment for these disorders is to teach active
coping skills, consciously applied strategies that help assure adaptive in-
terpretations or reactions to emotional stimuli. . . . The habits and skills
we develop to guide these internally generated emotional events are criti-
cal. Recent research on the neural systems of emotion regulation explores
the mechanisms underlying the ability to use cognitive and active coping
strategies to alter emotional reactions. (Phelps & LeDoux, 2005, p. 183)

The strategy of cognitive regulation exists in relation to the
presumed disregulation of the emotional brain systems. However,
coherence psychology emphasizes and coherence therapy demon-
strates that across a wide range of clinical symptoms, subcortical,
emotional brain systems are fully coherent in their internal know-
ings and functioning. Symptoms seem irrational and out of con-
trol only from the point of view of cortical consciousness, which
is not privy to the subcortical knowings that are coherently gener-
ating those symptoms. Symptoms, as a rule, are beyond conscious
control not because subcortical brain systems are malfunctioning,
but only because a lack of neural and psychological integration
keeps the cortex disconnected from the knowings and the agency
actually governing the person's responses, as illustrated in our
case examples in the previous two articles (Ecker & Toomey, 2008;
Toomey & Ecker, 2007).

There is a great deal of empirical, neurodynamic evidence
concerning cognitive regulation. Whereas the long-term memory
of a learned fear is stored in the amygdala itself (Maren, 1999a,
1999b), a subsequent, counteractive learning is stored elsewhere,
in a particular region of the cortex (Milad & Quirk, 2002; Phelps,
Delgado, Nearing and LeDoux 2004; Quirk, Likhtik, Pelletier &
Paré, 2003). This leaves the amygdalar fear memory unchanged
and pits one brain system against another with the hope that the
cortex reliably prevails. This structural opposition is the case in
classical extinction learning as well as for diverse forms of cog-
nitive regulation of emotion, ranging from orthodox behaviorist
therapy to the current "third wave" of cognitive-behavioral thera-
pies, such as acceptance and commitment therapy (ACT; Hayes,
Strosahl, & Wilson, 2003).

In one line of cognitive regulation research, subjects first
viewed a photo or a word that elicited an emotional arousal and
then carried out a cognitive reappraisal of that stimulus, designed
to diminish the arousal. Successful reappraisal is found to result

in decreased subjective ratings of emotional arousal and a measurable decrease in both physiological responses and activation of the amygdala (Gross, 2002; Ochsner, Bunge, Gross & Gabrielli, 2002; Schaefer et al., 2002). Brain imaging in such studies has identified specific cortical regions recruited in the cognitive control of emotion (Oschner & Gross, 2005; Ochsner et al., 2004). The images show an increase in cortical (specifically prefrontal) activation coupled with a decrease in amygdalar activation, both of which are correlated with subjects' reports of reduced emotion. These observations support the cognitive-behavioral tenet that the cognitions attached to emotionally laden stimuli and behaviors can amplify or weaken emotional responses.

Oschner and Gross (2005) defined a spectrum of cognitive regulatory strategies, listed here with benefit and effectiveness increasing from top to bottom:

- attentional control via deliberate suppression,
- attentional control via distracting secondary task,
- attentional control via attending to nonemotional features of stimulus,
- cognitive change via classical extinction learning,
- cognitive change via the placebo response, and
- cognitive change via cognitive reappraisal.

Contrasting the two ends of that spectrum in light of their brain imaging studies, these authors stated that deliberate suppression "of negative emotions might limit expressive action but does not dampen unpleasant experience, worsens memory, and increases sympathetic nervous system activation" (Oschner & Gross, 2005, p. 243). At the more effective end of the spectrum, "using reappraisal to regulate emotions is associated with healthier patterns of affect, social functioning, and well-being than is using suppression" (John & Gross, 2004, p. 1301). The meta-strategy of some therapies of cognitive regulation is to help clients develop a more adaptive repertoire of strategies across that spectrum.

Ochsner and Gross (2005) reported that, with minor variance, the same cortical regions are observed to activate across the spectrum of cognitive control strategies listed above. Phelps and LeDoux (2005) made the same point:

> [The cortical] region that has previously been linked to extinction learning in humans (Phelps et al., 2004) also showed a similar pattern of response when conditioned fear was diminished with a cognitive strategy, suggesting that overlapping neural mechanisms for amygdala inhibition/regulation may support both cognitive emotion-regulation strategies and extinction learning. (p. 183)

For all practical purposes then, and in light of current brain science, therapies of cognitive regulation can be regarded as variations on extinction learning—that is, they all use approximately the same region of the prefrontal cortex to counteract and suppress the responses of subcortical implicit memory.

In summary, it is well established that cognitive counteracting of emotional reactions has some degree of effectiveness and verifiability under the short-term conditions of brain studies.

A somewhat different picture is encountered in the context of psychotherapy, however. Outcome studies have shown that cognitive-behavioral therapies, which follow cognitive regulation strategies, do have efficacy, but they are only as effective as placebo treatments that are structurally equivalent (Baskin, Tierney, Minami & Wampold, 2003). This can be interpreted to mean that the real-life effects of cognitive counteracting are moderate. Indeed, there are several known, built-in weaknesses of the cognitive regulation strategy.

Prone to relapse. In an authoritative review of extinction learning, Bouton (2002, p. 982) commented that because "the original learning is not destroyed by a retroactive interference treatment, and if relapse is therefore always possible, we need to know how to optimize the new learning so as to prevent the phenomena of relapse." Relapse (recurrence of symptoms after counteracting/extinguishing seems effective) is fairly easily produced by (a) an established trigger (conditioned stimulus) occurring in a new context (termed *reinstatement*); (b) an established trigger occurring in an old context covered by counteractive training (termed *spontaneous recovery*); (c) relatively high levels of stress; or (d) passage of time since the counteractive/extinction learning.

Inherent, unending struggle. The main source of unwanted emotional reactions, the amygdala, carries out its own appraisal and reacts independently of and far faster than any conscious, cortical reappraisal or other type of regulation can possibly be

implemented (Whalen et al., 1998). This means that a person us-ing a cognitive regulation strategy is forever on the defensive, and that full suppression of emotional reactions is usually impossible, even when the amygdala's response is conscious. When it is uncon-scious, as all amygdalar *implicit* memory is, cognitive regulatory leverage is particularly weak. By design, cognitive counteracting prepares a person to perpetually oppose symptoms that will con-tinue to tend to arise from an amygdala that still harbors the same reactive, implicit memory.

Inherent ineffectuality. Amygdalar fear reactions observed in brain-imaging studies of cognitive regulation were relatively mild responses to photos or words in a safe, experimental set-ting. Among therapy clients presenting raw, intense anxiety or panic symptoms, cognitive antidotes are generally ineffectual, for these reasons: (a) Cognitive antidotes lack compelling re-alness and fail to alter the emotional root of the reaction; and (b) the stronger the emotional reaction, the weaker and less available is the person's cognitive attention to any cog-nitive antidote. (Behavior therapists have therefore sought a pharmaceutical "cognitive enhancer" to increase the strength of new cognitive learning during exposure therapy for phobias. Tests have been conducted on D-cycloserine, a compound pre-viously found to increase the strength of new synapses formed during extinction learning in rats. In an initial trial [Ressler et al., 2004], administering D-cycloserine to therapy clients just be-fore each session of exposure therapy for fear of heights resulted in two sessions being about as effective as eight sessions without the drug [Travis, 2004]. Long-term effects and relapse rates have not yet been reported.)

In the case example of Carol in the first of the articles in this series (Toomey & Ecker, 2007), a cognitive regulation approach to her distaste and coldness toward marital sex could have taken several different forms. For example, the therapist could have worked along any of these lines:

- Cultivate and make vivid the ways in which Carol "truly wanted to enjoy the sexual dimension of her marriage."
- Teach Carol to notice and interrupt negative self-talk around sex, and replace it with positive thinking and a focus on antici-pating the positive effects of physical intimacy with her husband.

- Guide Carol to identify the situational variables that constitute her key conditions for relaxing comfortably into sexuality, and coach her in setting up those conditions.
- Guide Carol and her husband to engage in sensate focus exercises that would allow Carol to expect a well-defined, positive experience.

Any of these or other methods of counteracting might have partially ameliorated Carol's sexual aversion, at times. If we consider, however, the power and urgency of the implicit, subcortical knowings underlying her aversion—the revulsion toward her mother's voyeuristic, incestuous interest in her own sexual behavior as a young girl, and her expectation that surrendering to sexual feelings would make her perpetrate the same mortifications on her own daughter—it is obvious that the cognitive antidotes listed above could not possibly have enough emotional strength to override that intense emotional schema.

However, if Carol's symptom-requiring emotional schema were dissolved, ending symptom production, then the same methods listed above would no longer be counteractive and could effectively help cultivate new knowings and behaviors for a satisfying sex life. This true dissolution of symptom-producing implicit schemas is the strategy of coherence therapy.

In the recent discovery that the amygdala's emotional memory is not indelible—that the brain can indeed dissolve a specific implicit memory or subcortical emotional appraisal—neuroscience has recognized a way of dispelling ingrained emotional reactions that is a fundamental and clearly superior alternative to cognitive regulation. The viability of the option of implicit memory depotentiation removes the main reason for resorting to cognitive regulation and becomes the psychotherapeutic strategy of choice for these reasons:

- Relapse is impossible and effectiveness is decisive when the implicit memory driving symptom production no longer exists.
- Removing the cause of symptom production means that the symptom ceases to occur, with no need for symptom-counteracting measures, cognitive or behavioral.
- An ongoing conflict of one part of the personality against another is not created and perpetuated.

Our previous article (Ecker & Toomey, 2008) details the theoretical and clinical evidence that coherence therapy's methodology achieves implicit memory depotentiation.

SUMMARY AND CONCLUSION

The staggering complexity of the brain and mind will forever allow for a rich diversity of valid approaches to alleviating suffering and fostering change, growth, and well-being. However, despite the proliferation of psychotherapeutic models and methods, so far the field has been constrained by a curious ceiling on efficacy. We have argued in this and the previous two articles in this series that the marriage of neuroscience and psychotherapy could help identify more reliable, effective methodologies and techniques. The brain sciences can guide psychotherapists to aim their creativity and skills in directions that work most effectively and parsimoniously.

Here we have attempted to help map this new territory. Our findings, based on extensive clinical experience and a review of the research literature, suggest that to counteract a symptom, whether pharmaceutically or cognitively, is to settle for an efficacy no better than is achieved by placebo treatments that have structural equivalence. The reason, we have suggested, is that these counteractive methods—drugs and cognitive regulation—strive to compete against and override the powerful, coherent, adaptive, prosymptom constructs in implicit memory. This is an internally oppressive strategy that is inherently limited in effectiveness and reliability because it does not actually eliminate the roots of symptom production. In contrast, we have described coherence therapy as an internally unifying methodology for fundamentally depotentiating the implicit, symptom-requiring constructs, eliminating the need for struggling against them. We have argued that this transformation represents an optimal therapeutic use of neuroplasticity and an optimal outcome for most clients. Why aim to compete against the cause of symptom production when the cause can usually be eliminated? Shouldn't psychotherapy reduce rather than increase the amount of internal conflict?

We also have argued that a neuroscientifically informed understanding of symptom causation by implicit memory implies that the therapeutic strategy of reparative attachment, with its

primary focus on use of the client–therapist relationship, needs to be selectively applied and is contraindicated in a sizable fraction of cases. Coherence therapy provides a meta-psychology and meta-methodology within which (a) the suitability or unsuitability of reparative attachment work can be determined; (b) reparative attachment work can be carried out, when suitable, in service of depotentiating an attachment schema in implicit memory (prosymptom position); and (c) either the client–therapist relationship or a range of methods other than use of the client–therapist relationship is available for transforming attachment schemas. Furthermore, the achieving of both narrative and neural integration is intrinsic to the methodology.

The arguments and analyses we have offered regarding coherence therapy require verification through controlled studies. The putative advantages of coherence therapy relative to pharmaceuticals and to psychotherapies based on cognitive regulation, such as CBT, could be tested by two types of investigation: (a) controlled comparative study of outcome efficacy with clients randomly assigned to the three modes of treatment and (b) functional imaging studies of regional changes in brain activity before, during, and after treatment.

Brain-imaging studies of the reduction of depression by SSRIs and by CBT show that those two types of treatment have quite different effects on more than a dozen brain regions (Goldapple et al., 2004), yet the two treatments are known to have essentially equal efficacy in producing symptom relief (DeRubeis, Gelfand, Tang & Simons, 1999; Hollon et al., 1992). The post-CBT brain scans show regional effects that are consistent with the psychological model of counteractive change posited by CBT, lending support for that model. A corresponding study carried out for coherence therapy could indicate whether coherence therapy's models of symptom production and symptom cessation are likewise consistent with changes of brain activation resulting from coherence therapy. Distinct differences should be apparent between posttreatment brain scans of CBT and coherence therapy responders. For example, as noted earlier, a hyperactive subgenual cingulate (Brodmann area 25) is a key characteristic of depressed persons (Mayberg et al., 1999). An important component of cortico-limbic pathways, this region has been shown to be a main neural correlate of the feeling of sadness

(Liotti et al., 2000). Curiously, CBT, even when effective in reducing depressive symptoms, does not diminish the activation of this region (Goldapple et al., 2004). We have conjectured an explanation based on coherence psychology: Hyperactivation of area 25 may be caused by chronic activation of specific, unconscious, sadness-inducing personal constructs held in subcortical implicit memory. CBT by design does not access or depotentiate such deeply unconscious material, but we believe coherence therapy will prove to do precisely that, and will therefore yield posttherapy brain scans of responders that show diminished activity in area 25.

In conclusion, we hope to have provided perspectives that are useful to clinicians and researchers seeking to bring psychotherapy to new levels of effectiveness.

Notes

1. Symptoms that have been dispelled by coherence therapy include depression, anxiety, panic, agoraphobia, low self-worth, attachment problems, sequelae of childhood abuse, sexual problems, food/eating/weight problems, rage, attention deficit, grieving, fidgeting, codependency, underachievement, procrastination, and a wide range of interpersonal, couple, and family problems.
2. Monoamines are a class of neurotransmitters that include serotonin, dopamine, and norepinephrine.
3. This efficacy alone does not indicate Lipitor as a treatment of choice. For instance, using a dietary intervention based on whole plant foods, Jenkins et al. (2003) found no difference between the effects of their regimen and a drug of the same class as Lipitor. The dietary intervention was also accompanied by a range of positive effects, in contrast to the array of negative side effects and medical risks attendant to the drug.
4. An example of depression caused by implicit memory was given in the example of Tina in our prior article (Ecker & Toomey, 2008) and is described more fully in Ecker and Hulley (2002a). For numerous examples of anxiety and panic caused by implicit memory, see Ecker (2003) and Ecker and Hulley (2002b).
5. It is when the client–therapist relationship *is* subjectively relevant to an attachment schema that a transference projection tends to arise. Use of transference remains central to therapies of the psychoanalytic school, but not to the more recently developed, experiential, neuroscience-oriented therapies of reparative attachment, as represented by Fosha (2001) and by Lewis, Amini, and Lannon (2000). The latter emphasize the healing effect of the client's experience of the therapist's noninterpretive, accurate empathy, regardless of the presence or absence of a transference projection. Coherence therapy likewise can use, but does not rely on, the arising of client transference. Coherence

therapy also differs significantly from reparative attachment therapies regarding the curative role of the therapist's empathy.
6. A video example of imaginal work on an attachment-defining prosymptom position can be viewed, along with transcript and commentaries, in an online short course available at www.coherencetherapy.org/training/courses.htm. Scroll to Course 700: Obsessive Attachment to Former Lover.

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