

*Ernest L. Rossi, Ph.D.*

# **The Memory Trace Reactivation and Reconstruction Theory of Therapeutic Hypnosis: The Creative Replaying of Gene Expression and Brain Plasticity in Stroke Rehabilitation**



*ERNEST L. ROSSI, Ph.D. is the author of *The Psychobiology of Gene Expression: Neuroscience and Neurogenesis in Hypnosis and the Healing Arts* (W.W. Norton Professional Books, 2002). He received the Lifetime Achievement Award for Outstanding Contributions to the Field of Psychotherapy from the Erickson Foundation in 1980 and The American Association of Psychotherapy in 2003. Rossi teaches his new approaches to therapeutic hypnosis in workshops at ZIST in Penzberg, Germany, lutz.bessel@zist.de*

## **ABSTRACT**

I tell the story of how I used my dreams and early training in therapeutic hypnosis with Milton H. Erickson to facilitate my own creative rehabilitation from a stroke at the age of sixty-nine. I explore how currently emerging neuroscience research on memory trace reactivation and reconstruction may be foreshadowing a new theory of the basic ideodynamics of therapeutic hypnosis on a molecular genomic level. I propose how the creative replay of activity-dependent gene expression, protein synthesis, and brain plasticity in the reconstruction of fear, stress, and traumatic memories and symptoms is the essence of therapeutic hypnosis and psychotherapy. A new generation of clinical researchers will be required to update Milton H. Erickson's view of the "neuro-psycho-physiological process" of therapeutic hypnosis on all levels from the experiences of consciousness and dreaming to the creative replay of the gene expression/protein synthesis cycle and brain plasticity.

## **ZUSAMMENFASSUNG:**

Ich erzähle die Geschichte, wie ich meine Träume und meine frühe Ausbildung in therapeutischer Hypnose durch Milton H. Erickson verwendet habe, um meine eigene kreative Rehabilitation nach einem Gehirnschlag im Alter von 69 Jahren zu fördern. Ich gehe der Frage nach, wie die gegenwärtig erstarkende Forschung auf dem Gebiet der Neurowissenschaft über die Reaktivierung von Gedächtnisspuren in der Rekonstruktion der Vorbote einer neuen Theorie der grundlegenden Ideodynamik der therapeutischen Hypnose auf einem molekularen genomischen Niveau sein könnte. Ich stelle zur Diskussion, wie die kreative Wiederholung des aktivitätsabhängigen Genausdrucks, der Proteinsynthese und der Gehirnplastizität bei der Rekonstruktion von Angst, Stress und traumatischen Erinnerungen und Symptomen das Wesen der therapeutischen Hypnose und

**KEY WORDS:** Creative replay, brain plasticity, dreaming, gene expression/protein synthesis cycle, ideodynamic, memory trace, mind-body healing, neuro-psycho-physiological, stress, stroke rehabilitation, trauma.

Psychotherapie ist. Es bedarf einer neuen Generation klinischer Forscher, um Milton H. Ericksons Sichtweise des „neuro-psycho-physiologischen Prozesses“ der therapeutischen Hypnose auf allen Ebenen auf den neuesten Stand zu bringen, angefangen bei Erfahrungen von Bewusstheit und des Träumens bis hin zur kreativen Wiederholung des Genausdrucks/Proteinsynthesekreislaufs und der Gehirnplastizität.

## SAMMANFATTNING

Här berättar jag om hur jag använde mina drömmar och mina tidiga lärdomar från Milton Erickson i min kreativa rehabilitering efter ett slaganfall vid 69 års ålder. Jag utforskar hur den senaste neuropsykologiska forskningen om reaktiverade minnesspår och återuppbyggnad kan ge oss en ny teori om hypnosens verkningsmekanismer på en molekylär genetisk (genomic) nivå. Jag föreslår att essensen i terapeutisk hypnos och psykoterapi är det kreativa uppreparandet av aktivitetsberoende genetiska uttryck, proteinsyntes, och hjärnans plasticitet när rädsla, stress, traumatiska minnen och symptom reaktiveras.

Det kommer att behövas en helt ny generation av kliniska forskare för att uppdatera Milton Ericksons syn på den ”neuro-psyko-fysiologiska processen” i hypnos, på alla nivåer, från upplevelser av medvetenhet och drömmande, till kreativt återupplevande av genetiska uttryck /proteinsyntetiska cykler och hjärnans plasticitet.

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### Correspondence Address:

Ernest Lawrence Rossi, Ph.D.  
125 Howard Ave  
Los Osos, CA 93402, USA  
Ernest@ErnestRossi.com

## Stroke, Brain Trauma, and Creative Replay in Dreams

I awakened early one morning about 4 am with a strange sluggishness, stiffness and awkwardness of movement. I could not stand steady on my feet. It was a struggle to dress. My right leg was so weak and limp that I could hardly put on my trousers. It wasn't till my wife, Kathryn awakened a few hours later, that I realized I had badly slurred speech. A quick check of my face in the mirror revealed that the right side of my face was pulled down out of its normal symmetry. I now knew I was experiencing a brain attack – a stroke. Kathryn called the doctor and rushed me to the hospital. From that time to the present I have had a series of dreams that are clearly related to my stroke and my recovery that utilized Milton H. Erickson's approach to daily creative rehabilitation.

After a day and night in the hospital I had this nightmarish dream in a restless sleep.

*I am driving on a freeway overpass but there is an accident and pile-up of cars ahead. Some people got out of their cars and went to the railing to get out of the way. But I fear I cannot stop my car because of some weakness in my leg and I may run into them. I greatly fear that a tragic accident is about to take place.*

This dream relates to the real weakness I feel throughout my body – especially on the right side where the stroke damage is most manifest. I tell this dream to my physical therapist explaining how it probably related to my physician reporting my stroke to the Department of Motor Vehicles and having my driver's license canceled. She looked at me softly and sympathetically but quietly questioned, “Have you considered the dream also could be reflecting your cerebral-vascular accident where the freeway was a blood vessel in your brain accidentally piling-up blood corpuscles?” “Well, yes,” I replied with a crooked

little smile doing my best to make my face more symmetrical just to please her, “how come I did not think of that?”

There has been a long history of psychiatric reports about dreams replaying stressful and traumatic experiences as metaphorical dramas and symptoms that Freud and his students described as manifestations of the “repetition compulsion” and “regression in the service of the ego.” Current neuroscience updates the significance of these clinical observations with the *trace reactivation theory of memory consolidation*: after salient, novel, surprising, unusual, unexpected, or stressful and traumatic experiences the neuronal networks in the brain *replay the memorable event during the “offline” periods of rest, recovery, quiet time, sleep, and dreaming*. This reactivation and potentially creative replay of heightened psychophysiological arousal during such later offline periods integrates and reconstructs memory and learning. Hoffman and McNaughton (2002) describe how the *concurrent reactivation or replay between brain cells encoding different parts of memory is essential for linking the correct pieces of memory together into a coherent whole as follows*.

“Neural ensembles in the rat hippocampus & neocortex show *memory trace reactivation during “offline periods” of quiet wakefulness, slow-wave sleep, and in some cases REM sleep [dreaming]*. *Reactivation of recent memory traces is also observed during sleep in motor areas of the zebra finch brain. ...neuro-imaging in humans reveals that brain areas with increased signal during a task have continued or reappearing activity after the task is completed.* (p. 2070, italics added)

The important implication for hypnosis and psychotherapy is that the accessing, reactivating, and replaying of any part of a memory tends to reactivate a possible therapeutic reconstruction of the entire original traumatic experience. Memory is made by implicate (unconscious) replay during offline periods of quiet wakefulness, sleep, and dreaming. I hypothesize that these creative offline periods, which Erickson called “The common everyday trance” in daily life, may in some cases include fantasy and daydreaming, wondering, meditation, and prayer.

## **Emergency Radar and Self-Reflection: Second Week of Rehabilitation**

During the second week of my recovery I had a dream and memories of how Erickson described his lifetime of self-directed rehabilitation from polio that began to guide my own efforts at stroke rehabilitation.

*A small, lonely, emergency radar on an infinite, desolate, and dusty plane seemingly coughs, sputters a bit and then begins sweeping the sky rapidly in a circular pattern – frantically but futilely seeking a response. Gradually, in the far distance, I see a response beginning to develop as an output image on a computer screen. The image is a 3-D wire mesh outline of a human head circulating so one can get a complete 360-degree view of the interior of the head. With growing excitement I realize that it is a MRI image of my own head that may enable me to see the exact area of my brain that was damaged by the stroke. I start to go lucid in the dream: I realize that I am dreaming and if I can slow down the rotating image enough I will be able to see exactly where the damage is to better guide my rehabilitation program. The rotating image does indeed slow down and I am just about to see the damaged area in my left cerebral hemisphere when I realize, “Oh, no, I am waking up!”*

This dream may be a metaphor of how neural networks of the brain and body are continually sending each other molecular messengers (neurotransmitters, hormones, immune system and growth factors etc.) to direct development, adaptation, and repair. The desolate plane of the dream is the damaged area of my brain where molecular signals from dead and still struggling neurons are thrashing about seeking contact and connection to reestablish lines of communication for my recovery. The dream inspires me to redouble my daily rehabilitation efforts in speech, memory, eye-hand coordination, balance and mildly strengthening physical exercises. I begin to go on little unsupervised walks around the block but I am not allowed to try to cross the street by myself.

My many occupational and medical therapists congratulate me on my positive attitude toward rehabilitation. But they really don’t know half the story. My body is still very weak and unco-

ordinated. I have to hold onto the banister with two hands to get up and down stairs haltingly. If I try to walk backwards I fall down. I cannot skip or whistle any more. My swallowing reflex is precarious; I believe that if I am to die it will be because of choking on my food that gets clogged in my cheeks and throat no matter how long I chew. My driver's license has been taken away.

My wife winces with worry as she watches me try to use both my arms to lift and haul my somewhat limp right leg into and out of our van when she drives me every day to my therapists. Paradoxically her worried wince evokes a grim satisfaction in me as I suddenly recall the analogous situation about 30 years ago when I lifted Erickson out of his wheel chair into his family's old station wagon to take him for a drive through the deserts around Phoenix to visit rock shops, which we both enjoyed. On those occasions the situation was reversed since I was the one who winced with worry lest I hurt Erickson as I hauled him about.

Erickson on the other hand, ever the healing mentor, would reassure me with his jaw firmly set in grim satisfaction as he told me yet another story about his efforts at self-rehabilitation from his lifetime of coping with polio. Life, he would explain, actually is a continuous process of rehabilitation. Every day and every moment when you consciously choose to work cheerfully and creatively with your handicap rather than complain – literally gives you a leg up. Getting in and out of an auto is utilized as another opportunity to gain yet another increment of muscle coordination and strength. He called this the essence of his *naturalistic or utilization approach whereby the process of actively coping with one's symptoms and problems* guides and facilitates real physical healing as well as further psychological development (Erickson 1958/1980, 1959/1980). This is the source of my positive attitude toward rehabilitation. This is the source of Erickson's greatest legacy of healing to us all although I could not appreciate its significance 30 years ago.

## Creative Reconstruction via Brain Plasticity: Third Week of Rehabilitation

During my third week of recovery a neurologist filling out a routine medical form asks me if my stroke has resulted in a loss of physical strength so that I can no longer do my job. I grimly grin at him with a plucky Ericksonian attitude and humorously respond, "Well, I'm not exactly an iron foundry worker you know." That night I have the following dream.

*A huge Paul Bunyan type man is using gigantic iron pliers and tongs to manipulate small metal objects. He is going to teach me how to do it skillfully. I am experiencing great awe that he notices me and I feel very grateful about the prospect his help.*

I interpret this dream figure to be analogous to my occupational therapist who is facilitating the recovery of my damaged hand-eye coordination by giving me many tasks involving puzzles, picking up small metal objects with tweezers etc. I tell him this dream and explain my interpretation: Paul Bunyan is a metaphor of an inner implicit healing process operating via activity-dependent gene expression and brain plasticity (the growth and reorganization of synapses and neurons in the brain) that hopefully are now being activated by all this occupational therapy to repair my brain. He has never heard of this new neuroscience concept of activity-dependent gene expression and Erickson's lifetime of creative rehabilitation. During this period I read and re-read Erickson's early paper (1970/1980) about facilitating recovery from trauma by encouraging patients to replay their dreams in a more therapeutic fashion in this way.

*"Dream the same dream with the same meaning, the same emotional significance, but with a different cast of characters. This time maybe it won't be so dark. Maybe you can see a bit more clearly. It won't be pleasant, but maybe it won't hurt so much. So go ahead as soon as you can and have your dream.' Within four minutes the dream developed; 20 minutes later, streaming with perspiration, Edward said, 'It was bad. It was awful bad. But it didn't hurt so much. . . Again he was asked to dream the same dream, but to dream it with less pain, less discomfort, and to*

*dream more clearly – to see the characters more plainly. His fingers tightened on my hand, and the dream developed immediately. The observed behavior was essentially the same. The duration was again about 20 minutes.*” (pp. 62-63)

Such intense states of psychobiological arousal that we now know could evoke gene expression, brain plasticity and recovery from trauma were characteristic of many of Erickson’s (1948/1980) early case histories, which he described as follows.

*“Therapy results from an inner resynthesis of the patient’s behavior achieved by the patient himself... this experience of re-associating and reorganizing his own experiential life that eventuates in a cure, not the manifestation of responsive behavior, which can, at best, satisfy only the observer. . . .Not until sometime later did the therapist [Erickson] learn by what train of thought he had initiated the neuro-psycho-physiological process.”* (pp. 38-39, italics added).

I awaken one morning with grateful tears when I imagine a powerful figure pounding a huge glowing gold ingot on a mighty anvil with flashing lightening leaping about. I feel empowered as I witness his methodical pounding whenever I call him forth in my imagination. On one level it is an awesome experience – a surprising drama that I feel to be deeply healing. On another level, I recognize that this positive emotion is good for me so I try to replay it as long as I can. This inner drama wherein I am both healer and healed reminds me of Erickson’s emphasis on the value of such multi-level states interacting during psychological development and healing.

Neuroscience research documents how the classical process of Pavlovian fear conditioning requires the recall and re-activation of a conditioned memory before it can be extinguished and/or reconstructed at the level of gene expression and protein synthesis as demonstrated experimentally by Nader et al. (2000 a & b). Nader et al. (2000a) summarize their research with these words, *“Our data show that consolidated fear memories, when reactivated, return to a labile state that requires de novo [gene expression] and protein synthesis for reconsolidation.* These findings are not predicted by traditional theories of memory consolidation (p.723, italics added).

Dudai comments on the potentially therapeutic implications of this finding in a paper titled, “The Shaky Trace,” (2000).

“The current textbook version, in a nutshell, goes like this. Training modifies proteins at synapses in the neuronal circuit that acquires the new memory. This alters synaptic efficacy and thus the encoding of information in that circuit. *But protein molecules survive only for periods of minutes to weeks, whereas many memories are destined to live longer. It seems that at least part of the immunity of memory to this molecular turnover is achieved by training-induced modulation of gene expression in the modified neurons. The new gene products promote long-lasting remodeling of the activated synapses, in a process that involves cross talk between the synapses and neuronal cell bodies. It takes a few hours for the new pattern of gene expression and the synaptic change to be consolidated.* During this time, the process can be halted by inhibitors of protein synthesis.... So it seems that *fear-associated memories become temporarily labile on retrieval.* Why should the brain invest so much energy in the original consolidation and then risk losing the trace by interference each time it is used? One can come up with teleological explanations – for example, that the brain prefers plasticity at the expense of stability – or mechanistic ones, suggesting in-built constraints on the *synaptic machinery*... More generally, might these results apply to different types of memory?... *Consider, for example, the prospect of intentionally recalling the memory of a traumatic experience and then selectively erasing it. What such a possibility would mean for psychoanalysts on the one hand, and poets on the other, is quite a different matter.*” (p. 686, italics added).

These experimental findings have important implications for understanding the long and controversial tradition of using hypnosis to facilitate memory recall and emotional re-experiencing in the so-called “cathartic cure.” The implication is that *“intentionally recalling the memory of a traumatic experience and then selectively erasing it”* is precisely what took place during many historical approaches to reactivating traumatic memory recall and their “Mental Liquidation” (Pierre Janet’s original words, 1925/1976, pp. 589) via therapeutic hypnosis. Extending the implica-



tions of Dudai's remarks, *I hypothesize that this activity-dependent process of reactivating a fear memory in order to extinguish and re-construct it on the level of gene expression, protein synthesis, and brain plasticity (including synaptogenesis and neurogenesis) is the psychobiological essence of creative replay in the practice of therapeutic hypnosis and psychotherapy.* We can generalize this mind-body essence of therapeutic suggestion to the many alternative and complementary approaches to medicine as well as the creative process in cultural rituals (Greenfield, 1994, 2000) and the humanistic arts in general. They all typically engage the ideodynamic recall and replay of memory in the therapeutic reconstruction of human learning and behavior (Rossi, 2002a, 2004). This appears to be the neurobiological substance of the popular psychotherapeutic metaphor, "Every Replay is a Reframe."

The concept of positive, creative, therapeutic replay during offline psychological states as potentially important periods for the transformation of psychological experience and mind-body healing finds further support in the research of Lisman & Morris (2001).

"...newly acquired sensory information is funneled through the cortex to the hippocampus. Surprisingly, only the hippocampus actually learns at this time — it is said to be online. *Later, when the hippocampus is offline (probably during sleep), it replays stored information, transmitting it to the cortex. The cortex is considered to be a slow learner, capable of lasting memory storage only as a result of this repeated replaying of information by the hippocampus.* In some views, the hippocampus is only a temporary memory store — once memory traces become stabilized in the cortex, memories can be accessed even if the hippocampus is removed. *There is now direct evidence that some form of hippocampal replay occurs... These results support the idea that the hippocampus is the fast online learner that "teaches" the slower cortex offline.*" (p. 248-249, italics added)

Research by Shimizu et al., (2000) provides more detail about how repetition, recall, and creative replay required for the transformations of consciousness, memory, and behavior on the cellular level. They found that the NMDA (N-

Methyl-D-Aspartate) receptor on brain cells in the CA1 region of the hippocampus serves as a "gating switch" in the construction and reconstruction of memory.

"Our results indicate that memory consolidation may require multiple rounds of site-specific synaptic modifications, possibly to reinforce plastic changes initiated during learning, thereby making memory traces stronger and more stable. Recent studies report that the learning-induced correlation states among CA1 neurons are reactivated spontaneously in a post-learning period. Such a co-activation of these neurons might suggest the existence of the natural condition within the hippocampus by which recurrent synaptic strengthening can occur during memory consolidation. We hypothesize that such synaptic re-entry reinforcement (SRR) process can also be applied to explain how the hippocampus transfers newly created memories to the cortex for permanent storage. As the hippocampus undergoes reactivation during consolidation, it may also act as a coincidence regenerator for activating neurons in the cortical area such as the association cortex. This would allow cortical neurons previously corresponding to the different sensory modalities to be reactivated together, leading to the strengthening of the connections between them through SRR. Indeed, such a coordinated reactivation of hippocampal-cortical neurons after learning has been observed recently... Once these cortical connections are fully consolidated and stabilized, the hippocampus itself becomes dispensable for the retrieval of the 'old memory'.. Therefore, we postulate that the hippocampus, by serving as a coincidence regenerator, may induce the reinforcement of synaptic connection within the cortex during memory consolidation as the cellular means to convert short-term memories into long-term memories." (Pp. 1172-1173, italics added)

A recent review of these psychobiological dynamics of the "*multiple rounds of site-specific synaptic modifications, possibly to reinforce plastic changes initiated during learning*" described above by Shimizu et al. (2000) is illustrated by Cohen-Cory (2002) who also provides more detail about the time parameters of activity-dependent synaptogenesis and brain plasticity in the central nervous system as well as the body.

“During development, more synapses are established than ultimately will be retained. Therefore, the elimination of excess synaptic inputs is a critical step in synaptic circuit maturation. Synapse elimination is a competitive process that involves interactions between pre- and postsynaptic partners. The dynamics of synapse formation and of synapse elimination may be much more rapid in the CNS than at the NMJ [Neuro Muscular Junction], where synapse elimination has been well characterized. At the vertebrate NMJ, a single muscle cell is initially innervated by multiple motor axons. *The transition from multiple innervations to innervation by a single motor axon occurs gradually as some terminal branches retract from each muscle fiber before others, a process requiring about 24 hours for withdrawal of the presynaptic terminal...* In the CNS, as with the NMJ, a developmental, activity-dependent remodeling of synaptic circuits takes place by a process that may involve the selective stabilization of coactive inputs and the elimination of inputs with uncorrelated activity. *The anatomical refinement of synaptic circuits occurs at the level of individual axons and dendrites by a dynamic process that involves rapid elimination of synapses. As axons branch and remodel, synapses form and dismantle with synapse elimination occurring rapidly, in less than two hours... hippocampal neurons in which glutamate receptor function was altered demonstrated that synapse disassembly in the CNS occurs rapidly, within 1.5 hours after synapses are no longer functional* (p. 771)... *Studies investigating the effects of long-term synaptic plasticity have generally used experimental paradigms in which repetitive, high-frequency stimulation gives rise to synaptic potentiation [called long-term potentiation, LTP] that is accompanied by structural and molecular changes at the level of single synapses...* Recent imaging experiments reveal that both NMDA and AMPA receptor activation are indeed involved in synapse formation and maturation.” (p. 773, italics added).

Notice how the time frame of 1.5 to 2 hours required for brain plasticity via synaptogenesis, as reviewed above by Cohen-Cory (2002), appears to be identical to Kleitman’s 1.5 to 2 hour Basic Rest-Activity Cycle (BRAC), which is the fundamental ultradian time parameter of the REM

dream cycle where it was originally discovered (Rossi, 2002a, 2004). *The time frames of the basic chronobiological life processes of homeostasis, adaptation, stress and trauma, memory, learning, and neurogenesis as well as the dynamics of neuroendocrinology and psychoimmunology are similar! The time parameters of this broad psychobiological perspective suggests that Milton Erickson’s typical 90-120 sessions of therapeutic hypnosis may have been efficacious, at least in part, because of their association and utilization with the natural chronobiology of Kleitman’s BRAC.* From this neuroscience perspective, brain plasticity in general and synaptogenesis, in particular, is the most recent addition to the list of complex adaptive systems of the BRAC that is evident on all levels from the molecular-genomic to the cognitive-behavioral that we facilitate via the psychosocial genomics of activity-dependent therapeutic hypnosis and psychotherapy (Rossi, 2002a, 2004).

One of the most interesting lines of research on the natural dynamics of replay during REM dreaming indicates a possible psychosocial genomic mechanism for mind-body healing by replaying traumatic memories with a more creative and therapeutic script as illustrated above by Erickson and in my own dream experience as follows.

### **A Dream of Numinous Beauty and Clarity: Fourth Week of Rehabilitation**

*I enjoy the numinous beauty and wonderment of looking through a new clear crystal cover on our swim spa seeing the delightful light blue, clean water in the sparkling sunlight.*

This dream was a profoundly moving and deeply surprising experience for me. Of course, we actually don’t have such a new crystal cover over our swim spa – it is a surprising, creative fiction of the dream that appears to be a metaphor for some sunlight clarity coming into the waters of my brain. Light is a frequent symbol of healing in the spiritual literature. A battery of psychological tests administered to me at this time tells the story of my mental status in a stark manner that clearly outlines my assets and deficits. The good news is that my *abstract reasoning* is at the 99th percentile level and my capacity for *mental*

organization is at the 97th percentile. The bad news is that I am way below normal in *perception and discrimination* at the 45th percentile level and, even worst, is my *short-term memory*, which is down to the 37th percentile.

Experiences of shock, surprise and the active, creative replay of problems and traumatic memories in dramatic psychodramas was a typical approach practiced frequently by Milton H. Erickson (Rossi, 1973). The role of surprise and expectancy in memory and learning has been noted by Waelti and Dickinsen (2001) as follows.

“Classical theories assume that predictive learning occurs whenever a stimulus is paired with a reward or punishment. However, more recent analyses of associative learning argue that simple temporal contiguity between a stimulus and a reinforcer is not sufficient for learning and that a discrepancy between the reinforcer that is predicted by a stimulus and the actual reinforcer is also required. This discrepancy can be characterized as a “prediction error.” *Presentations of surprising or unexpected reinforcers generate positive prediction errors, and thereby support learning . . . Expected reinforcers do not generate prediction errors and therefore fail to support further learning even when the stimulus is consistently paired with the reinforcer.*” (p. 43.)

Such research is a challenge and direct contradiction of the cognitive-behavioral view that that hypnosis is nothing but “expectancy” (Rossi, 2002a&b). The important experience of surprise and fascination in the psychophysiology of therapeutic hypnosis was long ago noted by Braid (1855/1970), however, in his classic text, “The Physiology of Fascination.”

“With the view of simplifying the study of reciprocal actions and reactions of mind and matter upon each other... the [hypnotic] condition arose from influences existing within the patient’s own body, viz., the influence of concentrated attention, or dominant ideas, in modifying physical action, and these dynamic changes re-acting on the mind of the subject. I adopted the term “hypnotism” or nervous sleep for this process . . . And finally as a generic term, comprising the whole of these phenomena which result from the reciprocal actions of mind and matter upon each other, I think no term more appropriate than ‘psychophysiology.’” (pp. 369-372)

Further, it has been found that when experimental animals have surprising and arousing experiences of novelty, environmental enrichment and physical exercise (Rossi, 2002a, 2004), the *zif-268 gene* is expressed during their REM sleep (Ribeiro, 2003; Ribeiro et al., 1999, 2002, 2003). *Zif-268* is an *immediate-early gene* and *behavioral-state related gene* that is associated with the generation of proteins and growth factors that facilitate brain plasticity. Ribeiro et al (2004) recently summarized the complementary role of “*neural replay*” during the stage of REM dreaming versus deep slow wave sleep in the consolidation of new memories as follows.

“The discovery of experience-dependent brain reactivation during both slow-wave (SW) and rapid eye-movement (REM) sleep led to the notion that the consolidation of recently *acquired memory traces requires neural replay during sleep*... Based on our current and previous results, we propose that the 2 major periods of sleep play distinct and complementary roles in memory consolidation: pretranscriptional recall during SW sleep and transcriptional storage during REM sleep... In conclusion, *sustained neuronal reverberation during SW sleep, immediately followed by plasticity-related gene expression during REM sleep, may be sufficient to explain the beneficial role of sleep on the consolidation of new memories.*” (p. 126–135, italics added.)

Two recent papers provide new details of how the reactivation of fear, stress, and traumatically encoded memories in a therapeutic context can be the first step in initiating a molecular-genomic of reconstructing them at the levels of gene expression, brain plasticity, and behavior. Lee et al. (2004) summarize their research as follows.

“The idea that new memories undergo a time-dependent consolidation process after acquisition has received considerable experimental support. More controversial has been the demonstration that established memories, once recalled, become labile and sensitive to disruption, requiring “reconsolidation” to become permanent... *We show that consolidation and reconsolidation are doubly dissociable component processes of memory. Consolidation involves [gene transcription and expression of] brain-derived neurotrophic factor (BDNF) but not the [gene] transcription factor Zif-268,*

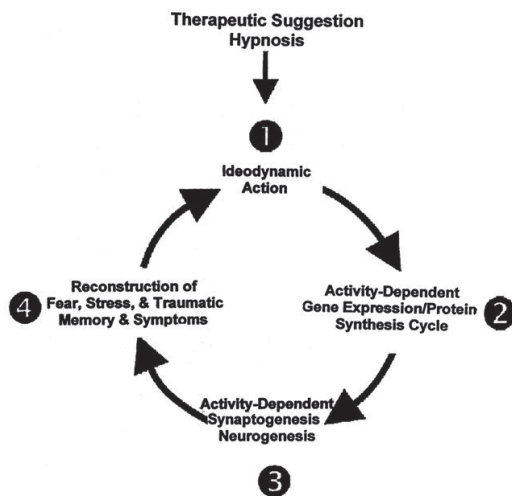


whereas reconsolidation recruits Zif268 but not BDNF. These findings confirm a requirement for BDNF specifically in memory consolidation and also resolve the role of Zif-268 in brain plasticity, learning, and memory. (pp. 839, italics added).

A related paper by Frankland et al. (2004) extends these findings by imaging activity-dependent gene expression in the anterior cingulate cortex during the activation of fear memories. These findings have particular significance for therapeutic hypnosis because the anterior cingulate cortex has been implicated in hypnotic susceptibility (Rainville et al. 1997, 1999).

“Although the molecular, cellular, and systems mechanisms required for initial memory processing have been intensively investigated, those underlying permanent memory storage remain elusive. We present neuroanatomical, pharmacological, and genetic results demonstrating that the anterior cingulate cortex plays a critical role in remote memory for contextual fear conditioning. Imaging of activity-dependent genes shows that the anterior cingulate is activated by remote memory and that this activation is impaired by a null -CaMKII mutation that blocks remote memory. Accordingly, reversible inactivation of this structure in normal mice disrupts remote memory without affecting recent memory.” (pp. 881, italics added).

From the neuroscience perspective, the (1) apparently simple process of recalling and (2) creatively replaying fear, stress, and traumatic memories within new, positive, and surprising therapeutic perspectives can (3) initiate the molecular-genomic dynamics of Erickson’s neuro-psycho-physiological process of healing. I propose that the psychosocial genomics of gene expression and brain plasticity is operative within the natural 4-stage ultradian creative process of deconstructing the old neural networks that a encoded posttraumatic stress disorder (PTSD) and re-synthesizing new neural networks capable of effective problem solving and symptom resolution (Rossi, 2002a, 2004). An outline of this 4-stage psychosocial genomics core of brain plasticity in the reconstruction of fear, stress, and traumatic memories and symptoms via the “ideodynamic action hypothesis” of therapeutic hypnosis (Weitzenhoffer, 2000) is illustrated in figure one.



**Figure 1.** The proposed 4-stage psychosocial genomics core of the ideodynamic action hypothesis of therapeutic hypnosis. Note how this creative cycle engages feedback loops whereby ideodynamic action generates “far more complex responses than the one originally called for” as described by Weitzenhoffer (2000,

We may regard figure one as an update of Weitzenhoffer’s (2000) description of the ideodynamic action hypothesis of hypnosis as follows.

“Few formulations regarding what the suggestion process is, exist that can be called a theory. The most widely accepted and influential so-called theory, still really a hypothesis, is known as the *ideodynamic action theory*, often being improperly referred to as the “ideomotor theory” and as a theory of hypnosis. Strictly speaking, it pertains directly only to suggested behavior. It has nothing to do with hypnosis, but of course, indirectly it does. Of all the hypotheses that have been proposed regarding the production of hypnotic effects (understood as suggested effects), it is the one that comes closest to being a theory and more workers in the field have ascribed to it than any other hypothesis (p. 123)... If the responses involved here [e.g. automatism, postural sway suggestion, Chevreul explorer pendulum etc.] are to be viewed as being ideodynamic in their entirety, they call for a more complex picture of the neuromotoric mechanisms in-

volved (130)... Furthermore, *many automatisms are self-correcting processes in which feedback plays an important part*. This last is important because this, combined with the capacity for self-termination, probably makes some complex automatisms appear like intentional behavior (p. 128)... *Last, far more complex responses than the one originally suggested might be expected to take place through ideodynamic action because one idea often leads to another idea, indeed to a whole chain of ideas, with loops and side branches. Not only is it reasonable to posit that a number of associated and interlinked ideodynamic responses can thus take place, but also that the more complex composite "idea" thus formed may have the capacity to produce its own specific ideodynamic effect*. Indeed, the overall response to a suggestion has the potential for being something quite different than what was originally proposed" (p. 129, italics added).

What, precisely, is the *action* of the ideodynamic *action* hypothesis of suggestion that Weitzenhoffer describes here? While the *Ideodynamic action hypothesis* of suggestion has played an important role toward the development of a theory of hypnosis for over 100 years, current neuroscience does not recognize this historical priority. Neuroscientists are now rediscovering many phenomena previously subsumed under the *ideodynamic action hypothesis* and, without any regard for the history of hypnosis, give these phenomena different names derived from recent experimental research on *activity-dependent gene expression, behavior state-related gene expression, and brain plasticity* in the molecular dynamics of memory, learning, behavioral adaptation, sensation, perception, emotions, dreaming, stress, trauma, and healing. There has been little or no communication between the three disciplines of therapeutic hypnosis, neuroscience, and psychosocial genomics. This paper is an effort to integrate the current neuroscience trace reactivation theory of memory and learning with the proposed molecular-genomic mechanisms of the ideodynamic action hypothesis of therapeutic suggestion, hypnosis, and psychotherapy (Rossi, 2002 a, 2004).

My dream series replaying dramas of stroke rehabilitation utilizing Erickson's creative approach continues even today almost three years

later in ever more surprising and delightful ways. In the early days of our relationship when I would drive him about through the deserts to the rock shops we liked to visit, I always dreamt that Milton was driving and I was the passenger. It gave me great pleasure at the time when I told Erickson of this peculiar reversal in my dreams, which certainly reflected the deeper truth that Erickson was in fact the driver in our relationship. With a twinkle in his eyes Erickson quietly murmured, "That may change in time." Indeed, it has. In my most recent dream I am the driver, for the first time, as Milton smiles at me from the passenger seat.

My wife tells me that in fact she has noticed over the past few weeks that I really am driving well after passing my driver's test for a new license. She can no longer see any lingering after effects of the stroke. A recent retest of my psychological functioning documented that after 15 months of rehabilitation both of my major stroke induced cognitive deficits improved dramatically: my *perception and discrimination* improved to the 90th percentile from the 45th; my *short-term memory* improved to the 66th percentile from the 37th.

## SUMMARY

The memory trace reactivation and reconstruction theory of therapeutic hypnosis proposes that the creative replaying of the gene expression/protein synthesis cycle, which generates brain plasticity and mind-body healing, is the "neuro-psycho-physiological" basis of Ericksonian psychotherapy. A series of dreams is used to illustrate the psychosocial genomics of facilitating stroke rehabilitation via occupational therapy and active imagination. The unfortunate reality at the present time, however, is there is as yet no comprehensive program of experimental research on the psychosocial genomics of therapeutic hypnosis. The hypnosis community urgently needs to network with those researchers on the molecular-genomic level who are interested in understanding the significance of their work for the profound issues of how the mind can heal the brain – how psychological experiences can facilitate genuine mind-body healing and rehabilitation.

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