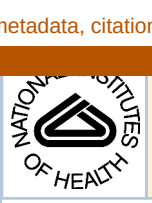

Published in final edited form as:
Health Soc Work. 2009 August ; 34(3): 191–199.

Neuroplasticity, Psychosocial Genomics, and the Biopsychosocial Paradigm in the 21st Century

Eric Garland, Ph.D., LCSW
College of Social Work Florida State University Tallahassee, FL 32308

Matthew Owen Howard, Ph.D. [Frank Daniels Distinguished Professor of Social Policy]
School of Social Work University of North Carolina - Chapel Hill

Abstract

The biopsychosocial perspective is a foundation of social work theory and practice. Recent research on neuroplasticity and psychosocial genomics lends compelling support to this perspective by elucidating mechanisms through which psychosocial forces shape neurobiology. Investigations of neuroplasticity demonstrate that the adult brain can continue to form novel neural connections and grow new neurons in response to learning or training even into old age. These findings are complemented by the contributions of psychosocial genomics, a field of scientific inquiry that explores the modulating effects of experience on gene expression. Findings from these new sciences provide external validation for the biopsychosocial perspective and offer important insights into the manifold means by which socioenvironmental experiences influence neurobiological structure and function across the life course.

Keywords

biopsychosocial; neuroplasticity; psychosocial genomics; gene-environment interaction

Introduction

Social work professionals in the 21st Century have adopted the biopsychosocial paradigm. This paradigm, first articulated by the physician, George Engel, holds that humans are dynamic systems whose functioning depends on the holistic integration of biological, psychological, and social factors (Engel, 1977); indeed, according to the biopsychosocial model, these factors are fundamentally interrelated and interdependent. Although Engel rejected the reductionism of the dominant biomedical model of his era, which assumed that molecular biological processes (e.g., genes and biochemistry) immutably dictated physiology and behavior, a simple-minded biological determinism nonetheless took root and became widely, if uncritically, accepted. At its inception, there was scant evidence to support Engel's biopsychosocial perspective; however, scientific discoveries of the past decade have provided important new findings validating and elaborating the biopsychosocial paradigm.

Over the past decade, two fields of empirical investigation, neuroplasticity and psychosocial genomics, have offered important findings that may lead to a paradigm shift in our conceptions of psyche and soma and the modes of their interrelationships. These two fields mutually inform one another, depicting interpenetrating biopsychosocial relationships on different scales:

Please send correspondence to: Eric Garland, PhD, LCSW, College of Social Work, Florida State University, University Center, Building C, Tallahassee, Fl 32306, egarland@fsu.edu.

neuroplasticity research describes how neurons within the brain proliferate and grow new connections across the lifespan, whereas psychosocial genomics describes the processes by which psychological and social experiences activate or deactivate genes, thereby driving the development of new neural pathways. The interplay of these sciences reflects a vision of humans as inherently resilient; psychosocial factors appear to stimulate gene expression within neurons resulting in alterations to the structure and function of the brain. Discoveries from both fields reveal that experience and learning can contribute to positive change, even at the neurobiological and structural levels.

Social work academicians have embraced the biopsychosocial perspective; yet, many are perhaps not fully aware of recent developments in genomic and neurobiological research with implications for social work and the biopsychosocial perspective. This research provides insights into the very substrates of biopsychosocial change. Thus, we review recent neuroplasticity and psychosocial genomics research and its implications for current understanding and application of the biopsychosocial perspective.

Neuroplasticity

Basic neurotransmission

The human brain is a complex, self-organizing, biological system, consisting of trillions of interconnected nerve cells called neurons. The operation of neurons results in two distinct forms of information processing: signaling and integration. Each neuron propagates signals via action potentials, electrochemical currents that travel the length of its axon. This current leads to the release of neurotransmitters which traverse synapses, the gaps between neurons. These chemical messages are received via specialized receptor cells at the ends of numerous, tree-like branches of the receiving neuron, called dendrites. The stimulation of dendritic receptors by neurotransmitters leads to integration, whereby large amounts of information from many neurons is summed up before reaching a threshold to fire the action potential down the next axon. In this manner, perceptual information from the external environment and the internal milieu of the body is transmitted and processed in the brain, leading to cognition, emotion, and behavior, the essence of human experience.

Origins of neuroplasticity research

The brains of infants and children are known to be plastic, undergoing spurts of neuronal development in response to stimulus exposure during critical periods (Mundkur, 2005). This development consists of the genesis of neurons, increased connectivity between extant neurons, and the routing of new synaptic connections between previously unrelated neurons. However, before 1998, it was widely accepted that neuronal connections in the adult brain were immutable; the neurons that populated a given brain area were thought to be fixed in accordance with whatever form and function the genetic code prescribed for that region (Begley, 2006). In addition, the conventional wisdom at the time--that no new neurons could be generated after injury or insult to the brain—was held with conviction on the part of leading neuroscientists.

However, upon discovery of the growth of new neural tissue, or neurogenesis, in the adult human hippocampus, a brain region responsible for memory (Eriksson et al., 1998), the dogma of the “hardwired brain” was formally repudiated. This finding complemented earlier evidence from primate studies demonstrating that novel sensory experience and learning new behaviors triggers neuronal growth in the somatosensory and motor cortices, areas of the brain subserving tactile perception and limb movement (Jenkins, Merzenich, Ochs, Allard, & Guic-Robles, 1990; Nudo, Milliken, Jenkins, & Merzenich, 1996). Subsequent to the discovery of neurogenesis in the adult human brain, neuroscience has pursued this line of investigation with vigor, aided by advances in brain imaging techniques such as magnetic-resonance imaging.

Neuroplasticity research findings

The growth of neurons has been documented in the brains of adults exposed to a variety of experiences. For instance, violinists evidence neural growth in the portion of their somatosensory cortex devoted to their fingering hand through hours of musical practice (Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995), as do persons engaged in the practice of juggling (Draganski et al., 2004). In addition to such physical training, mental practice may promote neuroplasticity: neurogenesis can occur in the motor cortex just by imagining playing the piano (Pascual-Leone, Amedi, Fregni, & Merabet, 2005). Similarly, taxicab drivers develop the areas of their brains involved in spatial relationships by memorizing the labyrinthine streets and avenues of the cities in which they work (Maguire et al., 2000). While the underlying mechanisms are different, neuroplasticity research suggests that challenging learning experiences can lead to the development of brain tissue analogous to the way physical exercise can lead to the development of muscle tissue.

One area of research that has found significant evidence of mental training leading to neuroplastic modifications in brain activity focuses on the study of meditation. Meditation, while greatly varying in technique and purpose across the diverse spiritual and cultural traditions where it is employed, may be generally defined as the intentional practice whereby one grasps “the handle of cognition” to cultivate a competent use of his or her own mental capacities, gaining agency over thought and emotion (Depraz, Varela, & Vermersch, 2003). Such intentional mental training has been shown to induce functional neurobiological changes.

A study by Lutz and colleagues found marked alterations in the synchronization of neurons as an effect of long-term training in Buddhist loving-kindness meditation, a practice which is thought by some practitioners to promote a state of unconditional compassion and benevolence (Lutz, Greischar, Rawlings, Ricard, & Davidson, 2004). Neural synchrony of the type observed in this study may be indicative of coherent and integrated psychological functioning (Williams et al., 2005). The synchronization of brain activity found in some of the practitioners sampled, whose experience ranged between 10,000 and 50,000 hours spent in meditation, was higher than any previously reported in the literature. Such increased neural synchrony was observed not only during the meditative state, but also when the practitioners were not meditating, suggesting that long-term mental practice can induce lasting, trait-level changes possibly mediated by structural modifications to the brain (Begley, 2006).

Other research has documented changes in neurobiological function as a result of mindfulness meditation, the practice of cultivating a present-centered, metacognitive awareness, “a naturalistic state wherein consciousness transcends its content to rest upon the dynamics of its own processes” (Garland, 2007). A recent study by Slagter et al. (2007) compared attentional performance of a group of experienced meditators participating in a 3-month mindfulness meditation retreat to that of a novice control group who received a 1-hour meditation class and were asked to meditate 20 minutes daily for one week. Relative to controls, experienced meditators evidenced significant improvements in attentional performance that correlated with alterations in brain activity. This cognitive enhancement was maintained 3 months after formal meditation practice, providing suggestive evidence that mental training can stimulate neuroplastic changes in the adult human brain (Slagter et al., 2007).

While the work of Slagter et al. and Lutz et al. provide tentative support for meditation-induced neuroplasticity, neither study examined structural brain changes per se. However, two structural MRI investigations comparing the brains of experienced meditators to control subjects matched in sex, age, race, and years of education found that years of meditation experience correlated with increased cortical thickness in brain areas where visceral attention (e.g. right anterior insula) and self-awareness (e.g. left superior temporal gyrus) have been

localized (Holzel et al., 2008; Lazar et al., 2005). These empirical investigations of meditation suggest that mental training may stimulate structural alterations reflective of neuroplasticity.

Clinical implications of neuroplasticity research

The finding that experience and training can lead to the development of new neural connections has key implications. For example, persons suffering from what was once thought to be permanent brain injury can heal through rehabilitation designed to stimulate the damaged area, such as in the case of stroke (Taub et al., 2006). However, although largely speculative, it is possible that neuroplasticity may undergird not only rehabilitation of physical illness but that of select psychological disorders as well, mediating natural recovery from mental illness in some cases as well as improvements related to psychosocial interventions. At present, it has been demonstrated that psychotherapy can induce functional changes in brain activation. For example, a brain imaging study found that persons with obsessive-compulsive disorder who were treated with a mindfulness-oriented form of cognitive-behavioral therapy (CBT) exhibited functional changes in the orbital frontal cortex and striatum, two brain structures found to be overactive in OCD (Schwartz & Begley, 2002). Other studies have demonstrated psychotherapy-related alterations in brain circuits involved in depression (e.g. Goldapple et al., 2004; Martin, Martin, Rai, Richardson, & Royall, 2001). CBT has also been associated with changes in frontal and temporal brain regions of persons suffering from panic disorder (Prasko et al., 2004). Such intervention-related changes in both psychosocial function and neural activity may correlate with neuroplastic alterations to the brain; critically, a combined functional and structural magnetic resonance imaging study of practice-induced increases in gray matter found that increased task-specific brain activation led to the remodeling of one of the same neural structures (i.e. dorsolateral occipital cortex) that was activated by the practice and learning of the task (Ilg et al., 2008).

Neuroplasticity research of psychosocial interventions has just begun. A recent longitudinal study of cognitive-behavioral therapy for women with chronic fatigue syndrome found increases in gray matter of the lateral prefrontal cortex after 16 sessions of CBT (de Lange et al., 2008). Increases in gray matter volume correlated with enhanced cognitive processing speed, suggesting that the neuroplasticity evoked by psychotherapy played a causal role in rehabilitation of cognitive performance after cerebral atrophy resulting from chronic fatigue.

Indeed, neuroplasticity may be the biological mechanism through which psychosocial interventions exert at least some of their therapeutic effects. During psychotherapy, when the client recalls negative or painful life experiences, the clinician may assist in reframing the context so that the experience gains new meaning (de Shazer, 1988). For instance, in treating persons who have experienced traumas such as rape, therapy may help clients to envision themselves as a survivor rather than as a victim. Such reframing or reappraisal may be a critical component of successful biopsychosocial outcomes (Folkman, 1997; Penley, Tomaka, & Wiebe, 2002). Some theorists hypothesize that the process of recalling, reconstructing, and reframing memories of past trauma during psychotherapy is mediated by the reorganization and genesis of neurons (Centonze, Siracusano, Calabresi, & Bernardi, 2005; E.L. Rossi, 2005). This hypothesis is founded on evidence that the formation of new long-term memories results from neuroplastic changes in the brain structure known as the hippocampus. Hippocampal changes appear within hours of significant learning experiences (McGaugh, 2000), such as those that can occur during psychotherapy.

Neuroplasticity is mediated at the cellular level through activity-dependent gene expression, the mechanism by which neurons secrete growth factors leading to the “activation of gene transcription in the nucleus that support[s] synaptic connections…Thus, with every new experience, the brain slightly rewires its physical structure and this rewiring is mediated

through the signaling cascade" (Mundkur, 2005). Hence, in order to understand neuroplasticity, we must consider the domain of psychosocial genomics.

Psychosocial Genomics

Basic epigenetics

In the 21st century, there is broad agreement that the genome is the basis of human life and a precondition for psychosocial experience. Nevertheless, the question of the respective roles of nature and nurture in human experience and the manner of their interaction in select contexts remains contentious, despite the more than half-century that has transpired since Watson and Crick (1953) identified DNA as the building block of biological processes.

The DNA code of the human genome does not determine protein synthesis in a one-toone fashion; instead, genes are subject to epigenetic processes (i.e. modifications that do not occur due to changes in the basic genetic sequence of amino acids but that instead result from biological and environmental influences on the expression of genes as proteins) (Eisenberg, 2004). During gene expression, the genetic code serves as a "blueprint" that guides the construction of proteins from amino acids. However, this construction process is modulated by signals from the internal and external environments, which steer and modify the manner in which basic organic molecules are organized into anatomy and physiology. Although genes prescribe protein synthesis, there is substantial variability in the manner in which they are expressed.

A single genotype, the genetic blueprint of an organism, can be expressed in a multiplicity of distinct physiological and behavioral forms, known as phenotypes. This is evident in Eisenberg's (2004) example of phenylketonuria, a disorder that when untreated may lead to severe mental retardation, psychosis, and seizures. If children with this genetic abnormality are kept on a postnatal diet low in the amino acid phenyalanine, they do not develop these disorders. Hence, although the genotype for phenylketonuria does not change, its phenotypic expression is modified by the environment (i.e., nutrition) to which the individual has been exposed. The mechanisms by which such different phenotypes are expressed are just beginning to be understood, but appear to involve the regulatory effect of internal and external environmental signals on stress hormones, which in turn modify gene transcription processes (Kandel, 1998; E. L. Rossi, 2004).

Learning and other psychosocial experiences may modulate gene expression

In addition to physical environmental forces, learning experiences in the social environment can alter gene expression (McCutcheon, 2006). The bi-directional relationship of nature and nurture, genes and environment, was first demonstrated in a series of path-breaking studies of maternal care in rats (Francis, Champagne, Liu, & Meaney, 1999; Liu et al., 1997). In these studies, an inverse relationship was found between the number of stress hormone receptors in a rat's hippocampus and its tendency to exhibit stress reactions. The number of these receptors is dictated by the genotype of the rat. Highly stress-reactive rats give low levels of maternal care to their offspring, who, in turn, exhibit high stress reactivity and later provide low levels of maternal care to their offspring. However, these studies revealed that hormonal and behavioral stress reactions of rat pups as well as the number of their stress hormone receptors are modulated by the licking, grooming, and nursing behaviors of their mothers. Even if a rat were born with a genotype coding for fewer stress hormone receptors, if it was reared by an adoptive mother providing high levels of maternal care, the rat's genes produced more stress receptors, making it calmer, less reactive to stressors, and more apt to provide maternal care to its offspring. These findings offer some evidence that social behavior may be inherited and

transduced via gene expression into neuroplastic alterations in brain structure, leading to psychobiological learning and change.

The notion that social experience can lead to changes in gene expression was voiced most prominently by Nobel laureate, Eric Kandel, who regarded this observation as the core component of a new paradigm for psychiatry (1998). Kandel summarized the current state of biological thinking with regard to the relation between social experiences and neurobiology, observing that:

> The regulation of gene expression by social factors makes all bodily functions, including all functions of the brain, susceptible to social influences. These social influences will be biologically incorporated in the altered expressions of specific genes in specific nerve cells of specific regions of the brain. These socially influenced alterations are transmitted culturally (Kandel, 1998, p. 461).

This powerful claim, while supported by over a decade of rigorous research, has rarely been directly tested. However, advances in psychoendoneuroimmunology, the study of how mental processes affect the immune system, have clearly shown the effects of psychological and social factors on human physiological functions that indirectly involve the genetic replication of cells (Ray, 2004). Such alterations of biological function may be mediated through experience-dependent gene expression, the process whereby social-environmental signals turn genes “on” and “off,” leading to alterations in protein synthesis which ultimately result in physiological changes (Pinaud, 2004).

Psychosocial genomic hypotheses

Although our genes provide a basic outline for development, environmental influences such as social experiences shape gene expression and ultimately make us unique individuals. This interaction is the essence of what Rossi (2002) has termed “psychosocial genomics,” the interdisciplinary study of the processes by which gene expression is modulated by psychological, social, and cultural experiences. Practitioners might profit from knowing more about this new science, for according to Kandel:

> Insofar as psychotherapy or counseling is effective and produces long-term changes in behavior, it presumably does so though learning, through producing changes in gene expression that alter the strength of synaptic connections and structural changes that alter the anatomical pattern of nerve cells of the brain (Kandel, 1998, p. 460)

Thus, it is conceivable that psychosocial interventions, the tools of social work practice, may produce alterations in gene expression leading, in some cases, to measurable neurobiological changes. Since can stress affect neurogenesis through alterations in gene expression and transcription (Glaser et al., 1990; Warner-Schmidt & Duman, 2006), ultimately leading to dysregulation of affect (Post, 1992), psychosocial interventions designed to reduce distress and improve mood may affect brain structure and function through this pathway. Muenke (2008) has recently suggested that the therapeutic effects of stress-reduction techniques might be mediated by changes in gene expression. In line with this hypothesis, a recent study of a meditative breathing practice found increased gene expression of the immune factors glutathione S-transferase, Cox-2, and HSP-70 in practitioners relative to controls (Sharmaa et al., 2008). While this study supports the psychosocial genomic hypothesis, its cross-sectional design does not allow for confident inferences vis-à-vis causality. However, in light of this potential shortcoming, a longitudinal study examined gene expression before and after exposure to eight weeks of meditation training (Dusek et al., 2008), and found alterations in the expression of 1561 genes after the intervention. Among these changes were increases in the expression of genes associated with the stress response, suggesting that learning to engage

the relaxation response through meditation may attenuate the deleterious impact of stress on cellular processes.

Although controlled psychosocial genomic research is uncommon, there are a growing number of psychosocial intervention studies that do measure physiological outcomes such as blood levels of cortisol or immune factors. For instance, stress reduction interventions have been shown to increase numbers of immune cells and decrease numbers of cells associated with allergic reactivity (Castes et al., 1999), and improve antibody response to the flu vaccine (Davidson et al., 2003). Intervention-related changes in such biological markers may serve as indirect measures of alterations in gene expression.

The new scientific paradigm outlined above provides a perspective on how the biopsychosocial constitutions of practitioners and clients might interact in the act of therapy:

> When a therapist speaks to a patient and the patient listens, the therapist is not only making eye contact and voice contact, but the action of neuronal machinery in the therapist's brain is having an indirect, and, one hopes, long-lasting effect on the neuronal machinery in the patient's brain; and quite likely, vice versa. Insofar as our words produce changes in our patient's mind, it is likely that these psychotherapeutic interventions produce changes in the patient's brain. From this perspective, the biological and sociopsychological approaches are joined. (Kandel, 1998, p. 466)

The union of neuroplasticity and psychosocial genomics research represents a synthesis of the social and biological sciences that is non-reductive: it does not dismiss human experience as the product of a neural machine, pre-determined by its genetic blueprint. Instead, it is integrative, inclusive, and holistic; this unitary approach reveals the power of thought and emotion, society and culture to affect not only our phenomenological experience but our very neurobiological structure and function. In sharp contrast to genetic determinism, this new paradigm envisions individuals as having the innate potential for agency over the tripartite dimensionality of their biopsychosocial selves.

Implications for Social Work

The social work profession's historical emphasis on the social environment as the context for individual well-being is supported by research over the past decade. Neuroplasticity and psychosocial genomic research indicate that socioenvironmental forces have the potency to alter human well-being through their effects on neurobiology. Social experience may be transduced through the activation of neurons, leading to modifications in the phenotypic expression of genes and eventuating in structural changes to the brain. While genes and neurobiology may be the substrates of vulnerability to environmental stressors, they are also, in all likelihood, the substrates of resilience (D Cicchetti, 2003; D. Cicchetti & Blender, 2006).

The sciences of neuroplasticity and psychosocial genomics may provide new empirical bases for social work interventions. Biological measures of change can and should be used to enhance the evaluation of social intervention research. Given the current funding climate and priorities of the National Institutes of Health, research programs designed to evaluate social work practice might be more likely to obtain grant support if interventions studied were evaluated with physiological outcome measures including those assessing gene expression and neuroplasticity. In time, a given practice may be deemed “evidence-based” when, among other criteria, it is shown to result in plastic brain changes or altered gene expression associated with improved biopsychosocial functioning.

Currently, there is a paucity of empirical support for this new paradigm in studies with humans. An abundance of research on higher mammals indicates that experience can trigger gene

expression leading to neuroplasticity. As referenced earlier in this paper, several studies on humans indicate that learning and training led to neurogenesis and the reorganization of neural networks. Despite developments in these lines of research, science has only begun to examine the effects of psychosocial interventions on brain structure and function. More research must be conducted in this emerging field, and the social work profession, with its expertise in addressing social problems and enhancing human well-being, can make a vital contribution to this endeavor.

Brain imaging and gene assays may be utilized to detect the neuroplastic and genomic effects of psychosocial interventions. Technologies such as magnetic resonance imaging (MRI), functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) are capable of assessing the neurophysiological changes associated with psychosocial interventions (Kumari, 2006). Reductions in psychiatric symptoms may be reflected in the alterations in brain metabolism and structure revealed by these imaging technologies. DNA microarray technologies, which can evaluate messenger RNA production in cells and thereby determine which genes are activated (Mirnics, Middleton, Lewis, & Levitt, 2001; Raychaudhuri, Sutphin, Chang, & Altman, 2001), have been used to assess alterations in gene expression related to post-traumatic stress disorder (Segman et al., 2005), social aggression (Berton et al., 2006), and depression (Evans et al., 2004). DNA microarrays may become more widely used to measure biological effects of psychosocial interventions in the not-too-distant future (E. L. Rossi, 2005).

Nevertheless, the funding and specialized training necessary to perform brain imaging and DNA microarrays decreases the likelihood that social work researchers working in isolation could leverage these technologies for biopsychosocial research. Consequently, future psychosocial intervention research could involve interdisciplinary teams of social workers, neuroscientists, and molecular biologists, where data from the biological sciences could be complemented by the insights of social work research. Alternatively, other more accessible biological markers, such as stress hormone levels in saliva, could be measured as a proxy for physiological change induced by psychosocial interventions. For example, salivary cortisol assays are a relatively inexpensive form of assessment that can be done by many university laboratories. Social work investigators could add this measure to their intervention research protocols.

Whether the impact of psychosocial interventions can be traced at the neuronal, genomic, or grosser levels of physiological response, biological markers will only be meaningful as a complement to self-report and collateral measures of change. Indeed, Engel's biopsychosocial paradigm is rooted in the philosophical principle of complementarity (Freedman, 1995); instead of the “either/or” mentality of dualistic reductionism, biopsychosocial research should embrace a “both/and” logic, where reports of subjective experience garnered through validated instruments and qualitative interviews are correlated with biological and behavioral data. Such research can add value to Social Work as a primary mental health and allied-health profession and lead to the implementation of interventions with demonstrable physiological, psychological, and behavioral benefits.

Conclusion

Over the past decade neuroplasticity research has enriched the biopsychosocial perspective by demonstrating that psychosocial experiences not only influence neurobiological processes but may actually change the structure of the adult brain. These structural changes consist of increased arborization of neurons, enhanced synaptic connectivity, and even the genesis of new neural tissue. Although neuroplasticity research is in its infancy, recent findings suggest that the effects of psychosocial experiences such learning and mental training on cognitive,

emotional, and behavioral functions may be mediated by alterations to the architecture of the brain.

In turn, experience-dependent modifications to neural tissue may be driven by epigenetic processes (i.e., changes in gene expression produced by environmental determinants). The human environment is constantly conditioned by social experiences, which, when transduced by the nervous system into electrochemical signals, may modulate protein synthesis in the nuclei of nerve cells, ultimately leading to changes in the replication and growth of neurons. Social experience can change gene expression, leading to the restructuring of the brain through neuroplasticity. While tentative at present, empirical investigations of the psychosocial genomic hypothesis will likely proliferate over the next decade.

These new biopsychosocial sciences are consistent with a view of human beings as holistic, recursive systems structurally coupled with their environments in a process of mutual change (Maturana & Varela, 1987). Intentionality and volition can generate changes in the structure of the brain, the very organ assumed to produce such mental phenomena (Schwartz & Begley, 2002). With this finding it is evident that human experience is not driven solely from the bottom-up by neurobiology and genetics. Instead, there is growing evidence that psychosocial experience can exert a macrodeterministic, top-down force upon our biology. In the philosophy of emergent interactionism, Roger Sperry, Nobel laureate neuroscientist, described macrodeterminism as a higher-order, molar level of organization that determines and conditions the activity of lower-order, nested sub-components (Sperry, 1987). Hence, human beings, who are at one level assemblies of organ systems comprised of aggregates of cells, in turn composed of organic molecules made up of sub-atomic particles, are not merely the summation of these physical elements. Instead, the consciousness that emerges from the interaction of these components can act back upon its physical substrate. Thought, emotion, and action trigger neural activity, which can lead to a re-organization of the brain, shaping future psychosocial experience. From this perspective, we are not the passive products of neurophysiology and heredity; rather, through our behavior in the social environment, we become active agents in the construction of our own neurobiology, and ultimately, our own lives.

This new paradigm may reveal the empirical foundation of that most central of social work principles, the idea that people have the power to transcend and transform their limitations into opportunities for growth and well-being.

Acknowledgments

ELG was supported by Grant Number T32AT003378 from the National Center for Complementary and Alternative Medicine.

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