Chapter 5
Brain Development, Early Childhood, and Brain-Based Education: A Critical Analysis

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Introduction

The last several decades have witnessed significant developments in neuroscience and in technology that allows researchers to monitor brain function while students are reading, solving mathematical problems, or performing other educational tasks. Accompanying this has been an effort to link these advances in neuroscience to education and the process of teaching. Jensen (2008) has called this "a new paradigm," commonly referred to as brain-based education. Some educators quickly joined the bandwagon and attempted to provide a cloak of respectability in this new paradigm by linking a number of techniques to these new developments, even though these new curricula did not originate in neuroscience research. As Bruer (1999a) has noted, brain science is said to "support Bloom's Taxonomy, Madeline Hunter's effective teaching, whole-language instruction, Vygotsky's theory of social learning, thematic instruction, portfolio assessment, and cooperative learning." Note that none of these theories or approaches originates from neuroscience.

We assume, and most readers would probably agree, that teaching methods which have been demonstrated to be effective via evidence-based approaches will also be supported by research on neural mechanisms and the neurobiological basis of learning. However, the demonstration of teaching and learning effectiveness is not dependent on neuroscience; rather, it must be demonstrated independently. Evidence of effectiveness does not come from efforts to link an educational

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technique to neuroscience research without an independent demonstration of educational effectiveness through careful research studies. Thus, while this new paradigm has given rise to a number of claims that techniques are "brain-based" and therefore presumed to be effective without such independent documentation of their effectiveness, the claim of a link to neuroscience is superficial at best. Rather than being driven by neuroscience research, such claims appear to be made in the hopes that such claims will provide a cloak of respectability to the uncritical eye.

Neural Development

Prenatal Development

Before one can understand the implications of early childhood experiences on later cognitive development, it is critical that a basic understanding of neural development be obtained. The human brain does not simply turn on and begin to experience sensations and have perceptions and even cognitions at birth. It is critical to understand that important changes in utero occur in conjunction with and in response to environmental experiences which then set the tone for later cognitive development.

The central nervous system (the brain and spinal cord) as well as the peripheral nervous system are composed primarily of specialized communication cells called neurons. These neurons are supported by glial cells, which both help hold the position of neurons and assist in neural transmission. Neurons are specialized cells composed of dendrites, a soma or body, and one or more axons. The dendrites are composed of hairlike projections that end at the synapse, the space between neurons. These dendrites have specialized receptors on their cell walls that allow them to capture the chemical (a neurotransmitter) released from other neurons. Axons are long projections off of the cell body. A neuron may have one axon or many axons. These axons are critically involved in sending messages between neurons via neurotransmitters. Further, axons lengthen to form pathways and connections to other nearby neurons as an organism experiences the world. In fact, this lengthening and creation of neural pathways, or neural circuits, form the neural underpinnings of learning (Garrett, 2011).

During the embryonic stage (14 days to 8 weeks postconception), the full formation of the embryonic disc begins (Kalat, 2007). This includes the mesoderm, which will develop into the nervous system (Rosenzweig, Breedlove, & Leiman, 2002). The nervous system begins as a hollow tube that later becomes the brain and spinal cord. This neural tube begins when the surface of the embryo forms a groove and the edges of this groove curl upward until they meet, forming a tube. The neuroectoderm forms when the foundation for the three main brain structures has been developed: the hindbrain, the midbrain, and the forebrain (Rosenzweig et al.). The closed neural tube will then become the spinal cord, central canal, and ventricles of the brain. The central canal and ventricles will form the irrigation system for the

brain, while the spinal cord is the major pathway for sensory input and motor output for the body (Kalat). From this point, development of the nervous system proceeds through six distinct stages: (1) cell proliferation through mitosis, (2) cell migration, (3) differentiation, (4) synaptogenesis or initial circuit formation, (5) cell death or circuit pruning, and finally (6) synapse rearrangement.

Cell proliferation is the first of these developmental stages. Once the neural tube is formed, many new cells are produced through mitosis or neurogenesis in the ventricular (i.e., ventricle) zone, the area surrounding the hollow tube (Rosenzweig et al., 2002). One cell division leads to the formation of a daughter cell, with additional divisions forming an immature neuron. The cells that will become neurons divide and multiply at the rate of 250,000 new cells every minute. During this stage, most of this proliferation occurs in the ventricular zone. This ventricular zone then develops into the actual ventricles and central canal. Cells in the hindbrain, or lowest portion of the brain, and the midbrain or middle brain then begin rapid proliferation and division.

Migration is the next stage. The newly formed neurons migrate from the ventricular zone to their final location in the brain (Kalat, 2007). This is how the cortex and higher brain areas are formed. To do this, the cells are aided by specialized radial glial cells. These glial cells provide the structure upon which the neurons can migrate to their final location. *Filopodia*, or tiny cytoplasm projections, assist the neurons in finding their location after leaving the radial glial cells (Mattli & Lappalainen, 2008). Finally, these glial cells will also provide the necessary structure to "hold" the neurons in place.

Once the newly formed neurons have migrated to their final location, *cell differentiation* begins. This process gives rise to specific types of neurons and glial cells (Kalat, 2007). During synaptogenesis, or neural maturation, there is an elongation of the axons, with growth cones forming on the ends of the axons (Marin & Rubenstein, 2001). Terminals, the area from which neurotransmitter is released, are then established at the ends of the axons. Dendrites also elongate, and the neuron begins to express its neurotransmitter. A neuron can express one or more types of neurotransmitter. Special proteins called neurotrophic factors help stimulate this cell growth and maturation of the neurons (Kalat). Any disruption in the combination of proteins and chemicals during this process can result in brain deficits, including mental retardation and developmental delays, and have even been linked to schizophrenia (Crossin & Krushel, 2000; Poltorak et al., 1997).

Synaptic rearrangement and circuit formation occur once the cells have formed. Here the axons of developing neurons grow toward their target cells to form functional connections (Rosenzweig et al., 2002). These functional connections will provide the pathways for not only basic brain functions but also cognitive functions. To do this, special growth cones form at the tip of the axons. The growth cones allow the neuron to sample the environment for directional cues and help the axons to find their way along the glial cells. That is, chemical and molecular signposts attract or repel the advancing axon, coaxing it along the way until the neuronal axons reach their final destinations. There is quite a bit of pushing and pulling and hemming in of the neurons from the sides via these chemical and molecular changes.

The chemical and molecular forces guide the neuron to intermediate stations and guide them past inappropriate targets (Rosenzweig et al.). This stage is very critical for appropriate development of basic life and cognitive functions.

Pruning is the final stage of neural development. This actually involves the *elimination* of excess neurons and synapses. Neurons that are unsuccessful in finding a place on a target cell or that arrive late die off (Oppeneheim, 1991). Thus, normal cell death occurs during synaptogenesis, typically through apoptosis. Apoptosis is the process of active cell death, while necrosis is passive cell death due to injury. The circuit formation, then, is critically dependent on this pruning. Which neurons die and which neurons survive is dependent on the interaction between apoptosis and environmental stimulation. Cells that are part of an active circuit are kept; cells that are not used may die (Rosenzweig et al., 2002).

While scientists have extensively studied early brain development, one disorder, *Fetal Alcohol Syndrome* (*FAS*), demonstrates the importance of early experiences on the developing brain. FAS, often characterized by mental retardation and behavioural dysfunction, is typically the result of a mother's use of alcohol during a critical period of brain development. Symptoms include low birth weight, a small head circumference, failure to thrive, developmental delays or disabilities, and poor organ development. Facial anomalies are the hallmark of FAS, including smaller eye openings, flattened cheekbones, and an underdeveloped philtrum, or groove between the nose and upper lip. This philtrum is the location of the final fusing of the facial features, and underdevelopment is suggestive of delayed and muted cranial development.

Interestingly, FAS brains are often small and malformed, with the neurons dislocated compared to typically developing brains (Garrett, 2011). These dramatic brain changes appear to occur during migration, where cortical neurons fail to line up in columns as they normally would. While neurons in the normal brain tend to line up along vertical axes, in the alcohol-exposed brain, neurons line up randomly (Gressens, Lammens, Picard, & Everand, 1992). The radial glial cells appear to revert to their more typical glial form prematurely, failing to contain neurons in their appropriate location. Thus, many neurons may continue migrating beyond the usual boundary of the cortex. Because of the disruption in neuron location and lack of appropriate circuit formation, many of these children develop significant cognitive delays or disabilities and seizure disorders.

Postnatal Development

While these six stages of neural development occur in utero, circuit formation and pruning are also critical throughout a child's life. As the child experiences the world, they will continue to experience circuit formation and pruning throughout their lifetime. Circuits are strengthened or weakened depending on a child's life experiences, including academic and social experiences. Certainly neural development occurs most rapidly during prenatal and then postnatal periods, but it is important to recognize that neural development is a lifelong process.

Between birth and age 6, the brain continues to undergo its last major wave of neurogenesis and massively increases synaptic connectivity and circuit formation. The proliferation of glial cells is critical in this postnatal development. At birth, the brain weighs approximately 25 % of the full adult brain weight. By age 6, this increases to 95 % of adult weight. This increased weight is due primarily to myelination.

Importance of Myelin

Myelin is of critical importance in postnatal brain development. Myelin, consisting of specialized glial cells, is formed from cholesterol. Myelin provides a guide tube for the sprouting end of a newly formed neuron to grow through, thus allowing the extending axon to be guided to its destination. At birth, the brain is myelinated through the thalamus. However, the postnatal process of myelination of the cortex, or thinking area of the brain, is largely based on experience. For example, a premature baby will have significantly more myelin than a full-term baby of the same gestational age, because that premature baby will have had life experiences outside the womb at an earlier age than a full-term baby.

Interestingly the peripheral nervous system has myelin formed from much more rigid glial cells, such as Schwann cells. In contrast, the myelin of the central nervous system does not have a rigid structure. This allows more flexibility in the formation of neural circuits but also makes neural repair a much more difficult endeavor: When the axon is injured, the myelin does not remain rigid but may collapse and actually block the path as the axon regenerates. Thus, central nervous system regeneration is much more difficult than peripheral nervous system regeneration.

As noted above, the nervous system refines its organization and continues to correct errors by eliminating large numbers of excessive synapses. Forty percent of active neuronal death occurs during the first two years of life. This neuronal death is critical because it eliminates unconnected or useless neurons. Failure to eliminate unused circuits or damage to critical circuits may result in development delays or disabilities. For example, there is evidence of apoptosis dysfunction in postmortem brains of children with autism, particularly in the cerebellum, midbrain, and hippocampus. This suggests an insufficient degree of circuit formation or synaptic connectivity of neurons in the brains of these children.

Remember, however, that reorganization will continue throughout a child's life. Indeed, brain development occurs in waves until approximately age 21 (Gogtay et al., 2007). The cortex and in particular the temporal, parietal, and frontal lobes as well as the limbic system are refined through these waves of development. Large episodes of increased circuit formation occur from birth to three, with other bursts from ages 7 to 9, and again in mid-adolescent years (ages 13–17). The temporal or language areas of the brain show the largest increase in synaptic connectivity from birth to three, and again in mid-adolescence. Changes in the parietal-temporal areas for higher cognitive functioning show a large increase in synaptic connectivity from

birth to three, with continued smaller bursts throughout the childhood years. The limbic system, integral for emotional growth and attachment, shows large episodes of synaptic connectivity from birth to three, again at approximately ages 7–9, and then in mid to late adolescence, or ages 15–17. Finally, frontal lobe development bursts are found from birth to three, with a slight increase in middle childhood. However, the largest burst of frontal lobe development occurs during the late adolescent years, ages 17–21. This may explain why adolescents are more impulsive and are more likely to engage in risk-taking behaviour: Their frontal lobe development is incomplete, and the neural circuits necessary to control these behaviours are not fully formed.

Life Span Changes in the Brain

While early childhood is critical for a large portion of cognitive, language, and emotional development, the brain undergoes additional critical development during the middle childhood and adolescent years. Importantly, frontal lobe functions such as impulse control and critical thinking do not appear to reach full development until late adolescence (Gogtay et al., 2007). Further, synaptic connectivity and circuit formation occurs throughout the life span.

How does this reorganization occur? Synapses between neurons are strengthened or weakened depending on whether the presynaptic neuron and the postsynaptic neuron fire together. Those that fire together are strengthened in a process called Long-Term Potentiation (LTP). LTP involves an increase in synaptic strength following repeated high-frequency stimulation (Garrett, 2011). There is an increase in dendritic growth as well as changes in the number of receptor sites in the synapse. Conversely, neurons that fail to fire together are weakened, a process called Long-Term Depression (LTD). LTD involves a decrease in synaptic strength when an axon of a neuron is active, but the postsynaptic neuron is not stimulated (Garrett). This, then, may result in decreased dendritic growth as well as a reduction in the number of receptor sites at the synapse. Specialized chemicals that enhance the development and survival of the neurons, called neurotrophins, are critical in this process. Recent research suggests that the postsynaptic neuron sends feedback to the presynaptic terminals via these neurotrophins. Neurotrophins decrease the plasticity, or ability to be modified, of these synapses. Thus, the synapses become more permanent via the action of neurotrophins.

Experience does indeed affect neural development (Bennet, Diamond, Krech, & Rosenzweig, 1964; Gottleib, 1976; Rosenzweig & Bennet, 1977, 1978). Neural activity due to environmental experience appears to regulate gene expression that directs the synthesis of cell adhesion molecules (Kalat, 2007). Further, neural stimulation regulates the release of neurotrophins (NGF) that are released from dendrites after synaptic connectivity. NGF stimulates the foundation neurotransmitter and promotes subsequent reorganization and synaptic connectivity (Garrett, 2011).

A child's experiences in the world continue to shape synaptic construction and reorganization throughout the individual's life. As noted above, this reorganization or a shift in connections that change the function of an area of the brain may occur at any point in the life span. Of course, the older the individual is, the more difficult reorganizing the brain becomes. This is not because the brain becomes "inflexible" but is due to the increased complexity of the neural circuits resulting from environmental interactions as the child grows. An analogy may be made with remodelling a house. A house undergoing its first remodel will have a relatively straightforward and timely remodel. Not much has been changed from the original plans, modifications have been minor, and changes may be easily made. However, a house that has been remodelled numerous times over the years is much more difficult to change, as the many restructuring and reforming of walls, wiring, plumbing, etc., make it a much more complex and less straightforward endeavor. So is true of the brain. A 20-year-old brain has many more circuits, and those circuits have many more extensive connections and reorganizations than a 2-year-old brain. Reorganizing or repairing the older brain is, by definition, a much more complicated process.

Repairing an Injured Brain

Neurogenesis, or the formation of new neurons, was once considered impossible in the older brain. Typically, neurons do not reproduce or replace themselves. Once a neuron is killed, it is often irreplaceable. However, newer research has shown that the nervous system does have some ability to repair itself by growing new neurons. The adult mammalian brain produces some new neurons, but research has only found these new neurons in two crucial areas: the hippocampus and near the lateral ventricles which supply the olfactory bulb, which is responsible for our sense of smell. Interestingly, both of these areas are critical for memory function. Thus, perhaps memory does indeed continue to "grow" (Kalat, 2007).

Recent research suggests that there may be several strategies for inducing self-repair following damage to the brain (Garrett, 2011; Kalat, 2007; Rosenzweig et al., 2002). First, neuron growth enhancers have been found which counteract the chemical forces that inhibit regrowth. These neural enhancers provide guide tubes or scaffolding for axons to follow in a manner highly similar to that found during the migration stage of neural development. Stem cells may also be critical for neural repair. Stem cells are undifferentiated cells that can develop into specialized cells such as neurons, muscle, or blood. Recent research has demonstrated that placing embryonic stem cells into an adult nervous system encourages new neurons to differentiate into neurons appropriate to that area. While this research is still in its infancy, it again demonstrates that the brain is a continuously changing and growing organ.

What is the take-home message here? Brain circuits are formed and pruned throughout the life span. While the biggest burst of neural development occurs prenatally and then in the first three years of life, middle childhood and adolescence

are also critical time points for brain development. Finally, human brains continue to change throughout an individual's life, and these changes are highly dependent on experiences in the world. Thus, brain development must be considered a lifetime endeavor. The old adage of "use it or lose it" takes on significant meaning for the brain. An individual who is active physically and mentally will be an individual whose brain continues to organize and reorganize, and perhaps even grow, throughout their entire life.

Practical Applications

Advances in neuroscience over the past several decades have resulted in a variety of proposals that claim to improve education. Ostensibly, these practical applications are based on advances in our understanding how the brain is linked to learning or to other processes that impact classroom performance. There are a variety of ways in which the term "brain based" has been applied (Sylvan & Christodoulou, 2010), ranging from simply labelling a practice as brain based to actually attempting to apply research in neuroscience to educational practices. There are reasons to view all such claims with skepticism unless there is direct evidence of educational gains that can be specifically tied to that practice through well-designed research (Alferink, 2007; Alferink & Farmer-Dougan, 2010).

The Brain, Educational Policy, and Critical Periods

As described above, research has demonstrated age-related changes in the brain and some have linked these changes to educational policy. From birth to around age 3, there is a period of very rapid synapse development such that the brains of very young children are densely packed with synapses. These high-density levels continue until about age 10. After age 10, synaptic pruning occurs and density declines to adult growth levels by around age 15 (Bruer, 1999a, 1999b). Brain volume increases until around age 14 and then shrinks over the remainder of the life span (Courchesne et al., 2000). In addition, there is some evidence indicating that the brains of young children use more glucose than adults, with glucose uptake levels following a similar time course as synaptic density. For example, Chugani, Phelps, and Mazziotta (1987) found that glucose metabolism in the brain increases from about age 4 to age 10 and then declines to adult levels at around age 16.

Based on these age-related changes in synaptic density, glucose uptake, and levels of neurotransmitters, Shore (1997) suggested that the brains of young children might be primed for learning. Indeed, Jensen (1998) and Kotulak (1996) suggest that it is during the early school years, the ages between approximately 4 and 10, when we learn material quickly and easily. Chugani (1998) suggested that there may be a critical period when learning occurs at its highest rate. Sousa (1998) suggested that critical period is between the ages of 4 and 11.

While there is a critical period for the development of vision (Bruer, 1999b; Fox, Levitt, & Nelson, 2010) or when we are more likely to learn specific tasks such as a language (Bruer, 1999b; Fox et al., 2010; Kotulak, 1996; Sousa, 2001), the observations cited above are said to suggest a critical period for learning in general (Bruer). This critical period is a window of opportunity, a window that closes if one fails to take advantage of it. As a matter of public policy, this implies that resources should be shifted significantly from funding high schools and universities to preschool and elementary education.

In contrast to current educational trends, there is no evidence either linking the number of synapses or glucose uptake as direct causal factors for rate of learning or indicating that 5-year-olds are better at learning than are students who are 15 (Bruer, 1999b). Learning is based on the formation of new synapses (Garrett, 2011), not on the number of existing ones. Children who do not learn to read by the third grade can still learn to read in adolescence, and adults can certainly learn numerical skills typically learned in childhood (Bruer, 1999b; Tokuhama-Espinosa, 2011). Further, critical thinking and analytic skills appear to develop later in childhood, and attempts to teach such skills in early childhood have met with failure. Appropriate levels of funding are important for children at all ages and well-designed early childhood education is strongly supported by the evidence in helping provide the foundation for future educational success. Importantly, though, it is not appropriate to single out one age group for especially high levels of funding based on an overinterpretation of the neuroscience research.

"Right" Versus "Left" Brain

The interest in brain-based education may have started with research on brain lateralization (Jensen, 2008). The cortex of the brain is divided into two hemispheres that are joined by a band of fibers, the corpus callosum. This band of fibers permits electrical impulses to travel between the two hemispheres. When the corpus callosum is severed, this communication is no longer possible.

In cases of severe epilepsy, the corpus callosum allows inappropriate electrical impulses to travel between the two hemispheres, and this can result in uncontrolled seizure disorders. In an effort to control these seizures, the corpus callosum has often been severed in these patients and the two hemispheres operate independently. Research with these individuals clearly demonstrates some degree of lateralization of function across the two hemispheres (Gazzaniga & Sperry, 1967). While these split-brain patients seem perfectly normal, careful testing showed that subjects would name objects that they could "see" with their left hemisphere and point to objects they could "see" with their right hemisphere (Gazzaniga, 1972). This research suggested that each hemisphere had specialized functions, with the left hemisphere linked to language and the right to spatial functions.

Continued research in neuroscience strongly supports this lateralization of function but would also note that these lateralized functions are integrated and occur

simultaneously in individuals with an intact corpus callosum (Carlson, 2010). That is, in typical individuals, information is processed differently but simultaneously by both hemispheres.

Right- versus left-brain teaching approaches suggest that the different brain hemispheres control different academic functions. According to right- versus left-brain theorists, the "left brain" is said to be the "logical" hemisphere, concerned with language and analysis, while the "right brain" is said to be the "intuitive" hemisphere concerned with spatial patterns and creativity (Sousa, 2001). "Left-brain" individuals are said to be verbal, analytical, and good problem solvers, while "right-brain" individuals are said to be good at art and mathematics. Thus, brain-based learning came to mean that teachers should teach to each specific hemisphere. To teach to the left hemisphere, teachers should have students engage in reading and writing. To teach to the right hemisphere, teachers should have students create visual representations of concepts (Sousa).

In the intact human, there is little evidence to support these teaching methods. True separation of function occurs only in individuals without an intact corpus callosum or those with specific brain damage, a group certainly representing a very small percentage of the student population. Thus, it is neither accurate nor realistic to believe that individuals may selectively use one hemisphere of their brain at a time for separate academic functions. It is highly improbable that any given lesson, regardless of analytic or spatial type, only stimulates activation of one hemisphere. Further, analytic and spatial functions are not as localized as is promoted by many of those developing such curricula (Garrett, 2011). Whether a visual-spatial task involves the right or left hemisphere depends on details of the task (Chabris & Kosslyn, 1998). The development of left-brain/right-brain curricula was debunked 25 years ago but continues to shape school curricula (Lindell & Kidd, 2011).

Brain Lateralization and Gender Differences

Recent research also suggests differences between the brains of boys and girls. Brain scans reveal structural differences between the genders and also suggest that different brain areas may develop at different times for boys and girls (Gurian & Stevens, 2005; Whitehead, 2006). In addition, a variety of research investigations have found significant differences in language and spatial processing between the two genders (Benbowa, 1988; Burman, Bitan, & Booth, 2008; Garai & Scheinfeld, 1968; Witelson, 1976). This research on specialized skills has been interpreted to support differences in academic performance and in brain lateralization. Boys were said to be "right-brain" dominant, while girls were said to be "left-brain" dominant (Gurian & Stevens, 2010). Schools were supposedly left-brain institutions, favoring girls over boys, supposedly explaining the academic achievement gap between the genders and the greater difficulty in managing the behaviour of boys in the classroom (Sousa, 2001). Based on brain differences between boys and girls, one school in Owensboro, Kentucky, even separated boys and girls into different

classrooms so that it would be possible to teach to these brain differences. Of course, again the problem with this is that few students have had their corpus callosum severed and as noted above, attempting to teach to one hemisphere is misguided. In addition, brain differences between the genders are generally small and have not been shown to have broad practical importance (Bruer, 1999a; Eliot, 2010). Such differences reflect group differences, not necessarily individual variation. Addressing brain differences simply by segregating the genders into different classrooms without direct measurement of differences such as lateralization is likely to result in two classrooms that have a mixture of "right- and left-brain" individuals but of different genders.

One may argue that increased academic progress and higher test scores result from gender-based classrooms, but the evidence does not support this argument (Eliot, 2010; Halpern et al., 2011). Halpern et al., after a review of the evidence, argue that claims of the advantages of sex-segregated education may be due to other uncontrolled factors and that no evidence exists from carefully controlled studies supporting these claims. They also note that gender segregation has its own problems. For example, increasing the number of boys in a group increases violence and aggression particularly in the early school years. Gender differences, then, must be interpreted cautiously, and isolating children by gender for education purposes does not appear to improve learning.

Brain-Compatible Teaching

Several educators have attempted to link educational techniques to recent progress in neuroscience, suggesting that some instructional techniques are brain based (Jensen, 2008; Laster, 2008), brain compatible (Ronis, 2007; Tate, 2003, 2004, 2005, 2009), brain friendly (Biller, 2003; Perez, 2008), or brain targeted (Hardiman, 2003). One prominent proponent of brain-based education, Tate (2003), not only provides examples of the brain-compatible activities but she suggests that some educational practices "grow dendrites" and others do not.

Proponents of brain-compatible instruction emphasize that only some forms of instruction are brain compatible. Indeed, these authors suggest that teaching practices such as drill, practice, and memorization do not "grow dendrites," while the techniques they support do (Tate, 2003, 2004, 2005, 2009). They suggest that instructional methods that are brain compatible follow constructivist approaches which involve open-ended, process-based, and learner-centered activities. This is where the applications of neuroscience may have jumped beyond the data. Tate (2003) provides no data indicating that the methods she disparages do not in fact grow dendrites, or that her preferred methods do. Further, she provides no evidence that dendritic growth is most critical for learning and education.

"Growing dendrites" is, at best, an incomplete picture of neural changes over time and inaccurate as a description of the neural mechanism for learning. Indeed, the literature suggests that it is long-term potentiation (LTP) that is critical for

learning and memory formation (Freeberg, 2006; Garrett, 2011). As described above, LTP is an increase in synaptic strength that allows for the development of neural circuits which underlie memory and cognitive processing. It is not necessarily having more dendrites that are critical, but it is the increased number and strength of connections between neurons within the newly formed neural circuits.

Any instructional technique that produces learning must necessarily be accompanied by changes in the neural bases that support learning. Claiming that some instructional techniques produce these neural changes while others do not is jumping beyond the data provided by neuroscience research. The data suggest that *repetition-based activities* such as *memorization* and *mastery learning* appear to strengthen and solidify the formation and maintenance of these circuits (Freeberg, 2006; Garrett, 2011). Data strongly support the use of precision teaching, mastery learning approaches, and programmes such as DISTAR or direct instruction approaches (Kim & Axelrod, 2005; Kirschner, Sweller, & Clark, 2006; Mills, Cole, Jenkins, & Dale, 2002; Ryder, Burton, & Silberg, 2006; Swanson & Sachse-Lee, 2001) as effective instructional techniques.

Ignoring the neuroscience literature on memory formation, Perez (2008) provides "more than 100 brain-friendly tools and strategies" for teaching reading and developing literacy. She reviews several findings from brain research, suggesting that neuroscience research and instruction have never been so closely linked. For example, she indicates that research shows that reading originates and relies on the brain systems for spoken language. While this should not be surprising since reading aloud so that others can assess reading skills is an important component of increasing reading skills (Rayner, Foorman, Perfetti, Pesetsky, & Seidenberg, 2001), it is unclear what this finding tells us about how to teach children to read. Perez indicates that this means reading must be taught actively, not passively. It is unclear how this suggestion is based on neuroscience and whether any evidence-based reading programme would suggest that children will learn to read by simply passively looking at the word on the page. Independent from brain research, we know that advocating that children read silently without feedback, rather than aloud with feedback, does not improve reading (National Reading Panel, 2000), or that training teachers not to correct reading mistakes results in unacceptably large numbers of children who can't read (Rayner et al.). It would seem that the neuroscience research on reading cited by Perez follows from how children learn to read in effective reading programmes rather than the opposite. It could be argued that neuroscience may tell us something about why ineffective programmes result in many nonreaders; the critical issue is how does one design reading programmes that work based specifically on that neuroscience. Here, Perez does not provide us with any guidance.

Brain-compatible education is perhaps an unfortunate outcome of the "Decade of the Brain." Linking educational practices to neuroscience, however superficial that linkage might be, provides a false sense of credibility for those that are in awe of advances in neuroscience. Thus, one must interpret untested "neuro-based" curricula with caution. Purporting to link educational practices to the brain really adds nothing to how one understands effective teaching practices. Instead, obtaining direct evidence of the effectiveness of various educational practices would be far

more useful and likely provide greater support for neuronal changes. There is little basis to doubt that effective practices will be brain compatible, but there are reasons to be skeptical of claims that studying the brain will guide us to develop new effective practices.

Brain-Compatible Teaching, Learning Styles, and Multiple Intelligences

Attempts have been made to arrange classrooms so that they are "brain compatible." These authors suggest that this can be accomplished by teaching to different learning styles or a child's multiple intelligences (Ronis, 2007; Sprenger, 1999; Tate, 2003, 2004, 2005, 2009). Following Gardner's (1983) hypothesis, they suggest that children learn best through teaching methods that are compatible with their specific individual intelligence profiles. One way to accomplish this is to teach to the child's preferred modality. This preference is determined on a self-report questionnaire, and the teacher uses this information to determine whether the child learns best visually, auditorily, kinesthetically, or a combination of modalities (Dunn, 1987; Keefe, 1982; Ronis, 2007). In other words, based on this measurement, the teacher matches instruction to that preferred modality.

However, whenever an assessment instrument is developed, users must be concerned with its psychometric properties. Unfortunately, learning style inventories are known to have problems with both reliability and validity (Dembo & Howard, 2007; Kratzig & Arbuthnott, 2006). Although other ways of categorizing learning styles have been developed, the problem of reliable measures persists or has not been tested.

More critically, it is easy to find studies that show that students differ on their preferred learning style on inventories but difficult to find studies that show that teaching to individual learning styles actually makes a difference in student learning outcomes specifically due to this practice. However, there is evidence that shows that teaching to learning styles is not an effective method (Dembo & Howard, 2007; Kratzig & Arbuthnott, 2006). Many student guides to textbooks advise students who know their learning styles to seek instructors that teach to that style. The evidence indicates that this does not make a difference in class performance (Dembo & Howard). For example, auditory learners that select instructors that emphasize that modality do not perform better than students with other preferred modalities. A recent comprehensive review of the evidence regarding learning styles (Pashler, McDaniel, Rohrer, & Bjork, 2009) found that claims about the effectiveness of teaching to learning styles are not based on the critical methodology they conclude is necessary to justify those claims. Further, some newer ways of categorizing learning styles lack any evidence of educational effectiveness at all. Pashler et al. conclude that for the learning styles hypothesis to have any credibility, well-designed studies must show that students with a particular learning style will perform better in a class tailored to their preferred modality than students with a different preferred

modality. That is, research must demonstrate that children learn and perform better when instruction is matched to their learning style. Curiously, few such studies exist. As noted above, matching instruction to learning styles failed to produce this outcome in the Dembo and Howard study.

Related to the teaching to learning styles is the concept of teaching to multiple intelligences. Multiple intelligences are said to be another way in which students may differ in how they input and process information. Gardner (1983) originally identified seven different intelligences, including linguistic, musical, spatial, logical-mathematical, bodily-kinesthetic, interpersonal, and intrapersonal. There have been numerous articles published describing the educational implications of Gardner's model and many teachers have adapted multiple intelligence in the same way that they use learning styles as a means of addressing individual differences. Thus, just as one might teach to multiple learning styles, one also can teach to multiple intelligences.

Unfortunately, just as is true for learning styles, questions exist about a reliable and valid way of measuring Gardner's multiple intelligences (Lubinski & Benbow, 1995). In addition, questions have been raised about whether asserting the existence of multiple intelligences adds any explanatory power over more traditional psychometric approaches that emphasize a single factor (Waterhouse, 2006a). In addition, while the number of articles exploring how the model can be implemented is extensive, the model has not been adequately tested through empirical research (Lubinski & Benbow, 1995; Waterhouse, 2006a, 2006b). Unfortunately, brain science is said to validate many other teaching techniques besides learning styles and multiple intelligence. Yet whatever evidence there is for the effectiveness of those techniques does not come from research on neuroscience (Bruer, 1999a; Eliot, 2010). Changing teaching methods or educational policy based on a model that lacks adequate empirical support carries with it a risk of time and resources being diverted from methods with stronger empirical support. The data on learning styles and multiple intelligence should alert the reader that skepticism about these claims is warranted.

Exercising the Brain

The development of executive function is a cornerstone for not only academic learning but development of important social behaviours. Several clinical disorders appear related to poor executive function development, including attention deficit hyperactivity disorder and autism (Lyon, 1996; Naglieri, 2003). Emergence of executive functions influences a child's ability to apply knowledge as well as influencing the child's ability to know when and how to act in social situations. A child who is unable to plan, update his or her working memory, or shift attention from one task to another will have difficulty in not only academic settings but social settings as well. Barkley (1996) suggests that executive attention may even influence the development of imagination, empathy, creative thought, and self-evaluation. So,

what environments or activities support and influence the development of executive functioning in childhood?

A large body of research has shown that organisms exposed to enriched environments are better at object exploration and recognition and are more prone to explore novel stimuli (Mitani, 1993; Rose, Dell, & Love, 1987; Walasek, Wesierka, & Werka, 2002). Studies have also found that enriched environments promote heavier brain weights (Susser & Wallace, 1982; Walasek et al., 2002), lasting changes in the brain including a thicker hippocampus (Susser & Wallace), and increased synaptic transmission in the hippocampus (Port, Murphy, Magee, & Seybold, 1996). These changes in the hippocampus may be critical given that the hippocampus is known to be directly connected to information processing and executive function. Further, several research investigations suggest that the *type of contact* with environmental stimuli may be important (Kiyono, Seo, Shibagaki, & Inouye, 1985; Mohanty & Behera, 1997; Ruiben, et al., 2001).

What kinds of environments appear to increase learning and cognition in children? Video game experiences appear to enhance performance on several tasks including multiple-object tracking task (Green & Bavelier, 2006b), identifying target objects embedded in a distracting background (Green & Bavelier, 2006a), and faster temporal characteristics of visual attention. Children who play video games may also have enhanced mental rotation abilities (Feng, Spence, & Pratt, 2007). However, video games do not appear to enhance every perceptual, attentional, and/or visuomotor skill (Green & Bavelier, 2008). The beneficial effects of video game play appear relatively constrained to attentional and motoric tasks.

Music lessons have also been shown to result in larger increases in IQ scores. Schellenberg (2004) found larger increases in IQ for children who received musical training. Rauscher et al. (1997) found increases in spatiotemporal reasoning for children who received keyboard training, even compared to those receiving computer training. However, there is no magical Mozart effect of music that increases IQ or cognitive abilities for all children in all settings (Green & Bavelier, 2008; Waterhouse, 2006a). Reading to a child has been well established as a factor in developing cognitive abilities (Bus, Van Ijzendoorn, & Pellegrini, 1995; Whitehurst & Lonigan, 1998). Reading to young children provides a variety of unique domains of stimulation including interactive language opportunities, picture-based stimulation, forms and cadences of written language, and sequencing.

Finally, athletics and physical exercise result in significant brain changes. In a study conducted by Kioumourtzoglou et al. (1998), basketball players showed superior selective attention and eye-hand coordination, volleyball players were better at estimating speed and direction of moving objects, and water polo players showed faster visual reaction times and better spatial orienting abilities. Aerobic exercise has been shown to improve a wider range of cognitive abilities and particularly dual-task performance (Colcombe & Kramer, 2003). In early learning, free play may be more beneficial for cognitive development than organized physical activity (Burdette & Whitaker, 2005). Physical activity exhibited during free play differs from typical physical activity in several important ways. Free play often involves gross motor play but also involves activities such as role-playing, manipulating and building with

objects, and pretend play. In their review of the effects of free play, Burdette and Whitaker found improvements in attention, social skills such as affiliation, emotional affect, as well as the cognitive effects found by other researchers.

Why are these particular activities so beneficial to cognition and learning? These activities all appear to increase brain activity. Vanyman, Ying, and Gomez-Pinilla (2004) found that exercise and exploration increased levels of brain-derived neurotrophic factor (BDNF) and promoted increased performance and learning on spatial learning tasks in rats. They found that rats with higher levels of BDNF, resulting from the opportunity to explore an enriched environment, were better at learning and recall. The authors suggested that exploration in an enriched environment gave rats multiple routes for exercise, promoting multiple opportunities for making spatial connections, and thus promoted increases in BDNF production and cognitive function. Similar studies show that glutamate transmission in the prefrontal cortex, which appears to play a role in spatial working memory, is deficient in rats reared in an impoverished condition (Melendez, Gregory, Bardo, & Kalivas, 2004).

One current concern is whether too much enrichment may be overstimulating and result in detrimental effects. While few investigations have examined the effects of a too rich environment, a recent study conducted by Lakin & Farmer-Dougan (2007) examined learning and motivation differences in rats reared in highly enriched, modestly enriched, and impoverished environments. Significant differences were found between three housing groups of rats. Rats in the highly enriched condition learned faster and grew faster than rats reared in the other two housing condition. However, these same rats showed *lower* sensitivity to changes in reward. That is, they were unable to effectively shift their behaviour as the reward ratio changed. They also showed lower concentrations of dopamine (DA) than solitary reared rats, indicative of less brain reactivity and motivation to the learning task. Dopamine is a neurotransmitter that is highly critical in the identification and response to reinforcement. Other researchers report similar findings. Bowling, Rowlett, and Bardo (1993) found that rats reared in an enriched condition showed lower baseline locomotion but greater locomotion in response to amphetamine than impoverish reared rats. Rats in the enriched condition also showed greater DA synthesis in the striatum in response to amphetamine in vivo but lower DA tissue concentrations than the impoverished rats. Finally, van der Harst, Baars, and Spruijt (2003) found that rats reared in enriched environments showed a weaker anticipatory response for sucrose reward when compared to a standard housed rat.

These results parallel investigations into enriched versus impoverished environments in humans. Early research with children with mental retardation (Balla, Butterfield, & Zigler, 1974; Butterfield & Zigler, 1970; Zigler, Balla, & Butterfield, 1968; Zigler, Butterfield, & Capobianco, 1970) found that children with Down syndrome who were institutionalized and came from a high-SES environment showed decreases in IQ and academic performance after 1 year of institutionalization. In contrast, children with Down syndrome who were institutionalized and came from low-SES environment showed increases in IQ and academic performance.

Why might over-enriched environments have these effects? One hypothesis is that animals reared in complex and enriched environments have lower basal DA concentrations and may metabolize DA faster Lakin & Farmer-Dougan (2007; van der Harst et al., 2003). Individuals raised in highly enriched environments may learn to "expect" high-value rewards and, when engaged in learning tasks that are not as stimulating, may not react with the same motivation as individuals reared in less enriched to impoverished environments. Individuals reared in highly enriched environments apparently show less attention and sensitivity to tasks that take place in environments that are less enriched than their home environment. This suggests that moderation of enrichment may be a key for early childhood. Opportunities such as reading to a child, exercise, musical training, free play, and even video game play appear very beneficial to the learning environment of young children. Learning settings should certainly promote these types of activities. However, overstimulation and over-enrichment may affect a child's motivation for less enriched settings such as a classroom.

While many different experiences contribute to the development and strengthening of neural circuits, one must again be skeptical of claims of products that are aggressively marketed and attempt to benefit from the glow of advances in neuroscience research. One example of a product that merits such skepticism is Brain Gym®. Presumably developed based on neuroscience research and particularly with respect to issues related to brain lateralization, Brain Gym® attempts to rebalance and integrate the hemispheres of the brain, makes claims of extraordinary gains in academic and sport performance, and has an evidence base that relies heavily on testimonials and on articles that have not been peer-reviewed (Spaulding, Mostert, & Beam, 2010). They report that they were unable to find any empirical studies based on sound methodology that support the use of Brain Gym®. There is no evidence that Brain Gym® has any benefit beyond that of normal play (Tokuhama-Espinosa, 2010).

Summary

Spaulding et al. (2010) echoes an important theme. It is not enough that products, instructional practices, or hypothesis about learning purport to be based on neuroscience or claim to be brain based. Such claims may overreach, be an overinterpretation of existing data, or, in some cases, have no link whatsoever to neuroscience research or be debunked by that research. A sound background in neuroscience may be helpful in evaluating extraordinary claims and battling the illusion of credibility that proponents attempt to gain by labelling something as brain based. While advances in neuroscience are clearly both exciting and impressive, evidence of significant improvements in educational practices based on these developments is not yet evident, and claims to the contrary should be examined with skepticism. The best evidence that an educational practice works is empirical evidence based on sound methodology of significant educational gains.

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