Modifiability and mediated learning in the light of neuroscientific evidence of ecological plasticity

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Abstract

This article explores the concept of ecological plasticity in relation to cognitive modifiability and mediated learning. Although direct neuroimaging evidence on the effect of Mediated Learning Experience is lacking up till now, there is some indirect evidence. The brain's development is not finished at birth, but cognitive development is contingent on the development of multiple brain networks. The brain is shaped by experience. The development of mind results from constant building of new synaptic connections as a result of learning activities, generated by a complex puzzle of stimuli: interaction with people, inclusive environments (home, school, leisure,), intervention programmes, socio-emotional experience, and mediated learning experience. Current evidence is reviewed how "environmental enrichment", a well-studied phenomenon from animal studies, can be transferred to human beings. Ecological plasticity is a characteristic of brain development : ecology has "external " as well as "inner" factors, whereby "external" means the whole of the stimuli a person receives from the outside world (information, activities) triggering emotional and learning experiences, whereas by "inner" factors we mean conscious and unconscious experiences. We criticize reductionist and deterministic discourse in much of the literature on brain and behaviour connection. Ecological neuroplasticity thus can be regarded as the neurobiological basis of Structural Cognitive Modifiability theory, which was anticipated by Reuven Feuerstein 50 years ahead of his time. However, at present, no distinction on neuroimaging can yet be made between mere activation and human-mediated activation. The specific action of mediated learning on neurobiological reorganisation of plasticity remains as yet to be researched.

Keywords

cognitive modifiability, ecological neuroplasticity, developmental disability, mediated learning experience

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Introduction

Modifiability is defined as the capacity of the human being to become durably modified by elaborating stimuli, so as to become able to adapt to new and changing situations. Feuerstein's Structural Cognitive Modifiability (SCM) theory (Feuerstein ea., 1979) states that a human being's cognitive development is generated by mediated learning experiences (MLE), gradually accumulated during life by cultural transmission from parents, teachers or other significant people in a child's life. Feuerstein postulated that all human individuals are modifiable, despite brain or genetic damage, or other causes which impair functioning. This postulate is based on extensive clinical data with a variety of individuals with various kinds and aetiologies of neurological impairments (Feuerstein, 2006). In this article we will explore the neurobiological basis of modifiability and mediated learning within a conceptual framework of the ecology of development, more specifically regarding the development of mind.

Donald Hebb (1949) anticipated that all learning must have structural consequences in brain organisation. Countless research studies about what has become known as neuronal plasticity, have confirmed Hebb's thesis. With the availability of neuroimaging technology at the end of the 20th century, knowledge about brain functioning and structural plasticity in human beings has been exponentially growing.

Examples of modifiability despite brain impairments

In our clinical work we have come across about 700 children and adults with some form of neurological impairment (brain injury, autism, genetically based syndromes, intellectual impairment, learning disabilities, etc.), who showed various degrees of modifiability, relative to the kind and degree of brain functioning impairment. It is important to distinguish *modifiability* from *normality*. There is no such as thing as a normal child, writes Roberta Garbo (2009; 18): "To recognize a new image of the norm, is a key to a cognitive restructuring of society, which is needed to adopt a mature vision to real inclusion"

Though all cases are remarkable, the following three examples are particularly illustrative of modifiability despite neurological impairments.

Peetjie Engels, now a young woman with Down syndrome, was in special education until the age of 9, where her educational programme was a low profile, with little attempts at teaching reading or writing, because in the eighties of the 20th century, it was considered either impossible or "not needed" to teach children with Down syndrome literacy or numeracy. At the age of 9 she switched to regular education. With the help of many people, in the first place her parents, the school team, sister, friends, and a thinking skills' programme (Feuerstein's Instrumental Enrichment), she reached a hitherto unknown level of cognitive development. She obtained a high school degree, a scooter's driver's licence and she lives an independent life. She describes her life as "a nice life" and stresses the need to work actively with children with Down syndrome from a young age and send them to regular schools (Engels, 2006). There are no indications that Peetjie is a mosaic trisomy 21, which is often advocated in the case of people with Down syndrome with unusual high degree of cognitive development. There are many other examples of people with Down syndrome who have attained high levels of cognitive development, which were unthinkable in the sixties. Although academic as well as general cognitive development is not always so spectacular, the case of Peetjie and others demonstrates that modifiability is a reality and a possibility. Interestingly, she was not an exceptional case "in the beginning". We are only beginning to understand the complex processes which lead to higher cognitive development.

Another example is Michael, who was a normally developing child until he was operated for a brain tumour (microcellular astrocytoma) at 4 years of age and a second time at the age of 8. While epilepsy and behaviour disturbance greatly improved after brain surgery, he remained with a huge frontotemporal brain damage (figure 1), leaving him with a spastic left hemiplegia, serious cognitive difficulties, wheelchair-bound and reading and writing difficulties. Thanks to committed parents, therapists and teachers, he relearned to walk again, to read and to write and finished middle school with reasonable success, having delays in certain topics such as math, language and sciences. At the age of 15, he still had substantial deficits in verbal as well as performance components of intelligence, in abstract thinking and problem resolution, with a WISC-R score of 49. Contrary to the advice to go to a school for manual executive labour, his parents and therapist were not ready to accept these low expectations. An evaluation with Feuerstein's LPAD battery (Lebeer, 2005) gave a different perspective and indicated a high capacity for ab-

stract thinking, no inherent memory defect, but a deficit in memorization and retrieval for lack of strategies, which could be resolved by teaching him better ways to organize. With the help of an intensive school- and family-oriented coaching program, inclusive education, cooperation with teachers, individual coaching by a rehabilitation team and a cognitive education program, he finished a scientific high school with success and was able to study at university. His IQ had risen to 95 in 4 years' time.

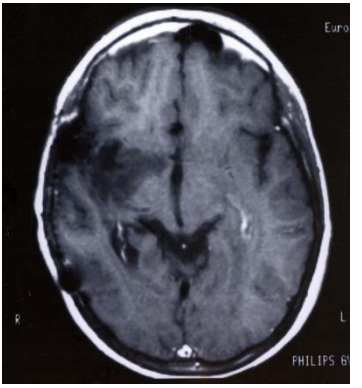

Figure 1. Michael, post-operative MRI showing extensive right frontotemporal cortical damage

A third example is Alex, a boy who was born with Sturge-Weber disease and unilateral multiple epileptic foci. Until the age of 9 years, he did not develop any speech, his language understanding remained at a low level; he was also behaviourally disturbed and functioned on a low cognitive level in the IQ range of 40-50. Because no combination of drug therapy was able to stop his epileptic spells, he was operated with a hemispherectomy at the age of 9 years. Despite a bad prognosis regarding speech development, because the assumed critical period of language development had been passed, he started to speak after the brain surgery (Vargha-Khadem e.a., 1997). His mother was intensively involved in his rehabilitation process. Though becoming quite fluent in speaking, he remained with a right sided hemiplegia. He had some difficulties in walking and fine motor coordination as well as serious learning and social difficulties. Despite attempts to teach him to read, write and to calculate, he was in a special school until the age of 16 making little

progress in academic skills. Alex became a resident pupil at the International Centre for the Enhancement of Learning Potential in Jerusalem (ICELP), where he was given more opportunities and challenges in academic and social learning. With intensive investment in mediated learning, described in detail by Sharma (2002), he made remarkable progress. He went to a regular school and he obtained a diploma in business administration in a vocational programme in a special school. At the age of 33, he is cheerful very social young man, managing his own life (with some assistance) and volunteers for the Red Cross, having been unable to find a paid job.

Several other examples of modifiability despite extreme brain lesions have been described earlier (Lebeer, 1998). Children with very extensive damage to the cortex recover fairly well or completely in some cases: e.g. in hydrocephalus (Smith & Sugar, 1984; Bigler, 1995); porencephalic cysts (Zhang & Sheng-Yu, 1984; Blackman, 1991) or absence of the small brain or Dandy-Walker syndrome (Maria e.a., 1987). Also in hemispherectomy, where an entire hemisphere is taken away, in 90 % of the cases epilepsy improves and sensorial and behavioural disturbances improve (Devlin e.a., 2003). Other authors have reported full recovery in adult hemispherectomized patients (Arnott, 1982; Damasio, 1975) as well as in children (Goodman & Whitaker, 1985). One of the most spectacular cases is a Hungarian 22-year old man, who was accidentally discovered to have extensive damage to his brain in both parts, which he must have had from birth. He had studied industrial engineering and had no symptoms at all (Leel-Ossy, 2006). Lorber described the case of a man with hydranencephaly (i.e. extreme hydrocephaly with absence of cortex), who was estimated to have only 5 % of brain cortex; yet this man studied mathematics on university level and was not aware of his brain damage until a scan was made at the age of 18 because of lasting headache (Lewin, 1980).

What might be a neurobiological basis of modifiability?

Brain plasticity after damage and in learning

The human body has an impressive healing capacity. Wounds close, blood clots, broken bones grow together again. A human being can survive with one quarter of a liver, with one quarter of only one of both kidneys, without spleen and only one lung. The whole continues to function in spite of dam-

age to its parts. Similarly the brain, its most complicated organ, has this capacity to adapt its structure and functioning, as a response to learning or to damage, although it does not seem to be as easy as restoration of other organs.

The brain never stops developing

At birth the brain is estimated to contain over a 100 billion neurones. Each neuron has hundreds of dendrites, receiving messages from other neurons, adjacent or far away, via the synapses. This gives innumerable connection possibilities. The construction of the brain during nine months of intrauterine growth is a very delicate process, in which many things can go wrong (. It is a miracle that the brain can function as a co-operative whole, starting off with only two cells (Eccles & Robinson, 1985).

The construction of the brain's hardware, is not terminated at birth. It continues to evolve into adulthood. There is much more than a simple growth in size, or in myelinisation. Synapses are continuously created and destroyed. Synaptic density peaks during the early years, until reaching a stable level at adult age (De Graaf-Peters & Hadders-Algra, 2006). There is programmed cell death, due to an excessive production during pregnancy, which continues after birth. The final network structure of the neuronal connections is established years after birth, not in the first place as an unfolding of a genetically determined structure, but it is *caused by experience* (Aoki & Siekevitz, 1988; Johnston, 2009). Activity drives synaptic plasticity and brain development. Functions that are not activated (motor, language, sensory, social, cognitive) will not lead to the creation of the proper brain structures. Neuronal group selection theory (Edelman, 1989) states that the brain is "shaped" through the process of developmental and experiential selection among the infinite connection possibilities.

This has enormous consequences for early intervention (Hadders-Algra, 2000). These relatively new findings support Feuerstein's mediated learning theory, which holds that experiences, mediated through human intervention, with a specific quality of interaction, are responsible for the creation of higher cognitive functions.

Natural healing system

As a reaction to damage, nerve axons grow out to make new connections. This phenomenon is called axonal sprouting and synaptic plasticity. The number of dendrites increases, as does the number of synapses (Klintsova & Greenough, 1999). It has long been believed that the number of neurones does not increase after birth, but many studies have shown that new neurones are formed after damage (Rakic, 2002). In human beings there is also evidence of synaptic reformation through dendrites: hemiplegic children have been found to develop compensatory non-crossing neuronal pathways on the affected side, provided they are actively stimulated (Farmer e.a., 1991; Carr, 1996).

There are also neuro-chemical changes. Denervation hypersensitivity, a phenomenon indicating that the post-synaptic plate becomes more sensitive to the stimuli of the neurotransmitters, is an example. Neurotransmitter production is increased (Marshall, 1985). Trophins, substances promoting nerve and axon growth, acutely regulate synaptic plasticity. Activity-driven experience activates specific gene promoters (BDNF –brain derived neurotrophic factor), leading to enhanced transcription, elevated trophin levels, postsynaptic receptor activation and increased synaptic transmission, constituting an epigenetic mechanism of gene expression (Black, 1999; Alder e.a., 2002). This might provide a major explanation of the mechanism of cognitive development, illustrated in the case examples above.

The same phenomena happen in the brain not only after damage, but also in learning. Each time we learn something new, the brain undergoes a structural change in its synapses. The synapses of the hippocampus react to learning situations with an increased long-term potentiating (LTP) effect: the synaptic plate, which receives the message, becomes more sensitive to chemical substances (Teyler & Fountain, 1987; Morris, Kandel & Squire, 1988; Squire, 2004).

Plasticity and activity: principles of experience-dependent neuroplasticity

There is overwhelming evidence, from animal studies as well as in human beings, that the development of the young brain as well as the reorganization of the brain, after damage or just in natural learning circumstances, is contingent on experience. This is called experience-dependent neural plasticity (Van Praag e.a., 2000).

Epigenetic influences

It is clear that the construction of the brain is influenced by genetic programming. But this is not enough. There are many instances where the natural capacity of neuronal networks to react and adapt are interrupted by a genetic defect. Rett syndrome is such an example, where it is believed that the gene MeCP2 hampers plasticity of early migration (Shabazian & Zhogbi, 2002). Similarly, in Fragile X syndrome – which is the most frequent cause of intellectual impairment in boys after Down syndrome, it has been shown that something is wrong in the "fingers" at the end of the synapses, which causes difficulties in the construction of the neuronal network. The neurochemical mechanisms, which lead the "stuttering" fragile-X gene to transcribe into a disturbed protein formation, are fairly well known. However, brains of animals with a Fragile-X-simulating condition, which are raised in active, enriched conditions, are more plastic (Ramakers, 2002). A similar mechanism applies in Huntington's disease (Nithianantharajah e.a., 2008). There is now evidence that not only genes drive the brain construction, but that activity and experience drive the gene expression mechanism. It looks as if Lamarck's first idea, that life experience may have genetic effects, is being rehabilitated.

Effect of age

Y*oung children* absorb rapidly enormous quantities of information and their capacity seems much higher than in adults: learning a second language, a musical instrument, reading and writing, skating and cycling all seem to work better. Children with frontal lesions, which in adults would certainly lead to

devastating symptoms, may have good outcome (Lebeer, 1998). Young children in whom the visual or acoustic areas of the brain are damaged recover quite well in most cases (Hécaen e.a., 1984). Hearing-impaired children who had an early cochlear implant, before the age of 1 year, have almost normal language development. When it's done later, there is a need for a much more intensive speech therapy, which is less successful (Geersa e.a., 2013). Children with hemispherectomy recover generally better than adults (Austin, 1955).

The plasticity of the brain to reorganise and take over functions from neighbouring areas seems to be better as long as the brain area is not too specialised. The immature brain compensates for neuronal injury better than the adult brain. However, cellular plasticity and nerve cell regeneration have been shown to exist in adult animals as well. Plasticity occurs over an entire lifetime (Kaplan, 1988).

Location and size

The degree of damage and subsequent recovery also depends on the location of the lesion. Small, scattered & asymmetric lesions are worse than symmetric lesions (such as compression in hydrocephalus or Dandy-Walker syndrome), but large unilateral lesions such as a porencephalic cyst may compensate well. Lesions in the brain cortex are better tolerated than deeper lying brain lesions. Whether there is a left- or right sided lesion also makes a difference: children where the left hemisphere had been removed had a normal language development (Smith & Sugar, 1975), apart from some syntax difficulties, and difficulties with understanding metaphoric language and reading. Children with right hemispherectomy had no problem at all. One of them obtained a university degree. This is also confirmed by a recent study in individuals with Sturge-Weber disease: those with small lesions as well as those with very large lesions have good cognitive outcomes, whereas those with intermediate lesions have worse cognitive development, which is suggestive of a mechanism of inhibition (Behen e.a., 2011)

Activity

Activity is of key importance in driving plasticity. Animals growing up together in stimulating environment have a better recovery and more plasticity. Rosenzweig & Bennet (1996) examined the difference in learning behaviour in brain damaged rats that were raised in standard conditions - an isolated position in a cage with just giving subsistence care - and others that were raised in an "enriched" environment - animals in a large cage with ample opportunity to exercise with various devices. Rats educated in *environmental enrichment* were better learners than those in isolation. Under conditions of environmental enrichment, neurone survival time increased, cortical thickness, neurone size, the size of the synaptic contact areas, neuronal metabolism, the number of synapses and the number of dendritic spines.

Not only in animals but also in human beings this has now been demonstrated. Maguire e.a. (2000) found that brain memory centres of *London taxi drivers* were thicker when the drivers had more experience and memorized street maps, than in the young ones with less experience and using navigation technology. Similarly, pianists' temporal lobe areas (related to auditory memory) have been shown to enlarge proportionally to the amount of time practised (Bangert & Altenmüller, 2003). Visual experience has been shown to have a profound effect on the maturation of the visual system in mammals, including humans (Kaplan, 1988).

It has been shown that cognitive activation and physical activity may compensate for function loss and even delay the onset of neurodegenerative diseases such as Alzheimer Disease, Parkinson and Huntington's disease (Hannan, 2014).

Deprivation and brain development

The opposite of activation is deprivation. Deprivation of sensory and motor stimuli has a negative effect on neurological development. Nobel Prize winners Hubel & Wiesel (1963) found that cats raised in a cylinder where they were exposed to diagonal patterns, were able to detect only diagonal patterns , and not vertical or horizontal - at adult age. When they grow up with one eye covered, the visual cortex that receives the information from the uncovered eye grows thicker to compensate. This finding is the basis of amblyopia treatment of covering the stronger eye in squinting children. This phenome-

non is similar to the effect of compensatory muscle growth in paralysis: unused potential atrophies (atrophy by lack of use). Deprivation of environmental stimulation occurs when young animals are separated from their mother. Examples of extreme environmental deprivation of stimuli in human beings, with lasting deleterious effects on intellectual, social and communicative development and only partial recovery, are some of the notorious cases such Victor, the wild boy of Aveyron (Malson & Itard, 1972), Genie (Skuse, 1984) and in the hospitalism and deprivation studies (Gunnar, 2001). On the other hand, the same stories also demonstrate the enormous plasticity despite early and long-lasting deprivation, when an intensive activation programme is set up (McVicker Hunt, 1986). In the semi-documentary "The Apple", Persian director Samira Makmalbaf relates the story of the Nadeeri twins, who had been locked up behind bars by their parents for fear of being abused. They could not speak, barely walked and were seriously cognitively retarded. Remarkably, by playing their own history in the movie, the twins started to talk and develop socially and cognitively.

Apparently there is a lot of plasticity possible even after the critical language period is over. Or perhaps the concept of "critical period" should be flexibilised. In summary: the brain grows by use, and it atrophies by deprivation. Use it or lose it.

Kleim & Jones (2008) summarize 10 principles of experience-dependent plasticity:

Table 1.
Principles of experience-dependent activity (after Kleim & Jones, 2008)

1. Use It or Lose It	Failure to drive specific brain functions can lead to functional degradation
2. Use It and Improve It	Training that drives a specific brain function can lead to an enhancement of that function
3. Specificity	The nature of the training experience dictates the nature of the plasticity
4. Repetition Matters	Induction of plasticity requires sufficient repetition
5. Intensity Matters	Induction of plasticity requires sufficient training intensity
6. Time Matters	Different forms of plasticity occur at different times during training.
7. Salience Matters	Training experience must be sufficiently salient/relevant
8. Age Matters	Training-induced plasticity easier in younger brains
9. Transference	Plasticity in response to one training experience can enhance the acquisition of similar behaviours, but it is not self-evident
10. Interference	Plasticity in response to one experience can interfere with the acquisition of other behaviours

It is not clear what kind of experience is needed to trigger brain development. Surely it is more complicated than just practice. What is meant in Kleim's summary of salient experience, time, intensity, repetition, and transference, can be found in Feuerstein's concept of "mediated learning experience". Feuerstein added a human element to the concept of "environmental enrichment" known from animal studies. This also includes a component of motivational-emotional activation, which we will discuss more in detail below.

Has every function its place in the brain?

Since Broca started to localize speech function in a specific temporal area of the brain, the brain has been mapped into detail. L*ocalisation theory* (Pribram, 1971) attributes specific functions to specific brain regions: Knowledge of this detailed localisation has been inferred from observations in patients who lack certain functions and appear to have defects in certain parts of the brain. With increasing resolution of the modern scanners, ever more defects are located. It is the basis of neuropsychology.

Pribram seriously criticizes the localization theory, however. In some cases there seems to be ample flexibility of localisation. Children's brain regions are not yet fully specialized and can change places. Memory seems to be distributed to large parts of the brain.

People with the occipital part of the brain lacking can learn to "see" with their parietal parts. Early –blind children learn to "read" Braille with their visual occipital cortex (Chen e.a., 2002). This means that the visual cortex which is specialized in visual recognition, can now decode sensory touch messages and translate them in a People with the occipital part lacking can learn to "see" with their parietal parts.

The speech centre which is usually on the left side, can go to the right side, but does so only after training, as is shown by PET scans of aphasic patients (Musso e.a. 1999). We described a girl with a left temporal porencephalic brain cyst with right-sided hemiplegia and normal speech). Despite serious cognitive problems (IQ 71), she was included in a mainstream school with an individualized programme and intensive parental support (Lebeer, 1998).

Cells of the temporal cortex, which normally "code" for auditory processing, can be trained to behave like visual cortical cells in recognizing patterns of orientation, when the visual cortex is damaged. In this experiment the brain had been rewired from the visual pathway to the auditory cortex, which led to specialisation of auditory cortex cells to recognize visual stimuli (Roe e.a. 1991; Merzenich, 2000). If it is possible to rewire the brain in experimental conditions, it can be imagined that similar mechanisms happen in natural situations.

Metaphors of brain functioning

It now becomes clear that the brain is a living system, constantly moving and recreating connections, with a capacity to learn and to react to damage with repair, subject to influences from outside and inside.

The compatibility of very large cortex lesions with a quasi-normal life, raises the fundamental question of *how much brain a mind needs* to function (Lebeer, 1998)? Apparently only 5%, which is far less than a chimpanzee. Certainly brain size is not related to intelligence, as is also demonstrated by Chugani's research on people with Sturge-Weber disease (Behen, 2011). But that raises another question: where does the brain store all this information, if 5% of cortex is left?

This calls for a different conceptualisation than the one commonly used as the brain as a sophisticated computer. A computer is prewired. Damage to its hardware is final; it is not self-repairing. Although sophisticated computers many times can perform operations much better than a man's brain, no artificial intelligence computer approaches the enormous capacity of plasticity of a human brain.

Looking at in vivo images of synapse formation in case of plasticity, one gets more the impression of the brain as a megalopolis: this is also a large complex whole consisting of many sub-entities, it is constantly moving, adapting, looking for better solutions and organization. It is constantly in evolution, with breakdown and rebuilding. It has a capacity to adapt to threats and damage and has lots of compensatory mechanisms. Yet, who is the mayor?

An ants' heap is also a better image, because ants' heaps are also capable of restoring damage, of communication via chemical substances, and a degree of self-reorganisation.

The brain is a very complex open system and as such has the properties of a "dissipative system" as described by Nobel Prize winner Ilya Prigogine (1984). Dissipative systems are in open connection with the world around (they absorb and give back energy). They have a capacity of self-organisation, of creating order out of chaos. According to Prigogine, open complex systems behave in an indeterministic way: one cannot predict whether there will be order or chaos. The brain apparently acts in a similar way.

Yet this does not answer the question where the information is stored when 90-95% of the cortex is absent, as in hydranencephaly. The old explana-

tion of *redundancy* (i.e. the idea that 90% of the brain is not used in normal conditions and constitutes a reserve) has been refuted since we know that after damage every brain region is immediately reused. The only plausible explanation can be found in Pribram's (1971) hypothesis of holographic storage, i.e. the brain is capable to store information in distributed wave interference patterns analogous to a holographic plate, which has the property to store information in a point-to-whole correspondence. That would mean that the memory storage in the brain is distributed (as Karl Lashley found) over the whole brain, and not contained in nerve cells (Speed e.a., 2010).

Metaphors of brain functioning have always existed and usually are based on current technological knowledge. Descartes e.g. had a mechanical metaphor of brain functioning: his theory proposed a complete separation of the brain as the material apparatus and the immaterial soul, known as the Cartesian worldview. Nowadays the most common metaphor of the brain is the computer. Metaphors, as cultural concepts, actually have an influence on creating culture as well. If one holds a computer-model of the brain, there is a tendency to view functioning still in a rather fixed way. Teachers, parents and health professionals, based on an unconscious metaphor of a "computer brain" will tend to think that "what is not there cannot be stimulated", when confronted with pupils with a damaged brain.

External and internal aspects in ecological plasticity

Analysis of remarkable developments, such as in our research and other published cases (Keller, 1903; Grandin, 1996, Feuerstein e.a. 1988), reveals a common pattern in their ecology. By "ecology" we mean the study of the relations between human beings and their environment (physical, psychological, social, information, etc.). A distinction can be made between external and internal aspects: e*xternal aspects* are *what* one does (stimuli, activities), *internal aspects* deal with *how* something is done on a level of experience (the energy level, e.g. enthusiasm, belief system). Our research has shown that the quality and quantity of active experiences is the most decisive in influencing outcome of children with neurological impairments *across* a variety of methods (Lebeer & Rijke, 2003). Children need to be actively stimulated, to be exposed to a rich variety of stimuli, to be mediated necessary steps leading to new functions by facilitating learning processes, to be provided new chal-

lenges and not stay in a status quo. There are many ways. The painter is more important than the paint he uses, in other words: it is *how* one interacts with the child which is the most important.

The way adults make intelligent use of their environment – in other words their ecology - substantially influences a child's intellectual, social and physical development. A simple decision e.g. to involve the child in its social environment rather than isolate it – which sadly is still often the case - to send it to a regular school, to go shopping, to play with animals, to visit places, etc. is a boost in experiences, leading to motivation, language, movement and social functions.

The environment *creates needs*, and it is the task of educators to take care that the child is sufficiently activated to give an answer to these needs. Peetjie's example mentioned above illustrates this: when she went to secondary school, the need rose to go by bicycle, as all children in the Netherlands do (the bicycle is part of the Dutch ecology). Her father (and Peetjie's luck to have such a father) taught her to ride a bike. He said it took him months to teach his daughter to cycle in traffic, to know where to stop, to look to the left and right to cross a street; to estimate the distances of the cars, to solve the complex traffic situations. That mediation lasted 3 months until Peetjie's sister urged him to let go, because she was convinced she could do it alone. We saw this kind of processes in many other children. Cycling is a motor as well as a cognitive process, with social learning components, because the need was generated by being in a normal school. Children attending a special school hardly ever get this experience, because they are taken by a special bus. A function only is generated when the opportunity in the environment is offered. Human caretakers take care that the child grasps the opportunities and accompany the learning processes. One needs to give the child sufficient learning opportunities. Children with special needs, whether they are in special schools or regular, often do not get sufficient opportunities to learn to read or write. First of all, it requires an environment where books are amply available, and where the need arises to learn to read; then sufficient mediation, a belief that they can learn it and the availability of a mediator.

Feuerstein's concept of an *Active Modifying Environment* (1988) describes four aspects which contribute to modifiability: (1) participation in everyday life and be exposed to a variety of experiences; (2) creating positive stress by causing disequilibrium; do not overprotect the child or keep it away from enriching experiences (3) offer challenging activities which provoke problem

solving and thinking and (4) offering adequate and adapted mediation. Placing an individual in a stimulating environment therefore is not enough. There is a difference between stimulation and mediation. One can overload children with stimuli, coming from television, electronic games, toys, voices, etc. This is not beneficial, because an overstimulated brain will also overreact, with e.g. ADHD or behavioural disturbance. The environment should be mediated, i.e. that the *stimuli should be adapted, selected and filtered* in such a way that the child is able to benefit from it in a structural, lasting way. How this can be done is explained elsewhere (Feuerstein, Klein & Tannenbaum, 1991). An example of language learning will make this clearer: one can live as a stranger in a foreign- language rich environment, yet not benefit from it for lack of an interpreter. This is the situation of children with Down syndrome in their own language environment, because they have an auditory perception and interpretation problem (Buckley, 2000). People in their environment should learn to speak clearly, slowly, in short and simple messages and with emphasis, so as to make sure that the child picks the message up and understands it. This can already make quite a difference in speeding up speech development. Visual support media such as using augmentative communication and later printed word might greatly enhance speech development.

Similarly, placing a child in a mainstream school (inclusive education) is a more enriching environment than a special school, but it is not necessarily more advantageous to the child, when no extra mediation is being offered by the people around the child, be it peers or adults. Only when inclusive education is accompanied by enhanced mediation, the child may really benefit (Lebeer, 2006).

Internal aspects are a decisive part of the human ecology and greatly influence modifiability. Inner experiences can be consciously aware or not. Feelings, emotions, awareness, motivation, will, experience of energy, vitality, drive, unity, love (or the opposites) operate primarily on an inner level (Rijke, 1993). They are hardly visible to inexperienced observers, but one can learn to observe them. Verbal declarations are unreliable. They are certainly inaccessible to questionnaires or superficial interviews asking opinions. Yet everyone knows that they are key to the more visible aspects of what one does. For example: it makes quite a difference to listen to a piece of music played by a skilled musician who plays "with heart and soul" or one who plays technically perfect but mechanically. Similarly, it makes quite a difference to work with a child who has motivation and energy, than to work with a

resistant child which does not really want. Or e.g. to work with a mediator using a programme such as Feuerstein's Instrumental Enrichment with or without "energy". The outcome will greatly depend on these inner factors, which are related to choice. On an inner level, even a small child chooses or not to engage in the interaction. In this way one can understand that sometimes children with autism may show high structural modifiability, while others are very difficult to modify. This is beautifully described by Temple Grandin, herself an individual with autism (Grandin, 1993).

These inner aspects render classic quantitative intervention studies highly problematic, because inner aspects are difficult to observe, to control and to manipulate and therefore are often dismissed as "placebo" effects. Yet they make all the difference between otherwise equal situations.

How the mind is built

The dominant paradigm in much of the scientific literature, as well as in medical-clinical practice and in school environments, still sees cognitive development as a property of the individual, given a more or less fixed capacity at birth, determined by genetic constitution, measurable by developmental tests and later intelligence tests (I.Q.). It is a basically linear-causal, maturational and mechanical model. However, more and more evidence becomes available, that the mind is not confined to the individual brain, but must be regarded in an ecological way. The idea of the "extended mind" has been forwarded by philosophers Andy Clark and David Chalmers (2010). It might help explaining why individuals with the same genetic damage such as trisomy 21 may display a wide variety in development. Mind is something what we do (Noë, 2009). The intellectual capacity of an individual is *not* a priori determined by genes or brain condition.

Ceci e.a. (1986) observed that some people, who otherwise carried a label of "mentally retarded" where capable of inventing highly complex solutions to predict better than others which horse was going to win the race at the horse race tracks. In other situations, however, such as in IQ tests, or in situations they did not have adequate experience, they were not nearly as efficient. The situation of complexity in their ecology, together with high motivation, helped them to develop complex operations. Ceci called this the con-

cept of *cognitive complexity*. Intellectual capacities to deal with cognitive complexity develop in circumstances of cognitive complexity.

The idea that the mind of a child, whether brain impaired or not, grows within the ecology in which the child lives, is compatible with the "embodied mind" theory (Varela & Maturana, 2000). Feuerstein's mediated learning experience theory added a specifically human component, in which the specific interaction between a mediator, the child, culture and the world, shapes the mind. Language acquisition plays herein a key role.

Affection triggers cognition

MLE theory however also has a strong *affective* component. The affective component is probably more important than anything in enabling brain functioning towards a development of higher cognitive functions (Cromwell & Panksepp, 2010). Typical babies intensely request communication with their mother. Trevarthen (1990) hypothesizes that this primary intersubjectivity is a condition for triggering gene expression and adequate neural development. In extremely deprived children absence of primary intersubjectivity hampers brain impairment. In children with congenital impairments, this affective intersubjectivity may be interrupted. When the baby does not smile back at the expected time, mothers may not exhibit the same patience to elicit a joyful mediational interaction (Mintzker, 1991). In Feuerstein's view this is also modifiable at any age. He stressed the importance of working with parents to restore the mediation process.

Mirror neurons and autism

Recently there has been a lot of popular attention to the role of the Mirror Neuron System (MNS) and its possible role in the explanation of autistic spectrum disorder. The MNS was discovered by co-incidence by Rizolatti et al (1996) as a part of the inferior frontal and temporal zone. The mirror neurons fire not only when someone is doing a particular action but also when that person is watching somebody doing the same action. The MNS has to do, not directly with imitation but with the understanding of actions, as a kind of automatic re-enactment, and with the interpretation of the intention of other people. It has been hypothesized - documented by fMRI and other

neuroimaging studies - that the MNS is involved in social cognition, learning through imitation, mind reading and empathy, which are all hampered in individuals with autistic spectrum disorder (Hadjikhani, 2007). However, the hypothesis is still controversial; some authors have found no impediment of action understanding in individuals with ASD and claim that multiple brain systems are involved in ASD (de Hamilton e.a., 2007). There is a wide-spread tendency of reductionist thinking in neurosciences, i.e. to reduce the complex functioning of a human being to its neurobiological functioning, and reversely, to think in simple causal interactions, i.e. to infer that a perceived neurobiological phenomenon is the cause of an associated function. There is yet little research about the plasticity of the MNS in relation to ASD. If the brain neurocircuitry is plastic as a result of ecological influences and experience, there is no reason to discard the idea that experience (positive or negative) may not influence the networking connections of the MNS. Autism is a multifactorial condition and may have multiple causes. There is preliminary evidence that intervention oriented at the mediation of understanding empathy, understanding emotions and the regulation of executive functions, when done in an ecological way has a positive effect on the modifiability of children with ASD (Kozulin e.a., 2010).

A central hypothesis: modifiability, plasticity and ecology

In conclusion, neurosciences offer ample evidence for Feuerstein's MLE and SCM theory. Although direct neuroimaging evidence on the specific neurobiological correlate of Mediated Learning Experience is lacking up till now, there is some indirect evidence: brain plasticity allows children to substantially become modified and adapt, to develop their cognitive, socio-emotional, motor, language and academic competences, despite genetic, brain or autistic conditions. Neuronal plasticity is ecological and experience-dependent. Development is the result of a complex puzzle of "ecological" influences. In those ecological influences, "outer" as well as "inner" factors are of fundamental importance. The outer factors are those that are readily visible: they are formed by a combination of interventions, environments, constituted by what people actually do; the intensity of learning, the opportunities for learning, practice time to learn some activities, etc. The inner factors

are more intangible, in the sense of attitudes, the "energy" between people, what people actually experience inside, what really drives them, the "spark" of how they mediate. The concept of MLE is sufficiently broad to encompass all these aspects.

However, some caution has to be expressed. First of all, plasticity may have adverse outcomes. Stimulation leading to synaptic connection expansion does not necessarily lead to positive functional outcomes. Second, mediated learning experience may seem a simple theory, but the complexity and unpredictability of a child's ecology make it difficult to reproduce. On the one hand unpredictability – as an inherent property of complex systems – gives sufficient theoretical grounding to Feuerstein's idea that development cannot be predicted on the basis of static tests (IQ tests, developmental tests, learning tests, brain examinations or other) and it justifies his vehement criticism of contemporary test practice. On the other hand, it makes intervention outcomes also unpredictable. This is the reason why comparative effect studies are bound to remain controversial. Almost any activation method, whether it is a specific cognitive activation programme like FIE, or another one, when it is done with mediation, might have a positive effect. Nevertheless current neuroscientific evidence has proven that Feuerstein with his bold hypothesis of modifiability and mediated learning was 50 years ahead of his time.

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