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Delayed Exposure to Second Language Acquisition: The Robustness of Critical Period Hypothesis and the Limitations of the Plasticity Theory

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***Abstract*—Studies of language acquisition have shown that language learning ability declines with age. It has been documented that the overwhelming majority of learners who begin the process of second language (L2) learning after passing the critical period — after a certain age – cannot attain native-like competence (underlying knowledge of language) at the end state. There is a universal folk belief, shared by almost all psycholinguists, that at an early age, the brain is plastic; it can modify its own structure, organization and function as a direct consequence of experiences and learning. According to this belief, the plasticity of the brain fades with age and with the increasing specialisation of the different hemispheres and areas of the brain. However, recently neuroscientists, equipped with the complementary aid of brain neuroimaging, have discovered that mature brain is far from being fixed as previously thought; it has the ability to continuously adapt its structure and function based on internal and external environmental changes and/or input such as experience, ageing, illness, injury and learning, meaning that the brain remains plastic throughout life. The purpose of this article, therefore, is to illustrate that the theory about the loss of brain plasticity during natural maturation is not valid in explaining the question why L2 learning is affected by delayed exposure to a language? It should be mentioned here that until now, the overwhelming majority of L2 researchers tend to think of plasticity as a phenomenon confined to early development; and therefore, evidence supporting plasticity is generally ignored.**

***Index Terms*—Second language acquisition, innateness hypothesis, incompleteness in second language acquisition, critical period hypothesis, plasticity theory**

I. Introduction: The Innateness Hypothesis and Critical Period Hypothesis

There is a universal folk belief, shared by many linguists, including Chomsky (1957, 1965, 1981); Lenneberg (1967); Pinker (1994); O'Grady (1997); Lightfoot (1999); Anderson and Lightfoot (2002); Fitch et al. (2005), that children come to the task of language acquisition with prior linguistic knowledge as part of a genetic endowment. This knowledge guides them in acquiring their native language(s) by placing limitations on grammar, constraining their forms as well as how they operate. This argument for some sort of biological basis to first language acquisition (L1A), referred to as the Innateness Hypothesis, is empirically supported by the observations that all normal children, from different cultures learning different languages, invariably acquire successfully a remarkably complex grammatical system with relative ease and without the benefit of instruction, and do so at roughly the same pace, following roughly the same developmental process, despite the fact that the speech input they are exposed to is very often not perfect, i.e., containing false starts, unfinished sentences.[1] For a more detailed discussion see, among many others, Thomas (2002); Sampson (2002); Lasnik and Uriagereka (2002); Scholz and Pullum (2002); Fodor and Crowther (2002); Schwartz and Sprouse (2013), and the references cited above.

However, many researchers (i.e., Birdsong, 1999; Birdsong & Molis, 2001; DeKeyser, 2000) argue that there is a limited developmental period when a child can acquire a language to normal, native-like levels. This view that a critical period characterizes language acquisition is referred to in the literature as the critical period hypothesis (CPH). This hypothesis states that the ability to acquire a language with native-like competence is related to the initial age of exposure.[2] If the process of language learning begins after a certain age, the child will never acquire the language

[1] The fact that native speakers' speech is not always perfectly grammatical led Chomsky (1965) to distinguish between competence and performance. Competence is ''the speaker/hearer's knowledge of his language'', whereas performance is ''the actual use of language in concrete situation'' (Chomsky, 1965, p. 4).

[2] It should be mentioned at this point that there is disagreement among researchers about the exact time when the critical period for language acquisition ends. The reported claims ranging from near birth (at approximately two years of age) to adolescence (around 13 years of age). For a detailed discussion, refer to Bialystok and Miller (1999); Birdsong (2004); Birdsong and Molis (2001); DeKeyser (2000); Hyltenstam and Abrahamsson (2003); Johnson and Newport (1989); or for a review of the literature that addressed this issue, see Singleton (2005). This disagreement among researchers may be explained by the idea that there may be multiple critical periods, related to different aspects of language learning. For

that he or she is exposed to natively; namely, he or she will never acquire the implicit and abstract knowledge of a language possessed by native speakers.

One piece of Evidence in support of the notion of a critical period for language acquisition initially came from cases of unfortunate young children who were raised in isolation.[3] One of the most famous instances of a child deprived of opportunities to hear and speak a language of their surroundings during childhood is the case of Genie, a young girl kept locked in a single room found in a Los Angeles suburb in 1970. Once freed from her captivity at the age of thirteen, linguists attempted to teach her English. However, even after extensive exposure to linguistic input, her subsequent language development was not normal; although she was quite successful in acquiring a large vocabulary, her syntax and morphosyntax never developed beyond a basic level - her production of sentences remained basically telegraphic (see Curtiss, 1977).[4] Genie's syntactic deficits suggest that the critical period for acquiring a native language 'holds for the acquisition of grammatical abilities, but not necessarily for all aspects of language' (Fromkin et al., 2013, p. 479). This finding suggests that grammatical ability appears to be more affected by delayed exposure to a language rather than semantic ability. For further discussion, see Birdson (1999); Granena and Long (2013a, 2013b); Hurford (1991); Smith (2004).

The same hypothesis, which assumes that language learning ability declines with age, predicts that learners who begin the process of L2 learning after passing the critical period cannot achieve native-speaker competence at the end state – the point at which learners' linguistic systems fossilise (cease to develop).[5] Studies of second language acquisition (SLA) lend empirical support to this observation that it is unlikely that adult L2 learners are capable of achieving completely native levels of proficiency (refer to, among many others, Coppieters, 1987; Singleton, 1989; Bley-Vroman, 1989; Schachter, 1989, 1990; Tsimpli & Roussou, 1991; Selinker, 1992; Ioup et al., 1994; Clahsen & Hong, 1995; Smith & Tsimpli, 1995; Hawkins & Chan, 1997; Long, 1997, 2003; Neeleman & Weerman, 1997; Han, 2004; Lardiere, 2007; Granena & Long, 2013a). It should be mentioned here that the critical period hypothesis does not stipulate that adult learners cannot reach very high levels of L2 proficiency; rather, it predicts that beyond a certain age, L2 acquisition becomes more difficult and is rarely entirely successful.

II. The Plasticity Theory

The discussion so far has shown that adult L2 learners, unlike children, often struggle to learn an L2, and even when they succeed, they do not achieve native-speaker competence at the end state. This is because, as most linguists take it for granted, that there is a period during which learners can acquire an L2 easily and implicitly and achieve native-speaker competence, but after which L2 acquisition becomes more difficult and is very rarely entirely successful. The preliminary question to be raised now, based on these documented empirical findings, is why is second language learning affected by late or delayed exposure to a language – maturation? In other words, what happens inside the brain at a certain age(s) that makes it to lose its innate ability that places limitations on L2 grammars, constraining their forms as well as how they operate? A plausible answer to these important questions comes from neurolinguistics – the science that investigates the neural mechanisms in the human brain that control comprehension, production and language acquisition. The CPH has been a theory in neurolinguistics that states that the brain is plastic at an early age, and it can modify its own structure, organisation and function as a direct consequence of experience and learning. Basic brain structure is established by our genes before birth; however, because of plasticity, the brain can develop by modifying its connections, changing its neurons and synaptic connections or even by rewiring itself. This process is known as neuroplasticity. As a result, the brain begins to specialise; specific brain functions become increasingly associated with certain areas of the brain. For example, certain regions of the left hemisphere (e.g., Broca's and Wernicke's areas) become specialised for some language functions (e.g., comprehension and production of speech). However, this adaptive capacity – the plasticity of the brain – is thought to fade with age and with the increasing specialisation of the different hemispheres and areas of the brain. See Bates et al. (1999); Bishop (1993); and Stiles et al. (2012) for reviews. For a detailed discussion, refer to Hart and Risley (1992); Huttenlocher et al. (1991); Lidzba and Staudt (2008); Sheehan and Mills (2008); Szaflarski et al. (2006).

An impressive empirical example that provides a unique window into the plasticity of language development comes from hemispherectomy – a procedure in which one hemisphere of the brain is surgically removed. For example, studies have shown that children who undergo a left hemispherectomy not only can reacquire language like that of normal children but also show many of the developmental patterns of normal language acquisition. This finding means that the right hemisphere, even though it is not innately specialised for language, can take over the injured areas' language functions in the removed left hemisphere. In other words, it can reorganise itself to perform the tasks that are innately

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instance, the ability to acquire native-like accent may end earlier than the ability to acquire grammar or vocabulary (cf. Ioup et al., 1994; Lardiere, 2007; Granena & Long, 2013a).

[3] Further evidence in support of the hypothesis came from Neurology and neurolinguistics. Lenneberg (1967) reviewed studies which show that when children underwent surgery to the left hemisphere, they rapidly recovered total language control, unlike adults who showed permanent language impairment after such operations. Lenneberg, therefore, concluded that the biological basis of language in children and adults is not the same.

[4] Telegraphic speech is the term used to refer to children's early two- or three-word utterances, where functional categories may be missing – strings of words (lexical morphemes) in phrases or sentences such as *shoe all wet*, *cat drink milk* and *daddy go bye-bye*.

[5] This end-state mental representation of language is also referred to in the literature as ultimate attainment, the steady-state, or the final-state.

performed by the left hemisphere. However, this dynamic developing ability of the brain's right hemisphere to take over the left hemisphere functions declines with age and fades by puberty. For example, such surgical removal of the left hemisphere in adults inevitably results in a complete loss of language function. (For detailed discussions about the plasticity of language development, see, among many others, Bates et al., 1999; Bishop, 1993; Demir et al., 2014, 2015; Elman et al., 1996; Huttenlocher, 2002; Lenneberg, 1967; Reilly et al., 2008; Rowe et al., 2009; Stiles et al., 2005, 2012; and Trauner et al., 2013).

Another example supporting the view that plasticity during childhood appears to be more pervasive than at other stages of life comes from studies of those unfortunate children with perinatal serious brain injury (Bates et al., 1997; Bates et al., 1999; Reilly et al., 2008; Stiles et al., 2012; Trauner et al., 2013). Such studies reveal that these children can acquire language even if the left hemisphere of the brain is seriously injured. Levine et al. (2016) surprisingly state that "children with unilateral perinatal lesions do not exhibit the marked aphasias that are common when anatomically comparable lesions are incurred during adulthood" (p. 969).[6] Similar findings were reported by Bates et al. (2001); Bishop (1993); Rowe et al. (2009).

III. Reflections and Critical Comments on the Plasticity Theory

It can be concluded from the discussion in the previous section that the brain is plastic only at an early age of development; once the structure of the brain develops during childhood, and, once organized in highly specialized critical zones (e.g., Broca's and Wernicke's areas), it became fixed and therefore it left no room for changes. Main evidence supports this claim comes from hemispherectomy; if focal injury occurs to the left hemisphere early in life and got surgically removed, the right hemisphere compensates by taking on the functions of the left hemisphere even though the right hemisphere is not innately predisposed to specialize for language. This ability of the right hemisphere to take over the functions of the left one is thought to decline with puberty. This obviously raises a crucial question: does the plasticity of the brain really fade with age – at or before puberty?[7] This question will be addressed in this section.

As the neurosciences have in general been descriptive, for many decades, this view of strict localization of function in the brain is held by almost all scientists. However, this viewpoint has recently begun to change; today neuroscientists know for sure that adult brain continuously adapts not only its function but also its structure. The various new brain imaging techniques (i.e., positron emission tomography, functional magnetic resonance imaging, magnetoencephalography, electroencephalography, transcranial magnetic stimulation – diffusion tensor imaging) reveal that mature brain remains constantly dynamic in response to internal and external environmental changes such as brain injury, hormones, illness, aging, learning and memory. For example, studies used these brain imaging and mapping techniques (i.e., Fernandez et al., 2004; Heiss et al., 1999; Karbe et al., 1998; Pedersen et al., 1995; Saur et al., 2006; Saur & Hartwigsen, 2012) show that mature brain can compensate for damage or lost function(s) by reorganising and forming new connections among intact neurons; if part of the brain is damaged due to a stroke for example, the healthy surrounding neurones– the intact ones – do take over some of the functions of the damaged area. Breier et al. (2009), Meinzer et al. (2004) and Fridriksson and Smith (2016) found that although the right hemisphere may support language recovery immediately after treatment, recruitment of perilesional regions is fundamental for prolonged, stable effects. All these studies support the view that there are structural and functional brain changes associated with recovery of language in patients with aphasia.[8] In fact, such changes in the brain structure not only can be mediated by changes in connectivity as new synapses form to join neurons to each other, but also can be mediated by the generation of new neurons. Until very recently, scientists believed that brain cells, once lost, could never be replaced; however, the recent discovery of stem cells in the brain has changed this belief. Stem cells are unspecialised cells grow into hundreds of particular types of neurons, each appropriate to its location and function. As they have the ability to become any of the specialized cell and as they are normally present across the lifespan, stem cells serve as a repair system for the brain, meaning that the damaged neurons can be replaced by new neurons generated within the brain itself (for more detail about stem cells cf. Bellin et al., 2012; Gonzalez-Perez, 2012; Nichols & Smith, 2012; Yamanaka, 2012; Nguyen & Cramer, 2016).[9] However, this is not a claim that the brain's ability to adapt is limitless. Normal plasticity cannot fully compensate for severe damage; so not everyone who has a stroke can recover full function. Indeed, the degree of recovery depends on many factors, including age (younger brains have a better chance of recovery), the brain area and the size of the area damaged, the rapidity of the damage and the treatments offered during rehabilitation. For a more

[6] Marked aphasia, also referred to as global aphasia, is severe impairment in all receptive and expressive language modalities.

[7] There is no clear consensus among researchers on about the exact time when child language acquisition ends and adult SLA starts. Some researchers claim that the boundary between them lies somewhere between the ages of seven and nine; some others argue for the age of eleven; and others claim that the boundary can be found at the age of fifteen (For a detailed discussion, refer to Bialystok & Miller, 1999; Birdsong, 2004; Birdsong & Molis, 2001; DeKeyser, 2000; Hyltenstam & Abrahamsson, 2003; Johnson & Newport, 1989).

[8] Readers interested in marvellous stories about stroke patients learning to speak are referred to Doidge (2007).

[9] It seems that stem cells offer great promise for new medical treatments. Stem cell-based therapies have been applied to treat a number of neurological conditions, including traumatic injuries to the brain or stroke, Parkinson's disease, and Alzheimer's disease. The outlook for this type of new medical treatment is promising despite the fact that this area of research is still in its infancy. For instance, Nguyen and Cramer (2016) suggest that ''stem cells may be found useful for improving language function. For example, several types of cell therapy appear promising in preclinical studies of diseases that affect language, such as stroke'' (p. 1087). For more detailed discussion about this type of treatment, see Brunt et al. (2012); De Feo et al. (2012); Heiss and Thiel (2006); Karussis et al. (2013).

detailed discussion about plasticity and structural and functional brain changes, refer to Allendorfer et al. (2012); Cramer (2008); Crosson et al. (2005, 2007, 2009); Epstein-Peterson et al. (2012); Fernandez et al. (2004); Fridriksson (2010); Heiss et al. (1999); Johansson (2000); Karbe et al. (1998); Kleim and Jones (2008); Kolb and Whishaw (1998); Leger et al. (2002); Marcotte et al. (2012); Menke et al. (2009); Musso et al. (1999); Ostry et al. (2010); Pedersen et al. (1995); Rijntjes and Weiller (2002); Thompson et al. (2010); Zatorre et al. (2012); Zipse et al. (2012).

Another example of neuroplasticity comes from studies that have shown that the brain never stops changing through learning. Throughout life, unless compromised by disease, whenever a person learns, behaves, stores memories and so on, the brain generates new connections between the neurons in response to stimuli, giving new neural networks a chance to flourish. This growth of new neural connections in brain and the emergence of new fibre tracts as well during adolescence and adulthood make the brain grow stronger, just as muscles expand and grow stronger with repeated exercise. Therefore, these developmental and neural connection mechanisms make the neural systems perform at a high level – specialized. This specialization creates a more complicated and therefore more sophisticated brain. This can explain why some areas of mental ability actually increase with age. For example, our linguistic abilities continue to develop into later life; with age we enjoys a larger vocabulary and sharpened syntactic skills (see Hayiou-Thomas et al., 2004; Huttenlocher et al., 2002; Leech et al., 2007; Nippold, 1998). In short, this ability that allows brain to establish new associations makes individual brain able to integrate new information and stores it until it is needed. For a detailed discussion about specialization and the emergence and formation of new fibre tracts during adolescence and adulthood refer to Brown et al. (2005); Holland et al. (2001); Levine et al. (2016); Perani et al. (2011) and Szaflarski et al. (2006).

IV. The Plasticity Theory and Second Language Acquisition: Concluding Remarks

The discussion in the previous section illustrated with some scientific evidence that the brain is not a computer that is wired forever during early childhood. Instead, studies have shown that this organ is far from being fixed as we tend to think of it; it is never resting throughout life. In fact, it continuously remodels and rewires itself throughout life and after injury to remain constantly dynamic - physically different from what it was in the preceding moment - in order to response to internal and external environmental changes such as brain injury, hormones, illness, aging, learning and memory. In fact, without the ability to be plastic, the brain would not be able to develop from infancy through to adulthood, recover from brain injury or absorb new information (e.g., learning a new skill). The new human brain mapping techniques have revealed that it is this plasticity that helps the normal brain quickly learn new things by making new connections among neurons and also helps the brains of patients with neurological and neuropsychiatric disorders to compensate for damage or lost function(s) by reorganising and forming new neurons and connections among intact neurons.

The discussion thus leads us to the conclusive conclusion that the theory about the loss of brain plasticity during natural maturation cannot explain why language learning is affected by delayed exposure to a language; it cannot explain why a second language can be complex for adults to learn and even when they succeed, native-likeness in L2A cannot be attained by the overwhelming majority of L2 learners. In fact, learning an L2 presents a challenge to the plasticity theory in particular. This is because plasticity is observed in the brains of bilinguals. Mechelli et al. (2004) and Costa et al. (2016) find that learning an L2 is only possible through functional changes in the brain; they argue that the left inferior parietal cortex is larger in bilingual brains than in monolingual brains.[10] There is no doubt that future research is required to explore this question in depth how: why is second language learning affected by late or delayed exposure to a language – maturation? Answer(s) to this question is/are central to the fields of second language learning and education; it/they can open various unexpected avenues of enquiries that will deepen our understanding of both language and language acquisition/learning.

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