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KEYNOTE ARTICLE

Rethinking the critical period for language: New insights into an old question from American Sign Language*

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The hypothesis that children surpass adults in long-term second-language proficiency is accepted as evidence for a critical period for language. However, the scope and nature of a critical period for language has been the subject of considerable debate. The controversy centers on whether the age-related decline in ultimate second-language proficiency is evidence for a critical period or something else. Here we argue that age-onset effects for first vs. second language outcome are largely different. We show this by examining psycholinguistic studies of ultimate attainment in L2 vs. L1 learners, longitudinal studies of adolescent L1 acquisition, and neurolinguistic studies of late L2 and L1 learners. This research indicates that L1 acquisition arises from post-natal brain development interacting with environmental linguistic experience. By contrast, L2 learning after early childhood is scaffolded by prior childhood L1 acquisition, both linguistically and neurally, making it a less clear test of the critical period for language.

Keywords: critical period for language, first language acquisition, second language acquisition, sign language, neurolinguistic processing

1. Introduction

Why would we need to know if there is a critical period for language acquisition? This information might be useful for educational policy to enable more children to become proficient in a second language. Likewise, the information might be useful in order to improve language programs for immigrants who speak other languages to help them integrate into their new countries more quickly. Clinical and rehabilitation language programs for children and adults often cite critical period (CP) research as a rationale. Last but not least, research into CP learning informs longstanding questions in cognitive science about language and brain development and how they affect one another, in addition to the role the environment plays in this development. Given that research on CP learning is vital to so many domains of inquiry, the next question is what kind of experiments and data are required to answer the question.

Generally speaking, CP phenomena are thought to reflect a unique type of learning when an animal or human is exquisitely sensitive to a particular stimulus in the environment during development. The main characteristic of this sensitivity is that it is limited to a temporal phase in development. Before the opening and after the closing of the CP, sensitivity to the stimulus is either diminished or absent, hence the notion of a PERIOD. CP learning is commonly observed throughout the animal kingdom with one frequently cited example being birdsong. The white crowned sparrow learns the song of its species beginning around day 10 after hatching. The CP window for learning its species' song closes around day 50 after hatching. A lack of exposure to adult song during this temporal window results in an abnormal song. The onset, closing, and duration of the CP for birdsong learning varies by species (Marler, 1989). Another example of CP learning is the discovery by Lorenz (1965) that baby geese imprint on the first moving stimulus they see beginning at 13 hours and ending around 16 hours after hatching. Typically the first moving object is the mother, and gosling survival depends upon learning to follow the gaggle, hence the notion CRITICAL. Socialization phenomena in animals have also been found to be governed by a CP. Developmental timing effects in dogs (Lord, 2013) and

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domesticated Siberian silver foxes (Trut, 1999) have been associated with enhanced abilities to interpret human signals of shared attention, such as gaze and pointing, both of which are prerequisites for human language acquisition (Hare, Brown, Williamson & Tomasello, 2002; Virányi, Gácsi, Kubinyi, Topál, Belényi, Ujfalussy & Miklósi, 2008). Mice that are isolated during the fourth and fifth weeks postpartum, a CP in their development, show decreases in myelination of neuronal axons related to behavioral and cognitive deficits (Makinodan, Rosen, Ito & Corfas, 2012). Some of the most well studied CP effects are found in the development of the visual system (Wiesel, 1982). Animal models provide a rich and detailed means to investigate CP effects on the development of perception, behavior and the underlying neural mechanisms of these effects (Hensch, 2005). However, there is no animal model with which to study a CP for language (CPL).

1.1 The Critical Period for Language

The existence of a CPL has been the subject of considerable debate. Skinner (1957) initially proposed that children learn language as a result of stimulus-response reinforcements emanating from the environment. Chomsky (1959) countered that features of the environment cannot explain language development, which he proposed to be the knowledge of linguistic structure, otherwise known as the human language faculty, and not linguistic behavior *per se* (Chomsky, 1965). Given the widespread findings of CP learning in animals, we might postulate that a CPL bridges these language domains, one centered in the environment and the other centered in the mind and brain. The CPL may function to link the experience of language present in the environment to development of the brain language system.

1.2 Early CPL proposals

For centuries the folk observation that children develop language quickly and effortlessly while adults often fail to learn a second language well enough to pass as a native speaker has been interpreted as evidence for the existence of a CPL. In 1894, the physician Itard (1962) concluded that the speechless *infant sauvage* he had tutored for two years, Victor, failed to learn French because the boy was simply too old. In 1967, Lenneberg marshaled evidence suggesting that language development co-occurs with brain development. He described several phenomena in language acquisition that appear to occur during childhood but not later. First, language development is stage-like in a fashion akin to the milestones of, for example, the development of walking by infants. Second, recovery of language ability after brain damage is possible for children but less so for adults. Third, the ability to acquire a second language (L2) spontaneously from mere exposure without conscious effort or a residual accent declines with age. Fourth, the language development of cognitively impaired children is delayed compared with that of typically developing children and appears to stop around puberty. And last, the effects of deafness on spoken language development are inversely related to its age-onset. Lenneberg (1967) argued that these linguistic age-related phenomena were not coincidental but instead are the effects of brain development and thus constitute evidence for a CPL. He further proposed that the closing of the CPL was marked by hemispheric lateralization for language, which he, along with other scholars at the time, erroneously believed to occur around puberty.

Since Lenneberg's seminal monograph, much research has been devoted to ascertaining the validity and scope of the putative CPL. Some research has focused on the interaction effects of the L1 syntactic and morphological structure on the acquisition of these structures in the L2 during childhood and beyond. This kind of cross-linguistic research is beyond the scope of the present paper, however. Other research has used an experimental paradigm measuring the ultimate outcome of L2 learning in relation to the AGE when the learning began, otherwise known as AGE OF ACQUISITION, AoA, although more accurate terms might be AGE OF EXPOSURE OR ONSET.[1] The results of this body of research may not yield the clearest insights into the veracity and nature of the putative CPL, however, for reasons we explain below.

1.3 Current Proposal

The main arguments we make here are, first, that conflating SECOND (L2) with FIRST language (L1) acquisition creates a confounded language learning situation that needs to be teased apart in order to illuminate the putative CPL. Logically, the CPL should govern the initial acquisition of language in early life, from both a behavioral and neural perspective, rather than the subsequent learning of an L2 after early childhood, after grammatical structure and its neural circuitry have been acquired and established. A childhood L1 and subsequent L2 acquired during early childhood have been shown to interact with one another in fascinating ways (Meisel, 2013). However, we argue here that sign languages, due to the unique environmental circumstances under which they are acquired, provide unique insights into the CPL that are hidden from the exclusive study of L2 spoken language acquisition. Indeed, comparing the outcome of post-childhood L2 acquisition with that of post-childhood L1 acquisition provides the necessary comparison for titrating the effects of the two learning situations. We begin by considering how CPL effects have been investigated

[1] Because AoA is the more traditional term found in this line of research, we adopt it here.

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with studies of ultimate attainment for L2 spoken language learning. Next we turn to the case of American Sign Language (ASL) and psycholinguistic studies of ASL ultimate attainment, and studies comparing late L1 with late L2 attainment. Case studies of late L1 acquisition provide the linguistic links necessary to understand the brain language processing effects associated with late L1 vs. L2 development. Together these diverse studies provide new insights into the scope and nature of the CPL, which we address last.

2. CPL effects on ultimate spoken L2 outcome

The structure of language consists of hierarchical layers of rules. Perceptual and motor processes link to phonological structure (Jackendoff, 2011), which is interleaved throughout the lexicon, morphology, and syntax. Two levels of linguistic structure in particular, phonology and morphosyntax, have each been hypothesized to be more sensitive to a CPL than other aspects of linguistic structure. In addition, the shape of the function between AoA and L2 proficiency has also been scrutinized with the goal of identifying the age at which a possible CPL closes, with the assumption that it opens at birth.

2.1 Phonological effects

Any language has a vast lexicon that is expressed and comprehended by way of a phonological system. Speaking with a non-native accent has long been cited as the most salient effect of learning language after early childhood (Lenneberg, 1967; Scovel, 1988). Infants are especially sensitive to the phonological system of the ambient language in the environment. During the first year of life, infants show perceptual learning of the vowel space and consonantal features of the environmental language (Kuhl, Williams, Lacerda, Stevens & Lindblom, 1992; Werker & Tees, 1984). This early perceptual-phonological learning has formed the basis of two related hypotheses about the nature of the CPL. One is that CPL effects arise from infant phonological learning, rendering the subsequent learning of another language difficult because phonemic categories have already been established in infancy for the L1 (Flege, Schirru & MacKay, 2003; Norrman & Bylund, 2016). A related hypothesis is that CPL effects observed in levels of linguistic structure other than phonology may in fact originate from early phonological learning of the L1. CPL effects at the level of L2 morphology and syntax, for example, have been postulated to be cascaded effects emanating from early phonological attunement (Werker & Tees, 2005), perhaps by way of working memory (Pierce, Genesee, Delcenserie & Morgan, 2017). The central role attributed to infant phonological discrimination with respect to the subsequent learning of linguistic structure in an L2 is reminiscent of models of reading in which phonological discrimination is posited to play a central and causal role in reading comprehension, both for successful and unsuccessful development (Dickinson & McCabe, 2001).

In a series of studies, Flege and his colleagues found robust AoA effects on the ability to speak English without an accent. They proposed that the main difficulty for L2 learning by older learners is adjusting the phonological categories of the natively acquired L1 to accommodate the altered or novel phonological categories of the L2. He proposed that the degree of phonological match and mismatch between the L1 and L2 causes varying degrees of L2 learning success (Flege et al., 2003). That said, there is evidence both for and against the notion of strict maturational limits on the possibility of attaining a native-like accent in an L2. Based on Lenneberg's (1967) original hypothesis, this cutoff is usually assumed to fall sometime during adolescence. Yet meticulously designed investigations of L2 phonology (Abrahamsson & Hyltenstam, 2009; Granema & Long, 2012; Moyer, 1999) provide equivocal evidence on this point. While there is some evidence for the presence of a discontinuity, albeit at different ages (age 6 in Granena & Long, 2012; age 12 in Flege, Yeni-Komshian & Liu, 1999; and age 16 in Abrahamsson & Hyltenstam, 2009), all these studies also report linear relationships between the degree of foreign accent and AoA up into the late twenties.

Another approach has been to determine if any late L2 learners can achieve native-like pronunciation. In a series of studies, Bongaerts and colleagues (Bongaerts, Mennen & van der Silk, 2000; Bongaerts, van Summeren, Planken & Schils, 1997; Palmen, Bongaerts & Schils, 1997) concluded that learners first exposed to an L2 after age 12 were still able to do so under certain circumstances. They also concluded that variables that provide "input enhancement" (Ioup, 1995), such as instructional support, motivation to sound like a native speaker, and biographical history (i.e., having a native speaker partner or family) also played a large role in achieving a native-like accent, as did the typological similarity of the L1 (e.g., either German or English) and the L2 (e.g., Dutch).

Individual case studies support these group studies. Ioup, Boustagui, El Tigi, and Moselle (1994) reported a case study of two women first exposed to (Egyptian) Arabic in their early twenties: one had had years of instruction and had done extensive graduate work in Standard Arabic, while the other was entirely self-taught. Both women had married Egyptian men. Eight out of 13 teachers of L2 Arabic judged their spontaneous spoken production to be native-like. A similar case study reported an exceptional learner with an AoA of 22 who was also largely self-taught, but whose pronunciation fell well within native speaker norms (Moyer, 1999). In addition to native speaker perceptions of accent, Birdsong (2003)

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examined both vowel duration and voice onset time (VOT) of initial stop consonants in L2 speakers of French first exposed to the language at 18 years of age or older. He reported two late learners out of 22 studied who fell consistently within native speaker norms on both the acoustic and the impressionistic parameters. Both of these late L2 learners also reported high levels of motivation. Another study reported an exceptional, advanced L2 learner of Spanish (out of five with advanced degrees in Spanish) with an AoA of 24 who performed consistently within native speaker norms on acoustic measures of Spanish stop-liquid clusters: VOT, rhotic quality, and vowel epenthesis (Colantani & Steele, 2006).

Clearly, L2 exposure that is delayed until as late as the third decade of life is not an absolute biological barrier to the acquisition of a native-like accent; certain exceptional and highly motivated individuals are still somehow able to surmount this impediment. If such a constraint were truly hard-wired, there could be no such exceptions. We are not claiming this to be the norm in L2 acquisition. To the contrary, everyone is anecdotally aware of the difficulty of achieving native-like pronunciation in a language acquired late in life. Nevertheless, by the same token, most people are also anecdotally familiar with some individual who, despite delayed exposure, seems to have been able to achieve a native accent in a second language. More importantly, with the exception of Granena and Long (2012), who studied Chinese speakers of Spanish, every other study that has drilled down on such individuals in order to determine their capabilities in the phonological domain has found at least one and up to four individuals who perform at native-like levels on both subjective (native speaker assessment) and objective (acoustic) measures of accent. Aside from Granena and Long (2012), who found none, the lowest percentage of L2 speakers deemed to exhibit native-like pronunciation in any study is a little over 4% of the population studied.[2]

2.2 Lexical, morphological, and syntactic effects

From the theoretical perspective of universal grammar and its variants, knowledge of the morphological and syntactic structure of a language is often taken as the *sine qua non* of the human language capacity. Like early phonological development, children acquire the linguistic features specific to the language they use early in life, and these features are thought to be difficult to reset at older ages (Wexler & Cullicover, 1980). For example, Curtiss and colleagues (Curtiss, 1977; Fromkin, Krashen, Curtiss, Rigler & Rigler, 1974) characterized the spoken language development of Genie (who was learning English at the age of 13 after being socially isolated from people beginning around the age of 20 months) as being deficient with respect to morphological and syntactic learning, but spared for lexical learning. This description of what Genie found easy and hard in language acquisition is consistent with the UG framework prevalent at the time, which considered lexical development to be unrelated to the acquisition of syntactic structure. The problem with this account of the CPL, however, is that Genie's lexical development was not systematically studied. Since this landmark study, lexical acquisition has been found to play a pivotal role in language development (Fenson, Dale, Reznick, Bates, Thal, Pethick, Tomasello, Mervis & Stiles, 1994; Bates & Goodman, 1997). Nonetheless, the idea that the CPL primarily affects morphological and syntactic development is a common hypothesis. Given this perspective, many studies have scrutinized AoA effects on L2 outcome with respect to morphological and syntactic proficiency.

2.3 AoA effects on L2 morphological and syntactic outcomes

A frequently used measure of morphological and syntactic knowledge is the grammaticality judgment task because it requires the detection of rule violations in these domains, which native speakers do unconsciously. In a seminal study using a grammaticality judgment task with Chinese and Korean native speakers who were L2 learners of English, Johnson and Newport (1989) found performance to decline as a linear function of AoA up to age 16, with no systematic relation to AoA afterwards. They interpreted this linear trend prior to puberty, and the lack of one afterwards, as evidence that a CPL governs language acquisition during childhood, but not afterward.

Shape of the AoA function

As described above, one of the main observable features of CP phenomena is a closing of the temporal learning window sometime during development. For this reason, researchers have searched for a closing of the CPL by scrutinizing the shape of the function between AoA and L2 performance at the level of morphology and syntax (and to some extent for phonology too, described above). Testing native Spanish L2 learners of English using the same task and stimuli as Johnson and Newport, Birdsong and Molis (2001) found few AoA effects until puberty, after which performance declined, suggesting that the linguistic similarity between the L1 and L2 modulates AoA effects. In a massive study using census data and a self-assessment of L2 proficiency, Hakuta, Bialystok, and Wiley (2003) found no break points in the linear function

[2] This rate of incidence, although low, is roughly equivalent to the mean percentage of individuals who claim same-sex attraction among the general population in studies worldwide. Clearly same-sex attraction is not the norm, and it would be folly to claim so. But it is equally implausible to claim the same-sex attraction is biologically impossible because it occurs with this rate of frequency, even if it is not the norm.

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for AoA effects, but rather a continuous decline in self-reported L2 English proficiency in native Chinese and native Spanish speakers, a decline that continued into the 7th decade of life.

DeKeyser, Alfi-Shabtay, and Ravid (2010) and Granena and Long (2012) both reported similar overall linear declines across the decades, but argued for the existence of two underlying points of discontinuity in this linear function, albeit at different ages of arrival. The results were again largely equivocal. In a study of Russian–English bilinguals in the U.S. and Canada, and of Russian–Hebrew bilinguals in Israel, DeKeyser et al. (2010) found robust linear correlations of AoA with morphosyntactic ability across the entire lifespan, as in Hakuta et al. (2003), albeit this time as measured by a grammaticality judgment task. Partial analyses of the dataset showed a linear correlation only up to an AoA of 40 years and not beyond. However, when age at time of testing was partialed out, only those with an AoA of up to 18 years still showed a linear correlation with proficiency. This correlation disappeared when this group was split at 12 years, but mean scores on the grammaticality judgment task still differed significantly between those with an AoA above or below 12. The important point for our line of argumentation here is that L2 proficiency declines with AoA, regardless of whether AoA is binned into separate age groups or the data are aggregated and regression functions are computed over the entire data set. The unanswered question is whether arbitrary cut offs in AoA reflect real breakpoints in the ability to acquire an L2 proficiently, which is where language aptitude and motivation come into play.

Based on Johnson and Newport (1989) and Flege et al. (1999), Granena and Long (2012) chose AoA breakpoints of 6 and 15 years in their study of Chinese–Spanish bilinguals living in Spain. Based on both grammaticality judgment and production measures, Granena and Long (2012) reported linear correlations with morphosyntactic and lexical ability only for those with an AoA between 7 and 15 years when the participants were grouped into three AoA categories spanning the first three decades of life. However, in the end, Granena and Long (2012:326-327) conceded that a model with two break points accounted for only 5% more of the variance in their data than a linear model with none at all, and that the latter (as in Hakuta et al., 2003) might in fact provide a more parsimonious, less complex fit.

Thus in both studies, linear correlations accounted for the data across multiple decades of first exposure to an L2, as in Hakuta et al. (2003), but more limited linear correlations up to age 15 or 18 were detected when the data were divided into discrete subsets of AoA ranges, as in Johnson and Newport (1989). In other words, much like the famous rabbit vs. duck optical illusion, these results leave it mostly to the observer to decide whether the linearity vs. discontinuity glass is half full or half empty in either direction.

Language aptitude and motivation

Both DeKeyser et al. (2010) and Granena and Long (2012) reported correlations between language aptitude and L2 proficiency primarily for learners exposed to an L2 later in life: between 18 and 40 for morphosyntax (DeKeyser et al., 2010) and between 16 and 29 for phonology and lexical knowledge (Granena & Long, 2012). These correlations were interpreted as indicating that language aptitude plays a role in successful L2 acquisition, but only for late learners. However, in a study of Spanish–Swedish bilinguals who were first screened for near-native proficiency, Abrahamsson and Hyltenstam (2009) reported a robust correlation between language aptitude and performance on a grammaticality judgment task testing fine points of Swedish grammar in early learners, with ages of arrival between 1 and 11. Those with higher language aptitude scores tended to be the ones who performed within native speaker norms. There was a similar trend in the late learners. This suggests that language aptitude plays a role in near-native L2 performance regardless of age of first exposure.

Case studies of high proficiency L2 learners have also found that aptitude and motivation modulate AoA effects. In a study described above, Ioup (1995) examined two high proficiency late learners of Egyptian Arabic. They were identified as such because they performed at near-native levels on a production task targeting intricate features of Egyptian Arabic morphology and syntax. Their high level of proficiency, in spite of having been first exposed to the language in their early twenties, was attributed to a desire to assimilate to the culture for family reasons along with language aptitude (with regard to the late learner who was self-taught), and formal instruction (with regard to the late learner who had done graduate work in Standard Arabic).

L2 use and education

Education in the L2 along with the amount of use of the L2 has also been found to exert robust effects on L2 outcome. Hakuta et al. (2003) found a significant effect of education level (independent of age of arrival) on self-assessed proficiency level in their analysis of U.S.A. census data. Birdsong and Molis (2001) also found robust effects of education and L2 use on L2 outcome. In their study of Korean–English immigrants to the U.S., Flege et al. (1999) found that individuals with more years of formal USA education outperformed those with less education on tests of rule-based morphosyntax, while those who spoke more English than Korean on a daily basis performed better than those who did not on tests of lexically idiosyncratic morphosyntax.

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AoA and L2 outcome

AoA effects on L2 outcome are robust under some circumstances, but also plainly interact with non-age related factors, such as the amount of linguistic experience with the L2, the typological relationship of the L1 to the L2, the amount of education received in the L2, the amount of L2 use, as well as learning factors such as motivation and aptitude. It is possible that these learning factors may contribute to some of the age-related decline in L2 outcome. For example, motivation to learn and use a new language, and amount of education received in the L2, may decline with age. If L2 outcome were fully under the control of a CPL, these learning variables should not predict L2 outcome, and the outcome of L2 learning would not be consistently observed to be so variable (Meisel, 2013).

Seidenberg and Zevin (2006) interpret AoA effects on L2 outcome as arising from what they call the "paradox of success." They propose that prior language learning itself alters the outcome of L2 learning. Within the framework of connectionist modeling, learning is accomplished by the creation of associative links among nodes within a network. These paths become weighted according to the frequency of their associations with other nodes within the network over time. Difficulties and limitations for L2 learning from a connectionist perspective thus arise from conflicts among the weights and associations of the originally developed language network with those of the new language. From this perspective, the linear AoA function often observed in relation to L2 outcome is due to the increasing entrenchment of the L1 language network with age. Note that this theoretical model of AoA effects on L2 outcome is somewhat related to the models discussed above that posit cascaded effects emanating from infant phonological attunement, which might be thought of as different weights to acoustic features of the speech signal. The difference is that a connectionist network accounts for morphological, syntactic, and lexical knowledge, as well as phonology.

Common to all these models is the assumption that infant L1 development is unlike L2 learning because L1 development begins from scratch in infancy while L2 learning is filtered through, and interacts with, L1 knowledge. In an abstract sense, all these theoretical proposals suggest that the putative CPL applies to L1 learning, and that L2 effects are a consequence of this prior learning. We turn now to a new source of data with which we can examine the CPL, AoA effects on sign language attainment.

3. AoA effects on ASL outcome

When considering AoA effects on ASL outcome, it is important to be mindful of the ways in which infant deafness radically alters the linguistic input from the environment necessary for language acquisition.

3.1 Infant deafness and language acquisition

The evolution of human social groups has conspired to create the ideal linguistic environment for language acquisition. Infants who hear normally are immersed in spoken language from birth and even before (Moon, Cooper & Fifer, 1993). The language spoken in the environment is sufficient for infants who hear to develop complex language by the ages of seven to nine without any overt instruction on the part of their caretakers, or any explicit practice on the part of the children (Ambridge & Lieven, 2011; Diessel, 2004). By contrast, infants born severely or profoundly deaf are isolated from the language spoken around them by virtue of their inability to hear it. Unfortunately, the visual signal of speech, as in lipreading, is too impoverished to support spontaneous language acquisition because most speech sounds are articulated inside the mouth.[3] However, deaf (and also hearing) infants do spontaneously develop sign language when the people around them sign. This fact alone indicates that the human capacity for spontaneous language development transcends the sensorimotor characteristics of the communication channel.

However, the majority of infants born deaf are not exposed to any sign language until ages well past infancy, when they first interact with signers. This initial exposure to sign language typically occurs outside the home in a school or social setting.[4] Many deaf infants in North America and Europe receive special intervention to promote spoken language development, but such intervention, which typically discourages the use of sign language, is not always available nor is it always successful. In the absence or paucity of prior spoken language development, a deaf child's first exposure to a sign language marks the initial onset of language acquisition, albeit at a late age. In addition, however, sign languages, like spoken ones, are also learned as second languages by many individuals, deaf or hearing, at a range of ages past infancy. These circumstances create naturally occurring variation in the age-onset of first- and second-language acquisition and thus provide a unique means with which we can investigate the postulated CPL.

Given that language is a complex system requiring several years of experience to fully develop, the next

[3] The intensity level of speech is typically described as being around 60 dB (spoken by an average size man standing one meter from the listener). The term *deaf* as used here is defined as *severe,* hearing loss greater than 70dB, to profound, greater than 90dB. Individuals with severe to profound hearing loss cannot perceive speech auditorily.

[4] Less than 10% of the population of deaf individuals is born into deaf families (Mitchell & Karchmer, 2004).

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question is what CPL effects might look like in signed languages.

3.2 AoA and ASL outcome

The first study to find that AoA affects ASL proficiency used narrative shadowing and sentence recall because these tasks yield insights into the psycholinguistic processing of natural language (Mayberry & Fischer, 1989). Shadowing is an on-line task where attention is split by comprehending while simultaneously reproducing a linguistic stimulus. Performance on this task is sensitive to linguistic structure (Cherry & Taylor, 1954; Marslen-Wilson, 1973). College students who were native signers (whose deaf parents signed to them from birth) and born deaf were more accurate shadowing ASL narratives than college students also born deaf who were non-native signers (whose hearing parents did not sign to them). The non-native signers began to learn ASL between the ages of 9 to 16 in school through interaction with peers who signed. Prior to this, they had attended "oral" schools, where sign language was discouraged. The second study used sentence recall, an off-line task where comprehension and expression are sequential and thus place a greater load on memory compared with the shadowing task. For the second study, another group of deaf college students participated whose AoA ranged from birth to age 15. Replicating and extending the first study results, ASL sentence recall accuracy declined as a linear function of AoA. However, because the participants of both studies were college students of a similar age, length/years of ASL experience was confounded with AoA; the two factors were inversely correlated.

Titrating the effects of AoA from linguistic experience is a design challenge for CPL studies. How much linguistic experience does a learner need to achieve maximum, or ultimate, proficiency? Some studies address the problem by recruiting only participants with high proficiency levels and then searching for AoA effects among these pre-screened L2 learners, as discussed above (Abrahamsson & Hyltenstam, 2009; Coppieters, 1987; White & Genesee, 1996).

The third study controlled linguistic experience by recruiting deaf signers who had used ASL for a minimum of 20 years and who, for the most part, were not college graduates. The task again was sentence recall but here the sentences were long and complex. Recall accuracy declined as a linear function of AoA, which ranged between birth and 13 years, with no correlation with length of experience (Mayberry & Eichen, 1991).

Across the three studies, AoA affected knowledge of ASL structure rather than cognitive processing constraints *per se*. For example, AoA showed no significant effects on the rate with which the signers produced signs, no effects on the overall number of signs the signers produced for each trial, and no effects on performance on non-verbal cognitive tasks such as block design. Rather, AoA showed differential effects on morphosyntactic processing. Later learners tended to strip inflectional morphology from the stimulus signs in complex ASL sentences to produce bare stems instead. By contrast, early learners tended to re-analyze and re-arrange them, all the while maintaining the overall meaning of the stimulus sentence.

Given the AoA effects described thus far, it comes as no surprise that native deaf signers show greater on-line sensitivity to violations of verb agreement compared with non-native deaf signers (Emmorey, Bellugi, Friederici & Horn, 1995), or that grammaticality judgment accuracy for ASL sentences, ranging from simple to complex, was found to decline as a linear function of AoA in a Canadian sample of highly experienced deaf signers (Boudreault & Mayberry, 2006). Similarly, Newport (1990) found a linear decline as a function of AoA and performance on tasks requiring knowledge of complex ASL verb morphology in a sample of highly experienced deaf signers with a minimum of 30 years' exposure to ASL. However, she found no AoA effect on tasks involving basic word order, suggesting that not all aspects of ASL morphology and syntax decline with AoA, an intriguing finding to which we return below.

To summarize AoA effects on ASL outcome, the results of several studies investigating the ASL outcome in diverse groups of deaf signers using varying proficiency measures concur to show AoA predicts ASL learning outcome. These effects show a linear relation to AoA from birth to adolescence in those studies that tested for this function. This indicates, first, that AoA effects are not unique to spoken L2 but instead transcend the sensorimotor modality of the communication channel. This is not surprising given that ASL is a language. Learning a manual-visual language does not circumvent AoA effects, suggesting that these effects do not originate from sensorimotor learning *per se*, or if they do, that all sensorimotor modalities associated with all language are affected. However, the fact that AoA effects on ASL learning outcome parallel those of AoA effects on L2 spoken language learning over the same age range does not address the question of L1 outcome in relation to AoA.

3.3 AoA effects on L2 vs L1 morphological and syntactic outcome

As explained above, not all signers who are deaf learn ASL as an L1. Some signers acquire another sign language in infancy and learn ASL later as an L2, although this situation has been little studied. Other deaf signers acquire spoken English to varying degrees before learning ASL later, also as an L2. A small proportion of L2 signers were

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not born deaf but instead suddenly became deaf due to viral infections.[5] Because such signers are indisputably L2 learners of ASL (because they fully acquired spoken English as hearing infants prior to learning ASL later), they provide an ideal test of the CPL. Comparing their ASL proficiency to signers who were born deaf, but who acquired minimal language prior to learning ASL at the same ages, provides a critical test, a means to ascertain the extent to which brain maturation alone predicts the outcome of language acquisition. If brain maturation alone affects language learning, then AoA will equally affect L1 and L2 outcome.

To this end, we matched signers who became deaf between the ages of 8 and 12 by age, sex, and length of ASL experience to signers who were born deaf and self-reported knowing minimal language prior to ASL exposure at the same AoA. All the signers were highly motivated to learn ASL and highly experienced, having 20 years or more of continuous experience. Native deaf signers served as the controls. Again, the task was ASL sentence recall. The results showed a marked advantage of infant language learning. The deaf L2 learners performed at near-native levels. By contrast, the deaf late L1 learners performed at low levels (Mayberry, 1993). These results provide initial evidence that L1 experience begun in infancy is necessary for later L2 learning to be successful. Note that these findings also confirm the amodal nature of language ability. Infant spoken language acquisition facilitates later sign language acquisition. Given that language ability is amodal, the facilitative effects of infant language acquisition should be bi-directional: that is to say, infant SIGN language acquisition should support later L2 learning of SPOKEN language (Mayberry, Lock & Kazmi, 2002).

Next, we turned to English as the target language to further probe this hypothesis using grammaticality judgment, as commonly used in L2 AoA studies described above, along with a sentence-to-picture matching task. To assess the amodal nature of AoA effects on L1 vs. L2 acquisition, we tested two kinds of L2 learners: one group was native deaf signers of ASL who learned English as an L2 in school; the other group was native hearing speakers of Urdu, Spanish, German, and French who also learned English as an L2 in school at similar ages. To verify what we have called the L1 TIMING HYPOTHESIS, we also recruited late L1 learners who were born deaf but who acquired minimal language in early childhood prior to learning ASL and English in school at the same ages as the L2 groups. As predicted, both groups of L2 learners performed at near-native levels on both tasks, despite the fact that one group had normal hearing and acquired a spoken language in infancy and the other group was born deaf and acquired ASL in infancy. The contribution of infant language experience to life-long language learning ability is demonstrably amodal.

Also as predicted, the late L1 learners, who had used ASL and English for the same length of time as the two L2 groups, but who also had acquired minimal language during early life, showed low levels of English proficiency on the two tasks, but not across all sentence structures. They performed at near-native levels on simple SVO structures, as Newport (1990) had previously found in her study testing late acquisition of ASL. As the morphosyntactic complexity of the English structures increased, their performance declined to chance levels on both tasks (Mayberry & Lock, 2003). These results were replicated in a study of deaf signers of British Sign Language (BSL) using the ASL grammaticality judgment task of Boudreault and Mayberry (2006) translated into BSL (Cormier, Schembri, Vinson & Orfanidou, 2012). The developmental timing of initial language experience during childhood clearly exerts robust effects on ultimate language proficiency across languages and sensorimotor modalities.

3.4 AoA effects on ASL phonology

Up to this point, we have focused exclusively on AoA effects on ASL outcome with respect to morphology and syntax. As described above, AoA shows robust effects on spoken L2 phonology, with some researchers proposing that infant phonological learning is the source of these effects. Like all languages, the linguistic architecture of ASL contains a phonological level of structure: signs, i.e., words are constructed from highly constrained bundles of articulatory features (Brentari, 1998; Perlmutter, 1992; Wilbur, 2011). In the above described studies employing shadowing and sentence recall tasks, AoA also showed effects on the signers' ASL phonological production. Specifically, the lexical errors made by the non-native signers were often phonological in nature. These phonological-lexical errors were real signs, not neologisms, that violated the morphosyntactic structure of the stimulus sentence, but, at the same time, they were clearly derived from the phonological structure of the original stimulus. An example of this kind of error in English would be like, "At Thanksgiving, I ate too much turkey *sleep* potato," where the verb "sleep" is substituted for the conjunction "and." The two signs vary in only one sublexical feature of sign, location, in ASL. These kinds of phonological errors suggest that the stimulus sentence was incompletely processed. Perhaps phonological pieces of the stimulus item were perceived, but the sentence was insufficiently processed to catch and rectify the error.

[5] Meningitis or measles epidemics are less common today in North America, but viral infections continue to be a major cause of post-infancy deafness in many developing countries (Morgan & Mayberry, 2012)

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Lexical-phonological processing errors

The fact that these phonological lexical errors typically violated the morphosyntactic structure of the stimulus corroborates the grammaticality judgment results of other studies (Boudreault & Mayberry, 2006; Mayberry & Lock, 2003; Mayberry et al., 2002). Moreover, the interpretation that these phonological-lexical errors reflect incomplete language processing was supported by the finding that they were negatively correlated with comprehension accuracy, both of which in turn were negatively correlated with AoA. These phonologically based lexical errors suggest that non-native deaf signers process the linguistic signal differently from native deaf signers. Their processing appears to be more shallow and often snagged at the surface level of lexical structure, leading to what we have called a PHONOLOGICAL BOTTLENECK in language processing (Mayberry & Fischer, 1989), a psycholinguistic phenomenon related to the results of subsequent neuroimaging work discussed below. These effects are reminiscent to the extra effort L2 speakers have to expend to comprehend speech in a noisy environment.

Other studies have also found AoA effects on phonological processing in sign language by comparing the performance of native vs. non-native deaf signers. For example, native deaf signers showed clusters of similarity judgments for movements extracted from ASL signs suggestive of phonemic categories, a kind of phonemic clustering not exhibited by sign-naïve hearing participants (Poizner, 1981). AoA effects have been found for lexical decision tasks in Spanish Sign Language and British Sign Language (Carreiras, Gutierrez-Sigut, Baquero & Corina, 2008; Dye & Shih, 2006). Native and non-native deaf signers appear to differentially weight phonological features in ASL and BSL (Hildebrandt & Corina, 2002; Orfanidou, Adam, Morgan & McQueen, 2010). These AoA effects on phonological processing in sign language are consistent with the widely observed AoA effects on phonological skills for spoken L2 learning. Given the differential and amodal AoA effects on L1 vs. L2 outcome on morphosyntactic processing in ASL, the next key question is whether similar differential effects are observed in ASL phonological processing.

Differential AoA effects on L1 vs L2 phonological processing

Some studies have found hints that phonological experience in early life facilitates rather than hinders later language learning, contrary to what has been proposed for spoken L2 learning described above. In a categorical perception study of an ASL handshape, Best, Mathur, Miranda, and Lillo-Martin (2010) found native deaf signers and hearing L2 signers to show category boundaries for the tested phonological feature. By contrast, non-native deaf signers, who perhaps were quasi-late L1 learners, were less categorical. In another study, native deaf and L2 hearing signers did not differ in their phonological similarity judgments for pairs of ASL signs. However, non-native deaf signers showed phonological similarity ratings that differed from those of both the native deaf and L2 hearing ASL learners, but not from those made by sign-naïve hearing participants (Hall, Ferreira & Mayberry, 2012). In a third study, native deaf and L2 hearing signers performed similarly on a gated sign recognition task (Morford and Carlson, 2011). By contrast, non-native deaf signers performed significantly less well, occasionally responding to stimuli containing only partial phonological information with gestures. As has been observed in other studies, the non-native deaf signers showed differential weighting patterns for phonological features of ASL sign. Morford and Carlson (2011) interpret these results to indicate that language experience during early life affects the organization of the mental lexicon. This interpretation was supported by the results of an eye-tracking study of ASL lexical recognition. Native deaf signers showed sensitivity to sign phonological structure during online lexical recognition whereas non-native deaf signers did not (Lieberman, Borovksy & Mayberry, 2016; Lieberman, Borovsky, Hatrak & Mayberry, 2015).

The studies comparing late L1 with late L2 acquisition in adults have mostly investigated phonological effects in the context of the lexeme. Recent studies with deaf children developing sign and spoken languages bilingually have corroborated the facilitative effects of early phonological experience cross-linguistically and cross-modally in children's language development (Davidson, Lillo-Martin & Chen Pichler, 2014; Hassanzadeh, 2012).

AoA effects on L1 outcome are thus clearly distinct from those for L2 outcome at the level of phonological processing. Like morphosyntactic learning, infant language acquisition facilitates later L2 learning at the phonological level, independent of sensorimotor modalities of the early L1 or the later L2. Also like morphosyntactic development, aspects of phonological development are clearly amodal. L1 phonology may interfere to some extent with successful L2 learning of a spoken language, even when the L1 is a sign language, which has not been studied yet. Nonetheless, it is equally clear that early-acquired L1 phonology facilitates L2 learning. A lack of language experience, including phonological experience, during early life renders language acquisition begun after childhood incomplete. Research investigating the trajectory of L1 acquisition begun after early childhood supports this interpretation and provides further insight into this phenomenon.

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4. The trajectory of late L1 acquisition

Infants born deaf who experience ASL in infancy spontaneously acquire it in a fashion similar to the spoken language acquisition of infants who hear (Bates, Marchman, Thal, Fenson, Dale, Reznick, Reilly & Hartung, 1994; Fenson et al., 1994). They babble with rhythmic arm movements aligned with ASL phonological features and prosody (Petitto, Holowka, Sergio, Levy & Ostry, 2004). The first 36 months of life is a period of rapid language development, and deaf infants exposed to ASL begin by learning more nouns then verbs, as is common for many languages. As their lexicon expands, they acquire more predicates. Vocabulary size rather than age predicts subsequent milestones such as word combination and morphosyntactic acquisition (Anderson & Reilly, 2002; Berk & Lillo-Martin, 2012). The development of ASL grammar extends over the next several years as children acquire complex syntactic structures such as question formation, conjoining, complementation, topicalization and others (Chen Pichler, 2012; Mayberry & Squires, 2006; Reilly, 2006).

4.1 Input and language acquisition

In their seminal study, Hart and Risly (1995) discovered that the amount of language spoken to children has robust effects on their language development. Since this landmark work, numerous studies have found that the amount and kind of linguistic input children receive affects the trajectory of their lexical and syntactic acquisition (Hoff, 2003; J. Huttenlocher, Vasilyeva, Cymerman & Levine, 2002). By any measure, the linguistic environment of infants born deaf is grossly impoverished compared to that of infants who hear when sign language is absent from the environment.

4.2 Homesign

Deaf children who are isolated from spoken and signed language provide another means, aside from retrospective outcome studies, to test the hypothesis that a CPL constrains L1 acquisition. Unlike cases of severe abuse where children are deprived of human contact and as a consequence are isolated from language (Curtiss, 1977; Fromkin et al., 1974; Fujinaga & Kasuga, 1990; Koluchova, 1972), deaf children do not typically suffer from social isolation. They are as nurtured as children who hear. In the absence of spoken language development, deaf children are observed to gesture for communication, known as HOMESIGN. Homesign has been shown to have some linguistic properties, such as ordering patterns, and these patterns are neither observed in the caregivers' gestures (Goldin-Meadow & Mylander, 1983), nor fully understood by them (Carrigan & Coppola, 2017). These findings have been taken to mean that any linguistic properties observed in homesign arise from within the child and not from the environment, suggesting that some features of the human language faculty require no linguistic input to emerge from the child (Goldin-Meadow, 2005). However, studies of L1 acquisition begun after childhood indicate that homesign does not function as an L1 for the deaf child.

4.3 Case studies of late L1 acquisition

Spoken language learning

The spoken Spanish development of a boy who was born profoundly deaf and fit for the first time with hearing aids at the age of 16 was studied by Grimshaw, Adelstein, Bryden, and MacKinnon (1998). Although EM could detect speech with amplification, his development of spoken Spanish was limited after 48 months of instruction. His mean length of utterance, MLU (the average number of morphemes or words combined in utterance expression) was less than 2.0. EM's minimal spoken Spanish development might be attributed to modality effects. Perhaps he could not learn spoken language because he had missed experiencing spoken phonology in infancy. However, we cannot assume that the amplified speech signal was of sufficient clarity for EM to discern the acoustic details of speech. His auditory system was damaged. Genie, whose auditory system was intact, was able to develop intelligible speech at the age of 13, although it is unknown how much spoken English she had acquired prior to being isolated from her family around the age of 20 months, or whether she overheard speech during her isolation (Curtiss, 1977). These language outcomes contrast sharply with a case studied by Vargha-Khadem (Vargha-Khadem, Carr, Isaacs, Brett, Adams & Mishkin, 1997) showing rapid acquisition by a 10-year-old after hemispherectomy. Unlike EM and Genie, the child had been exposed to spoken phonology from infancy.

ASL acquisition

Other studies have investigated ASL development begun at older ages. Berk and Lillo-Martin (2012) analyzed the spontaneous language of two children, Mei and Cal, who were born deaf and experienced ASL at the ages of 5;9 and 6;0 (years;months) with no prior language acquisition. After three months of immersion, they had learned proportionately more nouns than verbs and had begun to combine them into two-word utterances. The semantic content of their 2-word combinations was similar to that of 2-year old ASL deaf children and that of 2-year old hearing English learners reported in the literature. These results suggest that the beginning stages of late L1 development are similar to those of infant acquisition even after 5 to 6 years of delay. The question is whether this is true with an extreme delay in language experience.

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We followed the ASL acquisition of three adolescents who were born deaf, Shawna, Cody, and Carlos, and who were first immersed in ASL at the ages of 13;8, 14;7, and 14;8 respectively, after little or no prior language acquisition. After 12, 18, and 24 months of ASL experience, each adolescent had acquired a vocabulary that resembled that of young children, both in terms of the words they learned and the distribution of lexical types (nouns, predicates, and closed class items) as measured with the MacArthur ASL-CDI (Anderson & Reilly, 2002) and verified with analyses of their spontaneous language. The adolescents' rate of lexical learning was faster than that of infants, a likely effect of their being significantly more cognitively mature than infants. Similar to the 5- and 6-year-old late L1 learners, they also began to quickly combine signs into two-word utterances that were devoid of any inflectional morphology, as is the case for infant learners. Their utterance complexity, as measured by MLU, was related to the number of months each adolescent had been immersed in ASL (Ferjan Ramirez, Lieberman & Mayberry, 2013).

Like the 5- and 6-year-old Mei and Cal, the adolescents' language development showed promising beginnings. Both studies suggest that the ability to rapidly learn lexical items and combine them into two-word utterances is a latent human linguistic ability that is unperturbed by brain maturation in the absence of linguistic experience. Having no record of the adolescent's homesign, it is impossible to tell how it related to their initial ASL acquisition. However, their initial rapid lexical learning and word combinations indicate their ability to parse the linguistic signal into meaningful units remained intact despite their extremely late exposure to natural language. Although this skill might relate to early experience with gesture, it is important to note that Genie also displayed this skill, although not elaborated upon in the original reports of her language acquisition. However, this early linguistic parsing ability does not appear to develop into the ability to use sign phonological structure during language processing tasks, as demonstrated by the late L1 outcome studies described above.

Cross-sectional, longitudinal analyses of the word order acquisition of Shawna, Cody, Carlos, and Chris (who began learning ASL for the first time at age 13 with no prior language) from 12 months to 6 years of ASL experience revealed patterns that also resembled child ASL development. The patterns of word order acquisition for all four adolescent learners were initially variable, similar to that of 2- to 3-year-old native deaf learners reported in the literature. ASL uses variable word orders that are marked by morphosyntactic rules. Like young, native deaf learners, the adolescent learners progressed to a generalization stage of using SVO word order. However, unlike child ASL learners, they showed no indication of continued development of ASL word order beyond this stage, which involves complex sentence structure (Chen & Mayberry, under review).

Long-range outcome

Additional evidence that the trajectory of late L1 learner development becomes asymptotic at low levels of language development comes from a study by Morford (2003). She analyzed the ASL development of Maria and Marcus, two children who were born deaf and immersed in ASL at the ages of 12 and 13. Neither child had previously acquired any language; both were reported and observed to have used gestures with their hearing families prior to ASL exposure. She elicited language samples longitudinally from 1 to 32 months after ASL immersion with a wordless picture book. Analyses revealed that, by 7 to 9 months of ASL experience, they primarily used signs instead of gestures. They also began to quickly combine signs into utterances, as corroborated in the subsequent studies of late L1 acquisition described above. In a follow-up study conducted seven years later, however, Maria and Marcus both exhibited low levels of ASL comprehension on a sentence-to-picture matching task using utterances describing pictures from the original elicitation materials. They also made multiple phonological-lexical substitution errors on a sentence repetition task consistent with the adult, late L1 outcome studies described above.

The results of these longitudinal and cross-sectional studies of adolescent L1 acquisition converge, which is remarkable given the fact that these late L1 learners were born and raised in different countries and cultures, and first experienced ASL in a variety of home and school settings in the USA and Canada. Late L1 learners exhibit initial rapid learning of lexical items in different grammatical categories and subsequent word combinations that are reminiscent of the acquisition of young child language learners, but at a faster pace. At the same time, however, accumulating evidence suggests that two major characteristics of language acquisition begun for the first time at age 12 or older are, first, rapid initial language acquisition, and second, a subsequent protracted period of limited language development, despite rich linguistic environments and language instruction. The language development of adolescent late L1 learners does not progress to complex morphosyntactic structures, but remains limited to simple structures. Corroborating evidence for limited language development when language is not experienced in childhood comes from an ASL sentence comprehension study using a sentence-to-picture matching task. Individuals born deaf who experienced little or no language until the age of 12, with 10 years of experience, showed high accuracy on SV and SVO structures, but near chance performance on more complex structures (Mayberry, Cheng, Hatrak & Ilkbasaran, 2017).

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The trajectory of language acquisition begun after childhood is unlike infant language acquisition beyond the initial stages of word learning and word combinations. Very late L1 acquisition is characterized by protracted and limited acquisition beyond the initial stages of language acquisition. The unique trajectory of late L1 acquisition begins to explain the differential effects of AoA on L1 vs. L2 observed in ultimate attainment studies. L2 learners begin the task of new language learning with an already acquired and established linguistic system through which they can begin to learn and remember words and structures in the new language. By contrast, late L1 learners begin the task of language acquisition with no prior knowledge of words or of any linguistic structures. Next, we ask whether the asymptotic levels of language acquisition we observe in adolescent L1 learners relate to neurolinguistic processing. Specifically, we ask whether late L1 exposure affects development of the brain language system.

5. Late L1 vs. L2 effects on neurolinguistic processing

Before turning to the effects of late L1 and L2 learning on the neuroprocessing of language, it is necessary to first ask whether sign and spoken language are processed in similar brain areas under the typical circumstances of infant language acquisition. One obvious difference between sign and spoken language is the sensorimotor modalities through which they are sent and received. Instead of using the vocal tract, signers use the hands and arms in concert with movements of the head, torso, and face for articulation. Instead of listening to the language signal through the auditory system, signers watch the language signal through visual system. Many studies have investigated the question of whether the sensorimotor characteristics of the communication channel affect how language is structured and how the brain processes this structure. For example, unlike spoken English, ASL uses the spatial positions and orientations of the moving hands to mark verbal arguments, case and number, as well as prepositions, and syntactic categories, among other morphosyntactic phenomena (Lillo-Martin & Meier, 2011; Sandler & Lillo-Martin, 2006). Given the unique use of space by ASL to express linguistic structure, the question arises as to whether the brain processes it in a similar fashion to that of spoken language structure.

5.1 Neural processing of ASL in native learners

Initial research employed classic methods to investigate sign language in the brain. Poizner, Klima, and Bellugi (1987) discovered that the cognitive purpose to which space is put, linguistic vs. non-linguistic, determines which hemisphere processes it. Signers who were deaf and suffered lesions to the left hemisphere language areas exhibited language deficits involving morphosyntactic structures that are instantiated with spatial contrasts. These same left hemisphere damaged signers showed no deficits for non-linguistic spatial processing, such as recognizing pictures, making block designs, or arranging miniature furniture in a room. By contrast, right hemisphere lesioned deaf signers showed the reverse deficit pattern, namely, no deficits for comprehending spatially marked morphosyntax, but significant difficulty recognizing and reproducing pictures or block designs. The extent of left temporal lobe damage was further found to correspond to how patients performed on ASL comprehension tasks, which in turn resembled deficits exhibited by hearing English patients with left hemisphere lesions (Hickok, Love-Geffen & Klima, 2002). In another study, direct stimulation of the left temporal cortex of an epilectic deaf signer disrupted his sign expression in a fashion akin to what is observed for hearing epilectic patients (Corina, McBurney, Dodrill, Hinshaw, Brinkley & Ojemann, 1999). These pioneering studies show that the canonical language areas of the left perisylvian region are dedicated to the task of linguistic processing independent of the sensorimotor channel of language. Subsequent neuroimaging studies of healthy deaf adults, who acquired sign language from infancy, have corroborated these findings in several sign languages including ASL, BSL, Japanese Sign Language, and Swedish Sign Language (Cardin, Orfanidou, Ronnberg, Capek, Rudner & Woll, 2013; MacSweeney, Campbell, Woll, Brammer, Giampietro, David, Calvert & McGuire, 2006; Newman, Supalla, Fernandez, Newport & Bavelier, 2015; Petitto, Zatorre, Guana, Nikelski, Dostie & Evans, 2000; Sakai, Tatsuno, Suzuki, Kimura & Ichida, 2005).

Localization of sensory processing

The neural processing dissociation between sensory perception and linguistic processing was further demonstrated in a study using anatomically constrained magnetoencephalography, aMEG. In a picture-word priming task, the neural responses of native English hearing speakers who listened to spoken words were compared to those of native ASL deaf signers who watched ASL signs. Approximately 100 ms after presentation of the spoken word, the hearing English speakers showed activation in primary auditory cortex, as would be expected. Approximately 100 ms after presentation of the ASL sign, the deaf ASL signers showed activation in primary visual cortex, as also would be expected (Leonard, Ferjan Ramirez, Torres, Travis, Hatrak, Mayberry & Halgren, 2012). A bit later, around 400 ms after presentation of the spoken word, the hearing speakers exhibited expected activation in left superior temporal areas, showing the well-known pattern for semantic processing indexed by the N400 effect. Likewise, around 400 ms after presentation of the signed word, the deaf signers exhibited activation in the same

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superior temporal region, showing the same N400 effect. These results indicate that, although the initial stages of sensory processing for spoken and signed words occur in the cortical areas responsible for auditory vs. visual sensory processing respectively, the subsequent stage of lexico-semantic processing is the same regardless of sensory input. Given that the neural processing of spoken and signed languages, beyond the initial stages of sensory perception, is remarkably similar, we are now in a position to ask if there are differential effects of late L1 vs. late L2 learning on neurolinguistic processing.

5.2 Late L1 acquisition neural processing effects

fMRI studies

Using fMRI, Mayberry, Chen, Witcher, and Klein (2011) neuroimaged 22 signers as they performed an ASL grammaticality judgment task. The signers, all of whom were right handed and born deaf, learned ASL as an L1 in an immersion situation either at home or school between the ages of birth and 14. The non-native signers had used ASL daily for a minimum of 15 years. All the signers had begun school by the age of 6. Some non-native signers had acquired minimal spoken language prior to learning ASL. Other non-native signers, those who first learned ASL at ages older than 8 years, had begun their education in classrooms or schools where the use of sign language was actively discouraged. Their educational placement was subsequently switched to classrooms where sign language was used due to the fact that their spoken language was not functional for educational purposes.

The neural activation exhibited by the signers was analyzed with whole brain regression analyses to determine which brain areas were affected by AoA. Of the nine identified brain areas, seven were located within the language network, five in the left hemisphere and two in homologous right hemisphere areas. Two other identified brain areas were located in the left occipital-visual cortex (Mayberry et al., 2011). AoA for the L1 affected neural activation patterns along the anterior to posterior dimension of the left hemisphere. Anterior frontal and temporal language areas showed a significant negative relation to AoA. As the L1 AoA became older, the BOLD signal in the frontal and temporal language areas decreased. The reverse effect was observed in the visual processing areas of the left occipital cortex. Specifically, as the AoA for the L1 was older, the BOLD signal increased. In other words, signers who were born deaf and experienced ASL in early life showed robust neural activation patterns in the expected frontal and temporal language areas of the left hemisphere (and in two homologous areas in the right hemisphere). These same signers exhibited BOLD signals in left visual cortex that were below baseline (i.e., less neural activation when processing ASL sentences than for watching a still face). This suggests that when language is acquired in early life, the adult brain primarily allocates neural resources to the linguistic aspects of linguistic processing and requires minimal information from sensory-perceptual processing; perhaps because it is unnecessary for comprehension. Knowing a language means being able to predict with a high degree of probability the upcoming words in a sentence; this top-down prediction may require minimal perceptual information, suggesting that the language processing areas in frontal and temporal lobes may be in a feedback loop with the sensory-perceptual areas in visual cortex.

The neural processing results for the later L1 learners, despite their substantial linguistic experience, were quite different. Late L1 learners exhibited BOLD signals in left posterior visual cortex that were significantly greater than those they exhibited in the frontal and temporal language areas of the left hemisphere. Late L1 learners also showed greater neural activation patterns when watching ASL sentences than they did watching a still face (Mayberry et al., 2011). These results lead us to question whether the neural pathways connecting visual processing areas to language processing are fully developed when the onset of linguistic experience occurs late during brain development, a question to which we return below.

These neural results parallel some of the psycholinguistic effects of late L1 described above, in particular the finding that late L1 learners, but not early L1 and late L2 learners, tend to produce phonologically based errors divorced from lexical meaning and sentence structure when performing psycholinguistic tasks. Such late L1 learners also exhibit less evidence for phonologically organized lexical processing on a variety of tasks. These contrasting neural patterns for early vs. late L1 acquisition of ASL suggest that the brain language network requires linguistic experience during early life to develop fully. Subsequent studies provide evidence for this hypothesis.

aMEG studies

Using aMEG with a picture-sign priming task, we compared the neural correlates of lexical processing in hearing L2 learners of ASL with those of native deaf ASL signers. The L2 learners had acquired spoken English in infancy; they began to learn ASL in late adolescence to early adulthood. The L2 learners exhibited neural activation patterns for lexico-semantic processing of ASL signs that were nearly identical similar to those of native ASL deaf signers, with primary neural activation in left hemisphere perisylvian language areas and some additional activation in homologous right hemisphere and left parietal areas (Ferjan Ramirez, Leonard, Torres, Hatrak, Halgren & Mayberry, 2014).

These findings for L2 learners of ASL are consistent with studies of spoken language L2 neural

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processing. For example, native Spanish speakers showed neural activation patterns primarily in left hemisphere perisylvian language areas when listening to words in their native Spanish. When listening to spoken words in their less proficient L2, English, they exhibited neural activation patterns in the same left hemisphere perisylvian language areas but with some additional activation in left parietal and right occipitotemporal areas (Leonard, Torres, Travis, Brown, Hagler Jr, Dale, Elman & Halgren, 2011). Other studies have also found that a less proficient and/or a late acquired L2 engages language areas in the left hemisphere with the addition of some right hemisphere activation (Dehaene, Dupoux, Mehler, Cohen, Paulesu, Perani, van de Moortele, Lehericy & Le Bihan, 1997; Wartenburger, Heekeren, Abutalebi, Cappa, Villringer & Perani, 2003). This pattern for L2 neural processing was confirmed by the results of a meta-analysis analyzing 30 neuroimaging studies of spoken L2 processing (Indefrey, 2006). By definition, all L2 learners, whether hearing or deaf, learning a spoken or signed L2, share a common factor: infant language experience. Neurolinguistic studies of individuals who were bereft of language experience during childhood paint an entirely different picture.

5.3 Case studies of extremely late L1 acquisition

We studied the neural activation patterns of two cases of extreme late L1 acquisition described above, Carlos and Shawna, whose acquisition began at ages 13;8 and 14;7 respectively. When they were neuroimaged with aMEG using the same picture-sign priming paradigm mentioned above, they had 38 and 24 months of language experience respectively. The control groups were native deaf signers and hearing L2 signers (whose length of ASL experience was comparable to that of Carlos and Shawna). As expected, both the deaf native and hearing L2 signer control groups exhibited activation in frontal and temporal areas of the left hemisphere for lexico-semantic processing that was highly similar, as described above. Although Carlos and Shawna were nearly as accurate and fast as the hearing L2 learners when recognizing the signs in the scanner, they both showed strong activation in right occipital-parietal areas; Shawna showed some additional activation in right frontal and left temporal areas. We neuroimaged them a second time after they had accumulated 15 more months of ASL experience. Both Carlos and Shawna continued to show right occipital-parietal activation, but now they both also exhibited some additional neural activations in the temporal language areas, left for Shawna and bilaterally for Carlos, in response to signs with which they were most familiar, as indexed by response time (Ferjan Ramirez, Leonard, Davenport, Torres, Halgren & Mayberry, 2016). In the absence of childhood language experience, the adolescent brain exhibits radically altered neural processing patterns for lexical processing. At the same time, however, the results further indicate that the canonical language areas of the left hemisphere retain some capacity to process some language (familiar words) after three to four years of language experience late in life. The next question is whether late L1 learners exhibit more typical neurolinguistic activation patterns after decades of experience. Our previous fMRI study of highly experienced signers with late L1 acquisition (who were not as severely linguistically deprived as Shawna and Carlos) indicates that the answer is no (Mayberry et al., 2011).

We conducted another neuroimaging study to investigate the question more directly. We did so in another case study. Martin was born profoundly deaf and grew up as the only deaf person in his hearing family and community in rural Mexico. He attended no school in childhood and reported communicating with a sister through gesture. At the age of 21, he began to learn Mexican Sign Language and then, after immigrating to the USA at the age of 23, he began to learn ASL through immersion and classroom instruction. Martin had 30 years of continuous ASL experience when we neuroimaged him with aMEG using the same picture-sign priming paradigm mentioned above. Although he was as accurate and fast on the scanner task as the hearing L2 learner control group (and nearly as accurate and fast as the native deaf control group), Martin exhibited neural activation patterns that were primarily located in dorsolateral, superior parietal, and occipital areas bilaterally. This neural activation pattern was highly similar to the ones exhibited by Carlos and Shawna after they had 24 and 38 months of linguistic experience. Unlike the adolescent L1 learners, however, Martin, who was a young adult L1 learner, showed almost no activation in either the left or right temporal language areas (Mayberry, Davenport, Roth & Halgren, under review).

These contrasting results using different imaging techniques and paradigms indicate that the neurolinguistic processing of late L1 learners contrasts sharply from that of native learners, who experienced language in infancy, as well as from that of L2 learners, who also experienced language in infancy. Late L1 learners show greater activation in visual perceptual areas compared with native and L2 learners. That is, even though L2 learners have similar visual exposure to ASL as late L1 learners, they process it in like language. Cases of extreme language delay show neural activation patterns more commonly associated with watching meaningless human actions than processing lexical items from a language (Decety & Grèzes, 1999). Although adolescent L1 learners show some activation in left hemisphere perisylvian language areas as they accrue more ASL experience, the young adult L1 learner with decades of experience did not.

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This suggests that the left hemisphere language areas retain some capacity to process language when language is first experienced in adolescence, but this capacity is lost by young adulthood. No such reduction or absence of linguistic processing capacity in left hemisphere language areas is ever observed for L2 learners of signed or spoken languages.

6. The scope and nature of the CPL

From the array of research discussed here, it should now be clear that AoA effects on the ultimate outcome of L1 acquisition differ substantially from those of L2 outcome, both from a linguistic and a neurolinguistic perspective. Linguistically, AoA effects on L1 ultimate attainment are much greater than those for L2 attainment across a variety of psycholinguistic tasks. Late L1 learners perform at significantly lower levels than do late L2 learners on measures of morphology and syntax, phonological processing, and comprehension. This attenuated language attainment is unrelated to overall non-verbal cognitive skills or motivation to learn ASL (Valli, Lucas, Farb & Kulick, 1992). Limited language structure is acquired when the onset of L1 experience begins in adolescence and young adulthood. The stages of initial adolescent L1 acquisition resemble infant language acquisition, minus a babbling stage. Unlike L2 learning, late L1 acquisition slows and then stops at the level of simple sentence structure. The circumscribed level of language attainment observed for cases of adolescent and adult L1 acquisition begins to explain the low comprehension levels found across the studies of late L1 attainment. Our understanding of whether and how these effects are modulated by linguistic input, both in and out of school, is an important question in need of further research.

Parallel effects for late L1 acquisition are found for neurolinguistic processing. Neurolinguistic processing patterns for the signed L2 are highly similar to those found for native signed L1 neuroprocessing, with some additional activation elsewhere in the brain. The neurolinguistic processing patterns associated with AoA effects on L1 outcome show attenuated activation patterns in the frontal and temporal language areas of the left hemisphere, accompanied by increased neural activation patterns in sensory-perceptual processing areas in the parietal and occipital cortex. Extreme delays in the onset of L1 experience are associated with unique neurolinguistic processing patterns in dorsolateral occipital, parietal and frontal areas, processing patterns not observed when language – any language in any sensorimotor modality – is acquired from infancy. Perisylvian language areas show limited activation when language is first experienced in adolescence and nearly none when it is first experienced at the end of brain maturation in young adulthood.

The unique effects of AoA on L1 acquisition, attainment, and neurolinguistic processing suggest that the hierarchical structure of language and the architecture of the brain language processing system arise from their interaction over the course of early childhood when brain maturation and language acquisition are temporally synchronized. Although hearing infants show neural activation in response to speech in canonical language brain areas from birth (Vannasing, Florea, Gonzales-Frankenberger, Tremblay, Paquette, Safi, Wallois, Lepore, Beland, Lassonde & Gallagher, 2016), their brain language network is not yet developed. The brain language system shows organizational shifts over the course of development from infancy through adolescence. Neural responses when processing language are more posterior in the young child's brain and become more anterior with maturation (Brown, Lugar, Coalson, Miezin, Petersen & Schlaggar, 2005; Schlaggar, Brown, Lugar, Visscher, Miezin & Petersen, 2002). Neural language processing is more bilaterally represented in children and becomes more localized to the left hemisphere with maturation (Berl, Mayo, Parks, Rosenberger, VanMeter, Ratner, Vaidya & Gaillard, 2014; Ressel, Wilke, Lidzba, Lutzenberger & Krägeloh-Mann, 2008). The L1 AoA effects on brain language processing discussed above add to this developmental picture by demonstrating that the brain language system requires linguistic experience in order to potentiate its development from infancy to adolescence. Moreover, the onset of linguistic experience needs to be synchronous with post-natal brain maturation.

There are multiple environmental effects on brain development that are only beginning to be understood. For example, complex environments induce greater proliferation of neuronal growth during the exuberant phase of brain development as compared to impoverished environments (Greenough, Black & Wallace, 1987). Synapses that survive the pruning phase of neural development are those that have been stimulated by environmental input (Hensch, 2005; P. R. Huttenlocher, 1990). As Hebb (1949) initially proposed, synapses that fire together, wire together. Synaptic firing prompts myelination (Ishibashi, Dakin, Stevens, Kozlov, Stewart, Lee & Fields, 2006). Myelination of the fiber pathways connecting language areas in the temporal lobe to those in the frontal lobe develop with age and with vocabulary development (Pujol, Soriano-Mas, Oritz, Sebastián-Gallés & Deus, 2006). The dorsal pathway, the arcuate fasciculus, has been found to correlate with the comprehension of complex sentence structure in children (Skeide, Brauer & Friederici, 2016). Rather than being a strictly biologically driven maturational constraint on language acquisition, current work in our laboratory suggests that development of this neural fiber tract is driven, in part, by language experience and acquisition. The arcuate fasciculus is significantly less developed in

cases of late L1 acquisition compared with the same fiber tract in native deaf signers and hearing L2 signers, for whom anatomical measures of this fiber tract do not differ (Chen, Roth, Halgren & Mayberry, under review). Given these structural findings about the brain language system, one explanation for the circumscribed level of language acquisition attained by very late L1 learners is that their brain language systems are incompletely developed due to a lack of linguistic experience during childhood brain maturation. In other words, language acquisition and development of the brain language system appear to reciprocally affect one another, but only when the onset of language experience is synchronous with the onset of post-natal brain development. Under this scenario, L1 acquisition and development of the brain language system can be considered an example of critical period learning. These factors are not so clearly at play in L2 learning where the acquisition of a linguistic system and its neural underpinnings are already established.

In conclusion, nearly half a century of scientific discovery has occurred since Lenneberg (1967) made his paradigm changing observations and hypotheses about the biological and developmental nature of language acquisition. Sign language was not considered to be a language at the time, and neuroimaging technologies existing at the time provided limited understanding of the healthy brain. Much information remains to be learned about how the remarkable human achievement of language acquisition and the neural systems that enable it develop, and how environmental language affects them both. Sign language research has changed our thinking about the role sensorimotor modalities play in language structure and brain language processing. It also promises to reveal the complex and intertwined processes of language acquisition.

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