



Age of acquisition effects on the functional organization of language in the adult brain

Rachel I. Mayberry^{a,*}, Jen-Kai Chen^{b,c}, Pamela Witcher^b, Denise Klein^{b,c}

^a University of California, San Diego, United States

^b Department of Neurology and Neurosurgery, McGill University, Montreal, Canada

^c Cognitive Neuroscience Unit, Montreal Neurological Institute, Montreal, Canada

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ABSTRACT

Using functional magnetic resonance imaging (fMRI), we neuroimaged deaf adults as they performed two linguistic tasks with sentences in American Sign Language, grammatical judgment and phonemic-hand judgment. Participants' age-onset of sign language acquisition ranged from birth to 14 years; length of sign language experience was substantial and did not vary in relation to age of acquisition. For both tasks, a more left lateralized pattern of activation was observed, with activity for grammatical judgment being more anterior than that observed for phonemic-hand judgment, which was more posterior by comparison. Age of acquisition was linearly and negatively related to activation levels in anterior language regions and positively related to activation levels in posterior visual regions for both tasks.

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1. Introduction

Whether a dearth of language acquisition during post-natal brain growth affects language processing in the adult brain is unknown. The question is germane to the critical period hypothesis for language acquisition (Lenneberg, 1967). Although the hypothesis is decades old, it has been difficult to investigate because spoken language is ubiquitous in the environment of infants. In the absence of brain damage, one situation isolates infants from spoken language and often has the effect of delaying the onset of language acquisition, namely congenital deafness. Infants who are born deaf cannot hear the languages spoken around them and the visual signal of speech conveys insufficient phonetic detail to support spontaneous language acquisition. For many such children, language acquisition begins after exposure to, and immersion in, a sign language at ages well beyond infancy (Mayberry, 2007, 2010). Here we ask whether variation in the age-onset of language acquisition affects language processing in the adult brain.

Research has discovered that the grammar of sign languages, like that of spoken ones, is hierarchically organized. Sign language utterances are structured at the sentence (syntax), word

(morphology), sub-word (phonology), and semantic (word and sentence meaning) levels (Brentari, 1998; Davidson, Capronigro, & Mayberry, 2008; Klima & Bellugi, 1979; Sandler & Lillo-Martin, 2006; Stokoe, Casterline, & Croneberg, 1965; Zeshan, 2006). Although the grammatical properties of sign languages are similar to those of spoken ones, their age-onset of acquisition is typically different. A small percentage (less than 10%) of deaf children acquire sign language from birth because they had deaf parents who signed to them (Schein, 1989). For the remaining 90% of deaf children, sign language acquisition begins at a range of ages beyond infancy depending upon their first exposure to it. No underlying biological anomaly causes this variation in the age of acquisition, AoA, of sign languages. Instead the reasons are socio-cultural. For example, the child's hearing loss may have been detected late, or the child may not have been enrolled in school until an older age. A school that used sign language may not have been accessible to the family. Alternatively, the family and/or professionals may have elected to isolate the child from sign language despite a notable lack of functional speech in the erroneous belief that doing so would impede spoken language development (Mayberry, 2007, 2010).

Variation in the age-onset of sign language acquisition has multiple effects on psycholinguistic processing in adulthood. As acquisition begins at older ages, morphological and syntactic abilities decline (Boudreault & Mayberry, 2006; Emmorey, Bellugi, Friederici, & Horn, 1995; Newport, 1990). Later AoA is associated with the

* Corresponding author. Address: Department of Linguistics, University of California, San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0108, United States. Fax: +1 858 534 4789.

E-mail address: rmayberry@ucsd.edu (R.I. Mayberry).

commission of lexical errors made during off-line processing tasks that are dissociated from syntactic structure and sentence meaning and instead linked to the sub-lexical form of signs. Sentence and discourse-level sign language comprehension decrease in tandem with these phonologically-based lexical errors (Mayberry & Eichen, 1991; Mayberry & Fischer, 1989).

Key to investigating a possible critical period for language is the finding that AoA effects on sign language processing are especially large in cases where little or no language was acquired prior to the acquisition of sign language at older ages. These effects are unlike the well-documented AoA effects for the outcome of second-language, L2, learning (Mayberry, 1993). For example, learning an L2 at older ages can lead to near-native proficiency depending upon factors such as the grammatical relationship of the L2 to the first language and the degree of education undertaken in the L2 (Birdsong & Molis, 2001; Flege, Yeni-Komshian, & Liu, 1999). Consistent with how a critical period might be hypothesized to affect language development, research with deaf signers has found that an early onset of language is associated with near-native language proficiency, as in L2 learning. However, a dearth of language acquisition during early life is associated with low levels of language proficiency across all languages subsequently learned independent of sensory-motor modality (Mayberry & Lock, 2003; Mayberry, Lock, & Kazmi, 2002). The crucial question is whether the unique and life-long psycholinguistic effects associated with a lack of language in early life reflect differential neural language processing by the adult brain.

In order to predict how AoA effects might appear in the results of an fMRI experiment, we turn to research investigating the neural processing of sign language. Converging evidence shows that the neural processing loci of sign languages largely overlap those of spoken languages. These findings come from a variety of neurolinguistic paradigms, including brain lesion and cortical language mapping studies, and the neuroimaging of healthy adults with PET and fMRI. The bulk of this research has been conducted with participants with an early age-onset of sign language acquisition.

Case studies of brain lesions in deaf adults, who are described as being “lifelong signers,” show a leftward asymmetry for sign language processing. Left hemisphere, LH, but not right hemisphere, RH, lesions have been found to disrupt sign language comprehension (Poizner, Klima, & Bellugi, 1987). Lesions in the LH temporal lobe affect the comprehension of single signs and complex sentences (Hickok, Bellugi, & Klima, 1998; Hickok, Love-Geffen, & Klima, 2002). Similar results were obtained in a cortical mapping study. Direct stimulation of Broca and Wernicke’s areas in the LH disrupted sign production in a deaf patient undergoing surgery for epilepsy (Corina et al., 1999). Two case studies, one in American and one in Japanese Sign Language, found that deaf adults with lesions in the left occipital cortex exhibited difficulty recognizing signs (Hickok, Klima, Kritchevsky, & Bellugi, 1995; Saito, Otsuki, & Uneo, 2007).

One controlled means of comparing the neural processing of signed and spoken languages is to neuroimage them in the same brain. Hearing native signers, (i.e., hearing adults with deaf parents who signed to them from birth) were scanned as they produced spontaneous autobiographical narratives, once in ASL and once in spoken English. The PET results revealed largely overlapping LH activation for ASL and spoken English, with ASL showing somewhat more dispersed activation patterns (Braun, Guillemin, Hosey, & Varga, 2001). PET activation patterns during sign (i.e., word) retrieval in deaf native signers (i.e., deaf individuals with deaf parents who signed to them from birth) performing tool and action naming tasks in ASL were largely indistinguishable from the PET activation patterns of hearing English speakers performing the same task (Damasio et al., 1996). Importantly, the activation patterns associated with sign retrieval were unaffected by any iconic

relationship between the sign’s phonological form and its meaning (Emmorey et al., 2003, 2004). This indicates that higher levels of linguistic processing are modality independent, although some lower levels of sign processing are both modality independent and dependent, such as the maintenance of sign items in immediate memory (Pa et al., 2008).

fMRI studies of deaf native signers have also found activation in the classic language areas of the LH with a trend toward bilateral activation in frontal and temporal lobes. These results have been found for distinct sign languages, namely American, British, and Japanese, using various tasks and stimuli (Kassubek, Hickok, & Erhard, 2004; MacSweeney et al., 2002; McCullough, Emmorey, & Sereno, 2005; Sakai, Tatsuno, Suzuki, Kimura, & Ichida, 2005). Although sign language is visual, activation in the occipital cortex is not routinely found across studies for linguistic processing in highly proficient signers. Occipital cortex activation has been reported in hearing signers for whom sign language is a non-dominant language, and in hearing non-signers (Klann, Kastrau, & Huber, 2005; MacSweeney et al., 2002). Activation in occipital cortex when the task involves higher level linguistic processing may be associated with lower proficiency. This is not entirely due to the subtraction of visual activation by way of a moving baseline task. Use of a moving baseline does not predict a lack of activation reported for occipital cortex in deaf native signers (Corina et al., 2007; MacSweeney et al., 2006; Sakai et al., 2005).

Some studies have compared the neurolinguistic processing of native and non-native signers with inconsistent results. While passively viewing ASL stimuli, hearing native signers of ASL showed activation in the right angular gyrus, whereas hearing L2 signers did not (Newman, Bavelier, Corina, Jezzard, & Neville, 2002). It is possible that the RH activation shown by the hearing native signers was elicited by ASL stimuli that were more discourse- than sentence-like. The RH is involved in the processing of prosody and inter-sentential relations (Baum & Pell, 1999; Caplan & Dapretto, 2001). Consistent with this interpretation are the results of an fMRI study that directly compared activation patterns for ASL at the discourse and sentence levels. Discourse-level stimuli with prosodic contours in sign language elicited RH activation patterns whereas sentence-level stimuli elicited LH activation in deaf native signers (Newman, Supalla, Hauser, Newport, & Bavelier, 2010). In a PET study of working memory in Swedish Sign Language, hearing native signers showed a left parietal bias which was not found when hearing L2 signers were included in the group analyses (Rönnberg, Rudner, & Ingvar, 2004).

On a phonological similarity task, where the participants decided whether the signs for pairs of line drawings shared articulatory parameters (akin to rhyme judgments for spoken words), deaf native signers showed less activation in the left, posterior inferior frontal gyrus compared to deaf non-native signers of British Sign Language (who acquired spoken English in childhood and BSL in late adolescence or adulthood; MacSweeney, Waters, Brammer, Woll, & Goswami, 2008).¹ Greater activation in these brain regions is sometimes reported for L2 learners of a spoken language relative to native speakers of the language (Indefrey, 2006). On a task requiring detection of reversed signs in German Sign Language dialogs, deaf non-native signers showed a variety of individual activation patterns. This could be due to the fact that AoA was uncontrolled. Notably, some participants with late AoA showed primarily left occipital activation (Meyer et al., 2007). Given

¹ It is important to note that being born deaf and learning a sign language at an older age does not necessarily mean that it is a *first* language acquired at a late age in the absence of earlier language. Some deaf individuals acquire spoken language in early childhood; their subsequent sign language learning is more akin to L2 learning, even though their L1 proficiency in spoken language is less than native-like (Mayberry et al., 2002).

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