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Research Report

Neonatal conductive hearing loss disrupts the development of the Cat-315 epitope on perineuronal nets in the rat superior olivary complex

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ABSTRACT

The critical period is a postnatal window characterized by a high level of experience-dependent neuronal plasticity in the central nervous system and sensory deprivation during this period significantly impacts neurological function. Perineuronal nets (PNNs) are specialized aggregates of the extracellular matrix which ensheath neuronal cell bodies, primary dendrites and axon hillocks and function in neuronal protection and stabilize synapses. PNNs are generally not present at birth, but reach adult-like patterns by the end of the third or fourth postnatal week. Their appearance is believed to mark the close of the critical period and sensory deprivation during this epoch disrupts development of PNNs. Here we investigate the postnatal development of two PNN markers (*Wisteria floribunda* agglutinin [WFA] and Cat-315) and the effect of neonatal conductive hearing loss (CHL) on their development. Our data indicates that these PNN markers are not present in the superior olivary complex (SOC) at birth, but develop over the first four postnatal weeks in different temporal patterns and also that neonatal CHL results in a significant decrease in the number of SOC neurons associated with Cat-315 reactive PNNs.

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1. Introduction

Perineuronal nets (PNNs) are specialized constructs of the neural extracellular matrix (nECM) that ensheath the soma, dendrites and initial axon segment of select neuronal populations (Atoji et al., 1989; Brückner et al., 1993; Lander et al., 1998) and function in a multitude of physiological processes (Celio et al., 1998; Dityatev and Schachner, 2003; Morris and Henderson, 2000). In the post-embryonic brain, PNNs appear to limit critical period plasticity and stabilize synapses; experimental digestion of PNNs in the adult brain induces a state of elevated plasticity (Hockfield et al., 1990;

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Abbreviations: BL, bilateral; CHL, conductive hearing loss; CL, contralateral; D, dorsal; IL, ipsilateral; L, lateral; LNTB, lateral nucleus of the trapezoid body; LSO, lateral superior olive; M, medial; MNTB, medial nucleus of the trapezoid body; MSO, medial superior olive; NDS, normal donkey serum; nECM, neural extracellular matrix; P, postnatal; PB, sodium phosphate buffer; PNN, perineuronal net; SOC, superior olivary complex; SPON, superior paraolivary nucleus; VNTB, ventral nucleus of the trapezoid body; WFA, *Wisteria floribunda* agglutinin

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Kwok et al., 2008; Pizzorusso et al., 2002; Sur et al, 1988) and leaves memory traces in the amygdala susceptible to erasure (Gogolla et al., 2009). Given the importance of these extracellular structures, the distribution of PNNs in the central nervous system is not uniform—PNNs are plentiful in the auditory brainstem by postnatal day (P) 21 (Cant and Benson, 2006; Friauf, 2000; Köppe et al., 1997; Lurie et al., 1997) but develop slightly later (P35) and are less common in cortical areas (Brückner et al., 2000; Guimarães et al., 1990; Köppe et al., 1997; McRae et al., 2007). The association of neurons with PNNs varies by cell type, age and sensory experience (McRae et al., 2007; Pantazopoulos et al., 2008; Schmidt et al., 2010; Wagoner and Kulesza, 2009; Wintergerst et al., 1996). Further, there is evidence that PNN distribution varies by species (Brückner et al., 2006). Indeed, it is well established that the nECM is incomplete at birth, but PNNs (in rodents) develop rapidly in sensory systems, reaching an adult-like pattern by the end of the 3rd–4th postnatal week (Brückner et al., 2000; Friauf, 2000; Köppe et al., 1997; Lurie et al., 1997; Seeger et al., 1994). Further, maturation of PNNs appear to mark the close of the critical period, a developmental window characterized by a high level of sensory experience-dependent neuronal plasticity (Lurie et al., 1997; McRae et al., 2007; Pizzorusso et al., 2002). In fact it has been hypothesized that the presence of PNNs inhibits formation of new synapses (Celio and Blümcke, 1994). This is significant, since sensory deprivation during the critical period is known to have a lasting impact on neurological function (Clopton and Silverman, 1977; Wiesel and Hubel, 1963) and inhibits the formation of some PNNs in the spinal cord (Kalb and Hockfield, 1988), somatosensory cortex (McRae et al., 2007), lateral geniculate body and visual cortex (Guimarães et al., 1990; Sur et al., 1988). Finally, it should be noted that certain disease states have been associated with deterioration of PNNs (Creutzfeldt–Jakob—Belichenko et al., 1999; schizophrenia—Pantazopoulos et al., 2010). Thus, the normal PNN pattern in a brain region/nucleus seems to indicate the normal progression through activity-dependent developmental processes and normal neurological function.

The superior olivary complex (SOC) is a conglomerate of brainstem nuclei which comprise the first major site of convergence of information from both ears and functions in the localization of sound sources, encoding temporal features of sound and descending modulation of the organ of Corti (see reviews by Heffner and Masterton, 1990; Oliver, 2000; Schofield, 2010; Schwartz, 1992; Spangler and Warr, 1991; Thompson and Schofield, 2000). The SOC includes the medial and lateral superior olives (MSO and LSO, respectively, both of which receive input from both ears), the superior paraolivary nucleus (SPON) and the medial, ventral and lateral nuclei of the trapezoid body (MNTB, VNTB and LNTB respectively). In the rat, the external auditory meatus is closed until about P12, but high intensity sounds can elicit responses as early as P7 by bone conduction; mature conductive hearing is achieved by P15 and adult-like auditory thresholds are achieved by P22 (Geal-Dor et al., 1993). Even though SOC neurons achieve mature membrane properties soon after hearing onset (Chirila et al., 2007; Harris et al., 2005; Magnusson et al., 2005), many features (e.g. synaptic kinetics, discharge rates, response latency) depend on sensory experience and continue to mature through the 4th postnatal week (Sanes and Rubel, 1988; Sanes et al., 1992; Scott et al., 2005; Sonntag et al., 2009; Walcher et al., 2011; Webster, 1983b). Normal maturation of auditory brainstem circuits requires an intact cochlea and auditory nerve: permanent lesions (e.g. cochlear ablation) result in atrophy of auditory brainstem neurons (Kitzes et al., 1995; Kotak and Sanes, 1997; Nordeen et al., 1983; Russell and Moore, 1999, 2002). Further, it is clear that transient conductive hearing loss (CHL) during the early postnatal critical period disturbs normal auditory function in the midbrain (Mogdans and Knudsen, 1993; Popescu and Polley, 2010; Silverman and Clopton, 1977) and auditory cortex (Popescu and Polley, 2010; Xu et al., 2007), and results in elevated auditory thresholds (Walger et al., 1989), an enhanced acoustic startle reflex and increased risk for sound induced seizures (Sun et al., 2011). Additionally, in the cochlear nuclei and SOC, neonatal CHL has been shown to cause significant reductions in neuronal cell body size (Coleman and O'Connor, 1979; Webster, 1983a, b; Webster, 1988; Webster and Webster, 1979) and altered dendritic arbors (Feng and Rogowski, 1980; Torrero et al., 1999).

It is clear that achieving an adult pattern of PNNs marks the closure of the sensory critical period (in visual and somatosensory cortex) and that normal and meaningful neuronal activity is required during this critical period for proper development and function. Further, if evoked activity is disrupted, normal sensory processing cannot be achieved. Based on these observations in other sensory systems, we propose that long-standing auditory dysfunction, resulting from early auditory deprivation, is related (at least in part) to disruptions in the nECM and specifically PNNs. We hypothesize that neonatal CHL (as a model of temporary hearing loss in children) will significantly alter the morphology of SOC neurons and reduce the number of PNNs in the SOC.

2. Results

2.1. *Normal postnatal development of PNNs in the SOC*

2.1.1. *Development of WFA-PNNs in the SOC*

Figs. 1A and B illustrate the pattern of WFA-labeling and a characteristic WFA-PNN in the SOC of the adult rat (P100–120). At P0 there are very few WFA-PNNs in the brainstem or cerebellum, although occasional PNNs are observed in the reticular formation and spinal trigeminal nucleus. At P0, labeling is often seen around blood vessels and within many cells along the 4th ventricle. At P12, a few SOC neurons have a distinct cell body coating of WFA-labeling and extra-somatic labeling is observed in the neuropil as sparse and scattered extracellular debris (Fig. 3A—see Table 1 for total number of neurons counted per nucleus and Fig. 4 for % of neurons with PNNs). By P16, there is a noticeable increase in the number of WFA-PNNs in the SOC, but most neurons have only a light extracellular covering (Figs. 2C and 3B). By P20, many SOC neurons have PNNs and by P28, the vast majority of SOC neurons are associated with PNNs (as in the adult SOC, Figs. 1A, 2D, and 4). Notably, in the SOC the largest increases in WFA-PNNs occur about 4 days after the onset of hearing (Fig. 4).

In the LSO, no WFA-PNNs are present until P8 (<1%; Fig. 2B). After P12 there is a steep increase in the number of

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